

The Prevalence of Trichomoniasis in Young Adults in the United States

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Background and Objectives: The prevalence of trichomoniasis in the general population of the United States is unknown. This study provides the first population-based prevalence estimates of trichomoniasis among young adults in the United States.

Methods: The National Longitudinal Study of Adolescent Health (Add Health) is an ongoing prospective cohort study. In a cross-sectional analysis of Wave III of Add Health (N = 12,449), we determined the prevalence of trichomoniasis using a polymerase chain reaction assay.

Results: The estimated overall prevalence of trichomoniasis in U.S. young adults was 2.3% (95% confidence interval [CI], 1.8–2.7%). The prevalence was slightly higher among women (2.8%; 95% CI, 2.2–3.6%) than men (1.7%; 95% CI, 1.3–2.2%). The prevalence increased with age and varied by region, with the south having the highest prevalence (2.8%; 95% CI, 2.2–3.5%). The prevalence was highest among black women (10.5%; 95% CI, 8.3–13.3%) and lowest among white women (1.1%; 95% CI, 0.8–1.6%). Among men, the prevalence was highest among Native Americans (4.1%; 95% CI, 0.4–29.3%) and blacks (3.3%; 95% CI, 2.2–4.9%), and lowest among white men (1.3%; 95% CI, 0.9–1.8%).

Conclusions: Trichomoniasis is moderately prevalent among the general U.S. population of young adults and disturbingly high among certain racial/ethnic groups.

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TRICHOMONIASIS, CAUSED BY THE PROTOZOAN *Trichomonas vaginalis*, is manifested by vaginitis and cervicitis in women and urethritis in men.^{1–3} Although many persons with trichomoniasis are mildly symptomatic or asymptomatic,^{1,4} the infection is associated with substantial morbidity. The total direct medical cost associated with trichomoniasis is estimated to be \$34.2 million annually.⁵

A potential role of trichomoniasis in amplifying HIV transmission also has been identified.^{6–9} Inflammation associated with trichomoniasis may facilitate HIV transmission.^{10–12} Among women, trichomoniasis increases the number of HIV-receptive cells in the genital tract.¹³ *T. vaginalis* has a direct cytopathic effect in vitro and causes punctate microhemorrhages, which could facilitate HIV acquisition.¹⁴ Finally, up to 20% of HIV infections might be attributable to *T. vaginalis* infection in populations in which both infections are prevalent.⁹

Despite these important consequences of trichomoniasis, our current understanding of the prevalence of trichomoniasis is largely limited to clinical settings and special populations. Among women, the prevalence of trichomoniasis has ranged from 3% in adolescent¹⁵ and student health¹⁶ clinics to over 45% in incarcerated women.^{17,18} Among men attending sexually transmitted disease clinics, the prevalence of trichomoniasis has ranged from 3%¹⁹ to 12%.²⁰

The prevalence of trichomoniasis in the general population of the United States is unknown. The National Longitudinal Study of Adolescent Health (Add Health) provides the first opportunity to examine the prevalence of trichomoniasis in the United States among young adults outside of the clinic setting. Using a nucleic acid amplification test of urine samples, we provide prevalence estimates of trichomoniasis for young adult men and women by region, age, and race/ethnicity.

Methods

Study Design and Sample

Add Health is a prospective national cohort study that has followed nearly 20,000 adolescents into adulthood.²¹ We con-

The authors appreciate the support of the Add Health project team, including Joyce Tabor, Francesca Florey, and Lani Cartier. The authors also appreciate the assistance of the laboratory personnel, including Jason Gratz, Natasha Harvey, Sarah Matthews, Susan Blake, and Kuldeep Rawat.

Support was provided in part by the UNC STD Cooperative Research Center (National Institute of Allergy and Infectious Diseases UO131496), the National Institute of Health (HD38210), and The Robert Wood Johnson Foundation Generalist Physician Faculty Scholar Award Program.

This research uses data from Add Health, a program project designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris, and funded by a grant P01-HD31921 from the National Institute of Child Health and Human Development, with cooperative funding from 17 other agencies. Special acknowledgment is due to Ronald R. Rindfuss, PhD, and Barbara Entwisle, PhD, for assistance in the original design. Persons interested in obtaining data files from Add Health should contact Add Health, Carolina Population Center, 123 W. Franklin Street, Chapel Hill, NC 27516-2524.

The results presented in this manuscript were presented at the ISSTD Congress, Abstract 0527, Ottawa, Canada, July 27–30, 2003.

