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Socioeconomic-related Risk and STI Infection among African-American Adolescent Females

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Abstract

Introduction—Virtually no studies have examined the potential role that chronic stress, particularly the stress associated with socioeconomic (SES) strain, may play on STI risk. This study examined the association between SES-related risk at baseline to STI acquisition and reinfection over 36 months of follow-up.

Methods—627 African-American female adolescents, ages 14–20 years, recruited from sexual health clinics in Atlanta, GA participated in a randomized controlled HIV prevention trial, and returned for at least 1 follow-up assessment. Following baseline assessment, 6 waves of data collection occurred prospectively over 36 months. Chronic SES-related risk was assessed as a sum of yes-no exposure to seven risk indicators. Laboratory confirmed tests for *C. trachomatis* and *N. gonorrheoea* were performed at each follow-up.

Results—In multivariable regression analysis, SES-related risk significantly predicted STI acquisition over 36 months (AOR = 1.22) and STI reinfection (AOR = 1.16) above and beyond other known correlates of STI.

Discussion—Findings demonstrate that SES-related risk was predictive of both STI acquisition and reinfection among young African-American females. They are consistent with propositions that some health disparities observed in adulthood may be linked to earlier chronically stress-inducing life experiences, particularly experiences associated with low SES conditions. Although

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various explanations exist for the observed connection between SES-related risk and subsequent STI acquisition and/or reinfection across 36 months of follow-up, these findings highlight the need for further research to elucidate the exact pathway(s) by which SES-related risk influences later STI acquisition in order to refine STI prevention interventions for this population.

Keywords

adolescents; females; African-American; STIs; SES; stress

INTRODUCTION

Adolescents, particularly females, are disproportionately affected by sexual transmitted infections (STIs).[1] One in four females 14–19 years of age in the United States (US) is infected with *Neisseria gonorrhoeae*, *Chlamydia trachomatis, Trichomonas vaginalis*, herpes simplex virus type 2 and/or human papillomavirus (HPV).[2] However, among same-age African-American females, nearly one in two (48%) had an STI.[2] STI prevalence is particularly high in the Southern US relative to other US regions, and African-American adolescent females in the South have Chlamydia and gonorrhea rates far above national and regional averages.[3]

There are several recognized risk factors associated with increased STI rates among adolescent females; many are closely linked to developmental trajectories (e.g., coping, sexual behaviors, physical/mental health) and life transitions (e.g., substance use, interpersonal stress) that affect females physically and behaviorally.[4] Biologic differences also increase their STI risk. During adolescence the ectocervix develops a large ectropian of columnar epithelium, which may be more susceptible to sexually transmitted organisms such as Chlamydia than the squamous epithelium that develops with maturation.[5]

African-American females are at disproportionate risk of STI compared to white and Hispanic peers, despite normative sexual behavior,[6] suggesting that the observed STI disparity may not be fully explained by the prevalence of sexual risk behaviors. Racial disparities in STIs typically reflect environmental and social differences between racial groups.[6–8] For example, high levels of disassortative mixing (i.e, partners with dissimilar levels of sexual risk) among African-Americans in terms of sexual risk behavior as well as high levels of assortative mixing (i.e., partners from similar racial backgrounds) according to race may help explain high STI rates among African-Americans and racial disparities in STI rates.[9] Thus, even if African-American women have low or moderate HIV sexual risk behaviors, they are vulnerable to STI because their sexual partners are typically African-American males, a population with high rates of STIs.[6]

In addition to heightened susceptibility to STI acquisition due to age-associated factors (e.g., cervical ectopy),[4,5] potentially higher exposure to STIs in their sexual networks,[6–9] African-American adolescent females, especially those in the Southern US, may also be at heighted risk for STI acquisition due to socioeconomic (SES) risk factors prevalent among African-American families in this region.[11] These SES-related risk factors include chronic poverty and limited occupation and educational opportunities, frequent housing relocation, and unstable employment, all of which have adverse consequences for adolescents[10] Living in

low SES conditions can cause significant chronic strain on an individual, which can have deleterious effects on the functioning of biological stress regulatory systems across the lifespan and, ultimately, on later health and well-being.[11]

