Current Issues and Considerations Regarding Trichomoniiasis and Human Immunodeficiency Virus in African-Americans

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INTRODUCTION

Trichomonas vaginalis is an important protozoan parasite transmitted principally through vaginal intercourse. The spectrum of clinical trichomoniiasis in women has been well described, ranging from vaginitis to an asymptomatic carrier state, with 50% or greater of all infected women becoming symptomatic within 6 months of infection. In men, the infection is usually asymptomatic, but those individuals who develop symptoms typically experience urethritis. Despite a relative paucity of studies of the prevalence and incidence of trichomoniiasis, recent publications suggest that T. vaginalis is one of the most common sexually transmitted infections (STIs) worldwide with over 170 million cases per year. Over 8 million new cases are estimated to occur annually in the United States alone (1, 12, 109). Although the organism appears to be highly prevalent and has a widespread geographic distribution, Trichomonas is not a locally or nationally reportable disease and has not been the focus of active control programs. This neglect is likely a function of the relatively mild nature of the disease (109), the perceived lack of effect on fertility despite the fact that recent studies have indicated that T. vaginalis can cause pelvic inflammatory disease in both human immunodeficiency virus (HIV)-infected and -uninfected women (66), and the historic absence of association with adverse birth outcomes (although recent data suggest a possible causal role in low birth weight and prematurity [14]). However, it is now recognized that Trichomonas may play a critical and underrecognized role in amplifying HIV transmission, and it may have a major impact on the epidemic dynamics of HIV in African-American communities.

BIological rationale: expanding the portals of entry and exit

T. vaginalis infection typically elicits an aggressive local cellular immune response with inflammation of the vaginal epithelium and exocervix in women and the urethra of men (43, 79). This inflammatory response induces the recruitment of leukocytes, including HIV target cells such as CD4+ -bearing lymphocytes and macrophages to which HIV can bind and gain access (51, 79). In addition, T. vaginalis can frequently cause punctuate mucosal hemorrhages, which potentially compromise the mechanical barrier to infection (26). Trichomoniiasis may also increase the risk of HIV-1 acquisition by increasing susceptibility to bacterial vaginosis or persistence of abnormal vaginal flora (65).

In an HIV-negative person, both the leukocyte infiltration and genital lesions induced by Trichomonas may enlarge the portal of entry for HIV by increasing the number of target cells for the virus and allowing direct viral access to the bloodstream through open lesions. Similarly, in an HIV-infected person, hemorrhages and inflammation can increase the level of virus-laden body fluids, the numbers of HIV-infected lymphocytes and macrophages present in the genital contact area, or both. The resulting increase of both free virus and virus-infected leukocytes can expand the portal of exit, thereby heightening the probability of HIV exposure and transmission to an uninfected partner. Increased cervical shedding of HIV has been shown to be associated with cervical inflammation (44, 101), and substantially increased urethral viral loads as well as increased HIV RNA concentrations in semen from men with Trichomonas infection have been documented (35). Furthermore, among women, it has been demonstrated that trichomo-
niaisis increases the number of HIV-receptive cells in the genital tract (51). In addition, *T. vaginalis* has been shown to degrade secretory leukocyte protease inhibitors, which can block HIV-1 attachment to cells (22). Other laboratory studies have further supported the role of trichomoniasis in HIV-1 transmission. Two biological mechanisms that could contribute to the epidemiological association of *T. vaginalis* with the sexual transmission of HIV-1 have been identified. First, *T. vaginalis* disruption of urogenital epithelial monolayers could facilitate the passage of HIV-1 to underlying layers. Second, activation of local immune cells by *T. vaginalis* in the presence of infectious HIV-1 might lead to increased viral replication (32). Moreover, unlike other STIs, most patients with Trichomonas infection are asymptomatic or mildly symptomatic (106); therefore, they are likely to continue to remain sexually active in spite of infection (42). Studies suggest that approximately 50% to 70% of persons with *T. vaginalis* have subclinical infection (106). This results in a large reservoir of asymptomatic individuals, with high rates of reinfection and a higher community prevalence (39).

