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Using Integrative Data Analysis to Examine Changes in Alcohol Use and Changes in Sexual Risk Behavior across Four Samples of STI Clinic Patients

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Abstract

Background—Patients in sexually transmitted infection (STI) clinics report high levels of alcohol use, which are associated with risky sexual behavior. However, no studies have examined how *changes* in alcohol use relate to *changes* in sexual risk behavior.

Purpose—We used parallel process latent growth modeling to explore how changes in alcohol use related to changes in sexual behavior across four samples of clinic patients.

Methods—Patients participating in HIV prevention trials from urban clinics in the Northeastern and Midwestern U.S. ($N = 3,761$, 59% male, 72% Black) completed measures at 3mo intervals over 9–12mo. Integrative data analysis was used to create composite measures of alcohol use across samples. Sexual risk measures were counts of partners and unprotected sex acts. Parallel process models tested whether alcohol use changes were correlated with changes in number of partners and unprotected sex.

Results—Growth models with good fit showed decreases that slowed over time in sexual risk behaviors and alcohol use. Parallel process models showed positive correlations between levels of

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($r_s = .17-.40$, $p_s < .001$) and changes in ($r_s = .21-.80$, $p_s < .05$) alcohol use and number of sexual partners across studies. There were strong associations between levels of ($r_s = .25-.43$, $p_s < .001$) and changes in ($r_s = .24-.57$, $p_s < .01$) alcohol use and unprotected sex in one study recruiting hazardous drinkers.

Conclusions—Across four samples of clinic patients, reductions in alcohol use were associated with reductions in the number of sexual partners. HIV prevention interventions may be strengthened by addressing alcohol use.

Keywords

sexual behavior; sexually transmitted infections; alcohol use; integrative data analysis

Sexually transmitted infections (STIs) present a critical health challenge, with more than 19 million new infections each year in the United States (1). Consequences of infection include infertility, ectopic pregnancy, pelvic inflammatory disease, birth complications, and increased risk of certain cancers (1, 2). Moreover, untreated STIs substantially increase the risk of both acquisition and transmission of HIV (2, 3). STIs cost the U.S. health care system an estimated \$17 billion annually (1), with new HIV infections costing an additional \$36 billion (4).

Patients attending STI clinics demonstrate high levels of risky sexual behavior, including multiple sexual partners, casual partners, and sexual concurrency (5, 6). Additionally, clinic patients are likely to experience repeat STIs (7-10) and are at increased risk of HIV relative to the general population (6, 11). The prevalence and cost of STIs and HIV make them a major public health problem, and STI clinic patients are a population of special risk.

For this population, developing and disseminating cost-effective behavioral interventions to reduce sexual risk behaviors is an important public health goal. Meta-analyses show clinic-based behavioral interventions (which most commonly comprise testing and counseling, education, skills training, and motivational components) are generally successful in increasing condom use, reducing number of partners, and reducing rates of STIs (12). However, intervention effects tend to be small and to deteriorate over time (12). One reason why effect sizes for behavioral interventions in this setting may be small is because patients often have co-morbid substance use problems. Studies have suggested that patients at STI clinics have higher levels of problematic alcohol use (as indicated by positive screenings on instruments such as the Alcohol Use Disorders Identification Test and CAGE questionnaire) than those in the general population or those in other medical settings (such as emergency rooms; 13, 14, 15). In one large study, one-third of patients at STI clinics demonstrated problematic alcohol use (13).

Research suggests that alcohol use contributes to sexual risk behavior. Cross-sectionally and prospectively, alcohol consumption is related to STI and HIV infection (16-20); one meta-analysis of prospective studies found that rates of HIV infection were 120% higher for binge drinkers vs. non-drinkers (17). Drinking is also associated with an increased probability of intercourse, risky partner choices, and decreased risk discussion (19, 21-23). Results related to condom use and unprotected sex are more mixed, with most reviews and meta-analyses

suggesting no consistent association (21, 23, 24). However, some studies have identified a link between alcohol consumption and unprotected sex, with drinkers or heavy drinkers being less likely to use condoms (19, 25-27). Additionally, experimentally, increases in blood alcohol content (BAC) are associated with increases in intentions to engage in unprotected sex (28). Studies focusing specifically on STI clinic populations, most of which have been cross-sectional or focused on one specific sexual event, have also supported these associations between alcohol use and risky behaviors and STIs (20, 29-33).

Theoretically, alcohol myopia theory posits that alcohol use interferes with decision-making by reducing cognitive capacity and causing individuals to focus on salient, impelling cues rather than distal, inhibitory cues (34). Alternatively, expectancies theory suggests that individuals act in line with their beliefs that alcohol will promote risky sexual behavior when intoxicated (35). In contrast to these causal explanations, third-variable explanations focus on factors that might influence both alcohol use and sexual behavior, such as situational features (36, 37), social network composition (38), socioeconomic factors (39-41), and personality characteristics (33). Regardless of the theoretical explanation, alcohol use is clearly an important factor with respect to sexual risk behavior.

Despite the large literature on alcohol use and risk behavior, the majority of studies in this area utilize cross-sectional data, single-event, or longitudinal data collected over a relatively brief time frame. Additionally, few longitudinal studies have focused specifically on STI clinic patients, who, relative to the general population, often demonstrate elevated levels of both sexual risk behavior and alcohol use. Better understanding the covariation among alcohol use and sexual risk taking in the STI clinic population can help in the design of more efficacious sexual risk reduction interventions for implementation in the clinic setting. Specifically, it is important to know whether clinic patients' intraindividual changes in alcohol use over time are associated with changes in sexual risk behavior. If this is the case, alcohol use may be an important target for clinic interventions.

Thus, to address this gap in the literature, we used parallel process latent growth modeling to explore how *changes over time* in alcohol use relate to *changes over time* in sexual risk behavior among STI clinic patients. Parallel process modeling, also known as associative latent growth modeling, is a sophisticated analytic technique that allows for the examination of correlations between developmental parameters (intercepts and slopes) for multiple constructs (42). By examining associations between these developmental parameters, multivariate latent growth models clarify how changes in one variable relate to changes in another variable. In this case, we examined how changes in alcohol use related to changes in number of partners and number of unprotected sex events.

A major innovation of the current research is the use of integrative data analysis (IDA), a technique for pooling multiple, independent datasets (43-47). By pooling multiple datasets, integrative data analysis allows for the creation of large, diverse samples with sufficient statistical power to test complex models. Notably, research has suggested that studies examining correlated change using parallel process latent growth models are often underpowered, which may result in failures to detect existing correlations between slopes (48). The current study overcomes this weakness by integrating data from four independent,

longitudinal studies enrolling patients from urban STI clinics to yield a sample of nearly 4,000 patients. In addition to providing sufficient power to examine associations between changes in alcohol use and changes in sexual risk behavior, this pooled sample allows us to compare patterns across data sets and draw broader conclusions about this relationship in STI clinic patients.

The current study used up to five assessments (at 3-month intervals across 1 year) from a pooled sample of STI clinic patients to test these hypotheses: (a) initial levels of alcohol use would be positively associated with the initial numbers of sexual partners and unprotected sex events, and (b) changes over time in alcohol use would be positively associated with changes over time in number of sexual partners and number of unprotected sex events. We tested these hypotheses using data from four studies with patients from urban, publicly-funded STI clinics, which allowed us to explicitly test whether associations differed across multiple studies. To our knowledge, this is the first integrated sample of multiple independent studies in the HIV prevention context.

Method

Participants and Procedures

Participants were patients at publicly-funded, urban STI clinics in the Midwest and Northeast who were recruited into four separate randomized controlled trials to evaluate sexual risk reduction interventions utilizing an information-motivation-behavioral skills (IMB; 49, 50) framework. Although detailed information is available in the primary sources (51-55), we summarize these studies briefly here.