Research suggests that coping with SES-related stressors, which tend to be chronic rather than acute, elicits a cascade of biological responses that are functional in the short term but over time may "weather" or damage systems that regulate the body's stress responses,[12] including the sympathetic adrenomedullary system (SAM), the hypothalamic-pituitary-adrenal (HPA) axis, lipid metabolism, fat deposition, indices of inflammation, and immune functioning.[12,13] When coping demands are elevated or prolonged due to exposure to chronic stressors, the body actively mobilizes resources in response to the stressor. Some individuals become less efficient at ceasing the multifaceted physiological response resulting from chronic stress exposure, even during periods of relative calm.[12,13] The inability to "turn off" the demand response results in physiological changes that can play a part in the development of chronic illnesses and poorer reproductive health outcomes, many of which are more prevalent among African-Americans, including hypertension, cardiac disease, diabetes, and pre-term and low weight births.[11,13–15] Chronic stress has also been associated with suppression of both cellular and humoral immune function, with resulting heightened risk for immune-related disease and infection susceptibility and severity among adult samples.[16–19]

Within the STI literature, chronic stress has been associated with HIV susceptibility and progression,[17,20] and increased incidence of bacterial vaginosis (BV) even when accounting for common risk factors such as douching, frequency of sexual intercourse, and lifetime sexual partners.[19,21] Interestingly, BV has a higher incidence among African-American women compared to Caucasians,[22,23] and its occurrence can place women at increased risk for acquisition of subsequent STIs, such as Chlamydia and gonorrhea.[24]

Despite the role that SES-related stress or other chronic stress exposure may play in the development of chronic diseases and health disparities,[11] virtually no studies have focused on this as a potential pathway by which adolescent African-American females may be at increased risk of STI acquisition. Specifically, given the potential for increased STI susceptibility due to reduced immune function and the reported increase in risk for predisposing infections such as BV,[17,19–24] the role of chronic stress, such as that resulting from exposure to high SES-related risk during childhood and adolescence on prospective Chlamydia and gonorrhea infection is warranted, and has yet to be empirically examined among a sample of African-American adolescent females.

Thus, the purpose of this study was to assess the association between SES-related risk exposure during adolescence (i.e., baseline) to STI acquisition and reinfection over 36 months of follow-up, controlling for other factors associated with STI acquisition (i.e., age, coping, interpersonal stress, sexual risk behaviors, mental and physical health, douching, boyfriend, and STI history), among a sample of African-American adolescent females residing in the Southern US.

METHODS

Participants

African-American adolescent females were recruited from June 2005 to June 2007 from three clinics providing sexual health services in Atlanta, GA. Individuals were approached by an African-American female recruiter who described the study and assessed eligibility. Eligibility criteria included self-identifying as African-American, age 14–20 years and unprotected vaginal sex in the past 6 months. Exclusion criteria included being married, pregnant or attempting to become pregnant. Eligible adolescents interested in participating were scheduled to return to the clinic to complete informed consent procedures and baseline assessments and to be randomized to trial conditions. Written informed consent was obtained from all adolescents; parental consent was waived for adolescents <18 years. Of eligible adolescents, 94% (N=701) enrolled and were randomized. Participants were reimbursed $75 to complete each assessment. The Institutional Review Board at the affiliated institution approved all study protocols.

Intervention Procedures

The parent study was a randomized controlled supplemental treatment trial.[25] All participants received the same “primary” treatment, a group-delivered HIV risk reduction intervention based on HORIZONS, which is an intervention with demonstrated efficacy among African-American adolescent females.[26] Participants were randomized into one of two supplemental treatments that consisted of phone calls from a health educator every 8 weeks for 36 months (18 total calls); the intervention condition received HIV risk-reduction calls and the control received nutrition counseling calls. The purpose of the trial was to assess whether supplemental treatment enhanced maintenance of the primary treatment effects over follow-up. Detailed descriptions of all study procedures are reported elsewhere.[25]

Summary of Trial Findings

There were no differences between conditions on sociodemographic characteristics, sexual behavior or STI prevalence at randomization, and retention between conditions was similar.[25] Pertaining to the efficacy of the trial on STI over follow-up; there were significant differences between conditions through 36 months with participants in the intervention condition having fewer incident Chlamydia infections and a greater number of condom-protected sex acts.[25]

