Trichomonas vaginalis, HIV, and African-Americans

Frank Sorvillo,* Lisa Smith,* Peter Kerndt,H Lawrence Ash*
*University of California at Los Angeles, Los Angeles, California; and
HDepartment of Health Services, Los Angeles County, Los Angeles, California

Trichomonas vaginalis may be emerging as one of the most important cofactors in amplifying HIV transmission, particularly in African-American communities of the United States. In a person co-infected with HIV, the pathology induced by *T. vaginalis* infection can increase HIV shedding. *Trichomonas* infection may also act to expand the portal of entry for HIV in an HIV-negative person. Studies from Africa have suggested that *T. vaginalis* infection may increase the rate of HIV transmission by approximately twofold. Available data indicate that *T. vaginalis* is highly prevalent among African-Americans in major urban centers of the United States and is often the most common sexually transmitted infection in black women. Even if *T. vaginalis* increases the risk of HIV transmission by a small amount, this could translate into an important amplifying effect since *Trichomonas* is so common. Substantial HIV transmission may be attributable to *T. vaginalis* in African-American communities of the United States.

Trichomonas vaginalis is a protozoan parasite transmitted principally through vaginal intercourse. Infection with the organism, while frequently asymptomatic, can cause vaginitis in women and urethritis in men. Despite a relative paucity of studies on the prevalence and incidence of trichomoniasis, recent publications suggest that *T. vaginalis* is one of the most common sexually transmitted infections (STIs) in the United States, with an estimated 5 million new cases occurring annually (1). Although the organism appears to be highly prevalent and has a widespread geographic distribution, *Trichomonas* has not been the focus of intensive study nor of active control programs. This neglect is likely a function of the relatively mild nature of the disease (2), the lack of effect on fertility, and the historic absence of association with adverse birth outcomes (although recent data suggest a possible causal role in low birth weight and prematurity [3]). However, *Trichomonas* may play a critical and underrecognized role in amplifying HIV transmission (4). We present the rationale to support the hypothesis that *T. vaginalis* may be an important cofactor in promoting the spread of HIV and, in some circumstances, may have a major impact on the epidemic dynamics of HIV in African-American communities.

Biologic 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 (5). This inflammatory response induces a large infiltration of leukocytes, including HIV target cells such as CD4+ bearing lymphocytes and macrophages to which HIV can bind and gain access (6,7). In addition, *T. vaginalis* can frequently cause punctate mucosal hemorrhages (8). 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 the 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 (9), and substantially increased urethral viral loads have been documented in men with *Trichomonas* infection (10). In addition, *T. vaginalis* has the capacity to degrade secretory leukocyte protease inhibitor, a product known to block HIV cell attachment; this phenomenon may also promote HIV transmission (11). Moreover, since most patients with *Trichomonas* infection are asymptomatic or mildly symptomatic (12), they are likely to continue to remain sexually active in spite of infection. Studies suggest that approximately 50%-70% of persons with *T. vaginalis* have subclinical infection (12).

Empiric Evidence Implicating Trichomonas in HIV Transmission

Data from studies conducted in Africa have shown an association between *Trichomonas* and HIV infection, suggesting a two- to threefold increase in HIV transmission

Address for correspondence: Frank Sorvillo, Department of Epidemiology, School of Public Health, UCLA, Box 951772, Los Angeles, CA 90024, USA; fax: 714-816-9099; e-mail: fsorvill@ucla.edu

(4,13,14). 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, 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 analysis (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., lack of information on whether *Trichomonas* infection preceded HIV, these preliminary findings were subsequently reinforced in a single prospective study from Zaire (4). This study, 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 muiltivariate analysis.

Data on the Prevalence of *T. vaginalis* among U.S. Women

Information on the occurrence of *T. vaginalis* infection in the United States is meager. Trichomoniasis is not a reportable condition in most health jurisdictions, and prevalence surveys for STIs often do not include attempts to recover *Trichomonas*. In addition, the relatively few published studies with information on the prevalence of *T. vaginalis* infection have generally been conducted among highly selected populations, typically included only women, or were limited by small numbers of participants. Frequently these studies were not conducted with the primary purpose of assessing the prevalence of *Trichomonas*. Moreover, many of these studies have often used diagnostic techniques with relatively low sensitivity such as wet mount, stained preparations, or Papanicolaou (PAP) smear. Wet mount, the most commonly used method, has an estimated sensitivity of 58% when compared with culture (15); the sensitivity of PAP smear is approximately 57%. The accuracy of these techniques is dependent on the experience of the microscopist, and sensitivities may vary widely (15). The sensitivity of culture when compared with polymerase chain reaction (PCR) has been estimated to be 70% (16). Such highly sensitive PCR and related techniques are not routinely used nor readily available for *Trichomonas* as for other STIs (17). As a result of suboptimal laboratory methods, studies of *T. vaginalis* have often substantially underestimated the prevalence of infection. 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 21% (18-37).