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Received for publication December 7, 2004, and accepted February 14, 2005.

ducted cross-sectional analyses using the most recent round of data collection of Add Health, Wave III (April 2, 2001, to May 9, 2002). The University of North Carolina Institutional Review Board approved all study procedures.

The Add Health study design has been described in detail elsewhere.^{22,23} Briefly, the study used school-based sampling to obtain a nationally representative sample of adolescents in grades 7 through 12 in 1994–1995. To increase the precision of estimates within certain racial/ethnic groups, blacks with college-educated parents and certain Latino and Asian subgroups were oversampled. In Wave III, original Wave I participants were recontacted and invited to participate.

Interviews and Specimen Collection

After obtaining informed consent for the interview, participants were interviewed in their home or other convenient location. Responses to the questionnaire were entered directly into a computer. Potentially sensitive questions such as sexual behavior were administered using computer-assisted self-interview (CASI).

After completion of the interview, consent was obtained to test urine for sexually transmitted infections. Participants received a \$10 incentive for providing a urine specimen. A toll-free number was provided to participants to call for chlamydia and gonorrhea test results. Because the trichomonas assay used in this study has not undergone evaluation by the U.S. Food and Drug Administration for diagnostic use, results of the trichomonas assay were not provided to the participants.

Interviewers provided the participants with verbal instructions for urine collection and a 30-mL specimen container with a mark placed at 15 mL. The target volume for testing was 15 to 20 mL of first-void urine. After collection, the specimens were maintained at approximately 4°C until shipped by overnight courier to the University of North Carolina at Chapel Hill.

We used a urine-based polymerase chain reaction (PCR) assay for detection of *T. vaginalis*.^{24,25} Briefly, urine specimens were processed within 2 days of receipt using the Amplicor CT/NG Urine Specimen Prep kit (Roche Diagnostic Systems, Indianapolis, IN) according to the manufacturer's instructions. Urine preps were stored at –70°C until PCR was performed. For PCR, 50 μ L of thawed urine prep was used as a template in a reaction containing 40 pmol each of primers TVK3 and TVK7 (digoxigenin-labeled).²⁶ PCR products were detected using the PCR DIG enzyme-linked immunosorbent assay (ELISA) detection kit (Roche Diagnostic Systems) with the biotinylated TVK probe and ELISA controls as described previously.^{24,25} Based on the published receiver-operating characteristic analyses of the test, ELISA absorbance values of 3.0 for female specimens²⁴ and 2.0 for male specimens²⁵ were used as cutoffs. A more detailed description of Add Health trichomonas testing is available elsewhere.²⁷ A separate aliquot of urine was prepared and tested for *Chlamydia trachomatis* and *Neisseria gonorrhoeae* using the ligase chain reaction assay (Abbott Laboratories), as described previously.²³

Statistical Analyses

Analyses were conducted using Stata, version 7.0 (Stata Corp., College Station, TX). Prevalences were estimated with 95% confidence intervals. Prevalence ratios with 95% confidence intervals were estimated using Poisson regression for survey data. Prevalence ratios provide a measure of the relative occurrence of trichomoniasis across subgroups. To ensure appropriate point estimates and confidence intervals, all analyses incorporated poststratification weights, stratification, and primary sampling units to account for the complex survey design of Add Health.²⁸

Assessment of Test Performance and Nonresponse Biases

We conducted a sensitivity analysis to assess the effects of 2 potential sources of bias of the prevalence estimates for *T. vaginalis*: 1) test performance and 2) nonresponse. To assess the potential impact of test imperfection on the prevalence estimates, we present the data from a realistic estimate of test performance (sensitivity = 0.889, specificity = 0.99). We also assessed published estimates of sensitivity (men: 0.889, women: 0.908) and specificity (0.934 [women], 0.945 [men]) of the PCR assay, which likely underestimate specificity.^{24,25} These analyses were performed in conjunction with the assessment of the effects of nonresponse.

In Wave III of Add Health, nonresponse bias is a potential concern. Of the original Wave I study population, 6% refused participation and 19% could not be located or were unable to participate for other reasons. Among the population enrolled, 13% did not have a urine specimen available for *T. vaginalis* testing. This nonresponse can potentially bias the prevalence estimates under 2 conditions. First, if the response rate varies by an observed attribute such as race or sex, which is associated with prevalence, the estimates may be biased. We used poststratification weights developed by the Add Health research team to ensure the proper race and sex distribution to minimize this potential bias. Second, the prevalence estimates may be biased if the nonrespondents have a different pattern of prevalence from respondents with similar observed attributes. In this circumstance, an unobserved attribute may influence both survey participation and level of risk. We addressed this second potential source of nonresponse bias through sensitivity analysis using the method described by Brookmeyer and Gail and as previously described for estimates of chlamydial infection prevalence in Add Health.^{23,29} We present results from 2 estimates of a prevalence ratio, *P*, 0.5 and 2.0, reflecting the circumstances in which the persons with missing assays are one half and twice as likely to have *T. vaginalis* infection.