Data Collection

Data collection occurred at baseline, prior to randomization, and at 6-, 12-, 18-, 24-, 30- and 36-months following primary treatment and consisted of self-collected vaginal swab specimens for STI assessment and audio computer-assisted self-administered interviews (ACASI). The primary treatment (one day-long session) occurred immediately after baseline assessment and randomization. The majority (50.5%) completed all 6 follow-up visits; with 15.3%, 12.9%, 8.8% and 12.4% completing 5, 4, 3 and 2 follow-up visits. Specimens were assayed for two bacterial pathogens, *C. trachomatis* and *N. gonorrhoeae* using the

BDProbeTec ET *C. trachomatis* and *N. gonorrhoeae* Amplified DNA assay (Becton Dickinson and Company, Sparks, MD).[27] Participants with a positive STI test were provided directly observed single-dose antimicrobial treatment, risk-reduction counseling per CDC recommendations, and encouraged to refer partners for treatment. The County Health Department was notified of these reportable STIs.

Measures

Primary predictor

SES-related risk: Seven risk indicators were assessed, with each risk factor scored dichotomously (0 if absent, 1 if present).[28–30] SES-related risk was captured by summing responses to 4 items regarding receipt of family aid (i.e., welfare, food stamps, WIC, subsidized housing) and 3 items about neighborhood conditions ("On your street are there: a. abandoned homes or apartments?, b. buildings with broken windows?, and c. homes with bars on the windows and doors? ") Neighborhood condition was included as it has been associated with STIs in prior research,[31] and it has been suggested that residing in a disordered physical environment is stress inducing. Resulting index scores ranged from 0 to 7, with higher scores indicating higher SES-related risk exposure.

Control variables assessed at baseline

Age: Age was assessed in years.

Study condition: Participants in the intervention condition were coded 1; control condition were coded 0.

Sexual behaviors: Those who indicated using condoms during every episode of vaginal sex in the past 6 months were defined as consistent condom users (yes/no). Proportion of condom-protected sexual acts in the past 6 months was calculated by dividing the number of times sex occurred with a condom by the total frequency of sex. Lifetime number of vaginal sex partners was also collected.

Baseline STIs: Baseline STIs included *C. trachomatis,* and *N. gonorrheoea.* Participants who tested positive for either were categorized as having an STI at baseline (positive/ negative).

Substance use: Current cigarette use (yes/no), and lifetime alcohol and marijuana use were assessed (yes/no).

Coping: A modified version of the COPE scale was used to assess reliance on avoidance-based coping.[32] Higher scores indicate more reliance on avoidance-based coping.

Perceived interpersonal stress: The 13-item African-American Women's Stress Scale measured perceived interpersonal stress.[33] Higher scores indicate higher levels of current, more acute stress.

Overall physical and mental health: One item assessed physical and mental health, "During the past 30 days, for how many days did poor physical or mental health keep you from doing your usual activities, such as self-care, work, or recreation?"[34] Higher scores reflect lower physical and mental health quality of life.

Boyfriend status: Current boyfriend status was assessed (yes/no).

History of douching: Ever having douched was assessed (yes/no).

Outcomes—*STI acquisition* was defined as a positive test result for either *C. trachomatis* or *N. gonorrheoea* at a follow-up assessment subsequent to a negative result or documented directly observed antimicrobial treatment at the previous assessment.

STI reinfection was defined as a positive test result for either *C. trachomatis* or *N. gonorrheoea* at multiple follow-up assessments subsequent to a positive test and receipt of directly observed antimicrobial treatment at the previous assessment.

Data Analysis

Analysis was restricted to data collected as part of the parent study from participants who completed ≥1 follow-up assessment through 36-months. Of the 701 randomized and providing baseline data, 627 (89.4%) completed ≥1 follow-up assessment. For analysis with reinfection as the outcome, only those who completed ≥2 follow-up assessments were included (n = 556). Descriptive statistics were calculated for all study variables, and bivariate analyses examined differences on the primary predictor and control variables between those who did or did not acquire an STI across 36 months of follow-up and those who were or were not reinfected across follow-up. Differences were assessed using independent samples *t* tests for continuous variables and Chi-square analyses for categorical variables. Variables significant at $p < .10$ in bivariate analyses,[35] were entered as predictors in multivariable logistic regression models predicting each outcome. Significant control variables were entered in the first block; SES-related risk was entered in the second block of each model. In addition to age, study condition and baseline STI, baseline predictor values were used as there were significant intracorrelations per variable across assessment points (e.g., baseline coping scores were highly correlated with follow-up coping scores), suggesting that baseline values serve as good approximations for each construct for analytic purposes.

