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# The Effect of High Rates of Bacterial Sexually Transmitted Infections on HIV Incidence in a Cohort of Black and White Men Who Have Sex with Men in Atlanta, Georgia

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## Abstract

Data reporting sexually transmitted infection (STI) incidence rates among HIV-negative U.S. men who have sex with men (MSM) are lacking. In addition, it is difficult to analyze the effect of STI on HIV acquisition given that sexual risk behaviors confound the relationship between bacterial STIs and incident HIV. The InvolveMENt study was a longitudinal cohort of black and white HIV-negative, sexually active MSM in Atlanta who underwent routine screening for STI and HIV and completed behavioral questionnaires. Age-adjusted incidence rates were calculated for urethral and rectal Chlamydia (CT), gonorrhea (GC), and syphilis, stratified by race. Propensity-score-weighted Cox proportional hazards models were used to estimate the effect of STI on HIV incidence and calculate the population attributable fraction (PAF) for STI. We included 562 HIV-negative MSM with 843 person-years of follow-up in this analysis. High incidence rates were documented for all STIs, particularly among black MSM. Having a rectal STI was significantly associated with subsequent HIV incidence in adjusted analyses (aHR 2.7; 95% CI 1.2, 6.4) that controlled for behavioral risk factors associated with STI and HIV using propensity score weights. The PAF for rectal STI was 14.6 (95% CI 6.8, 31.4). The high incidence of STIs among Atlanta MSM and the association of rectal STI with HIV acquisition after controlling for behavioral risk underscore the importance of routine screening and treatment for STIs among sexually active MSM. Our data support targeting intensive HIV prevention interventions, such as preexposure chemoprophylaxis (PrEP), for Atlanta MSM diagnosed with rectal STIs.

## Introduction

Men who have sex with men (MSM) continue to be the largest risk group for HIV infection in the United States, accounting for 63% of new infections in 2010.[1] In addition, rates of some bacterial sexually transmitted infections (STIs) are increasing among MSM,[2] although few community-sampled incidence rates have been published. STIs, including *Neisseria gonorrhoeae* (GC), *Chlamydia trachomatis* (CT), and syphilis, have long been shown to increase the risk of HIV acquisition, likely by causing loss of integrity of the mucosa, increasing inflammation and increasing HIV target cell availability.[3] The high per-act HIV transmission probability associated with unprotected anal intercourse (UAI)[4,5] coupled with the high prevalence of bacterial STIs reported among MSM[2,6] calls for increased investigation into the role of STIs in HIV acquisition among MSM. To combat these dual epidemics, the Centers for Disease Control and Prevention (CDC) recommends routine annual urethral, rectal, and oropharyngeal screening for STIs in sexually active MSM, but it is not known how implementation of this practice will affect STI or HIV incidence.[7]

The city of Atlanta, Georgia had the eighth highest rate of new HIV diagnoses and fourth highest number of new HIV diagnoses among MSM in the United States in 2011.[8] As in other large municipalities, black MSM are disproportionately affected, comprising about 60% of HIV-infected MSM,

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whereas blacks represent only about 30% of the overall Atlanta population.[9] Meta-analytic evidence demonstrates large racial disparities in STI diagnosis history between black and nonblack MSM. This finding suggests that STIs play a contributory role in the similarly large disparities in HIV infection, yet prospective examinations of this hypothesis using incident diagnoses are lacking.[10,11] In 2011, over 70,000 bacterial STIs were reported in Georgia, and Georgia ranks very high in the United States for rates of STIs (seventh for CT, sixth for GC, and third for syphilis), with the majority of cases occurring among blacks.[12] A better understanding of the local overlapping epidemics of STIs and HIV will be crucial for enhancing the design of HIV prevention interventions for MSM, and for understanding the causal contribution of STIs toward HIV incidence. However, the examination of an effect of STIs on HIV acquisition is confounded by high-risk behavior, which greatly complicates these analyses. In the absence of a randomized clinical trial, robust analytic methodologies must be employed to control for confounding by risk behavior.