**EMPIRICAL EVIDENCE IMPLICATING TRICHOMONAS IN HIV TRANSMISSION**

Data from studies conducted in Africa have shown an association between *Trichomonas* and HIV infection, suggesting a 1.5- to 3-fold increase in HIV transmission (29, 48, 59, 97). A cross-sectional study conducted among 1,209 female sex workers in the Ivory Coast found an association between HIV and *Trichomonas* infection in bivariate analysis (crude odds ratio, 1.8; 95% confidence intervals, 1.3 and 2.7). In another cross-sectional study performed in Tanzania among 359 women admitted to a hospital for gynecologic conditions, *Trichomonas* was more common in women with HIV infection in multivariate analyses (odds ratio, 2.96; no confidence intervals provided; \( P < 0.001 \)). While such cross-sectional studies are limited by the issue of temporal ambiguity, i.e., a lack of information on whether *Trichomonas* infection preceded HIV, these preliminary findings were subsequently reinforced in two prospective studies from sub-Saharan Africa as well as in a nested case-control study. The first study from Zaire, in which 431 HIV-negative female prostitutes were evaluated over time, found that prior *Trichomonas* infection was associated with a twofold-increased rate of HIV seroconversion in a multivariate analysis (48). The second prospective study, conducted among 1,335 HIV-negative women in Kenya, identified that after adjustment for potential confounders, trichomoniasis was associated with a 1.52-fold-increased risk of HIV-1 acquisition in a multivariate analysis (59). The third study, conducted in Uganda and Zimbabwe, was a nested case-control study of 213 HIV seroconverters from a total population of 4,450 study subjects and found an adjusted odds ratio for HIV acquisition of 2.74 (98). Finally, using a variety of analysis scenarios, Sexton et al. estimated the effect of *T. vaginalis* infection on the risk of HIV acquisition to be 1.59 to 2.10 (89).

Note that such prospective studies give an estimate only of the amplifying effect of *Trichomonas* through inducing greater susceptibility in an HIV-negative individual. It does not provide a measure of the HIV transmission that may result from expanding the portal of exit and increasing the level of infectiousness in an individual who is coinfected with HIV and *Trichomonas*. These increased rates of HIV transmission should therefore be viewed as underestimates.

**DATA ON THE PREVALENCE OF T. VAGINALIS AMONG U.S. WOMEN**

Since trichomoniasis is not a reportable condition in most health jurisdictions, and prevalence surveys for STIs often do not include attempts to recover *Trichomonas*, information on the occurrence of *T. vaginalis* infection in the United States is meager. In addition, the relatively few published studies with information on the prevalence of *T. vaginalis* infection have generally been conducted among highly selected convenience samples, typically included only women, or were limited by small numbers of participants. Even among those studies that were conducted with the primary purpose of assessing the prevalence of *Trichomonas*, many of them often used diagnostic techniques with relatively low sensitivity such as wet mount, stained preparations, or Papanicolaou smear. Wet mount, the most commonly used method, has an estimated sensitivity of 58% to 77% compared with culture and is highly dependent on the skill of the microscopist as well as the specimen examined (45, 71, 105); the sensitivity of Papanicolaou smear is approximately 57%. The sensitivity of culture compared with PCR has been estimated to be 70% (55). Such highly sensitive PCR and related techniques are not routinely used, nor are they as readily available for *Trichomonas* as they are for other STIs (100). Furthermore, recent studies have indicated that the viability of the organism has a profound effect on all testing modalities. When urine specimens are being collected, the sensitivity is affected by a loss of viability as early as 1 h after specimen collection (91). As a result of suboptimal laboratory methods, studies of *T. vaginalis* have often substantially underestimated the prevalence of infection. The new membrane flow OSOM assay from Genzyme is thought to have excellent sensitivity and specificity, with limited skill required to process and interpret the test, but the test is available for use only on female specimens and may be cost-prohibitive (11). In spite of this, levels of infection have typically been high, with reported overall prevalences ranging from 3% to 58% and an unweighted average across studies of 19.6% (4, 6, 7, 10, 15, 18, 20, 24, 33, 37, 60, 62, 64, 69, 70, 77, 90, 92, 95, 99, 108).