Results

Characteristics of the Study Population

Of the 18,924 participants in the weighted nationally representative sample of Wave I of Add Health, 14,322 (75.7%) were identified and agreed to participate in Wave III. Of these, 1130 (7.9%) refused to provide a urine specimen, 226 (1.6%) were unable to provide a specimen, and 517 (3.6%) specimens could not be processed because of shipping or laboratory problems. Thus, 12,449 (86.9%; 65.8% of Wave I participants) specimens were available for *T. vaginalis* testing.

The study sample, including only those with *T. vaginalis* results, comprised 5916 males (47.5%) and 6533 females (52.5%, Table 1). Most participants (54.3%) were white with substantial representation of Latinos (16.3%), blacks (21.5%), and Asians (7.0%). Native Americans (0.9%) were a small proportion of the study sample. The mean age of the study sample was 22.0 years (standard deviation 1.8 years).

Prevalence of Trichomoniasis

The estimated prevalence of trichomoniasis in young adults in the United States was 2.3% (95% confidence interval [CI], 1.8–2.7%), based on this nationally representative sample. Women (2.8%) were more likely to be infected than men (1.7%; prevalence ratio: 1.64; 95% CI, 1.25–2.15). The prevalence of trichomoniasis in the south (2.8%) was 2.01 (95% CI, 1.10–3.65) times higher than in the west (1.4%).

TABLE 1. Characteristics of the Wave III Add Health Study Population With *Trichomonas vaginalis* Assay Results (N = 12,449)

	No.	Unweighted %*	Weighted %†
Sex			
Male	5916	47.5	50.9
Female	6533	52.5	49.1
Race/ethnicity			
White	6742	54.3	67.6
Latino	2026	16.3	11.7
Black	2664	21.5	16.2
Asian	873	7.0	3.7
Native American	113	0.9	0.8
Age (years)			
18–20	2911	23.4	29.1
21–22	4361	35.0	33.1
23–24	4439	35.7	31.2
≥25	738	5.9	6.6
Region			
West	3161	25.4	17.2
Midwest	2917	23.5	29.3
South	4848	39.0	40.5
Northeast	1514	12.2	13.0

*Unweighted % reflects the percentage of the characteristic in the study sample.

†Weighted % reflects the representative proportion in the target U.S. population based on the Add Health sample with appropriate sampling weights.

Group numbers may not sum to total number as a result of a limited amount of missing data.

The prevalence of trichomoniasis varied significantly by age (Table 2). Among males, the prevalence was less than 1% among 18 to 20 year olds (0.8%; 95% CI, 0.4–1.4%), but greater than 2% in men 25 years and older (2.8%; 95% CI, 1.4–5.3%). A similar pattern was seen among women, but the prevalence was considerably higher. Among women 20 years or younger, the prevalence

was 2.2% (95% CI, 1.4–3.5%) and in women 25 years or older, the prevalence was 6.1% (95% CI, 3.5–10.2%).

Trichomoniasis was more than 5 times more prevalent among blacks (6.9%; 95% CI, 5.4–8.8%; prevalence ratio: 5.85; 95% CI, 4.13–8.29) as compared with whites (1.2%; 95% CI, 0.9–1.5). The prevalence was also high in Native Americans (4.1%; 95% CI, 0.8–18.0), although this estimate was imprecise. Intermediate prevalences were observed in Latinos (2.1%) and Asians (1.8%).

Considering gender and race, the prevalence of trichomoniasis was highest among black (10.5%; 95% CI, 8.3–13.3%) and Native American (4.2%; 95% CI, 0.6–25.3%) women (Table 3). The prevalence among black women was nearly 10 times higher than the prevalence among white women (1.1%; 95% CI, 0.8–1.6%; prevalence ratio: 9.54; 95% CI, 6.19–14.72). Among men, the prevalence of trichomoniasis was also highest in blacks (3.3%; 95% CI, 2.2–4.9%) and Native Americans (4.1%; 95% CI, 0.4–29.3%). White men (1.3%; 95% CI, 0.9–1.8%) had the lowest prevalence of trichomoniasis.

