

Prevalence of Sexually Transmitted Infections Among Female Adolescents Aged 14 to 19 in the United States

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KEY WORDS

sexually transmitted diseases, adolescent, prevalence

ABBREVIATIONS

STI—sexually transmitted infection
NHANES—National Health and Nutrition Examination Survey
HSV-2—herpes simplex virus type 2
HPV—human papillomavirus
HR/6/11 HPV—high-risk or type 6 or 11 HPV
CI—confidence interval
RSE—relative standard error
OR—odds ratio
aOR—adjusted OR

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WHAT'S KNOWN ON THIS SUBJECT: The combined prevalence of the most common STIs among female adolescents and the relationship of STI prevalence with sexual behavior in this population have not been comprehensively evaluated by using nationally representative data.



WHAT THIS STUDY ADDS: Among US female adolescents, STI burden is substantial, and HPV is the most prevalent STI. STIs appear to be acquired rapidly after sexual initiation, which reinforces the need for prevention efforts, including HPV vaccination, well before the onset of sexual activity.

abstract

OBJECTIVE: Most young women initiate sexual activity during adolescence; risk for sexually transmitted infections (STIs) accompanies this initiation. In this study we estimated the prevalence of the most common STIs among a representative sample of female adolescents in the United States.

METHODS: Data were analyzed from 838 females who were aged 14 to 19 and participating in the nationally representative National Health and Nutrition Examination Survey 2003–2004. After interview and examination, survey participants provided biological specimens for laboratory testing. The main outcome was weighted prevalence of at least 1 of 5 STIs: *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, *Trichomonas vaginalis*, herpes simplex virus type 2, and human papillomavirus (HPV) (any of 23 high-risk types or type 6 or 11).

RESULTS: Prevalence of any of the 5 STIs was 24.1% among all and 37.7% among sexually experienced female adolescents. HPV (23 high-risk types or type 6 or 11) was the most common STI among all female adolescents (prevalence: 18.3%), followed by *C trachomatis* infection (prevalence: 3.9%). Prevalence of any of the STIs was 25.6% among those whose age was the same or 1 year greater than their age at sexual initiation and 19.7% among those who reported only 1 lifetime sex partner.

CONCLUSIONS: The prevalence of STIs among female adolescents is substantial, and STIs begin to be acquired soon after sexual initiation and with few sex partners. These findings support early and comprehensive sex education, routine HPV vaccination at the age of 11 to 12 years, and *C trachomatis* screening of sexually active female adolescents. *Pediatrics* 2009;124:1505–1512

The majority of young women initiate sexual activity during adolescence,¹ and risk for sexually transmitted infections (STIs) accompanies this initiation. Although the course of many of these STIs is benign, even without treatment, some infections may lead to long-term sequelae, including pelvic inflammatory disease, infertility, and cervical cancer. A number of STIs also increase the risk for acquiring HIV infection. Community-based studies and national surveillance data suggest that at least some subpopulations of adolescents have high STI rates²⁻⁴; however, these data are not nationally representative.

Recent analyses of US population-based data have shown varying prevalences of several individual STIs among female adolescents,⁵⁻⁸ but there has been no in-depth analysis of data in this age group. Estimates of overall STI prevalence and its association with sexual behavior among female adolescents would help to quantify the magnitude of STI risk in this vulnerable population and inform prevention efforts. We undertook this study to estimate the prevalence of the most common STIs among a representative sample of females aged 14 to 19 in the United States.

METHODS

Study Population and Survey Design

We analyzed data collected from females who were aged 14 to 19 and participating in the National Health and Nutrition Examination Survey (NHANES) 2003–2004. The NHANES is conducted by the National Center for Health Statistics of the Centers for Disease Control and Prevention and uses a complex, stratified, multistage probability sampling design to sample the US non-institutionalized civilian population randomly.⁹ The NHANES 2003–2004 had 10 122 participants, reflecting a 79%

interview rate.¹⁰ Race/ethnicity categories were defined as non-Hispanic white, non-Hispanic black, and Mexican American using self-reported information.¹¹ Participants who did not fit into 1 of these categories were classified as “other.” Non-Hispanic black and Mexican American participants and adolescents aged 12 to 19 were oversampled. Poverty index ratio¹² values of <1 were considered below and values of ≥ 1 were considered at or above the poverty level.