Table 1 lists published reports on the occurrence of *T. vaginalis* infection among women conducted among U.S. populations from 1964 through 2007. Although not necessarily complete, a comprehensive search through MEDLINE and review of articles yielded only 20 reports during this 43-year period. Evaluated populations have included such groups as sexually transmitted disease (STD) clinic patients, inner-city populations, pregnant women, university students, adolescents, incarcerated populations, and women with HIV infection. Only two studies were truly population based, and one of those studies, Add Health, was restricted to adolescents (63, 64, 95).

**DATA ON THE INCIDENCE OF T. VAGINALIS IN THE UNITED STATES**

Even fewer studies have assessed the incidence of trichomoniasis in the United States. In a study conducted from 1992 to
1995 among a cohort of 212 women with HIV in Los Angeles County, *Trichomonas* infection was the most frequently identified sexually transmitted disease and was found in 37 (17.4%) women, representing a crude incidence rate of 14.1 per 100 person-years (93). The crude rate was highest in black women (69.0 per 100 person-years). A recent prospective study conducted from 1990 to 1998 in New Orleans, which monitored women coinfected with HIV and *T. vaginalis*, documented high reinfection rates (16.1 per 100 person-years) of *T. vaginalis* (68). Among a predominantly black group of HIV-infected and high-risk women monitored in New York City from 1990 to 1994, *T. vaginalis* was the most frequent incident STI (107). A recent prospective study conducted among African-American women in New York City who use drugs found an incidence of 35.1 cases per 100 person-years at risk (63).

### PREVALENCE OF *T. VAGINALIS* AMONG MEN IN THE UNITED STATES

Estimation of the prevalence of *T. vaginalis* in men has been difficult because most infected men are asymptomatic. Very few published studies have assessed the prevalence of *T. vaginalis* among men, and, as is the case for women, these studies typically have included relatively small samples from selected populations. Often, data on race-specific prevalences are not provided. Reported prevalence rates have varied depending on the population studied and the diagnostic technique used. Most previously published studies that have been conducted have been done with males visiting STD clinics and found prevalences as high as 25% among sexual contacts of women with trichomoniasis and as high as 6% among heterosexual men visiting STD clinics. One study conducted with young men (most or all of whom were black) enrolled in an inner-city residential youth training program found that the prevalence of trichomoniasis was 58% (81). Among men attending an STD clinic in Seattle-King County from 1987 to 1990, 6% of 300 randomly selected men were found to be infected with *Trichomonas* by culture techniques; 22% of 147 contacts of women with *T. vaginalis* were also positive (46). In a study published in 1995 conducted in Richmond, California, 12% of 204 male patients from an STD clinic were culture positive for *T. vaginalis* (8). Among 454 consecutive men attending an STD clinic in Denver in 1998, 2.8% were found to be infected by culture method (40). In a small-scale study published in 1991, among 16- to 22-year-old black men enrolled in an inner-city residential youth job-training program, *Trichomonas* was recovered from 55% of 85 participants and was the most common STI identified (81). Schwebke and Hook were also able to demonstrate a 17.3% prevalence among men attending an STD clinic, the majority of whom were asymptomatic (85). One study conducted on male partners of women infected with *Trichomonas* found 71.7% of men to be infected (88). Data on race-specific prevalences of *Trichomonas* infection among U.S. males are not available. Only one study, Add Health, has simultaneously assessed the prevalence in males and females. That study found that the prevalence was slightly higher in female adolescents than in male adolescents, 2.8% versus 1.7% (64). While that study, along with studies conducted individually with males and females, indicates that the prevalence may be higher in women than in men, the data are so limited and potentially biased that any such conclusions must be made cautiously.