At the time of the interview, the majority of infections were asymptomatic. Among men with trichomoniasis, only 2.3% (95% CI, 0.3–14.8%) reported urethral discharge or dysuria. Similarly, 2.0% (95% CI, 0.7–5.8%) of women with trichomoniasis reported vaginal discharge or dysuria.

A substantial overlap of trichomoniasis with chlamydial infection was observed. Although the overall prevalence of chlamydial infection was 4.2% in this population,²³ among persons with trichomoniasis, the prevalence of chlamydial infection was 12.7% (95% CI, 8.4–18.7%). This overlap was most pronounced for women with trichomoniasis, in whom the prevalence of chlamydial infection was 17.2% (95% CI, 11.4–25.0%). In men with trichomoniasis, the prevalence of chlamydial infection was 5.6% (95% CI, 2.2–13.7%).

Sensitivity Analyses

To assess the potential impact of nonresponse and diagnostic test performance on the prevalence estimates for trichomoniasis, we conducted several sensitivity analyses (Table 4).

TABLE 2. Prevalence of Trichomoniasis Among Young Adults in the United States

	Prevalence*	95% CI	Prevalence Ratio	95% CI
Sex				
Male	1.7	1.3–2.2	—	
Female	2.8	2.2–3.6	1.64	1.25–2.15
Race/ethnicity				
White	1.2	0.9–1.5	—	
Latino	2.1	1.3–3.4	1.76	1.04–3.00
Black	6.9	5.4–8.8	5.85	4.13–8.29
Asian	1.8	0.9–3.9	1.55	0.70–3.46
Native American	4.1	0.8–18.0	3.50	0.72–17.03
Age (years)				
18–20	1.5	1.0–2.3	—	
21–22	2.6	2.0–3.4	1.73	1.12–2.67
23–24	2.2	1.6–3.0	1.46	0.88–2.43
≥25	4.0	2.6–6.2	2.68	1.47–4.86
Region				
West	1.4	0.8–2.4	—	
Midwest	2.2	1.4–3.5	1.59	0.78–3.24
South	2.8	2.2–3.5	2.01	1.10–3.65
Northeast	2.0	1.2–3.3	1.46	0.70–3.04

*Prevalence is determined using appropriate sample weights from the Add Health study sample to provide representative prevalence estimates in the target US population. CI indicates confidence interval.

TABLE 3. Prevalence of Trichomoniasis by Sex and Race/Ethnicity Among Young Adults in the United States

	Prevalence	95% CI	Prevalence Ratio	95% CI
Male				
White	1.3*	0.9–1.8	—	
Latino	2.0	1.1–3.7	1.57	0.75–3.28
Black	3.3	2.2–4.9	2.60	1.47–4.60
Asian	2.3	0.8–6.0	1.80	0.61–5.29
Native American	4.1	0.4–29.3	3.23	0.34–30.62
Female				
White	1.1	0.8–1.6	—	
Latino	2.2	1.2–4.1	1.99	1.01–3.95
Black	10.5	8.3–13.3	9.54	6.19–14.72
Asian	1.3	0.4–4.2	1.17	0.33–4.12
Native American	4.2	0.6–25.3	3.83	0.52–28.18

*Prevalence is determined using appropriate sample weights from the Add Health study sample to provide representative prevalence estimates in the target US population. CI indicates confidence interval.

TABLE 4. Sensitivity Analyses Demonstrating Estimated Prevalence of Trichomoniasis Accounting for Nonresponse to the Survey and Diagnostic Test Performance