The InvolveMENt study was a prospective, community recruited cohort of HIV-negative black and white MSM conducted from 2010 to 2014 in Atlanta, Georgia designed to examine individual, dyadic, and community level determinants of racial disparities in HIV and STI incidence. The prevalence of HIV at the baseline study visit was extremely high—43% among black MSM and 13% among white MSM—and the prevalence of bacterial STIs was also high.[13] This longitudinal study of HIV-negative MSM undergoing routine screening for STIs and HIV provides the unique opportunity to define STI incidence and examine the co-occuring STI/HIV epidemics while controlling for behavioral risk factors to better understand drivers of the Atlanta MSM epidemic.

## Materials and Methods

### *Ethics statement*

This study was reviewed and approved by the institutional review boards of Emory University and Georgia State University (IRB # 42405).

### *Sampling, recruitment, and enrollment*

InvolveMENt was a prospective cohort study at Emory University designed to examine individual, dyadic, and community level factors that may contribute to disparities in HIV and STI prevalence and incidence between black and white MSM in Atlanta, Georgia. Detailed recruitment and enrollment methods have been previously published.[13] Briefly, between 2010 and 2012, MSM aged 18 years and above were recruited, regardless of HIV status, from the Atlanta community primarily using time–space venue sampling, with a sampling frame built upon that used in the Atlanta site for the second MSM cycle of the National HIV Behavioral Surveillance System (NHBS).[14] Facebook was included as a virtual ''venue'' in the venue sampling frame.[15] Eligible participants self-identified as black or white MSM who reported sex with another man in the previous 3 months, who were not in a mutually monogamous relationship, could complete survey instruments in English, lived in the Atlanta metropolitan area, were not enrolled in another HIV prevention study, and had no plans to relocate in the subsequent 2 years. Men who self-identified as Hispanic or of other/mixed race were not enrolled.

Study visits occurred at various university-based and community-based organization venues in Atlanta. All eligible men, including those who self-reported a previous HIV diagnosis, were tested for HIV and completed a detailed computer-assisted self-interview questionnaire to evaluate demographic, individual (e.g., number of sexual partners, number of UAI partners, condom use, drug/alcohol use), dyadic (e.g., partner demographics such as age and race, partnership characteristics), and community level (e.g., poverty, neighborhood violence) HIV risk. Men who were HIV negative at baseline were prospectively followed for up to 24 months and underwent HIV antibody testing at 3–6 month intervals. Study discontinuation occurred after the 24-month visit or HIV seroconversion. This report examines incident STI and HIV diagnoses from the longitudinal cohort.

### *HIV and STI testing*

Regardless of self-reported HIV status, all participants were screened for HIV antibodies with an FDA-approved rapid test. For men who had preliminary positive results on their HIV rapid test, additional specimens were collected by venipuncture for confirmatory Western blot, CD4, and HIV viral load testing. Among those with preliminary positive results, confirmatory testing was by Western blot; in one case, where additional specimens were not available for Western blot testing, two additional HIV rapid tests were performed.[16,17] All participants were tested for syphilis and urethral GC and CT at each study visit. Beginning in October 2011, participants were also tested for rectal GC and CT using self-collected rectal swab specimens.

Syphilis testing was conducted using the rapid plasma reagin (RPR)[18]; specimens that were reactive by RPR were reflexed to quantitative nontreponemal titers and treponemal IgG. New syphilis diagnoses were designated by experienced clinicians after reviewing all available data including previous RPR titers, if available, and treatment history. Testing for urethral and rectal GC and CT was performed using the Becton Dickinson ProbeTec ET *C. trachomatis* and *N. gonorrhoeae* Amplified DNA Assay (Sparks, MD).[19] All participants who tested STI positive were notified and referred to a community treatment provider with treatment costs paid by the study. An STI was considered ''incident'' if a positive diagnosis followed a visit in which the participant had tested negative for that STI, or had tested positive and was confirmed treated by the treatment provider.