Study 1, the HIP-R (Health Improvement Project-Rochester; 51), recruited patients from a clinic in the Northeast between 2004 and 2006. To be eligible for the study, patients reported sexual risk behavior (e.g., multiple sexual partners, inconsistent condom use). They were randomly assigned first to a brief (15 min.), in-clinic intervention (either an information-only or brief motivational intervention), and then to either one of two intensive interventions (information-only or IMB) or a control condition. The brief interventions and the intensive IMB intervention briefly addressed substance use as a potential trigger for risky sex, but interventions did not target reductions in alcohol use directly. Intensive interventions were 4 hours in length, and 56% of participants assigned to an intensive intervention attended. Brief and intensive interventions were crossed in a 2×3 design. Results showed decreases in sexual risk behaviors across all conditions.

Study 2, the HIP-R2 (52) recruited from this same clinic between 2010 and 2012. To be eligible for the study, patients again reported sexual risk behavior. Participants were randomly assigned to view either a brief (15 min) sexual risk reduction DVD, or a brief general health promotion DVD (control), which included a small amount of material related to alcohol use. Additionally, half of participants in each DVD condition were randomly assigned to complete a baseline sexual health assessment (containing a detailed sexual health history), while half completed a general health assessment (containing only a few sexual health items). Participants in all conditions reduced their sexual risk behavior.

Study 3, In the Know (ITK), (53) recruited patients from an urban STI clinic in the Midwest in 2001 and 2002. All individuals receiving STI diagnostic and treatment services were eligible for the study. Participants were randomly assigned to one of four intervention conditions: HIV/AIDS information only; information and motivational enhancement; information and behavioral skills building; or information, motivational enhancement, and behavioral skills building. Alcohol use was briefly addressed as a trigger of sexual risk in the motivational and behavioral skills components but was not targeted directly. Interventions were delivered in single 90-min counseling sessions, and 91% of participants attended their assigned intervention. The full IMB session was most effective for men, while motivational counseling demonstrated the most positive outcomes for women.

Study 4, Project ACHIEVE (Alcohol Counseling and HIV Intervention; 55), recruited patients from the same Midwestern clinic between 2002 and 2004. To be eligible for the study, participants reported sexual risk behavior in the past 3 months and showed evidence of an alcohol use disorder. Participants were randomly assigned to either IMB-based HIV test counseling (pre- and posttest, totaling 30 min + 45 min general health info); IMB-based counseling plus a brief motivation intervention for alcohol use disorders (totaling 75 min); or IMB-based counseling plus a brief motivational intervention for alcohol use disorders (totaling 75 min) plus a booster session related to alcohol use. In addition to the alcohol intervention, participants discussed whether they saw alcohol use as a trigger of risky sex. Participants in all conditions reduced their sexual risk behavior.

We pooled these four samples to form an integrative data analysis sample; see Table 1 for a summary of demographic characteristics of the individual and pooled samples. Collectively, the four studies assessed alcohol use and sexual risk behavior over a 1-year span for 3,761 individuals through 13,203 total individual observations. The sample had a mean age of 30.86 ($SD = 10.30$) and was 59% male. Participants primarily identified as Black (72%); other common racial and ethnic identities were White (18%), mixed/multiracial (5%), and Latino (7%). The majority of the participants self-identified as heterosexual (89%). Participants were generally of low socioeconomic status, with 69% having a high school education or less and 54% reporting unemployment at the time of study enrollment. Over three-quarters of participants (78%) were single, while 8% were separated, 7% divorced, 6% married, and 1% widowed. Roughly one-third of participants (36%) were randomly assigned to the most active intervention condition in their respective study.

Measures

Assessments in all studies were administered using audio-computer assisted structured interviews (ACASI).

Demographic variables—The demographic control variables included sex (0 = male, 1 = female); age in years; race (0 = non-White, 1 = White); sexual orientation (0 = heterosexual, 1 = sexual minority); and socioeconomic status. There were multiple indicators of socioeconomic status combined into an integrated measure (see *Measurement Integration* section).

Intervention condition—Intervention condition was included as a control variable (0 = control conditions/less active comparisons, 1 = most active intervention condition). The coding of most active conditions was based on study hypotheses, as follows: in the HIP-R, the intensive IMB intervention; in the HIP-R2, the sexual health promotion DVD; in ITK, the intervention including all components of the IMB; in ACHIEVE, IMB-based counseling plus motivational interviewing to reduce alcohol use plus the alcohol-related booster session. We also tested whether results differed with an alternative coding of intervention condition (0 = control condition, 1 = any active intervention). Under this coding scheme, the HIP-R intensive information intervention, the ITK interventions pairing information and motivation or information and behavioral skills, and the ACHIEVE intervention involving IMB-based counseling and the alcohol intervention but no booster session were also considered active interventions. Results did not differ dependent on the coding of intervention conditions.

Alcohol use—A composite, integrated measure of alcohol use was created across studies (see *Measurement Integration* section for details). Core indicators of alcohol use assessed across studies included frequency of alcohol use, engagement in heavy episodic drinking, and drinks per drinking day (56). Supplementary indicators included peak alcohol consumption, drinks per week, and frequency of intoxication.

Number of sexual partners—Participants reported the number of different partners they had sex with during the past 3 months in the HIP-R, HIP-R2, and ACHIEVE, and the number of partners over the past 1 month in ITK. Values were log transformed.

Number of unprotected sexual events—Participants reported the number of times they had engaged in vaginal or anal sex without a condom over the past 3 months in the HIP-R, HIP-R2, and ACHIEVE. Participants in ITK completed a partner-by-partner assessment of unprotected sex acts in which participants recalled their four most recent sex partners. For each partner, the total number of intercourse acts without condoms during the past 1 month was recorded, and these values were summed. Values were log transformed.

Data Analysis

Measurement Integration—To combine measures when some items or response options varied across studies, we used moderated non-linear factor analysis (MNLFA; 57), which we implemented in SAS using PROC NLMIXED (55, pp. 4375–4376). The measures that differed across studies included alcohol use and socioeconomic status. For details regarding the measurement integration process, see Electronic Supplementary Material 1.

Alcohol use—Indicators of alcohol use were correlated with one another across studies, $r_s = .49-.71$, $p_s < .001$, and factor loadings were positive and significant, $p_s < .0001$. We tested for moderation by sex, time point, and study membership. Results indicated no significant moderation by time, very little moderation by sex, and modest moderation by study membership. Some items were invariant across studies, giving us confidence that the construct was appropriately anchored and interpretable across studies (57).

Socioeconomic status—In general, indicators of socioeconomic status were correlated with one another across studies, $r_s = .17-.50$, $p_s < .001$ (in ITK, education and employment were not correlated, $r = .07$, $p = .11$), and factor loadings were positive and significant, $p_s < .003$. We tested for moderation by sex, age, race, and study membership. Results indicated modest moderation by sex, age, race, and study membership. Some items were invariant across studies, giving us confidence that the construct was appropriately anchored and interpretable across studies.

Missing Data and Data Management—Study attrition resulted in some missing data in all studies. There was also a modest amount of missing data due to skipped items. Overall, 100% of participants ($N = 3752$) had data on key variables at baseline, 76% ($N = 2845$) at 3 months, 74% ($N = 2777$) at 6 months, 42% ($N = 1588$) at 9 months, and 60% ($N = 2241$) at 12 months. (The HIP-R did not include a 9 month follow-up, and ITK did not include a 12 month follow-up.) On average, participants completed assessments of key variables at 3.51 time points ($SD = 1.36$). Participants with missing data on key variables were more likely to be male and non-Black and were younger and of higher socioeconomic status. Those with missing data also reported heavier drinking at baseline ($M = 0.08$, $SD = 1.01$) than those without missing data ($M = -0.05$, $SD = 0.99$), $t(3680) = -3.75$, $p < .001$. There were no differences in baseline sexual risk behavior for those with and without missing data. Further information on missing data in individual studies is available elsewhere (51-53).

We handled missing data in two ways. First, to address missing data on outcome variables, we used a full information maximum likelihood estimator in Mplus (the ML estimator; 58). The full information maximum likelihood approach provides reasonable estimates of standard errors with missing data (42) and allows for the use of all available data. Second, we used multiple imputation to replace values for missing covariates (59, 60). Multiple imputation avoids biases associated with using only complete cases or with single imputations (61). We imputed 100 datasets (62) in Mplus 7 (63). All baseline study variables were included in the imputation, which also accounted for study membership. Analyses were conducted with all 100 datasets, and parameter estimates were pooled using the imputation algorithms in Mplus 7.