	Sensitivity = 1.0; Specificity = 1.0 (Nonresponse Only)					
	MAR		P = 0.5		P = 2.0	
	Prevalence	95% CI	Prevalence	95% CI	Prevalence	95% CI
Overall	2.3	1.4–3.3	1.8	1.1–2.5	3.2	2.0–4.8
Male	1.7	1.3–2.2	1.3	1.0–1.7	2.5	1.8–3.3
White	1.3	0.8–1.8	1.0	0.6–1.4	1.8	0.9–2.6
Latino	1.9	0.7–3.5	1.4	0.5–2.6	2.9	0.8–5.2
Black	3.3	1.9–5.0	2.5	1.5–3.7	4.8	2.0–7.5
Asian	2.5	0.2–5.4	1.9	0.2–4.1	3.5	0.3–8.0
Native American	4.2	0.0–13.0	2.7	0.0–8.5	7.2	0.0–22.4
Female	2.8	2.3–3.3	2.2	1.8–2.6	4.0	3.3–4.7
White	1.1	0.7–1.6	0.9	0.6–1.3	1.5	0.7–2.2
Latino	2.2	1.0–3.7	1.6	0.7–2.7	3.3	1.1–5.5
Black	10.6	8.6–12.7	8.3	6.8–10.1	15.0	9.3–18.1
Asian	1.3	0.1–3.1	1.0	0.1–2.4	1.9	0.1–4.4
Native American	4.0	0.0–12.8	2.9	0.0–9.2	6.2	0.0–19.4
Sensitivity = 0.889; Specificity = 0.99						
	MAR		P = 0.5		P = 2.0	
	Prevalence	95% CI	Prevalence	95% CI	Prevalence	95% CI
	Prevalence	95% CI	Prevalence	95% CI	Prevalence	95% CI
Overall	1.4	0.8–2.5	0.9	0.6–4.3	2.5	1.2–8.3
Male	0.8	0.3–1.4	0.4	0.0–0.8	1.7	1.0–2.6
White	0.3	0.0–0.9	0.0	0.0–0.5	0.9	0.1–1.8
Latino	1.1	0.0–2.8	0.5	0.0–1.8	2.1	0.0–4.8
Black	2.6	1.0–4.5	1.7	0.5–3.1	4.4	2.0–7.4
Asian	1.7	0.0–5.0	1.0	0.0–3.5	2.9	0.0–7.9
Native American	3.6	0.0–13.7	1.9	0.0–8.6	7.1	0.0–24.4
Female	2.0	1.5–2.6	1.4	0.9–1.8	3.4	2.6–4.2
White	0.1	0.0–0.6	0.0	0.0–0.3	0.6	0.0–1.3
Latino	1.4	0.0–3.0	0.7	0.0–2.0	2.6	0.5–5.1
Black	10.9	8.6–13.4	8.3	6.6–10.3	15.9	12.6–19.5
Asian	0.3	0.0–2.4	0.0	0.0–1.6	1.0	0.0–3.9
Native American	3.4	0.0–13.4	2.2	0.0–9.3	5.9	0.0–20.9

MAR indicates missing at random; P = prevalence ratio of infection for the missing assays compared with the observed assays; CI = confidence interval derived from bootstrap analyses.

To provide a direct estimate of the potential effect of nonresponse bias, we estimated the prevalence under different nonresponse conditions without considering test performance (Table 4, top). If persons without urine specimens were missing at random, the prevalence estimate is minimally affected (2.3%). If the prevalence of *T. vaginalis* infection was twice as high among persons without a urine specimen as among those with a urine specimen ($P = 2.0$), the overall estimate for *T. vaginalis* infection would increase to 3.2%. If the prevalence of *T. vaginalis* infection was half as great among nonresponders ($P = 0.5$), the overall prevalence would decrease to 1.8%.

We then examined estimates under conditions that corrected for expected test performance (sensitivity = 0.889, specificity = 0.99; Table 4, bottom). In this circumstance, estimates were slightly lower than those obtained with adjustment for nonresponse alone (Table 4, top). However, estimates for blacks and Native Americans, especially for women, changed relatively little.

Discussion

Add Health provides the first national estimates of the prevalence of trichomoniasis in the United States. We observed a high prevalence of this protozoan infection among specific demographic subgroups, including blacks and Native Americans. The prevalence was intermediate among Latinos and Asians and lowest among whites. The prevalence was higher among women and, generally, increased with age. We also observed a substantial overlap between chlamydial infection and trichomoniasis, suggesting the potential for common sexual networks.

Unlike chlamydial infection and gonorrhea, reporting of diagnoses of trichomoniasis to health departments is not required. Therefore, previous data regarding the prevalence of this infection in the United States are limited. Most prevalence estimates were obtained in clinical settings using wet preparations, a relatively insensitive test. Many studies included women only. In these studies, the prevalence of trichomoniasis was generally much higher, often 10% to 30%,^{15,30–32} than we observed in the general population using a more sensitive PCR assay. This discrepancy is largely expected based on the nature of the study populations. Many clinic-based studies included primarily symptomatic women typically at high risk for trichomoniasis and other sexually transmitted infections.¹⁵ In contrast, Add Health provides an estimate among primarily asymptomatic women in the general population without requiring presentation to a clinic.