Survey participants were interviewed, underwent a standardized physical examination, and submitted biological specimens. Sexual history information was collected by using audio computer-assisted self-interview. “Sex” was defined as vaginal, oral, or anal sex. We defined “sexually experienced” as a “yes” response to the question, “Have you ever had sex?” The Centers for Disease Control and Prevention institutional review board approved NHANES 2003–2004 protocols. All survey participants who were aged ≥ 18 provided written, informed consent. Parents gave written consent for minors (aged <18), accompanied by the minors’ assent.

Laboratory Tests and Methods

In the NHANES 2003–2004, female participants who were aged 14 to 19 provided urine, sera, and self-collected vaginal swab specimens for analysis. Urine was tested for *Chlamydia trachomatis* (chlamydia) and *Neisseria gonorrhoeae* (gonorrhea) by using BDProbeTec ET (Becton Dickinson, Franklin Lakes, NJ).¹³ Sera were tested for herpes simplex virus type 2 (HSV-2) antibodies with a solid-phase enzymatic immunodot assay¹⁴ that was based on glycoprotein gG2. Vaginal swab specimens were tested for *Trichomonas vaginalis* (trichomonas) and human papillomavirus (HPV) by using separate polymerase chain reaction tests.

A TaqMan-based polymerase chain reaction method was used to detect trichomonas.¹⁵ HPV detection and typing were performed on swab DNA extracts by using the Roche prototype line blot assay (Roche Molecular Systems Inc, Pleasanton, CA) and additional techniques as previously described.⁵ We defined high-risk HPV types as types 16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 67, 68, 69, 70, 73, 82, 85, and IS39. Participants could obtain their test results by telephone by using a password-protected system. Those who did not call were sent up to 3 reminder letters. Participants with positive STI results were given information about their infection(s) and advised to seek medical care. Those without health care providers were given names of area clinics that provide STI services.

Statistical Analyses

We estimated the weighted prevalence of HPV, gonorrhea, chlamydia, trichomonas, and HSV-2 infections in female adolescents who were aged 14 to 19. Evaluation of HPV infection was restricted to HPV types that are most likely to cause clinically important disease: the 23 high-risk HPV types defined already, which are responsible for cervical and other cancers,¹⁶ and low-risk types 6 and 11, which are responsible for >90% of genital warts.¹⁷ We designated this HPV variable “high-risk or 6 or 11” HPV (HR/6/11 HPV), which was considered positive when any high-risk HPV type or type 6 or 11 was detected. To measure the overall prevalence of at least 1 of these 5 STIs, we defined a composite STI variable, “any STI,” as a positive test for HR/6/11 HPV or gonorrhea or chlamydia or trichomonas or HSV-2 infection. We limited analysis of this composite variable to participants with laboratory results for all 5 STIs. We calculated weighted prevalence estimates among

all female adolescents and among those who were sexually experienced.

We evaluated “any STI” prevalence by selected demographic and sexual behavioral characteristics including age, race/ethnicity, poverty level, number of lifetime sex partners, and duration of sexual activity. Duration of sexual activity was defined as current age in years minus year of age at sexual initiation. We also evaluated HR/6/11 HPV infection prevalence separately from prevalence of the remaining 4 STIs (gonorrhea or chlamydia or trichomonas or HSV-2) according to these characteristics.

