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Alcohol and STI risk: evidence from a New Zealand longitudinal birth cohort

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Alcohol use and STI risk

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Abstract

Background: The present study examined the associations between involvement with alcohol and risks of sexually transmitted infection (STI) during adolescence and early adulthood.

Methods: A 30-year prospective longitudinal study of the health, development, and adjustment of a birth cohort of 1,265 New Zealand-born individuals. Measures included repeated assessments of frequency of alcohol use and number of symptoms of alcohol disorder from ages 15-30 and rates of STI from ages 14-30. Conditional fixed effects regression models augmented by observed time-dynamic covariate factors were used to control for non-observed confounding in the associations between alcohol and STI risk.

Results: There were clear and consistent trends for increasing involvement with alcohol to be linked with increased risk of STI diagnoses. Adjustment of the associations for sources of non-observed confounding and time-dynamic covariate factors reduced the magnitude of these associations, but they remained statistically significant ($p < .05$).

Conclusions: The results of the current study support the notion of the existence of a causal pathway in which increasing levels of alcohol use and symptoms of alcohol abuse/dependence led to increased risks of STI exposure. There was little evidence to suggest that the links between alcohol involvement and STI risk could be fully explained by an underlying predisposing factor that increased the risks of both alcohol involvement and STI.

Keywords: alcohol, sexually transmitted infection, longitudinal study, fixed effects regression

1. Introduction

In recent years there has been increasing concern regarding the problematic use of alcohol amongst young people. In particular, there has been an increased focus on the extent to which problematic drinking patterns, including regular heavy consumption of alcohol and binge drinking, may lead to increased risks of a number of adverse outcomes (Courtney and Polich, 2009; Stolle et al., 2009).

One linkage that has been of continued interest is the possible link between alcohol consumption, risky sexual behaviour, and increased risk of sexually transmitted infection (STI) (Cook and Clark, 2005; Cooper, 2002; Halpern-Felsher et al., 1996; Harrison and Kassler, 2000; Kang et al., 2007). A number of studies have shown that increased rates of alcohol consumption are associated with increased risks of STI diagnoses (Boyer et al., 2008; Buffardi et al., 2008; Chesson et al., 2003; Ericksen and Trocki, 1994; Jonsson et al., 1997; Larsson et al., 2007; Staras et al., 2009). Additionally, a number of case-control studies, and studies using clinical samples, suggest that those individuals who receive treatment for STI show significantly higher rates of alcohol consumption, and higher rates of alcohol problems and alcohol abuse/dependence (Bjekic et al., 1997; Hutton et al., 2008; Patton et al., 2008; Scott-Sheldon et al., 2009).

In general, there are two main explanations for the links between increasing levels of alcohol use and increased rates of STI. First, the acute intoxicating effects of alcohol may increase impulsivity and cause disinhibition, altering normal patterns of sexual behaviour and contraceptive use (Abbey et al., 2007; Boyer et al., 1999; Crosby et al., 2008; LaBrie et al., 2005; Parks et al., 2009; Yan et al., 2007), resulting in increased exposure to STI. Alternatively, both higher alcohol intake and higher rates of risky sexual behaviour may reflect a general underlying predisposition to engage in reckless, impulsive behaviour (Halpern-Felsher et al., 1996; Kalichman and Cain, 2004; Messiah et al., 1998). It could therefore be argued that a more general predisposition to recklessness explains the links between alcohol intake, risky sexual behaviour, and STI, rather than a direct causal link between alcohol and STI, via risky sexual behaviour. One way of addressing these competing explanations is through the control of non-observed confounding factors.

Although a number of previous studies have controlled for observed sources of confounding, it could be argued that that any associations between alcohol consumption and increased risk of STI may be explained by non-observed residual confounding.

While it is commonly believed that epidemiological studies can control only observed factors, this is not strictly true. There are at least two approaches by which non-observed sources of confounding may be controlled using a correlational design. The first approach is via the discordant twin design, in which twins who are discordant for alcohol use would be compared on STI. Because monozygotic twins share common genes, and frequently share some component of common environment, this comparison could account for these non-observed factors.