The disparity in the prevalence of trichomoniasis between racial/ethnic groups is particularly worrisome, because the estimates are population-based. Racial/ethnic disparities have been observed previously in clinic-based studies.^{17,33–35} Disparities in clinic-based estimates are often the result of differences in health-seeking behavior or testing strategies. In contrast, all eligible persons in Add Health were tested. Thus, these disparities appear to be real and cannot be easily explained by selection or other forms of bias. Future efforts will be necessary to attempt to identify potential explanations for these differences.

The two most significant potential biases in these data are selection bias related to the study sample and measurement error associated with test performance. Wave III of Add Health captured 76% of the original study population and an additional 10% did not have urine specimens for testing. Much of the potential bias, at least in terms of broad demographic categories, is accounted for in the poststratification weights created for use with Wave III of Add Health. These weights ensure that the current study sample provides an appropriate representation of the target U.S. population. Furthermore, the magnitude of the bias as a result of loss to follow

up and nonresponse in Wave III Add Health data appears to be small.³⁶

Within the study sample, systematic differences between those for whom specimens were available and those without specimens could also bias the prevalence estimates. For example, if persons who did not have specimens available had lower prevalence, then our estimates would be high. However, nonresponse seems to have a relatively small effect on most of the prevalence estimates based on our sensitivity analysis (Table 4, top).

Test performance is another potential source of error in our estimates. We used a newly developed PCR assay to identify *T. vaginalis* in the urine specimens of our study population.^{24,25} Urine may not be the ideal specimen for identification of trichomoniasis, potentially leading to underestimation of the prevalence in this group.³⁷ Despite this limitation, this assay is a substantial advance over previously available diagnostic methods such as culture and wet preparation. This study would not have been logistically feasible if culture and wet preparations were required. In addition, the PCR assay has substantially increased sensitivity as compared with the traditional tests. Indeed, the low sensitivity of culture and wet preparations is problematic for the assessment of the specificity of the PCR assay.^{24,25} When we used the published estimates of sensitivity and specificity of the PCR assay in our sensitivity analyses, the prevalence of trichomoniasis was ≤ 0 for many subgroups (data not shown). This strongly suggests that the specificity of the PCR assay is actually higher than the published reports. Using plausible estimates to correct for PCR test performance, we observed some reduction in prevalence for the lowest prevalence groups, as expected. However, estimates for blacks and Native Americans were changed only minimally.

The prevalence of trichomoniasis is sufficiently high to raise concerns about the potential impact on trichomonas-associated morbidity. Trichomoniasis is more than just a “nuisance infection.” *T. vaginalis* exerts a direct cytopathic effect in vitro and recruits HIV-receptive cells to the genital tract, which may increase the risk of HIV acquisition for women.^{13,14} In men, symptomatic infection with *T. vaginalis* has been shown to increase the HIV viral load in semen 6-fold.³⁸ Whether this infection increases seminal HIV viral load in asymptomatic men is not known. However, 60% of men in rural Tanzania with asymptomatic trichomoniasis demonstrated a positive leukocyte esterase dipstick,³⁹ suggesting the presence of inflammation.

This potential association of trichomoniasis with increased transmission of and increased susceptibility to HIV infection is particularly concerning for this population of sexually active young adults. Among women aged 15 to 19 years, the incidence of HIV has increased.⁴⁰ Furthermore, HIV increased in women aged 20 to 25 years as a result of increasing heterosexual transmission.⁴¹ The HIV epidemic has increasingly involved young adult heterosexuals, emphasizing the need for reduction of modifiable risk factors, including sexually transmitted infections. The potential for overlap of trichomoniasis and HIV infection networks is particularly strong in blacks and in the southern region of the United States.

In summary, the prevalence of trichomoniasis in young adults in the United States is moderately high. The minimal attention given to trichomoniasis in screening and reporting may contribute to this high prevalence. In the past, the lack of convenient, highly accurate tests has limited the potential for screening. The development of *T. vaginalis* assays for urine^{24,25} and self-collected vaginal swabs⁴² should facilitate screening if these tests can be developed for widespread use. Because the potential consequences of this infection are significant, greater efforts are needed to reduce the prevalence. Future work must also address the marked disparity of

trichomoniasis, and other sexually transmitted infections, across racial/ethnic groups.