All prevalence estimates were weighted using NHANES 2003–2004 examination weights to represent the total US civilian, noninstitutionalized population and to account for oversampling and nonresponse to the interview and physical examination.¹⁸ Using weights with additional nonresponse adjustment for missing laboratory results produced prevalence estimates within 95% confidence intervals (CIs) determined by the original NHANES weights; therefore, we made no additional adjustments.¹⁹

Prevalence estimate CIs were calculated by using logit transformation. Variance estimates were calculated by using a Taylor series linearization that incorporated the survey’s complex sample design.²⁰ Relative SEs (RSEs) were computed for each weighted prevalence estimate as $(SE/estimate) \times 100$. A prevalence estimate with an RSE of $>30\%$ is considered unstable and should be interpreted cautiously.

Bivariate statistical associations were summarized with unadjusted odds ratios (ORs); corresponding *P* values were based on a Wald *F* statistic. We calculated adjusted ORs (aORs) for the main outcome, “any STI,” and for the other 2 outcomes, HR/6/11 HPV infection and gonorrhea, chlamydia, trichomonas, or HSV-2 infection, by us-

TABLE 1 Prevalence of 5 STIs Among Females Aged 14 to 19: United States, 2003–2004

STI	All (<i>N</i> = 838)		Sexually Experienced (<i>n</i> = 404) ^a	
	<i>n</i>	Weighted Prevalence, % (95% CI)	<i>n</i>	Weighted Prevalence, % (95% CI)
HR/6/11 HPV ^b	652	18.3 (13.5–24.8)	357	29.5 (22.6–38.4)
<i>C trachomatis</i>	793	3.9 (2.2–6.9)	396	7.1 (4.1–12.2)
<i>T vaginalis</i>	695	2.5 (1.2–5.0) ^c	371	3.6 (1.6–8.1) ^c
HSV-2	729	1.9 (1.0–3.5)	370	3.4 (1.7–6.6) ^c
<i>N gonorrhoeae</i>	793	1.3 (0.6–3.1) ^c	396	2.5 (1.0–6.1) ^c
Any STI ^d	590	24.1 (18.4–30.9)	328	37.7 (28.8–47.5)

^a Data are available on sexual experience for 750 participants. The weighted prevalence of sexual experience among these adolescents was 50.7% (95% CI: 46.1%–55.2%).

^b HPV: any of 23 oncogenic (high-risk) types (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 67, 68, 69, 70, 73, 82, 85, and IS39) or type 6 or 11.

^c RSE $> 30\%$, reflecting an unstable estimate. No RSE was $\geq 50\%$.

^d Any of the following STIs: HPV (high-risk or type 6 or 11), *N gonorrhoeae*, *C trachomatis*, *T vaginalis*, or HSV-2.

ing multivariate logistic regression models that included age, race/ethnicity, poverty level, number of lifetime sex partners, and duration of sexual activity. We tested for all pairwise interactions for the models presented. Satterthwaite adjusted *F* *P* values are reported. SAS 9.1 (SAS Institute Inc, Cary, NC) and SAS-callable SUDAAN (Research Triangle Institute, Research Triangle Park, NC) were used for all analyses.

RESULTS

Of 838 female adolescent participants in the NHANES 2003–2004, 820 (98%) were examined, 806 (96%) had at least 1 STI laboratory result available, and 590 (70%) had test results for all 5 STIs. Participants who were aged 14 to 15 were more likely to have missing test results than those who were aged 16 to 19 (37.0% vs 22.8%; *P* = .003), as were participants who had never had sex (34.3% vs 16.6% among the sexually experienced; *P* = .008).

Prevalence of STIs

The prevalence of any of these STIs was 24.1% (95% CI: 18.4%–30.9%; Table 1). The most prevalent STI was HR/6/11 HPV infection (18.3% [95% CI: 13.5%–24.8%]), followed by chlamydia infection (3.9% [95% CI: 2.2%–6.9%]). Among those who were sexually experienced, prevalence of any of these

STIs was 37.7% (95% CI: 28.8%–47.5%), prevalence of HR/6/11 HPV infection was 29.5% (95% CI: 22.6%–38.4%), and of chlamydia infection was 7.1% (95% CI: 4.1%–12.2%). Of female adolescents with at least 1 STI, 15.8% (95% CI: 11.2%–21.8%) had >1 . The vast majority (79.3%) of those with >1 STI had HR/6/11 HPV as 1 of their infections.