Outliers on the counts of number of partners and number of unprotected sex acts (i.e., those more than three times the interquartile range from the 75th percentile) were truncated to three times the interquartile range from the 75th percentile plus one (64).

Latent Growth and Parallel Process Models—To understand the associations between changes in alcohol use and changes in sexual risk behavior over time, we used parallel process (associative) latent growth modeling. We first fit individual latent growth models to alcohol use, number of sexual partners, and unprotected sex data over one year in order to describe the general pattern of change over time. These growth models were fit in Mplus 7 (63). Across models, we fixed slope loadings for the 5 time points such that a one-unit change represented change over 3 months (e.g., at 0, 1, 2, 3, and 4). We compared several models of change, including no-change (intercept-only) models, linear models, quadratic models, and piecewise growth models with transitions at 3, 6, and 9 months. We compared nested models (no-change, linear, and quadratic) using the chi-square difference

test, and non-nested models on the basis of the Bayesian information criterion (BIC; 65). Better fitting models are indicated by lower BIC values.

Following the choice of a best-fitting model, we fit multigroup quadratic latent growth models with study as the grouping variable to examine study differences in changes controlling for demographic characteristics and intervention condition. These models used the multiple imputation data to account for missing data on covariates. Covariates were grand mean centered. Demographic paths were constrained to be equal across studies, while all other parameters were free to vary. We compared growth parameters across the four studies using the Wald chi-square test (66) and an adjusted alpha rate of .01; this was necessary given the use of multiple imputation, which made chi-square comparisons of nested models inappropriate (63). For growth models involving quadratic trends, where the slope differed at each time point, we assessed the rate of change at each time point by reparameterizing the model to move the intercept.

Individual growth models were then combined to look at associations between changes in alcohol use and changes in (1) number of sexual partners and (2) changes in unprotected sex over time. Parallel process models included correlations between all growth factors. These models also incorporated demographic controls. We tested for study differences in the associations between growth factors using the Wald test in order to determine whether the relationship between alcohol use and sexual risk differed across studies. We constrained covariances between growth parameters across studies using equality constraints when doing so did not significantly undermine model fit; this was intended to increase parsimony as well as power to estimate these parameters, given that studies using parallel process latent growth models are often underpowered (48). For growth models and parallel process models, model fit was assessed using traditional fit indices, including the comparative fit index (CFI; 67), the Tucker-Lewis index (TLI; 68), and the root-mean-square error of approximation (RMSEA; 69). Good fit is indicated by CFI and TLI values greater than .95 and RMSEA values less than .05 (69, 70). These fit indices are averages over 100 imputations.

To convey the associations between growth factors, we report standardized correlation coefficients. For demographic associations, we report standardized coefficients; in the case of binary covariates, we report StdY estimates from Mplus, which can be interpreted as the change in the growth factor in standard deviation units when the covariate changes from zero to one (63).

Results

Sample Characteristics

Descriptive statistics for the alcohol use and sexual risk measures over time are included in Table 2 and Electronic Supplementary Material 2 (Table 1). Alcohol use was very common—62% of participants engaged in heavy episodic drinking, and 51% had 3+ drinks on the average drinking day. The integrated measure was scaled to have a mean of 0 ($SD = 1.00$) at baseline. At this level, during the past 3 months, most participants drank several times per month (50%) or several times per week (31%), drank 3 or more drinks per drinking day (56%), and engaged in some heavy episodic drinking (70%). ACHIEVE recruited

participants demonstrating hazardous drinking; the mean level of drinking was 0.92 ($SD = 0.78$) in this sample at baseline. At this level, during the past 3 months, nearly all participants drank several times per week (53%) or 4 or more times per week (29%), drank 3 or more drinks per drinking day (96%), and engaged in some heavy episodic drinking (96%).

At baseline, most participants (64%) reported multiple partners; the mean number of partners in the past 3 months (in the three studies with a 3-month measure) was 2.85 ($SD = 2.30$). Participants also engaged in high levels of unprotected sex. The mean number of unprotected sex acts in the past 3 months (in the three studies with a 3-month measure) was 18.88 ($SD = 23.78$).

Latent Growth Models

Alcohol Use—A quadratic latent growth model was the best fit to the alcohol use data, $\chi^2(10, N = 3745) = 83.52, p < .001$, CFI = .99, TLI = .99, RMSEA = .04. The quadratic model fit significantly better than a linear model, $\chi^2(4) = 130.01, p < .001$. Additionally, comparison of the BICs suggested this model fit better than did any 2-part growth models (quadratic BIC = 28680.63; 2-part BICs = 28700.64–28800.63). Separate growth models for each study showed that this quadratic model fit well in each case.

We next fit a multigroup quadratic latent growth models with study as the grouping variable. This model controlled for demographics and intervention condition. This model fit the data well, $\chi^2(119, N = 1534, 1009, 609, 591) = 221.18, p < .001$; RMSEA = .03; CFI = .99; TLI = .98. This model (Figure 1; Table 3; Electronic Supplementary Material 2 [Table 2]) showed a significant negative slope within each study, combined with a positive quadratic in three of the four studies. This indicated that levels of alcohol use were decreasing over time and that the rate of decrease was slowing over time. Reparameterization showed that significant decreases (as indicated by a significant negative slope) continued through 6 months in ITK and through 9 months in the other three studies. Initial levels of alcohol use were highest and decreases over time were largest in ACHIEVE, as would be anticipated based on enrollment requirements. For each study, there was significant variance in initial rates of alcohol use, in the slopes at the majority of time points, and in the quadratic component (in all but ITK), indicating differences between participants in baseline alcohol use as well as in the change in alcohol use over time. Initial levels of alcohol use were not associated with decreases over time.

Demographically, heavier alcohol use was reported at baseline by men ($\beta = 0.63$ – $1.21, SE = 0.06$ – $0.31, p < .001$), White participants ($\beta = 0.62$ – $1.18, SE = 0.08$ – $0.31, p < .001$), and sexual minorities ($\beta = 0.33$ – $0.63, SE = 0.08$ – $0.21, p < .001$). Decreases in alcohol use were greater for sexual minorities ($\beta = -0.98$ – $-0.34, SE = 0.17$ – $0.71, p < .05$). There were no differences in initial levels of alcohol use or in the change in alcohol use over time based on intervention participation, $\beta = 0.01$ – $0.02, SE = 0.05$ – $0.09, p = .85$ and $\beta = -0.37$ – $-0.13, SE = 0.10$ – $0.33, p = .16$, respectively. We also tested for interactions between study and intervention participation by separately estimating this parameter in each study and found no significant relationship in any study.

Number of Sexual Partners—A quadratic growth model was the best fit to the data for number of sexual partners, $\chi^2(10, N = 3759) = 71.21, p < .001$, CFI = .98, TLI = .98, RMSEA = .04. The quadratic model fit significantly better than a linear model, $\chi^2(4) = 275.56, p < .001$. Additionally, comparison of the BICs suggested this model fit better than did any 2-part growth models (quadratic BIC = 17383.70; 2-part BICs = 17450.62-17482.34). Separate growth models for each study showed that this quadratic model fit well in each case. However, the quadratic component was non-significant and had no significant variance in ITK; it was thus constrained to 0 in the multigroup model.

We next fit a multigroup quadratic latent growth model with study as the grouping variable. This model controlled for demographics and intervention condition. This model fit the data well, $\chi^2(123, N = 1537, 1009, 609, 604) = 244.57, p < .001$; RMSEA = .03; CFI = .97; TLI = .97. This model (Figure 2; Table 4, Electronic Supplementary Material 2 [Table 3]) showed a significant negative slope within each study, combined with a positive quadratic in three of the four studies. Thus, in one study, the number of partners was decreasing slowly over time, while in the other three studies, the number of partners was decreasing more rapidly but the rate of decrease was slowing over time. Reparameterization showed that significant decreases continued through 6 months in the HIP-R and through 9 months in the other three studies. Additionally, for each study, there was significant variance in the number of partners at baseline, in the slope at the majority of time points, and in the quadratic component (in all but ITK), indicating differences between participants in the initial number of partners as well as in the change in number of partners over time. Finally, this model showed that participants who had more partners at baseline decreased their number of partners more over time in ITK and ACHIEVE, $r_s = -.44$ (0.10) and $-.52$ (0.07), $p_s < .001$, but not in the HIP-R or HIP-R2.