References

- Jackson DJ, Rakwar JP, Chohan B, et al. Urethral infection in a workplace population of East African men: Evaluation of strategies for screening and management. *J Infect Dis* 1997; 175:833–838.
- Krieger JN, Jenny C, Verdon M, et al. Clinical manifestations of trichomoniasis in men. *Ann Intern Med* 1993; 118:844–849.
- Pastorek JG 2nd, Cotch MF, Martin DH, Eschenbach DA. Clinical and microbiological correlates of vaginal trichomoniasis during pregnancy. The Vaginal Infections and Prematurity Study Group. *Clin Infect Dis* 1996; 23:1075–1080.
- Paxton LA, Sewankambo N, Gray R, et al. Asymptomatic non-ulcerative genital tract infections in a rural Ugandan population. *Sex Transm Infect* 1998; 74:421–425.
- Chesson HW, Blandford JM, Gift TL, Tao G, Irwin KL. The estimated direct medical cost of sexually transmitted diseases among American youth, 2000. *Perspect Sex Reprod Health* 2004; 36:11–19.
- Ghys PD, Diallo MO, Ettiegne-Traore V, et al. Genital ulcers associated with human immunodeficiency virus-related immunosuppression in female sex workers in Abidjan, Ivory Coast. *J Infect Dis* 1995; 172:1371–1374.
- Laga M, Manoka A, Kivuvu M, et al. Non-ulcerative sexually transmitted diseases as risk factors for HIV-1 transmission in women: Results from a cohort study. *AIDS* 1993; 7:95–102.
- ter Meulen J, Mgaya HN, Chang-Claude J, et al. Risk factors for HIV infection in gynaecological inpatients in Dar es Salaam, Tanzania, 1988–1990. *East Afr Med J* 1992; 69:688–692.
- Sorvillo F, Smith L, Kerndt P, Ash L. *Trichomonas vaginalis*. HIV and African-Americans. *Emerg Infect Dis* 2001; 7:927–932.
- Kiviat NB, Paavonen JA, Brockway J, et al. Cytologic manifestations of cervical and vaginal infections. I. Epithelial and inflammatory cellular changes. *JAMA* 1985; 253:989–996.
- Kreiss J, Willerford DM, Hensel M, et al. Association between cervical inflammation and cervical shedding of human immunodeficiency virus DNA. *J Infect Dis* 1994; 170:1597–1601.
- Hobbs MM, Kazembe P, Reed AW, et al. *Trichomonas vaginalis* as a cause of urethritis in Malawian men. *Sex Transm Dis* 1999; 26:381–387.
- Levine WC, Pope V, Bhoemaker A, Tambe P, Lewis JS, Zaidi AA. Increase in endocervical CD4 lymphocytes among women with nonulcerative sexually transmitted diseases. *J Infect Dis* 1998; 177:167–174.
- Gilbert R, Elia G, Beach D, Klaessig S, Singh B. Cytopathogenic effect of *Trichomonas vaginalis* on human vaginal epithelial cells cultured in vitro. *Infect Immun* 2000; 68:4200–4206.
- Bunnell R, Dahlberg L, Rolfs R, et al. High prevalence and incidence of sexually transmitted diseases in urban adolescent females despite moderate risk behaviors. *J Infect Dis* 1999; 180:1624–1631.
- McCormack WM, Evrard JR, Laughlin CF, et al. Sexually transmitted conditions among women college students. *Am J Obstet Gynecol* 1981; 139:130–133.
- Shuter J, Bell D, Graham D, Holbrook KA, Bellin EY. Rates of and risk factors for trichomoniasis among pregnant inmates in New York City. *Sex Transm Dis* 1998; 25:303–307.
- Bell TA, Farrow JA, Stamm WE, Critchlow CW, Holmes KK. Sexually transmitted diseases in females in a juvenile detention center. *Sex Transm Dis* 1985; 12:140–144.
- Joyner JL, Douglas JM, Ragsdale S, Foster M, Judson FN. Comparative prevalence of infection with *Trichomonas vaginalis* among men attending a sexually transmitted diseases clinic. *Sex Transm Dis* 2000; 27:236–240.
- Borchardt KA, al-Haraci S, Maida N. Prevalence of *Trichomonas vaginalis* in a male sexually transmitted disease clinic population by interview, wet mount microscopy, and the InPouch TV test. *Genitourinary Medicine* 1995; 71:405–406.
- Harris KM, Florey F, Tabor J, Bearman PS, Jones J, Udry JR. The National Longitudinal Study of Adolescent Health: Research Design. Available at: <http://www.cpc.unc.edu/projects/addhealth/design>. Accessed August 24, 2004.