STI Prevalence According to Demographic Characteristics

Prevalence of any of the 5 STIs varied by age (Table 2). Among participants who were aged 14 to 15, prevalence was 14.1%, although this estimate should be interpreted with caution because of small numbers (RSE > 30). Prevalence among participants who were aged 18 to 19 was 33.8% (*P* = .01, versus those aged 14 to 15). Prevalence of any of these STIs was similar among non-Hispanic white (19.4%) and Mexican American (18.0%) participants but higher among non-Hispanic black participants (43.9%; *P* = .001, versus non-Hispanic white participants).

For the HR/6/11 HPV STI outcome, we found statistically significant differences in prevalence by age and race/ethnicity. HR/6/11 HPV prevalence was standard error 26.7% among those who were aged 18 to 19 vs 9.4% (RSE > 30) among those who were aged 14 to

TABLE 2 Prevalence of 5 STIs According to Selected Characteristics Among Females Aged 14 to 19: United States, 2003–2004

Parameter	Sample Size	Any STI ^a		HR/6/11 HPV ^b		Gonorrhea, Chlamydia, Trichomonas, or HSV-2	
		Weighted Prevalence % (95% CI)	OR (95% CI)	Weighted Prevalence % (95% CI)	OR (95% CI)	Weighted Prevalence % (95% CI)	OR (95% CI)
Overall	590	24.1 (18.4–30.9)	–	19.0 (13.9–25.2)	–	8.2 (5.5–11.9)	–
Age, y							
14–15	168	14.1 (6.9–26.8) ^c	1.0	9.4 (4.1–21.4) ^c	1.0	5.5 (2.2–13.2) ^c	1.0
16–17	196	22.0 (14.2–32.4)	1.7 (0.8–3.9)	18.6 (11.3–30.6)	2.2 (0.9–5.5)	7.4 (3.9–13.6)	1.4 (0.5–4.0)
18–19	226	33.8 (27.8–40.5)	3.1 (1.3–7.3)	26.7 (21.4–33.3)	3.5 (1.6–7.9)	10.9 (6.5–17.8)	2.1 (0.6–7.3)
Race/ethnicity ^d							
Non-Hispanic white	179	19.4 (12.7–28.6)	1.0	15.9 (10.1–25.1)	1.0	5.1 (2.8–9.1)	1.0
Non-Hispanic black	206	43.9 (33.7–54.6)	3.2 (1.7–6.1)	33.7 (23.2–49.0)	2.7 (1.3–5.6)	21.2 (13.6–31.4)	5.0 (2.2–11.2)
Mexican American	178	18.0 (11.1–28.0)	0.9 (0.4–1.9)	14.0 (8.4–23.2)	0.9 (0.4–1.9)	6.6 (2.6–15.8)	1.3 (0.5–3.6)
Poverty level ^e							
Below poverty level	210	34.4 (25.4–44.6)	2.3 (1.1–4.6)	24.4 (17.8–33.5)	1.7 (0.8–3.6)	15.0 (8.4–25.5)	3.2 (1.5–7.1)
At or above poverty level	351	18.8 (12.2–27.8)	1.0	15.8 (9.8–25.3)	1.0	5.2 (3.4–7.9)	1.0
Total No. of lifetime sex partners							
0	228	6.6 (2.7–15.0) ^c	0.3 (0.1–0.8)	4.6 (1.7–12.5) ^c	0.3 (0.1–0.7)	1.9 (0.7–5.3) ^c	0.3 (0.1–1.6)
1	122	19.7 (12.3–29.9)	1.0	16.4 (10.3–26.1)	1.0	5.5 (2.0–14.4) ^c	1.0
2	58	38.1 (20.4–59.6)	2.5 (0.9–7.0)	31.1 (16.2–60.1)	2.3 (0.9–5.8)	10.0 (4.4–21.2) ^c	1.9 (0.4–8.9)
≥3	147	53.5 (43.1–63.6)	4.7 (2.4–9.2)	41.2 (31.3–54.4)	3.6 (1.7–7.7)	20.8 (14.0–29.8)	4.5 (1.3–15.2)
Duration of sexual activity, y ^f							
Never had sex	228	6.6 (2.7–15.0) ^c	0.2 (0.1–0.5)	4.6 (1.7–12.5) ^c	0.2 (0.1–0.5)	1.9 (0.7–5.3) ^c	0.3 (0.1–1.0)
0–1	139	25.6 (16.3–37.8)	1.0	22.5 (14.3–35.3)	1.0	6.8 (3.5–12.8) ^c	1.0
≥2	189	49.2 (40.9–57.6)	2.8 (1.6–4.8)	36.7 (28.5–47.2)	2.0 (1.1–3.6)	19.3 (13.5–26.8)	3.3 (1.6–6.9)