The second approach for controlling non-observed confounding is through the use of the conditional fixed effects regression model (Cameron and Trivedi, 1998; Greene, 1990). Subject to the availability of longitudinal data, this model makes it possible to estimate the associations between alcohol measures and STI measures net of the effects of non-observed fixed sources of confounding. Using repeated measures of both the exposure and outcome variables, the fixed effects model accounts for fixed sources of variance that are correlated with both the exposure and outcome (see Methods for an account of this model).

Against this background, the present study employed repeated measures data from a longitudinal birth cohort in order to examine the links between alcohol and risks of STI, using fixed effects models in order to control for non-observed sources of confounding, and using multiple measures of alcohol consumption and its effects (amount of alcohol consumed; number of alcohol abuse/dependence symptoms). The general aims of the study were to determine the extent to which alcohol played a causal role in increasing STI risk during late adolescence and early adulthood.

2. Methods

2.1 Participants

The data were gathered during the course of the Christchurch Health and Development Study (CHDS). As part of this study, a birth cohort of 1265 young people (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977 has been studied at birth, 4 months, 1 year and annually to age 16 years, and again at ages 18, 21, 25, and 30 years (Fergusson and Horwood, 2001; Fergusson et al., 1989). The study has collected information from a variety of sources including: parental interviews, teacher reports, self-reports, psychometric assessments, medical, and other record data. All study information was collected on the basis of signed and informed consent from study participants.

2.2 Measures

2.2.1 Measures of alcohol (amount of alcohol consumption; alcohol abuse/dependence symptoms)

At ages 15, 16, 18, 21, 25, and 30 years, participants were questioned concerning their use of alcohol during each year of the period since the previous assessment, and their experience of problems related to alcohol use. This information was used to create two measures of involvement with alcohol over the period 15-30 years.

The first measure of alcohol use, an estimate of the amount of alcohol consumed in the 12 months prior to assessment, was created by using the information provided by participants at each assessment concerning both: a) the frequency with which they consumed alcohol in the year prior to each assessment (ranging from “never” to “almost every day”); and b) the amount of alcohol consumed in a “usual” drinking session during the period in question. For the purposes of the present investigation, the two sources of information were combined to create an estimate of the number of standard drinks consumed by each cohort member in the 12 months leading up to each assessment. Then, because the rate of alcohol consumption differed across assessment periods, percentile rankings on the consumption measure were used to classify participants on a three-point

scale with the following classifications of alcohol consumption over the previous 12 months, at ages 15, 18, 21, 25, and 30: up to 70th percentile; 71st to 90th percentile; and 91st to 100th percentile. Also, alternative classification schemes using four- and five-group classifications were also examined (see Supplementary Analyses section, below).

The second measure of alcohol, alcohol disorder symptoms, was creating by using information provided by participants at each assessment concerning the extent to which they experienced difficulties related to alcohol consumption. At ages 15 and 16 alcohol-related problems were assessed using the Rutgers Alcohol Problem Index (White and Labouvie, 1989), whereas at ages 18, 21, 25, and 30 the questioning was based on the Composite International Diagnostic Interview (CIDI)(World Health Organization, 1993) items relating to alcohol disorders (alcohol abuse/dependence). At each point of observation a scale score was constructed based on the number of symptoms of alcohol abuse or alcohol dependence that the participant met during each year of the assessment period, with this score ranging from 0 for those meeting no criteria to a maximum of 11 for those meeting all criteria. Then, because the rate of scores varied across assessment periods, these scores were used to classify participants in terms of the number of symptoms of alcohol abuse/dependence experienced over each of four assessment periods (14-18, 19-21, 22-25, and 26-30 years). The scores were converted into a three point classification scale for each assessment period, with the following values: those with no symptoms; those who reported symptoms but whose scores were below the highest decile on the measure; and those whose symptom score placed them in the highest decile on the measure. Again, alternative classification schemes using four- and five-group classifications were also examined (see Supplementary Analyses section, below). Pearson correlations between the two alcohol measures at each assessment ranged from .15 to .35.