### *Analytic methods*

For individuals with incident STI, person-time was calculated as the difference between the date of STI diagnosis and the date of HIV seroconversion or censoring. For individuals without an STI for the duration of the study, person-time was calculated as the difference between the enrollment date and the date of HIV seroconversion or censoring due to study completion or loss to follow-up. The date of HIV seroconversion was estimated as halfway between the date of the seroconversion study visit and the prior visit. Age-adjusted incidence rates per 100 person-years were calculated for each STI and HIV and stratified by race. Incidence rate

ratios for black vs. white MSM were calculated with exact 95% confidence intervals.

To assess the association between STI and HIV acquisition, crude hazard ratios (HR) were calculated using unadjusted Cox proportional hazards (PH) regression for the unadjusted effect of the exposure, incident STI, on the outcome, incident HIV. Because of the limited number of HIV outcomes, it was necessary to combine STIs by site of infection into three categories (urethral STI, rectal STI, and syphilis) in order to maximize statistical power to detect an association with HIV acquisition. In addition, we were underpowered to stratify these analyses by race, so HRs reported reflect the entire cohort.

To address the dual and potentially conflicting needs for confounding control and statistical efficiency in estimating effects of STI on HIV incidence with a limited number of incident HIV events, we used propensity-score-weighted Cox proportional hazards models.[20,21] By weighting using correctly specified propensity scores, the exposure groups (i.e., participants with and without an STI diagnosis) will have similar distributions of measured confounders, simulating a randomized clinical trial (RCT). Consequently, given the temporal association between the exposure and outcome of interest and its biological plausibility, the results of the weighted Cox proportional hazards model lend support to a causal association.

The steps in this method are as follows: first, a logistic regression model of STI incidence was fit as a function of race, age at STI diagnosis/censoring, age–race interaction, UAI in the interval of STI diagnosis/censoring, reported race of sexual partners in the interval of STI diagnosis/censoring, any reported receptive anal intercourse (RAI) for the study duration, and any census-tract level poverty, diagnosis of other STIs for the study duration, and any noninjection drug use for the study duration. Second, the results of the logistic regression models were used to calculate weights as the inverse stabilized propensity scores (i.e., probability of incident STI). Finally, using these weights, adjusted hazard ratios for incident HIV infection of those diagnosed with STI relative to those who were not were then calculated controlling for UAI in the interval of HIV diagnosis/censoring, age at HIV diagnosis/censoring, and reported race of sexual partners in the interval of HIV diagnosis/censoring. Propensity score methods were evaluated by calculating standardized differences for each variable in the propensity model. A standardized difference less than 0.25 is considered to indicate adequate specification of the propensity model and control of residual confounding.[22]

Population attributable fractions (PAF) were calculated using the weighted, adjusted HRs and proportion of seroconverters previously diagnosed with an STI ($p$), using the standard formula [$p$(HR – 1)/HR]. Confidence intervals for the PAF were estimated using the delta method.[23]

## Results

The InvolveMENt study enrolled a total of 803 MSM, of whom 562 (260 black and 302 white) tested HIV negative at enrollment and were included in the longitudinal cohort with 843.1 person-years of follow-up. Incidence rates, stratified by race, for STI and HIV are presented in Table 1. With the exception of urethral CT, STI rates for black MSM were significantly higher than for white MSM. Rate ratios were highest for urethral GC [RR 10.34; 95% CI (1.39, 458.7)] and syphilis [RR infinite; 95% CI (7.22, infinite)]. Overall, 90% of STIs were categorized as treated after their STI diagnosis based on documentation of treatment from a referring provider or by a negative STI test at the subsequent study visit.