^a Any of the following STIs: HPV (high-risk or type 6 or 11), *N gonorrhoeae*, *C trachomatis*, *T vaginalis*, or HSV-2.

^b HPV: any of 23 oncogenic (high-risk) types (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 67, 68, 69, 70, 73, 82, 85, and IS39) or type 6 or 11.

^c RSE > 30%, reflecting an unstable estimate. No RSE was ≥50%.

^d "Other" race/ethnicity is not shown because of the small sample size ($n = 27$).

^e Poverty index ratio was assigned by dividing total family income by the poverty threshold index, then adjusting for number of people in the household at the year of interview; <1.0 is considered below poverty level, and ≥1.0 is considered at or above poverty level.

^f Age in years at interview minus year of age at first sex; 0 indicates that the participant was interviewed at the same age as reported age of sexual initiation.

15 ($P = .005$). Non-Hispanic black female adolescents had a higher prevalence of HR/6/11 HPV infection (33.7%) than did either non-Hispanic white (15.9%; $P = .01$) or Mexican American (14.0%; $P = .01$) participants.

Prevalence of any of the other 4 STIs—gonorrhea, chlamydia, trichomonas, or HSV-2—varied significantly by race/ethnicity and poverty level. Prevalence of any of these 4 STIs among non-Hispanic black female adolescents (21.2%) was greater than among either non-Hispanic white (5.1%; $P < .001$) or Mexican American (6.6%; $P = .02$) participants.

STI Prevalence According to Sexual Behavioral Characteristics

STI prevalence varied by duration of sexual activity and by number of life-

time sex partners (Table 2). Among female adolescents whose age was the same or 1 year greater than their age at sexual initiation, prevalence of any of the 5 STIs was 25.6%; among those whose age was at least 2 years greater, prevalence was 49.2% ($P < .001$). Even for those whose age was the same as at sexual initiation (ie, <1 year of sexual experience), prevalence of any STI was 19.2%. Female adolescents who reported only 1 lifetime sex partner had 19.7% prevalence of any of the STIs; prevalence increased to 53.5% among those who reported ≥3 partners ($P < .001$, versus 1 lifetime partner).

Prevalence of HR/6/11 HPV infection was 22.5% among female adolescents whose age was the same or 1 year

greater than their age at sexual initiation; among those whose age was at least 2 years greater, prevalence was 36.7% ($P = .03$). HR/6/11 HPV prevalence for those who reported having had 1 lifetime sex partner was 16.4% and for those who reported ≥3 partners, 41.2% ($P = .003$, versus 1 partner).

The prevalence of gonorrhea, chlamydia, trichomonas, or HSV-2 infection was 6.8% (RSE >30) among female adolescents whose age was the same or 1 year greater than their age at sexual initiation and 19.3% among those whose age was at least 2 years greater ($P = .004$). The prevalence of any of these 4 STIs was higher for young women with more lifetime sex partners, increasing to 20.8% among those with ≥3 partners ($P = .02$, versus 1 partner).