Demographically, more recent sexual partners were reported at baseline by men ($\beta = 0.64$ -1.75, $SE = 0.09$ -0.23, $p < .001$), younger participants ($\beta = -0.37$ -0.15, $SE = 0.04$ -0.08, $p < .001$), sexual minorities ($\beta = 0.96$ -2.61, $SE = 0.13$ -0.34, $p < .001$), and participants with lower socioeconomic status ($\beta = -0.20$ -0.11, $SE = 0.05$ -0.08, $p < .05$). Decreases in the number of sexual partners were greater for men ($\beta = -7.10$ -0.47, $SE = 0.20$ -3.78, $p < .01$), White participants ($\beta = -10.50$ -0.66, $SE = 0.28$ -5.21, $p < .01$), sexual minorities ($\beta = -10.50$ -0.69, $SE = 0.31$ -5.62, $p < .01$), and participants with lower socioeconomic status ($\beta = 0.35$ -2.81, $SE = 0.15$ -1.48, $p < .01$). There were no differences in the number of partners at baseline or in the change in number of partners over time based on intervention participation, $\beta = -0.07$ -0.03, $SE = 0.06$ -0.16, $p = .65$ and $\beta = -0.10$ -0.01, $SE = 0.17$ -2.49, $p = .97$, respectively. We also tested for interactions between study and intervention participation and found no significant relationship in any study.

Unprotected Sex—A quadratic growth model was the best fit to the data for number of unprotected sexual events, $\chi^2(10, N = 3756) = 71.91, p < .001$, CFI = .98, TLI = .98, RMSEA = .04. The quadratic model fit significantly better than a linear model, $\chi^2(4) = 268.75, p < .001$. Additionally, comparison of the BICs suggested this model fit better than did any 2-part growth models (quadratic BIC = 42271.34; 2-part BICs = 42343.01-42463.02). Separate growth models for each study showed that this quadratic model fit well in each case.

We next fit a multigroup quadratic latent growth model with study as the grouping variable. This model controlled for demographics and intervention condition. This model fit the data well, $\chi^2(123, N = 1535, 1009, 608, 604) = 221.44, p < .001$; RMSEA = .03; CFI = .97; TLI = .96. This model (Figure 3; Table 5, Electronic Supplementary Material 2 [Table 4]) showed a significant negative slope combined with a positive quadratic in three of the four studies. Thus, the number of acts of unprotected sex was decreasing over time initially before beginning to increase. In the final study (ITK), which utilized partner-specific assessments of unprotected sex, there was a positive slope combined with a negative quadratic, indicating that the number of acts of unprotected sex was initially increasing before beginning to decrease. Reparameterization showed that significant decreases continued through 6 months in the HIP-R, HIP-R2, and ACHIEVE. Acts of unprotected sex were significantly increasing at 9 and 12 months in the HIP-R and by 12 months in ACHIEVE. In ITK, significant increases in the number of unprotected sex acts continued through 3 months; there was a trend toward decreases ($p < .10$) by 12 months. Additionally, for each study, there was significant variance in the number of unprotected sex acts at baseline, in the slope at all time points (except for ITK, where there was significant variance at only some points), and in the quadratic component (in all but ITK), indicating differences between participants in the initial number of partners as well as in the change in number of partners over time. Finally, this model showed little association between the number of unprotected sex acts at baseline and changes in unprotected sex over time, with no significant associations between intercepts and slopes and modest associations between intercepts and quadratic components in only two studies, $r_s = -.35$ – $-.34, p_s < .05$.

Demographically, more unprotected sex acts were reported at baseline by men ($\beta = 0.07$ – $0.40, SE = 0.04$ – $0.23, p < .05$) and younger participants ($\beta = -0.40$ – $-0.07, SE = 0.02$ – $0.16, p < .001$). Decreases in unprotected sex were greater for older participants ($\beta = -0.18$ – $-0.10, SE = 0.06$ – $0.13, p = .05$) and participants with lower socioeconomic status ($\beta = 0.15$ – $0.16, SE = 0.07$ – $0.11, p < .05$). There were no differences in the number of unprotected sex acts at baseline or in the change in unprotected sex over time based on intervention participation, $\beta = 0.03$ – $0.17, SE = 0.04$ – $0.21, p = .42$ and $\beta = -0.09$ – $-0.05, SE = 0.10$ – $0.19, p = .61$, respectively. We also tested for interactions between study and intervention participation and found no significant relationship in any study.

Parallel Process Models

Alcohol Use and Number of Sexual Partners—A multigroup parallel process latent growth model including the quadratic growth models for alcohol use and number of sexual partners and controlling for demographics and intervention condition fit the data well, $\chi^2(316, N = 1537, 1009, 609, 604) = 575.97, p < .001$; RMSEA = .03; CFI = .98; TLI = .97 (baseline fit; reparameterization resulted in alternative models with very similar fits). Across time, Wald tests suggested between 27 and 35 constraints on covariances between growth parameters; all constrained models were superior to unconstrained models based on BICs (BIC for unconstrained model = 43940.09; BICs for constrained models = 43696.18–43759.66). Supporting our first hypothesis, this model (Table 6; Electronic Supplementary Material 2 [Table 5]) showed consistent associations between levels of alcohol use and numbers of sexual partners. There were positive correlations between initial

levels of alcohol use and initial numbers of sexual partners in all studies, $r_s = .17-.18$, $p_s < .001$. Levels of alcohol use were also positively correlated with the number of sexual partners at all follow-up points in all studies, $r_s = .20-.40$, $p_s < .001$. This indicated that participants who drank more heavily also had more sexual partners.

Additionally, supporting our second hypothesis, there were positive correlations between the slope of alcohol use and the slope of number of partners across time and study. The initial rate of change in alcohol use was correlated with the initial rate of change in number of partners in three studies; this correlation was strongest in ACHIEVE, $r = .76$, $p < .001$, and also significant and positive in the HIP-R and HIP-R2, $r_s = .45$, $p_s < .001$. This correlation was not significant in ITK, $r = -.02$, $p = .93$. When the model was reparameterized, changes in alcohol use also correlated with changes in number of partners at 3 months ($r_s = .21-.65$, $p_s < .001$ in HIP-R and ACHIEVE, $p < .05$ in HIP-R2, and $p = .07$ in ITK), 6 months ($r_s = .27-.62$, $p_s < .001$), 9 months ($r_s = .33-.66$, $p_s < .001$ in HIP-R, HIP-R2, and ACHIEVE and $p < .01$ in ITK), and 12 months ($r_s = .30-.80$, $p_s < .001$ in HIP-R, HIP-R2, and ACHIEVE and $p < .01$ in ITK). This indicated that participants who reduced their drinking more over time also reduced their number of sexual partners more over time, and that this relationship was fairly similar across studies, although it was only evident at later follow-up points in ITK.

There was also a positive correlation between the quadratic change in alcohol use and the quadratic change in number of partners in three studies, $r_s = .47-.86$, $p_s < .001$ in the HIP-R and HIP-R2 and $p < .01$ in ACHIEVE. This indicated that participants who slowed their rate of change in alcohol use more over time also slowed their change in number of partners more.

Initial levels of alcohol use were not associated with initial changes in number of partners ($r_s = -.09-.05$, $p_s > .20$), nor was the initial number of partners associated with initial changes in alcohol use ($r_s = -.07-.06$, $p_s > .16$). Finally, there were positive residual correlations between alcohol use and number of sexual partners at each time point for each study ($r_s = .06-.34$, $p_s < .05$), indicating that when participants reported higher alcohol use than would be anticipated based on their personal alcohol use trajectory, they also reported a higher number of recent partners than would be anticipated based on their personal trajectory of number of partners (see Electronic Supplementary Material 2 [Figure 1]).