2.2.2 Self-reported sexually transmitted infection diagnoses (STI), ages 14-30

At ages 18, 21, 25 and 30, cohort members were questioned about a range of sexual activities and practices they had engaged in since the previous assessment, and the consequences of these activities and practices, including whether they had been diagnosed with a sexually transmitted infection (STI) at any time since the previous assessment. Cohort members who responded "yes" were then asked to provide details of the infection, including the age at which it was contracted, the type of STI contracted (i.e. according to a formal medical diagnosis), and the treatment (if any) provided. In addition to this questioning, at age 30 a comprehensive history of lifetime STI diagnoses was obtained, as a check on previous data. For the purposes of the present investigation, the measures of STI were defined using a combination of both prospective and retrospective reports. Using the information on timing and outcome for each reported STI, each cohort member's STI diagnosis history for any given period of interest (ages 14-18; ages 19-21; ages 22-25; ages 26-30) was operationalized as a count measure of the number of STI diagnoses reported in each assessment period.

2.2.3 Observed covariate factors

The regression models (see Statistical Analysis, below) employed a series of observed covariate factors, measured during the period 14-30 years, that were abstracted from the study data base and were selected on the basis that they were known on the basis of prior analyses to be associated with measures of alcohol use or sexual behaviour in this cohort. These measures included: stressful life events; unemployment; cohabitation with partner; caring for dependent children; frequency of cannabis use; lifetime prior history of major depression; and lifetime prior history of anxiety disorder.

2.3 Statistical analysis

The data analyses were conducted over several steps. In the first step of the analyses, the cohort was divided into three groups at each assessment period according to the two primary predictors: a) the three levels of the alcohol consumption measure; and b) the three levels of the symptoms of alcohol abuse/dependence measure. Then the mean rate of STI diagnoses for each group at each assessment period was calculated. In the case of the measure of amount of alcohol consumed, the present analyses employed a lagged model in which alcohol consumption was measured prior to the assessment period for STI (for ages 19-21, 22-25, and 26-30) or early in the assessment period for STI (for ages 14-18), in order to avoid issues of reverse causality (note that there were only two occurrences of an STI diagnosis at age 14, which were contemporaneous with the alcohol consumption measure at that age). In the case of the alcohol disorder symptoms measure, the present analyses employed a model in which symptoms were measured contemporaneously with STI diagnoses.

In the next step of the analyses, the estimates of the associations between the alcohol measures (amount of alcohol consumption; alcohol problems) pooled over the four time periods and STI diagnoses were estimated using Generalized Estimating Equation (GEE) models, in which the rate of STI at each of the four time periods was modelled as a function of the alcohol measure at each time period. These models were of the general form:

$$f(Y_{it}) = B_0 + B_1 X_{it} + B_2 A_{it} \qquad \text{(EQ1)}$$

where $f(Y_{it})$ was the log rate of STI reported by the ith subject in a given interval t, X_{it} represented the alcohol measure (amount; number of symptoms) during the interval t, and A_{it} represented the individual's age. In fitting this model observations from the same individual over time were permitted to be correlated with an unstructured correlation matrix. All models were fitted using Stata 10.0 (StataCorp, 2003).

In the next step, covariate-adjusted estimates of the associations between alcohol and STI were obtained by fitting conditional fixed effects Poisson regression models (Cameron and Trivedi,

1998; Greene, 1990) respectively to the observed annual rate data for the count measure of STI. Fixed effects models are used to control for all non-observed sources of variation that are correlated with both the exposure variable and the outcome variable, prior to the onset of exposure. Models were fitted of the form:

$$f(Y_{it}) = B_{0i} + B_1 X_{it} + B_2 A_{it} \quad \text{(EQ2)}$$

This model form is similar form to the unadjusted model in EQ1 above. However, in this instance the coefficients B_{0i} are individual specific intercept terms representing the effects of (non-observed) fixed confounding factors specific to the ith individual that are correlated with alcohol (X_{it}) and may also influence risk of STI.