TABLE 3 Multivariate Analyses of Prevalence of 5 STIs According to Selected Characteristics Among Females Aged 14 to 19, United States, 2003–2004

Parameter	Any STI, aOR (95% CI) ^{a,c}	HR/6/11 HPV, aOR (95% CI) ^{b,c}	Gonorrhea, Chlamydia, Trichomonas, or HSV-2, aOR (95% CI) ^c
Age, y			
14–15	1.0	1.0	1.0
16–17	0.8 (0.3–2.1)	1.0 (0.3–2.9)	0.9 (0.1–5.7)
18–19	1.0 (0.5–2.0)	1.3 (0.5–3.3)	0.7 (0.1–3.8)
Race/ethnicity ^d	^e		^f
Non-Hispanic white	1.0	1.0	1.0
Non-Hispanic black	3.6 (1.9–6.8)	2.5 (1.2–5.1)	5.2 (2.0–13.8)
Mexican American	1.2 (0.6–2.5)	1.2 (0.5–2.8)	1.4 (0.6–3.8)
Poverty level ^g			
Below poverty level	1.2 (0.6–2.4)	0.9 (0.5–2.0)	1.8 (0.8–4.2)
At or above poverty level	1.0	1.0	1.0
Total No. of lifetime sex partners	^e	^f	
0	0.3 (0.1–1.1)	0.3 (0.1–0.9)	0.2 (0.06–0.8)
1	1.0	1.0	1.0
2	2.3 (0.6–8.6)	2.4 (0.8–7.3)	1.3 (0.2–7.1)
≥3	4.9 (2.2–11.0)	4.4 (1.8–10.5)	4.0 (0.9–17.8)
Duration of sexual activity, y ^h			
0–1	1.0	1.0	1.0
≥2	1.3 (0.6–2.9)	0.9 (0.4–2.1)	1.7 (0.6–4.6)

^a Any of the following STIs: HPV (high-risk or type 6 or 11), *N gonorrhoeae*, *C trachomatis*, *T vaginalis*, or HPV-2.

^b HPV: any of 23 oncogenic (high-risk) types (16, 18, 26, 31, 33, 35, 39, 45, 51, 52, 53, 56, 58, 59, 66, 67, 68, 69, 70, 73, 82, 85, and IS39) or type 6 or 11.

^c aORs for all values are from a multivariate model that includes age, race/ethnicity, poverty index ratio, number of lifetime sex partners, and duration of sexual activity (sample size used in model = 529).

^d “Other” race/ethnicity is not shown because of the small sample size ($n = 27$) but is included in multivariate analysis.

^e Satterthwaite adjusted F; $P < .01$.

^f Satterthwaite adjusted F; $P \geq .01$ and $\leq .05$.

^g Poverty index ratio was assigned by dividing total family income by the poverty threshold index, then adjusting for number of people in the household at the year of interview; <1.0 is considered below the poverty level, and ≥ 1.0 is considered at or above poverty level.

^h Age in years at interview minus year of age at first sex; 0 indicates that the participant was interviewed at the same age as reported age of sexual initiation.

Multivariate Analysis

After adjustment for other factors, non-Hispanic black participants had almost 4 times the odds of having any of the STIs compared with non-Hispanic white participants (aOR: 3.6 [95% CI: 1.9–6.8]; Table 3). The odds of having any of the 5 STIs were also greater for those with more lifetime sex partners (aOR: 4.9 [95% CI: 2.2–11.0]; ≥ 3 vs 1 lifetime partner). In the multivariate analysis of HR/6/11 HPV infection, findings were similar (non-Hispanic black versus non-Hispanic white participants, aOR: 2.5 [95% CI: 1.2–5.1]; ≥ 3 vs 1 lifetime sex partner, aOR: 4.4 [95% CI: 1.8–10.5]). In the multivariate analysis that evaluated gonorrhea, chlamydia, trichomonas, or HSV-2 infection, the association

with race/ethnicity was pronounced (non-Hispanic black versus non-Hispanic white participants: aOR: 5.2 [95% CI: 2.0–13.8]). We found no statistically significant interactions between any factors in the models.