### RACE AND TRICHOMONAS

Table 2 presents data, where available, on the prevalence of *Trichomonas* among women, by race, in the United States. In each study that has presented information on race and/or ethnicity, the prevalence of *Trichomonas* has been highest in African-Americans (10.5% to 51%), ranging from approximately...
TABLE 2. Prevalence of *Trichomonas vaginalis* among women, by race, in the United States

<table>
<thead>
<tr>
<th>City</th>
<th>Overall <em>Trichomonas</em> prevalence (%)</th>
<th><em>Trichomonas</em> prevalence in blacks (%)</th>
<th><em>Trichomonas</em> prevalence in nonblacks (%)</th>
<th>OR*</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baltimore</td>
<td>8.6</td>
<td>12.8</td>
<td>3.3</td>
<td>3.9</td>
<td>74</td>
</tr>
<tr>
<td>Nationwide</td>
<td>3.1</td>
<td>13.3</td>
<td>1.3</td>
<td>10.3</td>
<td>95</td>
</tr>
<tr>
<td>Nationwide</td>
<td>2.8</td>
<td>10.5</td>
<td>1.1</td>
<td>9.54</td>
<td>64</td>
</tr>
<tr>
<td>New York</td>
<td>47</td>
<td>51</td>
<td>35</td>
<td>1.6</td>
<td>92</td>
</tr>
<tr>
<td>San Francisco</td>
<td>11</td>
<td>28</td>
<td>9</td>
<td>3.7</td>
<td>90</td>
</tr>
<tr>
<td>5 cities</td>
<td>13</td>
<td>23</td>
<td>6</td>
<td>4.4</td>
<td>15</td>
</tr>
<tr>
<td>Philadelphia</td>
<td>16</td>
<td>30</td>
<td>11</td>
<td>3.6</td>
<td>37</td>
</tr>
<tr>
<td>New York</td>
<td>38</td>
<td>Population 100% black</td>
<td>Population 100% black</td>
<td></td>
<td>63</td>
</tr>
<tr>
<td>Birmingham</td>
<td>12.9</td>
<td>Population 100% black</td>
<td>Population 92% black</td>
<td></td>
<td>16</td>
</tr>
<tr>
<td>New York</td>
<td>27</td>
<td>Population 92% black</td>
<td>Population 83% black</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>New York</td>
<td>20</td>
<td>Population 83% black</td>
<td>Population 96% black</td>
<td></td>
<td>107</td>
</tr>
<tr>
<td>Baltimore</td>
<td>26</td>
<td>Population 96% black</td>
<td>Population 90% black</td>
<td></td>
<td>70</td>
</tr>
<tr>
<td>New York</td>
<td>20</td>
<td>Population 90% black</td>
<td>Population 100% black</td>
<td></td>
<td>24</td>
</tr>
<tr>
<td>Baltimore</td>
<td>25</td>
<td>Population 100% black</td>
<td></td>
<td></td>
<td>62</td>
</tr>
<tr>
<td>Birmingham</td>
<td>21</td>
<td>Population 89% black</td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>Providence</td>
<td>3</td>
<td>Population 87% white</td>
<td></td>
<td></td>
<td>60</td>
</tr>
</tbody>
</table>

* OR, estimated odds ratio.

1.5 to nearly 10 times greater than that for other racial/ethnic groups. In several studies in which very high prevalences of infection were observed, the population consisted exclusively or predominantly of African-Americans. Since this racial finding is consistent across studies, it is unlikely to be artifactual.