- Resnick MD, Bearman PS, Blum RW, et al. Protecting adolescents from harm: findings from the National Longitudinal Study of Adolescent Health. *JAMA* 1997; 278:823–832.
- Miller WC, Ford CA, Morris M, et al. Prevalence of chlamydial and gonococcal infections among young adults in the United States. *JAMA* 2004; 291:2229–2236.
- Kaydos SC, Swygard H, Wise SL, et al. Development and validation of a PCR enzyme-linked immunosorbent assay with urine for use in clinical research settings to detect *Trichomonas vaginalis* in women. *J Clin Microbiol* 2002; 40:89–95.
- Kaydos-Daniels SC, Miller WC, Hoffman I, et al. Validation of a urine-based PCR-enzyme-linked immunosorbent assay for use in clinical research settings to detect *Trichomonas vaginalis* in men. *J Clin Microbiol* 2003; 41:318–323.
- Kengne P, Veas F, Vidal N, Rey JL, Cuny G. *Trichomonas vaginalis*: Repeated DNA target for highly sensitive and specific polymerase chain reaction diagnosis. *Cell Mol Biol* 1994; 40:819–831.
- Add Health Biomarker Team. Biomarkers in Wave III of the Add Health Study. Available at: <http://www.cpc.unc.edu/projects/addhealth/files/biomark.pdf>. Accessed October 26, 2003.
- Chantala K, Tabor J. Strategies to Perform a Design-Based Analysis Using the Add Health Data. Carolina Population Center, University of North Carolina at Chapel Hill. Available at: <http://www.cpc.unc.edu/projects/addhealth/files/weight1.pdf>. Accessed August /28, 2004.
- Brookmeyer R, Gail MH. *AIDS Epidemiology: A Quantitative Approach*. New York: Oxford University Press, 1994.
- Cu-Uvin S, Hogan JW, Warren D, et al. Prevalence of lower genital tract infections among human immunodeficiency virus (HIV)-seropositive and high-risk HIV-seronegative women. HIV Epidemiology Research Study Group. *Clin Infect Dis* 1999; 29:1145–1150.
- DeHovitz JA, Kelly P, Feldman J, et al. Sexually transmitted diseases, sexual behavior, and cocaine use in inner-city women. *Am J Epidemiol* 1994; 140:1125–1134.
- Pabst K, Reichart C, Knud-Hansen C, et al. Disease prevalence among women attending a sexually transmitted disease clinic varies with reason for visit. *Sex Transm Dis* 1992; 19:88–91.
- Cotch MF, Pastorek JG 2nd, Nugent RP, Yerg DE, Martin DH, Eschenbach DA. Demographic and behavioral predictors of *Trichomonas vaginalis* infection among pregnant women. The Vaginal Infections and Prematurity Study Group. *Obstet Gynecol* 1991; 78:1087–1092.
- Shafer MA, Sweet RL, Ohm-Smith MJ, Shalwitz J, Beck A, Schachter J. Microbiology of lower genital tract in postmenarchal adolescent girls: differences by sexual activity, contraception, and presence of nonspecific vaginitis. *J Pediatr* 1985; 107:974–981.
- Ipsen J, Feigl P. A biomathematical model for prevalence of *Trichomonas vaginalis*. *Am J Epidemiol* 1970; 91:175–184.
- Chantala K, Kalsbeek WD, Andraca E. Non-Response in Wave III of the Add Health Study. Carolina Population Center [web site]. Available at: <http://www.cpc.unc.edu/projects/addhealth/files/W3nonres.pdf>. Accessed August 28, 2004.
- Lawing LF, Hedges SR, Schwelbe JR. Detection of trichomonosis in vaginal and urine specimens from women by culture and PCR. *J Clin Microbiol* 2000; 38:3585–3588.
- Cohen MS. Sexually transmitted diseases enhance HIV transmission: No longer a hypothesis. *Lancet* 1998; 351(suppl III):5–7.
- Watson-Jones D, Mugeye K, Mayaud P, et al. High prevalence of trichomoniasis in rural men in Mwanza, Tanzania: Results from a population based study. *Sex Transm Infect* 2000; 76:355–362.
- Albrecht H. HIV incidence trends in young women. *AIDS Clinical Care* 2001; 13:97.
- Rosenberg P, Biggar R. Trends in HIV incidence among young adults in the United States. *JAMA* 1998; 279:1894–1899.
- Wiesenfeld HC, Lowry DL, Heine RP, et al. Self-collection of vaginal swabs for the detection of chlamydia, gonorrhea, and trichomoniasis: Opportunity to encourage sexually transmitted disease testing among adolescents. *Sex Transm Dis* 2001; 28:321–325.