Table 1 lists published reports on the occurrence of *T. vaginalis* infection among women conducted among U.S. populations from 1964 through 1999. Although not

Table 1. Studies of the prevalence of *Trichomonas vaginalis* infection in women, United States, 1964–1997

Year[a]	Location (ref)	N	Population	Trichomonas prevalence (%)	Diagnostic method(s)
1996-97	New York (18)	213	Incarcerated	47	culture
1995-97	St. Louis (19)	143	HIV clinic	11	wet mount
1993-95	4 cities (20)	1,285	HIV infected and high risk	11	wet mount
1994	New York (23)	1,404	Inner city	20	not provided
1992	Baltimore (24)	279	STD clinic	26	culture
1990-94	New York (37)	677	HIV and community clinics	22	culture
1901-93	Southeastern city (21)	650	Adolescent health clinics	3	culture
1986	5 cities (27)	13,816	Antepartum women	13	culture
1990-91	New York (22)	372	Inner city	27	culture
1989-90	New York (25)	1,401	OB/GYN clinics	20	culture
1989	Baltimore (26)	3,005	Cancer screening	25	wet mount
1987-88	Denver (36)	5,681[b]	STD clinic	11	wet mount
1984-86	Birmingham (28)	818	STD clinic	21	wet mount
1985	San Francisco (29)	171	Adolescent clinic	11	wet mount/PAP[c]
1982	Baltimore (30)	115	Pregnant adolescents	34	culture
1981	Seattle (31)	80	Juvenile detention	48	wet mount
1980	Providence (32)	500	Student health center	3	culture
1979-80	Storrs (33)	383	GYN clinic	19	wet mount/PAP
1971	Oregon (34)	338	State school/adolescents	35	Gram stain
1964	Philadelphia (35)	27,392	Cancer screening	16	PAP

[a]Year of study (or publication).

[b] Number of visits.

[c]Papanicolaou smear; STD = sexually transmitted disease; OB/GYN = obstetrics/gynecology.

necessarily complete, a comprehensive search through MEDLINE and review of articles yielded only 20 reports during this 35-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.

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' experience (38). 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 followed women co-infected with HIV and *T.* vaginalis, documented high rates (16.1 per 100 person-years) of *Trichomonas* re-infection (39). Among a predominantly black group of HIV-infected and high-risk women followed in New York City from 1990 to 1994, *T. vaginalis* was the most frequent incident STI (37).

Prevalence of *T. vaginalis* among Men in the United States

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. Among men attending an STD clinic in Seattle-King County from 1987 to 1990, 6% of 300 randomly selected men were infected with *Trichomonas* by culture technique; 22% of 147 contacts to women with *T. vaginalis* were also positive (40). 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* (41). Among 454 consecutive men attending an STD clinic in Denver in 1998, 2.8% were found to be infected by a culture method (42). 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 (43). Data on race-specific prevalences of *Trichomonas* infection among U.S. males are not available. We are unaware of any published reports that have assessed the prevalence of *T. vaginalis* in males and females. While the separate studies we have cited suggest that *Trichomonas* may be more common in women in the United States, 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/ethnicity, the prevalence of *Trichomonas* has been highest in African-Americans (23%-51%), ranging from approximately 1.5 to nearly 4 times greater than 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. This racial finding, consistent across studies, is unlikely to be artifactual.

Table 2. Prevalence of *Trichomonas vaginalis* among women, by race, United States

City (ref)	Overall *Trichomonas* prevalence (%)	*Trichomonas* prevalence in blacks	*Trichomonas* prevalence in non-blacks	OR[a]
New York (18)	47	51	35	1.6
San Francisco (29)	11	28	9	3.7
5 cities (27)	13	23	6	4.4
Philadelphia (35)	16	30	11	3.6
New York (22)	27	population 92% black		
New York (25)	20	population 83% black		
Baltimore (24)	26	population 96% black		
New York (23)	20	population 90% black		
Baltimore (26)	25	population 100% black		
Birmingham (28)	21	population 89% black		
Providence (32)	3	population 87% black		

[a] Estimated odds ratio.

Several factors may explain the apparent elevated rate of trichomoniasis in black women. This phenomenon may indicate a high prevalence of *Trichomonas* infection among the sex partners of these women. Although a study in Washington, D.C., observed a high prevalence of *T. vaginalis* (55%) among young, inner-city, black men (43), data on race-specific rates of *Trichomonas* infection in men are lacking. The association with black race may also reflect 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 (44) and a reported decrease in sexual fulfillment (45). Alternatively, it is possible that practices such as douching, which is reportedly more common in black women (46) and can increase susceptibility to other STIs (47), could predispose to trichomoniasis and explain the observed racial association. Increased prevalences of *Trichomonas* infection could also reflect 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. Drug use and its association with 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 (48), and unmarried status is itself a risk marker for STIs (49). It is also conceivable that a genetic or racial-based heightened susceptibility to *T.vaginalis* exists in African-Americans; however, such a

phenomenon has not been recognized. Finally, the observed racial disparity could 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.