Several factors may explain the apparently elevated rate of *Trichomonas* infection in black women. This phenomenon indicates a high prevalence of *Trichomonas* infection among the sex partners of these women. Although a study in Washington, DC, observed a high prevalence of *T. vaginalis* (58%) among young, inner-city, black men (81), data on race-specific rates of *Trichomonas* infection in men are lacking. The association with black race may also reflect a decreased use of barrier protection in this population. Studies indicate that African-American males are less likely to use condoms than men of other racial groups because of a higher frequency of condom breakage and slippage (30) and a reported decrease in sexual fulfillment (94). Crosby et al. recently reported that young African-American women reporting that a male sex partner had been intoxicated during sex were significantly more likely to have an STI. The nature of this phenomenon could be a consequence of the women’s selection of risky partners and lack of condom use, possibly stemming from their partners’ intoxication (17). If rates of drug and alcohol use are higher in the African-American community, this phenomenon could also contribute to the increased incidence and prevalence of *T. vaginalis* in the African-American community. Alternatively, it is possible that practices such as douching, which is reportedly more common in black women (3) and which can increase susceptibility to other STIs (82), could predispose a person to *trichomoniasis* and explain the observed racial association. Increased prevalences of *Trichomonas* infection could also reflect a lack of access to care and distrust of the health care system, which could manifest as failure to seek care, noncompliance with treatment recommendations, and hesitation to refer partners for treatment. Lichtenstein and Schwebke recently reported the unique issues surrounding preferences for partner notification for African-American men (52). If infected male partners in the African-American community are not made aware of the need for diagnosis and treatment, women will continue to get reinfected. Given that numerous studies have shown high reinfection rates of *T. vaginalis* up to a year following treatment in African-American populations, and metronidazole resistance was not thought to be responsible, failure to screen and treat partners is a significant issue contributing to the high prevalence of infection in this community (25, 72).

The association between drug use and high-risk sexual behaviors, including trading sex for money or drugs, may also explain the racial differences in the occurrence of *Trichomonas*. In addition, compared with other racial and ethnic groups, a greater proportion of blacks are unmarried, divorced, or separated (5), and unmarried status is itself a risk marker for STIs (2). It is also conceivable that a genetic or racial-based heightened susceptibility to *T. vaginalis* exists for African-Americans; however, such a phenomenon has not been recognized. The observed racial disparity could also reflect strain differences of *Trichomonas*. For example, if the strains that infect African-Americans are more likely to produce chronic, persistent infection of longer duration, higher prevalences would be observed. However, this hypothesis has not been studied, and additional research on the subject is needed.

**TRICHOMONAS COMPARED WITH OTHER STIs IN AFRICAN-AMERICAN WOMEN**

Table 3 lists studies comparing the prevalence of *T. vaginalis* infection with that of other STIs among black women in the United States. In all studies conducted before 2000, *Trichomonas* was the most commonly identified STI, exceeding both *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in prevalence. In studies conducted after 2000, *Chlamydia trachomatis* was more prevalent than *Trichomonas*. No hypothesis has been put forth to explain this switch; however, all of these studies were conducted in southeastern urban centers. It is possible that various sensitivities of the tests used for diagnosis could account for this observed phenomenon.
TABLE 3. Studies comparing the prevalence of *Trichomonas vaginalis* infection with that of other STIs among black women in the United States

<table>
<thead>
<tr>
<th>Yr</th>
<th>City</th>
<th>Prevalence (%)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td><em>Trichomonas</em></td>
<td><em>Chlamydia</em></td>
</tr>
<tr>
<td>2007</td>
<td>Atlanta</td>
<td>13</td>
<td>18</td>
</tr>
<tr>
<td>2004</td>
<td>Atlanta</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>2001</td>
<td>Birmingham</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>1996</td>
<td>New York</td>
<td>51</td>
<td>9</td>
</tr>
<tr>
<td>1994</td>
<td>New York</td>
<td>27</td>
<td>7</td>
</tr>
<tr>
<td>1994</td>
<td>New York</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>1992</td>
<td>Baltimore</td>
<td>26</td>
<td>21</td>
</tr>
<tr>
<td>1990–1994</td>
<td>New York</td>
<td>22</td>
<td>6</td>
</tr>
<tr>
<td>1985</td>
<td>San Francisco</td>
<td>28</td>
<td>25</td>
</tr>
</tbody>
</table>