Then, in order to examine the potential effects of time-dynamic covariate factors on the associations between alcohol and STI diagnoses, the models above were extended to incorporate the set of observed time dynamic covariate factors (Zijt) as follows, entered simultaneously:

$$f(Yit) = B_{0i} + B_1 X_{it} + B_2 A_{it} + \sum B_j Z_{ijt} \quad \text{(EQ3)}$$

The coefficient B_1 thus represents the pooled effects of alcohol (X_{it}) on the risk of STI diagnosis (Y_{it}) net of: a) non-observed fixed confounding factors; and b) observed time-dynamic covariate factors, including: stressful life events; unemployment; cohabitation; dependent children; cannabis use frequency; lifetime depression; and lifetime anxiety disorder.

To provide estimates of effect size, the parameter estimates for each alcohol measure, derived in each step above, were used to calculate incidence rate ratio (IRR) estimates and corresponding 95% CIs for the effect of varying levels of exposure to alcohol on rates of STI.

Finally, a series of supplementary analyses were conducted, using the procedures outlined above, but substituting differing alcohol-related measures. In the first analysis, in place of the three level-measure of alcohol consumption/symptoms, the analyses were repeated using four- and five-level classifications of each alcohol measure. In the second supplementary analysis, the count measure of STI diagnoses was replaced by a dichotomous classification measure indicating whether participants reported an STI diagnosis during a particular assessment period.

2.4 Sample sizes and missing data

The present analyses were based on samples ranging from 982 to 1025, representing 78% to 81% of the original cohort of 1265 participants, for whom data were available concerning alcohol use and symptoms of alcohol dependence, risky sexual behaviour and STI during the period 14-30 years.

To examine the effects of sample losses on the representativeness of the sample, the obtained samples with complete data at each age, were compared with the remaining sample members on a series of socio-demographic measures collected at birth. This analysis suggested that there were statistically significant ($p<.01$) tendencies for the obtained samples to under-represent individuals from socially disadvantaged backgrounds characterized by low parental education, low socio-economic status and single parenthood. To address this issue, the data weighting methods described by Carlin et al. (1999) were used to examine the possible implications of selection effects arising from the pattern of missing data. These analyses produced essentially the same pattern of results to those reported here, suggesting that the conclusions of this study were unlikely to have been influenced by selection bias.

3. Results

3.1 Prevalence of STI diagnoses

By age 30, 18.1% of cohort members had reported being diagnosed with at least one STI. In terms of the number of STI reported, 14.6% of the cohort reported only one STI, 2.7% of the cohort reported 2 STI, while 0.8% of the cohort reported three or more STI diagnoses.

The most common STI amongst the cohort was Chlamydia; 9.5% of the cohort reported at least one Chlamydia infection. Other common infections included herpes (4.4% of the cohort), and genital warts (4.3% of the cohort). Gonorrhea, urethritis, and Tricomenes were uncommon (0.3%, 0.5%, and 0.1% respectively), while there were no reported cases of syphilis or HIV infection. A further 2.2% of the cohort indicated having been infected with an unspecified STI.

3.2 Associations between measures of alcohol consumption/alcohol disorder symptoms and STI diagnoses, ages 14-30

Tables 1 and 2 shows the cohort divided into three groups across two alcohol-related measures: amount of alcohol consumed; and alcohol abuse/dependence symptoms. The measures represent increasing levels of alcohol use in the period just prior to the reporting of STI diagnoses, and increasing rates of self-reported alcohol disorder symptoms measured contemporaneously with the reporting of STI (see Methods). Table 1 shows the mean number of standard drinks per week in the twelve months prior to the assessments at age 15, 18, 21, and 25; and the mean number of alcohol disorder symptoms during each assessment period (14-18 years; 18-21 years; 21-25 years; and 25-30 years); for each level of classification.