RESULTS

Sample Description

Descriptive statistics are presented in Table 1 for all study measures. The majority was still in high-school or had only completed some high-school at enrollment (51.9%). 36.8% had a job, and many reported living with their mother only (44.0%) or mother and father (16.6%). At baseline, adolescents reported, on average, 27.3 episodes of vaginal sex in the previous 6 months.

Bivariate Analyses

SES-related risk, age, coping, and having an STI at baseline were significantly associated with STI acquisition across the 36 month follow-up period (Table 2). Along with SES-related risk, age, interpersonal stress, and having an STI at baseline were significantly associated with reinfection across follow-up (Table 3).

Multivariable Regressions Predicting STI Acquisition and Reinfection across Follow-Up

Overall, the 2-step model was significant for predicting STI acquisition over the follow-up period (Table 4). Younger age and more SES-related risk were significantly associated with increased odds of STI acquisition. The 2-step model was also significant for predicting reinfection over follow-up. Above and beyond age and STI at baseline, more SES-related risk significantly increased odds of STI reinfection.

DISCUSSION

Using a prospective design, we tested the hypothesis that SES-related risk measured at baseline assessment during adolescence predicts STI acquisition and reinfection over 36 months, while controlling for other factors associated with disparities in STI acquisition such as age, sexual risk behaviors, STI history, coping, other stressors (interpersonal stress), and mental and physical health, among a sample of African-American adolescent females residing in the Southern US. SES-related risk predicted both STI acquisition and reinfection in this study. Although multiple explanations exist for why SES-related risk may be associated with STI acquisition, these findings are consistent with propositions that some health disparities observed in adulthood may be linked to earlier life experiences, particularly experiences associated with low SES conditions which can cause significant chronic strain on an individual and thereby placing them at heightened biological risk for STI.[11–14,19–24]

Other plausible explanations exist for the association between SES-related risk and STI acquisition in this sample. From a developmental perspective, socioeconomic conditions early in life may influence adolescent or young adult sexual health directly and indirectly. Directly, low SES can reduce access to resources (e.g., money, access to medical care, STI knowledge) thereby increasing STI risk. Alternatively, circumstances surrounding SES-risk in early life and adolescence may increase exposure to and probability of experiencing adverse life events that can contribute to trajectories of behavioral pathology (e.g. adolescent delinquency, depression). These pathologies have been associated with increased engagement in high-risk sexual behaviors,[36,37] suggesting another mechanism by which SES-risk may increase STI acquisition.[38] This indirect path posits that adolescent adjustment (e.g., coping ability, mental health) may mediate the association between SES-related risk and later STI acquisition.[38] Wickrama et al. identified a direct association between community-level disadvantage during childhood and STI prevalence in a nationally representative sample of young adults.[38] Importantly, Wickrama's study also found that adolescent adjustment difficulties and risky sexual practices only partially mediated the relationship between SES-related factors, suggesting that other, undetermined mechanisms may also mediate this relationship.[38] In the current study, sexual risk, mental health ratings,

and coping were included in analyses to account for these demonstrated influential factors on STI acquisition, yet they were not significantly predictive of STI acquisition when SES-related risk was included in the models.

Alternatively, biological mechanisms may also explain the observed findings. Coping with the SES-related stressors on a constant basis may "weather" or damage the systems that regulate the body's stress response,[11–14] which results in physiological changes that heighten one's infection susceptibility. The connection between chronic stress and heightened infection susceptibility has been demonstrated among adult samples,[16–24] yet this biological explanation for how SES-related risk may increase susceptibility to STI infection in particular, among adults or adolescent samples, has been largely unexamined. The findings of the current study suggest that SES-related risk plays a role in subsequent STI acquisition and reinfection among a sample of young African-American females, above and beyond that of other known correlates of STI risk. Although this study did not collect biological and physiological data to fully test the aforementioned hypothesis, the findings lay the foundation for future studies to investigate this biologically plausible hypothesis.