Ten MSM were excluded from the analyses of the association between STI and HIV acquisition; six were acutely infected with HIV at enrollment and four were missing key covariates. The remaining 552 HIV-negative MSM contributed 809 person-years of follow-up, during which 26 MSM became HIV infected. (A full report of the HIV incidence rates and non-STI risk factors for HIV seroconversion in the InvolveMENt cohort has been submitted for publication).[24] For this analysis, 21 incident cases of syphilis, 29 cases of urethral CT, 9 cases of urethral GC, 37 cases of rectal CT, 32 cases of rectal GC, and 10 cases of concurrent rectal GC and CT were used. A description of the covariates used in logistic regression models to predict incident bacterial STI is presented in Table 2. MSM included in this analysis who became HIV infected during follow-up were younger, more likely to be black, and more likely to report black sexual partners than MSM who remained HIV negative.

There were no observed HIV infections in men with incident syphilis, urethral CT, or urethral GC during the study follow-up (Table 3). However, there was evidence of prior syphilis infection (e.g., positive RPR determined by a clinician to not represent a new syphilis diagnosis) at the baseline

Table 1. Rates of Sexually Transmitted Infection and HIV per 100 Person-Years Stratified by Race in a Cohort of Black and White Men Who Have Sex with Men in Atlanta, Georgia

| | *Black* | | *White* | | | |
|---|---|---|---|---|---|---|
| | *Cases/PY* | *(Rate/100PY, [Ex. 95% CI])* | *Cases/PY* | *(Rate/100PY, [Ex. 95% CI])* | *Rate ratio* | *[Ex. 95% CI]* |
| HIV | 24/369.2 | (6.5, [4.2, 9.7]) | 8/473.9 | (1.7,[0.7, 3.3]) | 9.56 | [2.2, 86.2] |
| Urethral CT | 17/361.0 | (4.7, [2.7, 7.5]) | 14/466.0 | (3.0, [1.6, 5.0]) | 1.57 | [0.73, 3.43] |
| Urethral GC | 8/366.7 | (2.2, [0.9, 4.3]) | 1/473.8 | (0.2, [0, 1.2) | 10.34 | [1.39, 458.7] |
| Rectal CT | 34/313.7 | (10.8, [7.5, 15.1]) | 22/402.3 | (5.5, [3.4, 8.3]) | 1.98 | [1.13, 0.56] |
| Rectal GC | 30/319.3 | (9.4, [6.3, 13.4]) | 15/407.3 | (3.7, [2.1, 6.1]) | 2.55 | [1.33, 5.10] |
| Syphilis | 22/361.4 | (6.1, [3.8, 9.2]) | 0/476.3 | (0, [0, 0.8]) | Inf. | [7.22, inf.] |

PY, person-years; CT, *Chlamydia trachomatis*; GC, *Neisseria gonorrhoeae*.

Table 2. Distribution of Covariates, Stratified by Incident HIV Cases, Used in Logistic Models to Predict Sexually Transmitted Infections from 552 Men Who Have Sex with Men from the Involvement Cohort Included in This Analysis

| *Characteristic* | *HIV seroconverter* | | p-*value* |
|---|---|---|---|
| | *No* (n = *526*) | *Yes* (n = *26*) | |
| | N *(%)* | | |
| Black race | 232 (44) | 19 (73) | 0.003 |
| Ever reporting RAI[a] | 372 (71) | 21 (81) | 0.19 |
| Noninjection drug use[a,b] | 287 (55) | 13 (50) | 0.40 |
| Report of black sexual partners[c] | 202 (38) | 16 (62) | 0.02 |
| Reported UAI[c] | 341 (65) | 21 (81) | 0.07 |
| | *Median (IQR)* | | |
| Age in years | 27.6 (24.3, 33.5) | 24.6 (22.5, 28.8) | 0.03 |
| Census tract poverty level | 13.9 (8.8, 26.3) | 18.8 (10.5, 29.7) | 0.25 |