DISCUSSION

We found a high burden of STIs among US female adolescents; ~ 1 in 4 was infected with HR/6/11 HPV, gonorrhea, chlamydia, trichomonas, or HSV-2. Applying the prevalence of these 5 common STIs to 2004 US Census data, we predict that nationwide nearly 3 million female individuals aged 14 to 19 have at least 1 of these infections. To our knowledge, this is the first published report of the overall prevalence of the most common STIs among US

female adolescents by using data from a nationally representative sample. On the basis of published estimates for each of the 5 individual STIs,^{5–8} the 24% STI prevalence found in this analysis was not unexpected; however, this aggregate estimate strengthens our understanding of STI epidemiology in female adolescents and highlights the need for enhanced prevention and treatment efforts in this group.

Our data suggest rapid STI acquisition soon after sexual initiation and among teens with few lifetime sex partners. Prevalence of any of these STIs was 26% among female adolescents whose age was the same or 1 year greater than their age at sexual initiation and 20% among those who reported only 1 lifetime sex partner. HPV infection was the predominant STI. Winer et al²¹ observed that college-aged women acquired HPV infection soon after sexual initiation. Our data suggest similarly high rates of HR/6/11 HPV infection soon after sexual initiation among a representative sample of female adolescents. Our findings are also consistent with a recent study of college-aged women that established high risk for HPV acquisition with a first sex partner.²² Although HPV infection was most prevalent, prevalence of the other 4 STIs also increased soon after sexual initiation. These findings emphasize the need to educate young people about sexual issues early, well before sexual initiation.

HPV infection accounted for nearly three quarters of the overall STI prevalence among female adolescents. The NHANES 2003–2004 cycle was the first to include HPV DNA testing.⁵ Findings from our analysis of these data further elucidate the epidemiology of HPV infections in female adolescents. It is important to recognize that 90% of new HPV infections clear without treatment within 2 years²³; however, a proportion of HPV infections will persist, leaving

infected women at risk for cervical cancer. A quadrivalent HPV vaccine, which prevents HPV types that are responsible for 70% of cervical cancers and >90% of genital warts, is now recommended for routine use by girls aged 11 to 12 and for “catch-up” vaccination by those aged 13 to 26.²⁴ Immunization is encouraged before sexual initiation²⁴ because the vaccine is most efficacious among HPV-naive women.^{25,26} Because a substantial proportion of female adolescents acquire HPV infection soon after sexual initiation and parents typically fail to predict the timing of their daughters’ sexual initiation,^{27,28} routine vaccination of preadolescent girls according to current recommendations is of critical importance.

Chlamydia infection was found among 7% of sexually active female adolescents. This treatable, primarily asymptomatic bacterial STI can result in potentially serious long-term consequences for women, including pelvic inflammatory disease and subsequent infertility. To decrease chlamydia prevalence and sequelae, routine annual screening is recommended for sexually active women who are aged ≤ 25 years.²⁹ Despite this, recent data indicate that only 42% of eligible young women who were enrolled in US commercial and Medicaid health plans received annual chlamydia screening in 2007.³⁰ Raising awareness among teens and their parents and health care providers about the need for routine chlamydia screening is a fundamental component of protecting young women from chlamydia sequelae.³¹