Figure 5 shows the anticipated trajectories of number of partners for the average participant in terms of baseline number of partners in each study, for a participant with alcohol use decreases -1 or $+1$ standard deviation from the mean.

Alcohol Use and Unprotected Sex—A multigroup parallel process latent growth model including the quadratic growth models for alcohol use and unprotected sex and controlling for demographics and intervention condition fit the data well, $\chi^2(317, N = 1535, 1009, 609, 604) = 535.74$, $p < .001$; RMSEA = .03; CFI = .98; TLI = .98 (baseline fit; reparameterization resulted in alternative models with very similar fits). Across time, Wald tests suggested between 31 and 37 constraints on covariances between growth parameters; all constrained models were superior to unconstrained models based on BICs (BIC for

unconstrained model = 69009.14; BICs for constrained models = 68742.10-68780.51). Supporting our first hypothesis, this model (Table 6; Electronic Supplementary Material 2 [Table 5]) showed consistent associations between levels of alcohol use and numbers of unprotected sex acts. There were positive correlations between initial alcohol use and initial unprotected sex acts in all studies, $r_s = .06-.27$ ($p < .001$ in ACHIEVE, $p_s < .05$ in other studies). Additionally, levels of alcohol use were positively correlated with the number of unprotected sex acts at most follow-up points for three of the studies, $r_s = .08-.17$, $p_s < .05$ for HIP-R and HIP-R2, $r_s = .25-.43$, $p_s < .001$ for ACHIEVE. They were correlated only at 9 months for ITK, $r = .13$, $p < .001$. These correlations indicated that participants who drank more heavily also reported more acts of unprotected sex.

Additionally, partially supporting our second hypothesis, there were positive correlations between the slope of alcohol use and the slope of unprotected sex across time in ACHIEVE. The initial rate of change in alcohol use was correlated with the initial rate of change in unprotected sex, $r = .24$, $p < .001$. When the model was reparameterized, changes in alcohol use also correlated with changes in number of partners at all follow up points, $r_s = .30-.57$, $p_s < .01$. This indicated that, in a study recruiting hazardous drinkers, participants who reduced their drinking more over time also reduced their unprotected sex more. In contrast, there was a positive association between changes in alcohol use and changes in unprotected sex only at 3 months in the HIP-R and ITK, $r_s = .23-.27$, $p_s < .01$. Contrary to our hypothesis, there were no associations between changes in the two behaviors in these studies at other time points or in the HIP-R2 at any time point. Additionally, there were no associations between the quadratic change in alcohol use and the quadratic change in unprotected sex in any study, $r_s = .03-.05$, $p_s > .70$.

Initial levels of alcohol use were associated with changes in unprotected sex in the HIP-R2 ($r = .31$, $p < .001$), indicating that participants in this study who were heavier drinkers decreased their levels of unprotected sex more slowly. There were no associations between initial alcohol use and changes in unprotected sex in the other studies ($r_s = .00$, $p_s > .99$), nor were initial levels of unprotected sex associated with changes in alcohol use in any study ($r_s = -.00-.02$, $p_s > .61$).

Finally, there were positive residual correlations between alcohol use and unprotected sex at each time point in the HIP-R2 and ACHIEVE ($r_s = .08-.28$, $p_s < .001$), indicating that when participants reported higher alcohol use than would be anticipated based on their personal alcohol use trajectory, they also reported more unprotected sex than would be anticipated based on their unprotected sex trajectory (see Electronic Supplementary Material 2 [Figure 1]).

Figure 6 shows the anticipated trajectories of unprotected sex for the average participant in terms of baseline unprotected sex in each study, for a participant with alcohol use decreases -1 or $+1$ standard deviation from the mean.

Discussion

Across four samples, in line with hypotheses, we found that STI clinic patients who reduced their alcohol use more also reduced their number of sexual partners more. Additionally, in one study recruiting hazardous drinkers, clinic patients who reduced their alcohol use more also showed greater reductions in unprotected sex. This study is the first use of integrative data analysis to combine data from longitudinal studies conducted in STI clinics as well as the first study to use parallel process latent growth modeling to explore how *changes* in alcohol use relate to *changes* in sexual risk behavior. Our results suggest that interventions aiming to reduce the number of partners in order to prevent HIV and other STIs may be enhanced by addressing alcohol use.

As anticipated, growth models showed decreases in sexual risk behavior over one year. Decreases in the number of sexual partners were evident in all four studies, while decreases in unprotected sex occurred in three of four studies. These decreases were described in the original investigations (52-55). The finding that decreases were similar across different intervention conditions may be attributable to the use of active comparison conditions, which generally included some pre- and post-test counseling by ethical necessity. Notably, integrative data analysis shows how similar patterns of change in both outcomes were for the three studies using 3-month measures, and how a 1-month partner-by-partner measure of unprotected sex (53) performs much differently. In the study utilizing this measure, there were no decreases in unprotected sex over time. Interestingly, this trial was the only one to show some significant differences between intervention conditions in unprotected sex (53). If future studies of similar populations include both 1-month and 3-month measures of sexual risk, a formal measurement integration process for these sexual risk measures would be possible; this process could prove informative with regards to risk assessments.

Less expected were the decreases over time in alcohol use, which were present in all studies. Participants engaged in high levels of alcohol use at study entry; heavy episodic drinking was quite common even in the three studies that did not specifically recruit hazardous drinkers. Participants in these three studies reduced their drinking by one-third to more than one-half of a standard deviation across one year of follow-up, despite the fact that alcohol use was not a primary target of the intervention or control conditions. Those participants recruited into ACHIEVE based on their hazardous drinking reduced their drinking by more than 1.5 standard deviations; by 12 months, their mean levels of drinking were not that dissimilar from those in the studies recruiting clinic patients in general. There are a number of potential explanations for these decreases. First, the experience of going for STI testing and waiting for test results may cue patients to reflect on the potential consequences of their risk behaviors (71); perhaps this consideration of risk behavior extends beyond sexual risk to also encompass substance use. Second, it is also possible that there are effects of repeatedly assessing alcohol use over time. For example, a study assessing the impact of a brief intervention for risky alcohol consumption in the clinic setting found that both intervention and control groups decreased consumption, suggesting that screening alone might influence behavior (14). Finally, related to the parallel process models discussed below, it is possible that changes in sexual risk behavior over time among these patients, prompted by the

process of STI testing, by supportive clinic staff, or by contents of the interventions, triggered the changes in alcohol use.

The primary substantive contribution of this study is the demonstration that changes in alcohol use are associated with changes in sexual risk behavior, particularly the number of sexual partners, and that these associations generalize across four studies. The parallel process models contribute additional evidence to studies that have shown associations between alcohol use and sexual risk behaviors among clinic patients using cross-sectional or event-level measures (20, 29-33) by specifically demonstrating associations between *changes* in the two risk behaviors. As hypothesized, changes in alcohol use were correlated with changes in number of sexual partners across time and study with few exceptions, indicating that participants who reduced their alcohol use more over time also reduced their number of sexual partners more over time. Given that changes in alcohol use relate to changes in number of partners, this association is unlikely to be due to relatively stable personality characteristics such as sensation-seeking (33).

There are a number of potential explanations for the relationship. First, in line with alcohol myopia theory (34) or theories involving alcohol expectancies (35), it is possible that alcohol use leads to sexual risk behavior (e.g., sex with multiple partners). This would lead to the expectation that as participants reduced their alcohol use, this reduction would contribute to a decrease in the number of partners. Alternatively, it may be that changes in the number of sexual partners inspire changes in alcohol use. For example, if patients are motivated to avoid sexual risk behavior by their experience seeking STI testing (and possibly treatment), they might choose to avoid contexts where they commonly seek out new partners and also consume alcohol, such as bars (36, 37). They may also terminate relationships with one or more of their sexual partners; sexual partners may influence substance use (72). Finally, these longitudinal associations might be driven by third variables, such as social network composition (38), socioeconomic factors (39-41), lifestyle changes, the repeated assessment of risk behavior, or demand characteristics. Overall, these results corroborate prior findings suggesting ties between alcohol use and number of partners (21, 23) and contribute additional evidence by showing associations between changes in the two.