**DISCUSSION AND IMPLICATIONS**

The HIV/AIDS epidemic is a heterogeneous one, impacting communities and subpopulations in disproportionate ways. HIV is increasingly affecting low-income groups, particularly African-Americans and women. The amplifying effect of *T. vaginalis* is likely to play a role in this phenomenon. Several aspects of the biology and epidemiology of *Trichomonas* suggest that this long-neglected protozoan may play an important role in HIV transmission dynamics. A compelling biological rationale, supported by laboratory studies, suggests that the pathology caused by *Trichomonas* enhances the efficiency of HIV transmission. In addition, *T. vaginalis* infection is often asymptomatic, and affected persons are likely to continue to engage in sexual activity. This strong biological plausibility is supported by empirical studies from Africa documenting that *Trichomonas* may increase HIV transmission by as much as threefold. Infection can persist at least a year, particularly in asymptomatic men (75, 103), and a long-term-carrier state may result in an increased duration of elevated infectiousness for trichomoniasis and HIV (38). Moreover, although imperfect, the available data suggest that *T. vaginalis* is a highly prevalent infection, particularly among African-American women in urban communities of the United States. Given the evidence that *T. vaginalis* likely promotes HIV infection, the apparently high level of *Trichomonas* infection in black women is cause for concern. Even if *T. vaginalis* increases the risk of HIV transmission by a small or modest amount, it translates into a sizable population effect since *Trichomonas* is so common. Using mathematical models, it was recently estimated that 746 new HIV cases among women in the United States can be attributed to the facilitative effects of trichomoniasis (13). The associated lifetime cost of treating these infections was estimated to be $167 million (13). This has important implications for HIV prevention and control. A reduction in the prevalence of *Trichomonas* could translate into substantial decreases in HIV transmission.

While convincing data suggest that other STDs, including both ulcerative and inflammatory infections, promote HIV transmission (102), available evidence suggests that *T. vaginalis* is often the most common STI in African-American women and therefore may play a more prominent role than other STIs in augmenting the spread of HIV in this high-risk group.

**DIAGNOSIS OF T. VAGINALIS**

Current information is compelling enough to warrant considering implementation of efforts to identify and treat persons with *T. vaginalis* infection, particularly African-Americans, in areas of overlapping HIV and *T. vaginalis* epidemics. Screening programs using self-collected vaginal swabs (86) for culture may be a reasonable method for such an effort. Diamond’s modified medium used to be the method of choice for culture, but it has been largely replaced by the Tv InPouch diagnostic system (BioMed Diagnostics, Inc., San Jose, CA) due to the ease of inoculation and examination (50). The recent development of sensitive and specific urine-based diagnostic techniques can enhance both the yield and ease of screening efforts (57); however, issues of cost and accessibility may limit the use of such methods for the average physician. In addition, the problems of the short-term viability of *Trichomonas* in urine and the dramatic reduction in sensitivity observed in specimens not processed within 30 min are also problematic (91). Inger- soll et al. recently suggested that the stability of *T. vaginalis* DNA in urine could be improved with the use of a urine preservative transport kit. This method, while useful only for molecular diagnosis, could serve to substantially improve the sensitivity of molecular testing (36). PCR for the diagnosis of *T. vaginalis* is also available, but unlike for infections such as gonorrhea and chlamydia, in which PCR appears to have greater sensitivity than culture methods, PCR for trichomoni- asis in women does not appear to offer a diagnostic advantage. This may be because *T. vaginalis* is much less fastidious to culture than *Neisseria gonorrhoeae* or *Chlamydia trachomatis* and requires only one viable organism, the same as is required for PCR (84). One promising development for the appropriate diagnosis and treatment of trichomoniasis is rapid point-of-care tests. Two such tests are now FDA approved, the OSOM *Trichomonas* Rapid Test (Genzyme Diagnostics), an immunochromatographic capillary-flow dipstick technology, and the Affirm VP III test (Becton Dickenson), a nucleic acid probe test that evaluates specimens for the presence of *T. vaginalis*, *Gardnerella vaginalis*, and *Candida albicans* (73, 104). The sensitivities of the tests are 76.7 to 90% versus culture, but results are rapid, occurring within approximately 10 min (47). The rapid assays are also more sensitive than wet mount but are slightly less specific. When PCR is used as the “gold standard,” the sensitivity of the test drops to 66.7% (73). The cost of rapid tests is comparable to that of PCR, with each test costing about $10. Additionally, Munson et al. recently evaluated the use of a transcription-mediated amplification-based *Trichomonas vaginalis* analyte-specific reagent (Gen-Probe, Incorporated) and found positive results with twice the frequency that was found using wet mount (14.5% versus 7%) (67). Clearly, all of these relatively recently developed diagnostic techniques could help facilitate appropriate screening, testing, and treatment for *T. vaginalis* infection, which could in turn serve as a cost-effective tool in HIV prevention (Table 4).