Prevalence estimates of HSV-2, trichomonas, and gonorrhea infection were relatively low among female adolescents, although precise estimates for trichomonas and gonorrhea were unreliable because of small numbers. Our HSV-2 seroprevalence estimates may not reflect the full spectrum of genital HSV infection in this population because HSV-1 may account for an in-

creasing proportion of symptomatic first-episode genital herpes, especially among youth.³² In addition, a recent analysis of HSV-2 infections in the United States revealed a five-fold increase in HSV-2 seroprevalence between females who were aged 14 to 19 and those aged 20 to 29 years.⁷ Thus, adolescents soon face markedly increased risk for HSV-2 infection as young adults. HSV-2 infection, the most common cause of genital ulcer disease, is chronic and lifelong and thus differs from the treatable infections trichomonas and gonorrhea; however, all 3 of these infections have been linked with HIV infection. A large body of evidence indicates that HSV-2 infection increases the risk of HIV acquisition³³; trichomonas and gonorrhea have also been associated with increased HIV risk.^{34,35}

We found substantial racial disparities in STI prevalence among US female adolescents. Our findings, like those of other studies,^{36,37} suggest that racial differences in STI prevalence cannot be fully explained by individual risk behaviors. Specifically, non-Hispanic black participants with the same duration of sexual experience or number of lifetime sex partners as their white counterparts were more likely to have at least 1 of the 5 STIs. Social, environmental, and population-level determinants, such as poverty, barriers to health care access, and the characteristics of sexual networks, may contribute to greater STI rates among non-Hispanic black individuals.^{38,39} High STI prevalence within a sexual network increases the risk for STI exposure with any given sexual encounter. Resources for broad strategies to close the racial gap need to be directed to communities at risk. Although STIs among adolescents of all races and ethnicities are a concern, increased efforts are needed to ensure that all subpopulations have the same opportunities for sexual health.

Reducing STIs among female adolescents and thereby protecting their reproductive health requires a comprehensive approach. Adolescents should be provided thorough skill-based education about prevention strategies to lower STI risk, such as delaying sexual initiation, minimizing numbers of sex partners, avoiding concurrent sexual partnerships,⁴⁰ and using condoms consistently and correctly.⁴¹ In addition, adolescents should be informed about the ability of those who are aged ≥ 14 to obtain medical care for STIs without parental consent in all 50 states.⁴² The recent increase in teenage birth rates⁴³ also highlights the importance of providing education about available contraceptive methods. Ideally, adolescents’ parents, educators, and health care providers all would participate in providing such education. Quality parent–adolescent communication about sexuality, sexual risk, and condom use can result in adolescents’ reporting more recent and consistent condom use.^{44,45} Comprehensive sex education programs can increase STI knowledge and prevent risky sexual behaviors.⁴⁶ Health care providers’ counseling about safer sex can result in fewer STIs.⁴⁷ In addition, health care providers can promote recommended primary and secondary STI prevention methods, including routine HPV vaccination of girls at ages 11 to 12 and screening all sexually active female adolescents for chlamydia.

Our analysis had several limitations. We may have underestimated overall STI prevalence because the composite STI variable did not include syphilis or HIV. Only female adolescents who were aged 18 to 19 were tested for syphilis and HIV; no cases of either infection were found (Centers for Disease Control and Prevention, unpublished data). Furthermore, we restricted our analysis of HPV to types that are more likely to cause disease.

Conversely, we may have overestimated STI prevalence because missing laboratory results were more common among lower risk younger and sexually inexperienced participants. However, even if all missing tests from examined adolescents were negative, the overall STI prevalence estimate would be 19.7%, within our estimate's confidence limits. Inaccuracies in self-reported data could have led to misclassification of sexual behavior categories. This may explain prevalent STIs among those who reported no sexual experience, although these estimates were unstable. False-positive test results or STI acquisition from non-penetrative sexual activity (especially for HPV)⁴⁸ could also account for these STIs.

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CONCLUSIONS

Our findings suggest that the STI burden is substantial among female adolescents, and the most common STIs begin to be acquired soon after sexual initiation. These findings highlight the importance of both primary and secondary STI prevention, including early, skill-based sex education; HPV vaccination of preadolescent girls; and chlamydia screening of all sexually active female adolescents. A comprehensive approach designed to decrease STIs among female adolescents would also include steps to lessen racial disparities and to ensure that all adolescents have access to such sex education and sexual health care well before they initiate sexual activity.

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