Evidence related to the hypothesized association between alcohol use and unprotected sex was more mixed. Although levels of alcohol use and levels of unprotected sex were correlated at most time points for 3 of 4 studies (i.e., those studies utilizing 3-month measures of sexual behavior), changes in alcohol use consistently related to changes in unprotected sex only in ACHIEVE, which specifically recruited hazardous drinkers. Conclusions from past research have also been mixed, with some studies identifying cross-sectional or event-level associations between alcohol use and unprotected sex (19, 25-27) and other studies failing to find these associations (21, 23, 24). In ACHIEVE, the positive association between changes in alcohol use and changes in unprotected sex may be attributable to several things. First, decreases in alcohol use may be leading to either increases in condom use or decreases in the total number of sex acts combined with maintenance of consistent levels of condom use. Both of these explanations align with alcohol myopia theory and expectancies theory (34, 35). The number of unprotected sex acts is a good measure of exposure to STIs and HIV (73, 74), so either of these possibilities

would be positive. It is also possible that changes in sexual behavior contribute to changes in alcohol use. For example, termination of a relationship with a heavy drinking partner might lead to reductions in both unprotected sex and alcohol use. Finally, third variables may contribute to both changes. That these associations existed in only one study may indicate that only very high levels of alcohol use lead to unprotected sex, as suggested in some past research (30, 75). Patients in ACHIEVE also made the most dramatic changes in their levels of drinking over time, which may have made the association between these changes and changes in unprotected sex more apparent. Replication of these findings with other samples of heavy-drinking clinic patients is needed.

Implications and Future Research

These results have important implications for interventions conducted in STI clinics. Given that heavy alcohol use is common, screening for alcohol misuse in the STI setting is recommended. One study has already demonstrated that such screening is feasible and acceptable (14). Also likely to be feasible is the provision of brief alcohol interventions or referral to more intensive programs. Indeed, the “Screening, Brief Intervention, and Referral to Treatment” (SBIRT) model has been successfully implemented in other primary care settings as well as emergency department and other community health settings (76, 77). Validated screening tools with 1 to 10 items are available, and brief interventions can involve 15-30 minutes of feedback on drinking behaviors, advice on recommended drinking limits, and goal setting (78). Given the growing interest in integrated care and the “bundling” of services, it is now more common for patients seeking one type of care to also receive treatment for other health problems (79). Because changes in alcohol use relate to changes in number of sexual partners across four studies of clinic patients, targeting alcohol use in sexual risk reduction interventions is warranted. Although we cannot be certain that the association between alcohol use and sexual risk behavior is causal, the other health consequences of heavy alcohol use alone (80) make it a valuable target.

Given the reductions in alcohol use observed across studies, it may be that measurement alone can prompt reductions in alcohol use (14). Because there is limited time available in most clinic interventions, future research should specifically test whether this is the case by including conditions where baseline alcohol use is not assessed (e.g., in a Solomon four-group design, 14). Additionally, future research might aim to identify subpopulations of patients who maintain high levels of alcohol use over time, because these patients may need more intensive intervention. Use of other recreational drugs may also impact sexual risk-taking (15, 81), so future research should explore associations between changes in drug use and changes in sexual risk, as well as interactions between alcohol use and drug use. Finally, given the variety of potential explanations for the association between changes in alcohol use and changes in sexual risk, research should attempt to determine the causal relationship between these behaviors and explore possible mechanisms for the association. For example, intervention trials in STI clinics could target alcohol use alone and examine reductions in sexual risk; this would supplement information from trials that have targeted both alcohol use and sexual risk (55). One previous study conducted in Australia (14) implemented a brief intervention for excessive alcohol consumption among clinic patients and found decreases in drinking among both intervention and control participants; additional studies

are needed. Parallel process latent growth modeling can also be used to explore mechanisms tying changes in alcohol use to changes in sexual risk (82).

Strengths and Limitations

This study was the first use of integrative data analysis on data collected in STI clinics and the first application of parallel process latent growth modeling to longitudinal alcohol use and sexual risk data in this population. It showed the feasibility of combining data from multiple, independent trials and demonstrated many similarities across trials. We were able to build an integrated measure of alcohol use that drew on multiple important dimensions of alcohol use (83) and functioned across studies. Results related to changes in alcohol use and changes in number of partners were replicated across trials, building confidence in this association. Additionally, research has suggested that most uses of parallel process models are underpowered (48); we overcame this obstacle by pooling data from multiple studies.

Despite these strengths, there were also limitations to the current study. First, differences across trials in alcohol use measures necessitated some simplification of items. Additionally, the sexual risk measures differed in one study, and we could not place these measures on a common metric because the studies had no behavior measure in common. Second, although the use of parallel process latent growth modeling contributes new and interesting information about the association between alcohol use and sexual risk, we cannot draw causal inferences from this research. In general, there were not associations between intercepts of one construct and slopes of the other; associations were instead between the slopes of the two behaviors. Third, although integrative data analysis helps to build a cumulative science (i.e., by demonstrating what replicates across studies), independent studies lack common measures of some constructs. For example, we did not test potential explanations for the alcohol-sexual risk association because there were not common measures of variables such as sensation-seeking or expectancies across datasets. Fourth, the quadratic patterns of change in the current study are challenging to interpret and add complexity to our analyses (84). Fifth, participants with heavier alcohol use at baseline were more likely to have missing data during follow-up, which may bias results. Finally, because we pooled studies with some commonalities, the four samples we report on are similar demographically (e.g., in terms of race and socioeconomic status) and in terms of sexual risk behavior. Practical considerations also meant that we pooled data from only two urban areas; future work should include data from other public clinics.

Conclusions

Reductions in alcohol use over time are associated with reductions in the number of sexual partners over time across four samples of patients from urban, publicly-funded STI clinics. Additionally, for patients recruited specifically based on their hazardous drinking, reductions in alcohol use are related to reductions in unprotected sex. Although future research is needed to explore reasons for this association, HIV risk reduction interventions may be strengthened by addressing alcohol use. Our combined use of parallel process latent growth modeling, a method for exploring how changes in one construct relate to changes in another construct, and integrative data analysis, a technique for combining multiple, independent

datasets, contributes additional support to the association between alcohol use and sexual risk behavior among STI clinic patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Fitted trajectories and observed means for alcohol use across four studies. $\chi^2(119, N=1534, 1009, 609, 591) = 221.18, p < .001$; RMSEA = .03; CFI = .99; TLI = .98.

Figure 2.

Fitted trajectories and observed means for number of partners across four studies. $\chi^2(119, N = 1534, 1009, 609, 591) = 216.82, p < .001$; RMSEA = .03; CFI = .99; TLI = .98.

Figure 3.

Fitted trajectories and observed means for unprotected sex across four studies. $\chi^2(123, N=1537, 1009, 609, 604) = 244.57, p < .001$; RMSEA = .03; CFI = .97; TLI = .97.

Figure 4.

Trajectories of number of partners for the average participant in each study, for a participant with alcohol use decreases -1 or $+1$ standard deviation from the mean. At baseline, changes in alcohol use were significantly correlated with changes in number of partners in three of the four studies (a, b, and d), $r_s = .45-.76$, $p_s < .001$.

Figure 5.

Trajectories of unprotected sex for the average participant in each study, for a participant with alcohol use decreases -1 or $+1$ standard deviation from the mean. At baseline, changes in alcohol use were significantly correlated with changes in unprotected sex in ACHIEVE (d), $r = .24$, $p < .001$, and marginally associated with changes in unprotected sex in HIP-R (a), $r = .17$, $p = .08$.