Table 2 shows the rates of self-reported STI diagnoses during each assessment period (ages 14-18 years; 19-21 years; 22-25 years; 26-30 years). Table 2 also shows the population averaged estimate, pooled across all four assessment periods, of the incidence rate ratio (IRR) for both the consumption measure and the alcohol symptoms measure. The estimate of the IRR was obtained through the use of generalized estimating equation (GEE) models of the associations between the alcohol measures and STI diagnoses (see Methods). The Table shows:

1. There were clear and consistent tendencies, across all assessment periods, for rates of STI diagnosis to increase as levels of alcohol consumption increased. The population averaged estimates suggested that those in the highest decile on the measure of alcohol consumption had rates of STI that were 2.50 (95%CI: 1.80-3.47) times higher than those in the lowest 70th percentile.
2. There were also clear tendencies, across most assessment periods, for rates of STI diagnosis to increase as the rates of self-reported alcohol disorder symptoms increased. The exception to this trend was during the age 26-30 assessment period, when those who were in the highest decile on the measure of alcohol disorder symptoms had rates of STI that were slightly lower

than those who reported alcohol symptoms, but were below the highest decile on the measure. The population averaged estimates across the four assessment periods suggested that those in the highest decile for alcohol disorder symptoms had rates of STI diagnoses that were 2.50 (95% CI: 1.81-3.45) times higher than those who reported no alcohol symptoms.

INSERT TABLES 1 AND 2 HERE

3.3 Associations between alcohol measures and STI diagnoses, ages 14-30, after adjusting for non-observed fixed effects and time-dynamic covariate factors

In the next step of the analyses, the models used to obtain the population-averaged estimates of association in Table 2 were adjusted in order to control the associations between alcohol and STI for non-observed fixed sources of confounding, using conditional fixed-effects regression models (see Methods). The results of these analyses are shown in Table 3, which reports the IRR for STI diagnoses for each level of the alcohol-related predictor (amount of consumption; number of alcohol symptoms). The Table shows that:

1. Adjusting the associations between the alcohol measures and STI outcomes for non-observed fixed confounding factors reduced the magnitude of the associations between alcohol and STI diagnosis; however, in both cases these associations remained statistically significant. Those in the highest decile on the measure of alcohol consumption had adjusted rates of STI diagnoses that were 1.85 (95% CI: 1.22-2.82) times greater than those in the lowest 70% on the alcohol consumption measure. Also, those in the highest decile on the measure of alcohol disorder symptoms had adjusted rates of STI diagnoses that were 1.60 (95% CI: 1.02-2.51) times greater. The results of these analyses suggest that the associations between alcohol and STI could not be fully explained by the effects of non-observed fixed confounding factors that were correlated with both alcohol consumption/alcohol disorder symptoms and STI diagnoses.

2. Extension of the conditional fixed effects models described above to account for the effects time-dynamic covariate factors, including: stressful life events; unemployment; cohabitation; dependent children; cannabis use frequency; lifetime depression; and lifetime anxiety disorder; did not materially alter the magnitude of the associations between the alcohol measures and STI diagnosis. After adjustment for the set of observed covariate factors, the associations remained statistically significant ($p < .05$).

INSERT TABLE 3 HERE

3.4 Supplementary analyses

In order to examine the extent to which the above findings were generalizable to variations in classification of the measures of alcohol consumption and alcohol problems, a number of supplementary analyses were carried out (see Methods). In these analyses, the three-level classifications of alcohol consumption and alcohol problems were substituted by a range of measures using 4- and 5-level percentile classifications with varying cutoffs (e.g. 0-60%; 61-75%; 75-90%; 90-100%), and were modelled using the same analytical procedure described in Tables 1 and 2 (above). The results of these analyses were congruent with those reported above; after adjustment for both non-observed fixed confounding and time-dynamic covariate factors, those participants in the highest classification level had rates of STI diagnoses that ranged from 1.52 to 1.81 times higher than those in the lowest classification level (median value IRR = 1.68).

4. Discussion

In this paper we have used data gathered over the course of a 30-year longitudinal study to examine the linkages between alcohol and rates of STI diagnosis. This analysis led to the following conclusions.