[a]For the duration of the study.
[b]Any reported noninjection drug use or positive baseline drug screen.
[c]In interval HIV diagnosis/censoring.
RAI, receptive anal intercouse; UAI, unprotected anal intercourse.

visit in one man who acquired HIV during study follow-up, and two men who acquired HIV had prevalent urethral CT at the baseline study visit. We did not include these prevalent STIs in the analysis as we could not accurately estimate person-time for the STI diagnosis. Of the 79 incident rectal STIs diagnosed, six (two CT alone, two GC alone, and two both CT and GC) occurred in men who acquired HIV. Rectal STI were associated with incident HIV in unadjusted analyses (crude HR: 3.6; 95% CI: 1.4, 9.2). Results from the rectal STI propensity models demonstrated good control of confounding with all standardized distances $<0.25$. In adjusted, weighted Cox-proportional hazards models, rectal STI had a reduced but significantly elevated association with incident HIV (adjusted HR: 2.7; 95% CI 1.2, 6.4). The PAF for rectal STI in the cohort was 14.6% (95% CI 6.8, 31.4).

## Discussion

We report incidence rates for STIs among black and white MSM undergoing routine STI screening and treatment, and show that rectal STIs are associated with HIV acquisition after robust control of behavioral confounders. A high prevalence of bacterial STIs[2] and high incidence of syphilis[25] have been reported for U.S. MSM, particularly among those attending STI clinics. However, our data reflect race-stratified incidence rates among community recruited HIV-negative MSM. These data show startingly high rates of STIs among black, HIV-negative MSM with approximately 10% of men acquiring rectal GC and/or CT per year. We have previously documented stark disparities in HIV prevalence and incidence from the InvolveMENt cohort as well as potential drivers of disparities at individual, dyadic, and community levels.[13,24] Clearly, factors such as high prevalence sexual networks, high rates of poverty, less access to healthcare, and other community level factors operate in parallel fashion to drive disparities in STI similar to HIV.

To our knowledge, this is the first study to report an increased risk of rectal bacterial STI on HIV acquisition in a community-recruited cohort of HIV-negative MSM undergoing routine screening and treatment for STI in the United States. After extensive control for confounding by behavioral risk with propensity score-weighted models, MSM diagnosed with rectal STI during study follow-up were more than 2.5 times as likely to acquire HIV as MSM not diagnosed with

Table 3. Unadjusted and Adjusted Hazard Ratios and Population Attributable Fraction for the Association Between Incident Sexually Transmitted Infection and Incident HIV

| *STI* | | *Counts* | | *Crude HR (95% CI)* | *Standardized differences <0.25* (n) | *Adjusted HR (95% CI)* | *PAF (95% CI)* |
|---|---|---|---|---|---|---|---|
| Urethral STI | | $STI^+$ | $STI^-$ | — | 8/8 | — | — |
| | $HIV^+$ | 0 | 26 | | | | |
| | $HIV^-$ | 37 | 489 | | | | |
| Syphilis | | $STI^+$ | $STI^-$ | — | 3/8 | — | — |
| | $HIV^+$ | 0 | 26 | | | | |
| | $HIV^-$ | 21 | 505 | | | | |
| Rectal STI | | $STI^+$ | $STI^-$ | 3.6 (1.4, 9.2) | 8/8 | 2.7 (1.2, 6.4) | 14.6 (6.8, 31.4) |
| | $HIV^+$ | 6 | 20 | | | | |
| | $HIV^-$ | 73 | 453 | | | | |

HR, hazard ratio; PAF, population attributable fraction; STI, sexually transmitted infection.

rectal STI. Based on the PAF, nearly 15% of HIV infections may have been prevented had the rectal STI not occurred. Ultimately, our findings support a causal association between rectal STI and HIV acquisition among MSM and call for further research into mechanisms of rectal mucosal HIV transmission, particularly in the setting of STI treatment.