Table 1

Descriptive Statistics by Study and for the Integrated Sample

	Integrated Sample	HIP-R	HIP-R2	ITK	ACHIEVE
<i>N</i>	3761	1537	1009	610	605
Male	59%	54%	56%	69%	69%
Age	30.9 (10.3)	29.1 (9.6)	28.5 (9.5)	35.6 (10.3)	34.5 (10.7)
Black	73%	64%	69%	85%	88%
White	18%	24%	19%	9%	8%
Mixed	5%	6%	7%	--	2%
Other	5%	6%	5%	3%	1%
Latino	7%	9%	8%	3%	5%
Sexual Minority	11%	12%	13%	8%	6%
Education High School	69%	62%	64%	81%	86%
Unemployed	54%	51%	52%	48%	74%
Socioeconomic Status	0.0 (1.0)	0.2 (0.8)	0.2 (0.8)	0.2 (0.7)	-1.0 (1.3)
Single (Never Married)	78%	78%	82%	--	74%
Most Active Intervention	36%	32%	50%	23%	35%
3 Month Follow-Up	76%	73%	82%	77%	70%
6 Month Follow-Up	74%	75%	78%	71%	67%
9 Month Follow-Up	42%	--	75%	70%	66%
12 Month Follow-Up	60%	71%	75%	--	65%
Total Number of Assessments	3.5 (1.4)	3.2 (1.1)	4.1 (1.4)	3.2 (1.2)	3.7 (1.7)
Missing Assessments	41%	43%	37%	39%	43%

Numbers are percentages or means (standard deviations). Socioeconomic status = a composite, integrated measure across studies which includes income, education, and employment. Missing assessments = did not complete any of the key measures (alcohol use, number of sexual partners, and unprotected sex) at an assessment point included in the study. Most active intervention = in the HIP-R, an intensive information-motivation-behavioral skills (IMB) intervention; in the HIP-R2, a sexual health promotion DVD; in ITK, an intervention including all components of the IMB model; in ACHIEVE, IMB-based counseling plus motivational interviewing to reduce alcohol use plus an alcohol-related booster session.

 $p < .001$

**
 $p < .01$

*
 $p < .05$

+
 $p < .10$

Table 2

Alcohol Use and Sexual Risk by Study and for the Integrated Sample

			Integrated Sample	HIP-R	HIP-R2	ITK	ACHIEVE
Alcohol Use (Integrated Measure)	BL	<i>M(SD)</i>	0.00 (1.00)±	−0.14 (0.90)	−0.04 (0.97)	−0.37 (0.99)	0.92 (0.78)
		<i>N</i>	3682	1532	1009	601	540
	3mo	<i>M(SD)</i>	−0.27 (1.01)	−0.37 (0.93)	−0.27 (0.99)	−0.63 (1.02)	0.33 (1.01)
		<i>N</i>	2839	1123	830	466	420
	6mo	<i>M(SD)</i>	−0.39 (1.06)	−0.45 (1.01)	−0.36 (1.06)	−0.69 (1.05)	0.07 (1.09)
		<i>N</i>	2772	1148	790	430	404
	9mo	<i>M(SD)</i>	−0.42 (1.19)	--	−0.46 (1.17)	−0.73 (1.14)	−0.01 (1.18)
		<i>N</i>	1575	--	760	421	394
	12mo	<i>M(SD)</i>	−0.49 (1.20)	−0.55 (1.16)	−0.50 (1.22)	--	−0.31 (1.26)
		<i>N</i>	2238	1086	758	--	394
Number of Sexual Partners [†]	BL	<i>M(SD)</i>	2.85 (2.30)	2.83 (2.23)	2.69 (2.02)	1.58 (1.73)	3.17 (2.81)
		<i>N</i>	3148	1536	1009	602	603
	3mo	<i>M(SD)</i>	2.08 (1.90)	2.10 (1.91)	2.21 (1.80)	1.43 (1.57)	1.75 (2.04)
		<i>N</i>	2372	1124	830	466	418
	6mo	<i>M(SD)</i>	2.02 (1.90)	2.09 (1.91)	2.16 (1.90)	1.49 (1.66)	1.54 (1.81)
		<i>N</i>	2341	1148	790	433	403
	9mo	<i>M(SD)</i>	1.72 (1.53)	--	1.97 (1.50)	1.31 (1.29)	1.24 (1.47)
		<i>N</i>	1156	--	760	429	396
	12mo	<i>M(SD)</i>	1.88 (1.88)	2.03 (1.92)	1.99 (1.85)	--	1.24 (1.70)
		<i>N</i>	2236	1088	758	--	390
Unprotected Sex Events [†]	BL	<i>M(SD)</i>	18.88 (23.78)	17.49 (21.84)	16.61 (20.95)	2.24 (4.11)	26.21 (30.61)
		<i>N</i>	3140	1529	1009	598	602
	3mo	<i>M(SD)</i>	13.92 (20.09)	12.54 (18.89)	15.09 (20.28)	3.06 (6.32)	15.32 (22.51)
		<i>N</i>	2370	1122	830	462	418
	6mo	<i>M(SD)</i>	15.31 (22.77)	14.39 (21.72)	16.47 (23.22)	3.90 (8.35)	15.68 (24.66)
		<i>N</i>	2338	1147	790	427	401
	9mo	<i>M(SD)</i>	14.40 (20.77)	--	15.25 (20.70)	3.40 (7.77)	12.77 (20.83)
		<i>N</i>	1156	--	760	421	396
	12mo	<i>M(SD)</i>	14.55 (21.97)	14.58 (21.82)	14.67 (20.92)	--	14.23 (24.35)
		<i>N</i>	2230	1085	758	--	387

For information related to individual alcohol use indicators, see Electronic Supplementary Material 2 (Table 1). ± The integrated alcohol use measure was scaled by setting the baseline mean to 0 and variance to 1 (see Electronic Supplementary Material 1).

[†] Measures covered the past 3 months in HIP-R, HIP-R2, and ACHIEVE and the past 1 month in ITK. The integrated sample means include only the three studies with past 3 month measures.

Table 3

Latent Growth Model Examining Changes in Alcohol Use and Sexual Risk Over One Year in Four Samples of Sexually Transmitted Infection (STI) Clinic Patients Participating in Randomized Controlled Trials for Sexual Risk Reduction

Alcohol Use (Past 3 Months): Growth Parameter Estimates (N = 3,743)					
		HIP-R	HIP-R2	ITK	ACHIEVE
Intercept	Mean	−0.15 (0.02) *** _c	−0.05 (0.03) ^b	−0.37 (0.04) *** _d	0.89 (0.04) *** _a
	Variance	0.50 (0.03) *** _{bc}	0.63 (0.04) *** _a	0.63 (0.06) *** _{ab}	0.33 (0.08) *** _c
Slope	Mean	−0.16 (0.02) *** _a	−0.20 (0.02) *** _a	−0.28 (0.05) *** _a	−0.49 (0.05) *** _b
	Variance	0.15 (0.03) ***	0.11 (0.03) ***	0.12 (0.09)	0.31 (0.08) ***
Quadratic	Mean	0.02 (0.01) *** _b	0.02 (0.01) *** _{ab}	0.05 (0.02) *** _{ab}	0.05 (0.01) *** _a
	Variance	0.01 (0.001) ***	0.01 (0.002) ***	0.01 (0.01)	0.01 (0.004) [*]
Number of Sexual Partners (Ln, Past 3 Months): Growth Parameter Estimates (N = 3,759)					
		HIP-R	HIP-R2	ITK	ACHIEVE
Intercept	Mean	1.21 (0.01) *** _a	1.18 (0.01) *** _a	0.80 (0.02) *** _b	1.18 (0.03) *** _a
	Variance	0.11 (0.01) *** _b	0.10 (0.01) *** _b	0.13 (0.01) *** _b	0.28 (0.03) *** _a
Slope	Mean	−0.19 (0.01) *** _c	−0.13 (0.01) *** _b	−0.02 (0.01) ^a	−0.31 (0.03) *** _d
	Variance	0.04 (0.01) *** _a	0.03 (0.01) *** _{ab}	0.01 (0.003) ^b	0.10 (0.03) *** _a
Quadratic	Mean	0.03 (0.003) *** _a	0.02 (0.003) *** _b	0.00 [†]	0.05 (0.01) *** _a
	Variance	0.002 (0.001) ^{**}	0.001 (0.001) ^{**}	0.00 [†]	0.003 (0.001) [*]
Number of Unprotected Sexual Events (Ln, Past 3 Months): Growth Parameter Estimates (N = 3,756)					
		HIP-R	HIP-R2	ITK	ACHIEVE
Intercept	Mean	2.25 (0.03) *** _a	2.20 (0.04) *** _a	0.76 (0.04) *** _b	2.39 (0.06) *** _a
	Variance	0.56 (0.06) *** _b	0.61 (0.06) *** _b	0.19 (0.05) *** _c	1.10 (0.13) *** _a
Slope	Mean	−0.43 (0.04) *** _c	−0.22 (0.04) *** _b	0.18 (0.06) *** _a	−0.50 (0.07) *** _c
	Variance	0.32 (0.09) ***	0.34 (0.08) ***	0.26 (0.14) ⁺	0.47 (0.14) ***
Quadratic	Mean	0.09 (0.01) *** _a	0.03 (0.01) *** _b	−0.04 (0.02) ^c	0.08 (0.02) *** _{ab}
	Variance	0.02 (0.01) ***	0.02 (0.004) ***	0.01 (0.01)	0.02 (0.01) ^{**}