Further research is needed to elucidate the pathway(s) by which SES-related risk exposure early in life influence later STI acquisition in order to identify new targets for STI prevention interventions and interrupt the effects of exposure on STI acquisition. From a public health perspective, understanding ways to optimize STI prevention interventions, such as by including content designed to enhance coping and reduce the effects of stress during childhood and adolescence, may be beneficial for reducing STI acquisition among those with high exposure to SES risk. Additional components, such as teaching developmentally appropriate stress-coping skills and cognitive behavioral management skills,[39] mindfulness training that would help youth learn relaxation techniques for managing uncontrollable stressors,[40] and strategies for building and accessing social support systems, could be integrated into existing STI prevention interventions to optimize their efficacy among youth exposed to high levels of SES risk or other chronic stressors.

Limitations

SES-related risk was assessed at baseline when participants were 14–20 years of age, thus may not accurately capture changes in SES-related risk exposure across childhood. Future research may consider collecting data earlier in adolescents' development, or collecting a detailed SES-related risk history. Further, participants were part of a larger STI prevention intervention trial and may not be representative of other adolescent African-America females not eligible or enrolled in the trial. Also, measurement of constructs was restricted to the variables assessed in the parent study and self-reported sexual behavior, especially over longer periods of recall, is subject to recall bias. While this intervention was intended to reduce sexual risk behaviors, which could influence direct risk of STI acquisition, both reported sexual risk and the intervention condition were included as potential confounders in all analyses.

Conclusions

Although various explanations exist for the observed connection between SES-related risk and subsequent STI acquisition and/or reinfection across 36 months of follow-up, there remains a gap in our empirical knowledge pertaining to the mechanisms by which SES risk influences STI acquisition. The ability to identify causal factors underlying the observed link between SES-related risk and STI acquisition may be an important step in refining, adapting, or designing new STI prevention interventions to optimize their efficacy for especially vulnerable youth.

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Implications and Contribution

SES-related risk, a proxy for chronic stress resulting from residing in low SES conditions, is related to STI acquisition, though there are many possible explanations for the connection between SES-related risk and subsequent STI acquisition and/or reinfection.

Table 1

Descriptive statistics for all study variables (N = 627).

	Mean (frequency)	SD (%)	Observed Range
Primary Predictor			
SES-related risk (mean/SD)	1.50	1.55	0–6
Baseline Control Variables			
Age (mean/SD)	17.61	1.69	14–21
Intervention group (frequency/%)	309	49.3	0–1
Smokes cigarettes (frequency/%)	97	15.5	0–1
History of alcohol use (frequency/%)	485	77.4	0–1
History of marijuana use (frequency/%)	453	72.2	0–1
Mental and physical health (mean/SD)	2.83	5.72	0–30
Interpersonal stress (mean/SD)	28.24	13.10	0–63
Coping (negative coping) (means/SD)	19.23	4.46	10–31
History of douching (frequency/%)	262	41.8	0–1
Boyfriend status (frequency/%)	496	79.1	0–1
Consistent condom use (past 6 mo.)[a] (frequency/%)	73	11.6	0–1
Proportion condom use (past 6 mo.) (mean/SD)	0.50	0.35	0.0–1.00
Lifetime number of sexual partners (means/SD)	8.26	12.78	1–200
STI at baseline (frequency/%)	125	19.9	0–1
Outcomes			
STI acquisition across 36 month follow-up (frequency/%)	236	37.6	0–1
Reinfection across 36 month follow-up [b] (frequency/%)	86	15.5	0–1

Note:

[a] Screening for inclusion criteria was conducted at the clinic when the young women were approached about the study. If eligible and interested, they were then scheduled to come back at a later date for the baseline visit/intervention session (which was typically within a month for most participants, but could have been longer). Thus, these young women, because of the later date of the actual baseline visit, could have screened eligible yet then reported no unprotected sex acts in the past 6 months.

[b] for the outcome variable reinfection across 36 month follow-up the n = 556.

Table 2

Differences in primary and control variables by STI acquisition status across 36 months of follow-up assessment (N = 627).