First, in agreement with previous research (Bjekic et al., 1997; Cook and Clark, 2005; Cooper, 2002; Halpern-Felsher et al., 1996; Harrison and Kassler, 2000; Hutton et al., 2008; Kang et al., 2007; Patton et al., 2008; Scott-Sheldon et al., 2009), there were clear and consistent links between increasing levels of involvement with alcohol and rates of STI diagnosis. Furthermore, these links were observed irrespective of the measure of alcohol involvement employed; increasing levels of both amounts of alcohol consumed and the number of symptoms of alcohol disorder were related to increased risks of STI. Those with the highest level of alcohol consumption/symptoms had rates of STI diagnoses that were 2.50 times higher than those reporting the lowest level of alcohol consumption/no symptoms.

One explanation of the associations between alcohol and STI diagnosis is that these links may be due to common confounding processes that influence both alcohol use and STI risk (Halpern-Felsher et al., 1996; Kalichman and Cain, 2004; Messiah et al., 1998). A key strength of the present study was that, because of the availability of repeated measures of both alcohol involvement and STI diagnosis, it proved possible to employ conditional fixed effects regression modelling techniques to control for non-observed sources of confounding that may contribute to the links between alcohol and STI. The results of these analyses showed that, after controlling for both non-observed sources of confounding and time-dynamic covariate factors, there remained a statistically significant association between the measures of alcohol and STI risk, suggesting that increasing levels of alcohol involvement played a causal role in increasing the risk of contracting an STI. The results suggested the existence of a causal sequence in which increasing levels of alcohol involvement amongst the present cohort led to increased risks of STI.

In general, the results of the present study provide support for the argument that the links between alcohol and STI are due to the disinhibiting effects of alcohol, resulting in increasing levels of impulsive sexual behaviour and unsafe sexual practices, and increased risk of exposure to STI (Abbey et al., 2007; Boyer et al., 1999; Crosby et al., 2008; LaBrie et al., 2005; Parks et al., 2009; Yan et al., 2007). However, the results of the present study, after controlling for non-observed

confounding factors via the fixed effects regression model, lend little support to the alternative explanation, that both higher alcohol intake and higher rates of STI may reflect a general underlying predisposition to engage in reckless, impulsive behaviour (Halpern-Felsher et al., 1996; Kalichman and Cain, 2004; Messiah et al., 1998). In general, if an underlying predisposition to impulsive or maladaptive behaviour was the primary explanation for both alcohol involvement and STI risk, controlling for non-observed fixed effects would have accounted for a far more substantial proportion of the association between alcohol and STI than was observed in the present study.

The results of the present study also suggest that young people who consume larger amounts of alcohol, or who engage in problematic drinking patterns, are at increased risk of contracting STI. There is a growing literature suggesting that one key component in reducing overall STI exposure may be reducing overall levels of alcohol consumption and problematic alcohol use that may lead to risky sexual behaviour (Boyer et al., 2008; Cooper et al., 2008; Hutton et al., 2008; Scott-Sheldon et al., 2009; Sieving et al., 1997; Yu et al., 2008). Further research is needed, however, to develop and assess interventions designed to reduce the risk of unsafe sexual behaviour following alcohol consumption.

As with any study, there are a number of caveats that need to be imposed upon the analyses and conclusions of the current study. First, it is clear that alcohol use and STI are complex outcomes that are likely to unfold over time in complex ways. This raises the important question of the adequacy of the statistical models and methods in capturing the complexity of the processes involved with increased STI risk. It is likely that, given this complexity, the statistical models we have employed give only an approximation to the true (but non-observed) state of affairs.

Second, the variables included in the analyses are based on report data that have been provided by the participants. As such these variables may be subject to various errors of measurement that may compromise the estimation of model parameters.

Third, it should be noted that the classification schemes for the measures of alcohol involvement that were employed in the present study represented a subset of a wide range of

possible classifications. It may be possible that estimates of the associations between alcohol and STI risk may vary somewhat as a function of the methods used to classify alcohol intake and alcohol problems.