Previous studies have highlighted the importance of rectal STI in HIV acquisition in U.S. MSM, but these studies were conducted in STI clinic settings, which could bias toward a higher behavioral risk population with more symptomatic STIs.[26,27] In addition, these studies relied on passive case finding and did not observe incident STIs and HIV diagnoses prospectively as did our study. One longitudinal cohort study conducted in Australia also showed an association between a combined exposure of self-reported and study-diagnosed rectal STI and HIV; however, it is not clear if these results apply to a sample of black and white MSM in the United States with substantially higher rates of both STI and HIV.[28] In addition, these previous studies used different methodologies to control for confounding behavioral risks. We believe that our use of propensity scores as weights in adjusted models represents a more robust control for behavioral confounding. Given that the majority of rectal STIs are asymptomatic among MSM,[6] underlying CDC's recommendation for routine screening in this population,[7] it is important to note that a significant association between rectal STI and HIV acquisition was still present among MSM undergoing routine screening and treatment.

Overall, the PAF for HIV acquisition associated with rectal STI was relatively modest in our study ($< 15\%$), suggesting that these infections are not large drivers of the HIV disparity between black and white MSM. The iPrex study, a clinical trial of preexposure chemoprophylaxis (PrEP) among MSM, recently reported the PAF associated with a prior self-reported diagnosis of STI was also low, $< 10\%$.[29] A low PAF may be partly driven by the numerous other contributing causes of HIV infection such as unprotected anal intercourse, given that both studies recruited general samples of sexually active MSM. Previous population-based interventions aimed at control of STIs as a means of HIV prevention have generally not been successful in reducing the incidence of HIV and have varied substantially in estimates of attributable risk associated with STI.[30,31] Some have suggested that populations with high-risk sexual behavior and high rates of STI may have a greater attributable risk associated with STI and benefit most from STI treatment interventions for HIV prevention, whereas populations with an advanced HIV epidemic may not.[32] While MSM comprise a group with high-risk sexual behavior and high rates of STI, the high prevalence of HIV in our cohort[13] shows that the HIV epidemic among MSM in Atlanta is certainly advanced. Nonetheless, our data support STI prevention as an important component of multifaceted HIV prevention interventions.

Our study has several limitations. We did not collect clinical symptom data from study participants, so we are unable to report the proportion of STI cases with symptoms. We did not test for oropharyngeal CT or GC due to budget constraints, and we were unable to test for acute HIV infection at all study visits. The overall number of incident HIV cases was small, and the analysis was underpowered to examine the effect of STI on HIV acquisition by race or by specific organism. We were unable to estimate the effect of incident syphilis or urethral STI on HIV incidence as no seroconversions were observed in this group. The iPrEx trial did show an association between syphilis and HIV acquisition among MSM, and we may have seen a similar effect with additional follow-up time in our study.[33] Our study enrolled only black and white MSM, was limited to one geographic urban area in the Southeast, and may not be generalizable to all U.S. MSM. Finally, it is important to note that we have not estimated the PAF associated with the increased risk of HIV transmission from HIV-positive partners with STI in this study.

Defining the contribution of STI to HIV acquisition in MSM in Atlanta where high rates of STI and HIV co-occur is important for the design of optimally effective HIV prevention interventions. MSM diagnosed with a rectal STI are at increased risk for HIV seroconversion, even in the setting of routine screening and treatment. While the PAF associated with rectal STI may be modest, prevention of rectal STI among MSM is a logical target for control of both STI and HIV. However, our data show that focusing solely on prevention of rectal STI may have a limited effect on HIV incidence and highlights the need for multifaceted HIV prevention interventions for MSM. In Atlanta, there is an urgent need to increase the availability of STI and HIV prevention services for MSM, including community-based STI screening and treatment and PrEP, given the alarmingly high incidence of STI and its effect on HIV incidence.

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## Author Disclosure Statement

No competing financial interests exist.

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