Average fit statistics over 100 imputations: for alcohol use, $\chi^2(119, N = 1534, 1009, 609, 591) = 221.18, p < .001$, RMSEA = .03; CFI = .99; TLI = .98; for number of partners, $\chi^2(123, N = 1537, 1009, 609, 604) = 244.57, p < .001$; RMSEA = .03; CFI = .97; TLI = .97; for unprotected sex, $\chi^2(120, N = 1535, 1009, 608, 604) = 221.44, p < .001$; RMSEA = .03; CFI = .97; TLI = .96. These multigroup latent growth models controlled for sex, age, race, socioeconomic status, sexual orientation, and intervention condition; all covariates were grand-mean centered. Alcohol use is a composite, integrated measure across studies. It has a mean of 0 and standard deviation of 1 for the integrated sample at baseline. The intercept is at baseline; for slope estimates following model reparameterization and correlations between growth parameters, see Electronic Supplementary Material 2 (Tables 2-4). Coefficients in the same line with different superscripts are significantly different according to the Wald test, $p < .01$.

 $p < .001$

**
 $p < .01$

*
 $p < .05$

⁺
 $p < .10$

τ^* = Constrained to 0.

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Table 4

Parallel Process Latent Growth Model Associations Between Changes in Alcohol Use and (a) Changes in Number of Sexual Partners (Ln, Past 3 Months) and (b) Number of Unprotected Sexual Events (Ln, Past 3 Months) Over One Year in Four Samples of Sexually Transmitted Infection (STI) Clinic Patients (Ns = 3,759; 3,756) Participating in Randomized Controlled Trials for Sexual Risk Reduction

			Alcohol Use				
			Intercept at BL		Quadratic	Intercept at 12mo	
			Intercept	Slope		Intercept	Slope
Number of Sexual Partners (Intercept at Same Time Point as Alcohol Use Intercept)	HIP-R	Intercept	.18 (0.03) ^{***a}	.06 (0.05) ^a	-.06 (0.05) ^a	.26 (0.03) ^{***a}	.08 (0.05) ^{+a}
		Slope	-.06 (0.04) ^b	.45 (0.11) ^{***b}	-.38 (0.11) ^{***}	.13 (0.05) ^{***b}	.30 (0.09) ^{***b}
		Quadratic	.06 (0.05) ^c	-.50 (0.13) ^{***c}	.48 (0.12) ^{***b}		
		Resid.	.09 (0.03) ^{***}			.10 (0.03) ^{***}	
	HIP-R2	Intercept	.18 (0.03) ^{***a}	.06 (0.06) ^a	-.07 (0.06) ^a	.27 (0.03) ^{***a}	.09 (0.05) ^{+a}
		Slope	-.05 (0.04) ^b	.45 (0.11) ^{***b}	-.37 (0.11) ^{***}	.13 (0.05) ^{***b}	.30 (0.09) ^{***b}
		Quadratic	.05 (0.04) ^c	-.48 (0.12) ^{***c}	.47 (0.12) ^{***b}		
		Resid.	.13 (0.02) ^{***}			.14 (0.02) ^{***}	
	ITK	Intercept	.15 (0.03) ^{***a}	.05 (0.05) ^a	-.06 (0.05) ^a	.31 (0.04) ^{***a}	.10 (0.06) ^{+a}
		Slope	-.09 (0.07) ^b	-.02 (0.20)	.29 (0.23)	.33 (0.13) ^{*b}	.80 (0.26) ^{***b}
		Resid.	.10 (0.03) ^{**}			.08 (0.03) [*]	
		Quadratic	.18 (0.03) ^{***a}	-.07 (0.05)	-.06 (0.05) ^a	.25 (0.03) ^{***a}	.08 (0.05) ^{+a}
Number of Unprotected Sex Acts (Intercept at Same Time Point as Alcohol Use Intercept)	ACHIEVE	Slope	-.06 (0.05) ^b	.76 (0.11) ^{***}	-.59 (0.16) ^{***}	-.50 (0.18) ^{**}	.39 (0.12) ^{***b}
		Quadratic	-.11 (0.13)	-1.03 (0.35) ^{**†}	.86 (0.31) ^{***b}		
		Resid.	.19-.2	6 (0.03-0.04) ^{***±}		.20-.22 (0.03) ^{***}	
		Quadratic					
	HIP-R	Intercept	.07 (0.03) ^{*d}	-.03 (0.06) ^d	.05 (0.06) ^d	.04 (0.03) ^d	-.07 (0.05) ^d
		Slope	.00 (0.05) ^e	.17 (0.10) ⁺	-.09 (0.09) ^f	-.10 (0.05) ^{*e}	-.03 (0.09) ^e
		Quadratic	-.02 (0.06) ^f	-.11 (0.10) ^e	.04 (0.09) ^g		
		Resid.	.05 (0.03) ⁺			.04 (0.03)	
	HIP-R2	Intercept	.06 (0.03) ^{*d}	-.03 (0.06) ^d	.05 (0.06) ^d	.04 (0.03) ^d	-.06 (0.05) ^d
		Slope	.31 (0.07) ^{***}	-.02 (0.10)	-.09 (0.09) ^f	-.09 (0.05) ^{*e}	-.03 (0.09) ^e
		Quadratic	-.34 (0.08) ^{***}	.08 (0.10) ^f	.04 (0.09) ^g		
		Resid.	.08 (0.02) ^{***}			.09 (0.02) ^{***}	
	ITK	Intercept	.11 (0.06) ^{*d}	-.06 (0.11) ^d	-.15 (0.13) ^e	-.02 (0.07)	-.12 (0.09) ^d
		Slope	.00 (0.05) ^e	.04 (0.10)	-.09 (0.09) ^f	-.10 (0.05) ^{*e}	-.03 (0.09)
		Quadratic	-.02 (0.05) ^f	.08 (0.10) ^f	.03 (0.08) ^g		
		Resid.	.04 (0.03)			.05 (0.03)	
	ACHIEVE	Intercept	.27 (0.07) ^{***}	-.02 (0.04) ^d	-.11 (0.09) ^e	.43 (0.07) ^{***}	.11 (0.07)
		Slope	.00 (0.07) ^e	.24 (0.07) ^{***}	-.12 (0.12) ^f	.17 (0.06) ^{**}	.30 (0.11) ^{**}
		Quadratic	-.03 (0.08) ^f	-.09 (0.08) ^e	.05 (0.13) ^g		
		Resid.	.20-.26 (0.03-0.04) ^{***±}			.20-.28 (0.03-0.05) ^{***}	

Within each column, parameters that share superscripts were constrained to be equal across studies without detrimental impact on fit.

$\pm$ = Alcohol use residual at baseline differed from residuals at other time points. For parallel process associations when intercept is positioned at 3, 6, and 9 months, see Electronic Supplementary Material 2 (Table 5).

$\hat{\gamma}$ = Standardized correlation coefficients can sometimes be greater than ± 1 in the presence of highly-correlated variables without this indicating any problem with the model (85).

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