Fourth, although we have attempted to control sources of confounding, it is possible that the analyses may have omitted some important time-dynamic confounding factors that were not measured. In addition, although the fixed-effects models used in this investigation control extensively for non-observed confounding, they may not explain all confounding, particularly for unmeasured confounding factors whose effects on alcohol involvement and STI risk vary with age, such as sensation-seeking. Due to this, it may be possible that present results are an overestimation of the causal linkages between alcohol and STI risk.

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Table 1. Rates of alcohol consumption/alcohol disorder symptoms for each alcohol consumption/symptom classification level, ages 14-30.

	Amount of alcohol consumed				Alcohol disorder symptoms		
	1st-70th percentile	71st-90th percentile	91st-100th percentile		None	Up to 90th percentile	91st - 100th percentile
Mean (SD) number of standard drinks/week in 12 months prior to assessment period				Mean (SD) number of symptoms during assessment period			
Ages 14-18	.01 (.01)	.13 (.05)	.61 (.64)	Ages 14-18	0.0	1.72 (0.79)	7.12 (3.74)
n	633	155	116	n	674	184	120
Ages 19-21	.94 (1.00)	7.25 (2.05)	27.49 (36.83)	Ages 19-21	0.0	3.05 (1.61)	12.36 (5.14)
n	673	190	97	n	669	202	92
Ages 22-25	1.51 (1.65)	10.42 (2.47)	50.82 (96.36)	Ages 22-25	0.0	2.61 (1.43)	12.57 (5.94)
n	688	174	101	n	720	144	107
Ages 26-30	1.17 (1.13)	7.26 (2.23)	37.32 (52.62)	Ages 26-30	0.0	1.92 (1.15)	10.84 (7.56)
n	678	187	106	n	812	64	111

Table 2: Associations between measures of alcohol consumption/alcohol disorder symptoms and STI diagnoses, ages 14-30.

	Amount of alcohol consumed				Alcohol disorder symptoms			
	1st-70th percentile	71st-90th percentile	91st-100th percentile		None	Up to 90th percentile	91st - 100th percentile	
Ages 14-18								
Mean (per 100) rate of STI	4.1	3.2	12.9		3.3	4.9	15.8	
n	633	155	116		674	184	120	
Ages 19-21								
Mean (per 100) rate of STI	5.5	8.9	12.4		5.5	8.9	10.9	
n	673	190	97		669	202	92	
Ages 22-25								
Mean (per 100) rate of STI	4.7	6.3	11.9		4.7	6.9	9.3	
n	688	174	101		720	144	107	
Ages 26-30								
Mean (per 100) rate of STI	4.0	8.6	8.5		4.4	12.5	7.2	
n	678	187	106		812	64	111	
Population-averaged IRR (95% CI)	1 -	1.58 (1.34-1.86)	2.50 (1.80-3.47)	p<.0001	1 -	1.58 (1.35-1.86)	2.50 (1.81-3.45)	p<0001

Table 2. Associations between measures of alcohol consumption/alcohol disorder symptoms and STI diagnoses, ages 14-30, after controlling for non-observed fixed sources of confounding and observed time-dynamic covariate factors.

		Amount of alcohol consumed				Alcohol disorder symptoms			
		1st-70th percentile	71st-90th percentile	91st-100th percentile	p[1]	None	Up to 90th Percentile	91st - 100th Percentile	p[2]
Model 1: Adjusted for non-observed fixed effects	IRR (95% CI)	1 -	1.36 (1.10-1.68)	1.85 (1.22-2.82)	<.01	1 -	1.26 (1.01-1.59)	1.60 (1.02-2.51)	<.05
Model 2: Adjusted for fixed effects & observed covariates	IRR (95% CI)	1 -	1.34 (1.09-1.67)	1.81 (1.18-2.78)	<.01	1 -	1.27 (1.00-1.60)	1.61 (1.01-2.57)	<.05

[1, 2] Statistically significant ($p < .05$) time-dynamic covariate factors included: dependent children; frequency of cannabis use; marginally significant ($p < .10$) covariate factors included lifetime history of major depression.