Another valuable screening method has been advocated by Schwebke and colleagues, who demonstrated that vaginal swabs may be stored briefly either in a small amount of saline or in a clean dry tube (15 to 20 min) while a wet-mount preparation is made and examined (87). If the wet mount is negative for *T. vaginalis*, the stored swab can then be processed.
for culture. If the wet mount is positive for *T. vaginalis*, no further culture of the specimen is needed, thereby reducing unnecessary costs. Given that the prevalence of this infection often exceeds 20% in high-risk populations, this approach can reduce costs substantially without compromising the accuracy of the tests. Such an approach may also be useful for combination with rapid test methods. Additionally, Swygard et al. recommend using one of the following three predictors to identify women who would benefit from targeted culture for *T. vaginalis*: any drug use, contact with trichomoniasis, or African-American race (96). Ultimately, any method that reduces the cost of diagnosis will advance further screening for trichomoniasis and promote the goal of implementing intervention efforts.

### CONTROL THROUGH TREATMENT

Effective, inexpensive, single-dose therapy (2 g oral metronidazole) is available for the treatment of *T. vaginalis* infection. The cure rate with this regimen is 97% (53). It may not be hyperbole to suggest that *Trichomonas* infection may be more readily modifiable than sexual behavior in some high-risk groups. Trials in Tanzania and in Kenya have demonstrated the benefit of reduced HIV incidence in communities receiving aggressive STD control intervention (31, 58). However, since recent data indicate that resistance to metronidazole is rising (9.6% of isolates in a recent study), judicious drug use is extremely important (83). Resistance to metronidazole is thought to be relative and can often be overcome with higher doses of the drug (54). Additionally, resistance to tinidazole, a drug that was recently approved for the treatment of *T. vaginalis*, has been reported, but this drug is in the same drug class as metronidazole (23). Currently, although rates of repeated infection have been found to be as high as 8% among women and as high as 30% among HIV-infected women, expedited partner treatment (the provision of medication to the index to provide to his or her partners) has not been recommended for *T. vaginalis* infection (19, 41, 56). However, the addition of metronidazole to the regimen for syndromic management has been shown to be effective. Price et al., in a randomized clinical trial in Malawi, showed that while adding 2 g metronidazole to the standard syndromic management of urethritis did not improve symptoms, it did clear 95% of culture-proven *T. vaginalis* infections in the treatment group, compared to 54% in the placebo group (76). In addition, among HIV-infected men in that study, the seminal HIV RNA concentrations in the treatment group were lower than those in the control group (76).

### CONCLUSION

Additional studies to evaluate the prevalence and incidence of *T. vaginalis* and to determine risk factors for infection in both men and women are needed. Moreover, given the paucity of data and the potential importance of *Trichomonas*, consideration should be given to requiring mandatory reporting of *T. vaginalis* infection. Efforts to further evaluate the interactions between *T. vaginalis* and HIV, particularly in an industrialized country setting, would also seem to be warranted. The control of *Trichomonas vaginalis* through appropriate testing and treatment also represents a potentially important step in the control of HIV transmission in the African-American community.

### REFERENCES