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 each study *Trichomonas* was the most commonly identified STI, exceeding both *Chlamydia trachomatis* and *Neisseria gonorrhoeae* in prevalence. While the optimal tests for detecting *C. trachomatis* and *N. gonorrhoeae* were not always used in these studies, neither were highly sensitive tests used for the diagnosis of *Trichomonas*.

Discussion and Implications

The HIV/AIDS epidemic is a heterogeneous one, impacting communities and subpopulations in disproportionate ways. In many jurisdictions in the United States, HIV is increasingly affecting low-income groups, particularly African-Americans and women. We suggest that part of this phenomenon may result from the amplifying effect of *T.vaginalis*. 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 biologic rationale 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 biologic plausibility is supported by empiric studies from Africa documenting that *Trichomonas* may increase HIV transmission by two- to threefold. 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 apparent 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 may translate into a sizable population effect since *Trichomonas* is so common. To illustrate this, we present population-attributable risk curves, or the level of HIV transmission that would be attributable to *T. vaginalis*, at varying prevalences of *Trichomonas,* given the assumption of an increased relative risk of HIV infection of 2 or 3 (Figure). As the figure illustrates, if *Trichomonas* amplifies HIV transmission by twofold and the prevalence of *T. vaginalis* in a community is 25%, one fifth (20%) of HIV transmission in that population would be attributable to *Trichomonas*. This has important implications for HIV prevention. Reduction in the prevalence of *Trichomonas* could translate into substantial decreases in HIV transmission. Effective, inexpensive single-dose therapy (2 g oral metronidazole) is available for the treatment of *T. vaginalis* infection. 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 have demonstrated the benefit of reduced HIV incidence in communities receiving aggressive STD control intervention (50).

While convincing data suggest that other STDs, including both ulcerative and inflammatory infections, promote HIV transmission (51), available evidence suggests that *T.vaginalis* is 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.

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

Table 3. Studies comparing the prevalence of *Trichomonas vaginalis* infection with that of other sexually transmitted infections among black women in the United States

Year	City (ref)	Trichomonas (%)	Chlamydia (%)	Gonorrhea (%)
1996	New York (18)	51	9	5
1994	New York (22)	27	7	2
1994	New York (23)	20	15	no data
1992	Baltimore (24)	26	21	14
1990 -94	New York (37)	22	6	1
1985	San Francisco (29)	28	25	no data

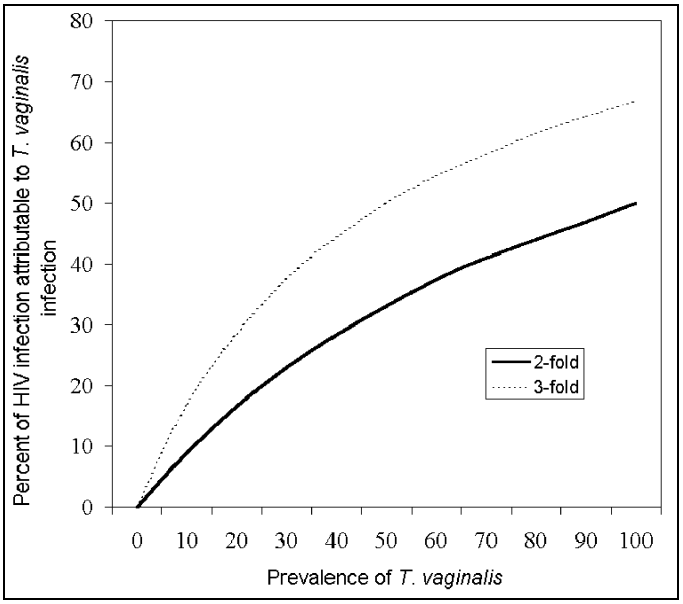

Figure. Hypothetical level of HIV transmission attributable to *Trichomonas vaginalis* at varying prevalences of *Trichomonas* infection and assuming that *T. vaginalis* infection amplifies HIV infection by two- or three-fold.

the interactions between *T. vaginalis* and HIV, particularly in an industrialized country setting, would also seem warranted. However, given the lower rates of heterosexual transmission, such studies would be expensive and require a large sample. Nevertheless, we believe that 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 (52) for culture may be a reasonable method for such an effort. An alternative approach would be to first use wet mount examination, which is relatively easy and inexpensive but lacks sensitivity, followed by culture for specimens that are negative on wet mount. Recent development of sensitive and specific urine-based diagnostic techniques can enhance both the yield and ease of screening efforts (53); however, issues of cost and accessibility may limit the use of such methods for the average physician.

Frank Sorvillo is an associate professor in-residence in the Department of Epidemiology at UCLA=s School of Public Health. His research interests include the epidemiology and control of infectious diseases, particularly parasitic agents.

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