Study variables	Acquired STI over follow-up (n = 236)	Did not acquire STI over follow-up (n = 391)	Test statistic	*p*
Primary predictor				
SES-related risk[b]	1.86 (1.70)	1.29 (1.42)	−4.30	.0001
Baseline controls				
Age[b]	17.31 (1.68)	17.80 (1.67)	3.55	.0001
Intervention group[a]	116 (49.2%)	193 (49.4%)	0.003	.960
Smokes cigarettes[a]	42 (17.8%)	55 (14.1%)	1.57	.211
History of alcohol use[a]	183 (77.5%)	302 (77.2%)	0.01	.930
History of marijuana use[a]	174 (73.7%)	279 (71.4%)	0.41	.520
Mental and physical health[b]	3.28 (5.96)	2.56 (5.57)	−1.53	.126
Interpersonal stress[b]	27.91 (13.77)	28.44 (12.70)	0.49	.626
Coping (negative coping)[b]	19.83 (4.37)	18.87 (4.49)	−2.63	.009
History of douching[a]	95 (40.3)	167 (42.7)	0.37	.546
Boyfriend status[a]	191 (80.9)	305 (78.0)	0.76	.382
Consistent condom use (6 mo.)[a]	25 (10.6%)	48 (12.3%)	0.41	.524
Proportion condom use (6 mo.)[b]	0.50 (0.35)	0.50 (0.35)	−.01	.994
Lifetime number of sexual partners[b]	9.21 (17.38)	7.69 (8.91)	−1.25	.214
STI at baseline[a]	57 (45.6%)	179 (35.7%)	4.22	.040

Note:

[a]Chi-square is test statistic and frequency (percent) are reported,

[b]t-test is the test statistic and mean (standard deviation) are reported

Table 3

Differences in primary and control variables by reinfection status across 36 months of follow-up assessment (N = 556).

Study variables	Reinfection over follow-up (n = 86)	No reinfection over follow-up (n = 470)	Test statistic	*p*
Primary predictor				
SES-related risk[b]	1.92 (1.56)	1.47 (1.56)	−2.45	.015
Baseline controls				
Age[b]	17.26 (1.61)	17.66 (1.71)	2.02	.044
Intervention group[a]	37 (43.0%)	238 (50.6%)	1.69	.194
Smokes cigarettes[a]	15 (17.4%)	67 (14.3%)	0.59	.444
History of alcohol use[a]	65 (75.6%)	367 (78.1%)	0.26	.608
History of marijuana use[a]	58 (67.4%)	339(72.1%)	0.78	.377
Mental and physical health[b]	2.69 (5.22)	2.85 (5.80)	0.16	.873
Interpersonal stress[b]	25.80 (13.87)	28.52(13.07)	1.75	.080
Coping (negative coping)[b]	19.81 (4.07)	19.19 (4.56)	−1.20	.230
History of douching[a]	32 (37.2%)	196 (41.7%)	0.61	.436
Boyfriend status[a]	74 (86.0%)	366 (78.0%)	2.94	.110
Consistent condom use (past 6 mo.)[a]	7 (8.1%)	55 (11.7%)	0.93	.335
Proportion condom use (past 6 mo.)[b]	0.48 (0.34)	0.49 (0.36)	0.37	.713
Lifetime number of sexual partners[b]	7.76 (12.16)	8.52 (13.63)	0.49	.628
STI at baseline[a]	27 (31.4%)	81 (17.2%)	9.32	.002

Note:

[a]Chi-square is test statistic and frequency (percent) are reported,

[b]t-test is the test statistic and mean (standard deviation) are reported

Table 4

Multivariable logistic regression models predicting STI acquistion and reinfection across 36 months of follow-up.

Predictors	β	SE	95% CI Odds Ratio	Lower	Upper	*p*
			STI acquisition across 36 month follow-up			
STEP 1:						
Age	−.13	.05	.88	.79	.97	.010
STI at baseline	.28	.21	1.33	.88	2.00	.171
Coping (negative coping)	.03	.02	1.03	.99	1.07	.124
STEP 2:						
SES-related risk	.20	.06	1.22	1.09	1.35	.001
Step 1 $\chi^2 = 19.68$						.001
Step 2 $\chi^2 = 12.62$						.001
Overall Model $\chi^2 = 32.30$						.0001
			Reinfection across 36 month follow-up			
STEP 1:						
Age	−.10	.07	.90	.79	1.05	.152
STI at baseline	.74	.27	2.10	1.24	3.56	.006
Interpersonal stress	−.02	.01	.98	.96	1.00	.045
STEP 2:						
SES-related risk	.15	.07	1.16	1.01	1.34	.046
Step 1 $\chi^2 = 15.34$						.002
Step 2 $\chi^2 = 3.88$						.049
Overall Model $\chi^2 = 19.22$						.001