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The influence of depressive symptoms on alcohol use among HIV-infected Russian drinkers

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Abstract

Background—Depressive symptoms have been linked to HIV progression through a number of biobehavioral mechanisms including increased alcohol use. Although research supports an association between alcohol use and depressive symptoms among HIV patients, there have been few studies that have examined whether depressive symptoms predict subsequent drinking, especially among heavy drinking HIV-infected patients.

Method—Heavy drinking Russian HIV-infected patients ($n = 700$) were recruited from addiction and HIV care settings for a randomized controlled trial of a risk reduction intervention [HERMITAGE]. GEE overdispersed Poisson regression analyses were conducted to assess the association between depressive symptoms and alcohol consumption 6-months later.

Results—In adjusted analyses, depressive symptom severity was significantly associated with drinks per day (global $p = .02$). Compared to the non-depressed category, mild depressive symptoms were significantly associated with more drinks per day [IRR = 1.55, (95% CI: 1.14, 2.09)], while moderate [IRR = 1.14, (95% CI: 0.83, 1.56)] and severe [IRR = 1.48, (95% CI: 0.93, 2.34)] depressive symptoms were not. Associations between depressive symptom severity and heavy drinking days were not statistically significant (global $p = .19$). Secondary analyses using the BDI-II screening threshold (BDI-II > 14) and the BDI-II cognitive subscale suggested an association between depressive symptoms and drinks per day over time but not heavy episodic drinking.

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Contributors

All authors contributed to the design of the study protocol and writing/editing of this manuscript. Authors Cheng, Coleman, and Palfai were responsible for data analyses. All authors have contributed to and approved the final manuscript.

Conflict of interest

All authors declare no conflict of interest related to this research.

Conclusions—Among heavy drinking HIV-infected patients, elevated depressive symptoms were associated with greater subsequent alcohol use. These findings suggest that depressive symptoms may be important to address in efforts to reduce alcohol-related risks among HIV-infected populations.

Keywords

HIV; Depression; BDI-II; Alcohol; Heavy drinking; Russia

1. Introduction

Depression represents one of the most common co-occurring conditions for those living with HIV infection (Simoni et al., 2011). Not only does depression represent a major source of disability for those living with HIV, but there is considerable evidence to suggest that depression may also influence the progression of HIV through a variety of biobehavioral mechanisms. Depressive symptoms have been associated with greater disease progression among those with HIV, as indicated by lower CD4 counts, more rapid progression to AIDS (Ghebremichael et al., 2009; Leserman, 2008), and poorer responses to antiretroviral treatment (Safren et al., 2012). Depression may influence a number of behaviors with potential to accelerate HIV progression. Adherence to antiretroviral treatment has been one of the most frequently studied, as those with depression appear to use ART less consistently however, other health-related behaviors (e.g., substance use, missing health-care appointments) may similarly be affected (Carrico et al., 2011; Traeger et al., 2012).

In addition to interference with treatment, depressive symptoms may increase disease progression through its association with other HIV-risk behaviors (Simoni et al., 2011; Zhan et al., 2012). One particular concern is its impact on alcohol use. Heavy drinking has been linked with HIV disease progression through both biological and behavioral mechanisms (Pandrea et al., 2010; Samet et al., 2007). Although there is no clear level of “safe” drinking among those who are HIV-infected, alcohol consumption at even moderate doses has been shown to increase behavioral risks for HIV progression (Grodensky et al., 2012; Fisher et al., 2007; Shuper et al., 2009).

Studies with a range of HIV populations have suggested that depression is associated with higher rates of heavy drinking and alcohol use disorders (e.g., Longmire-Avital et al., 2012; Rosenbloom et al., 2007; Sullivan et al., 2008, 2011), however, few have examined whether depressive symptoms may be associated with subsequent alcohol use (Cook et al., 2012). Cook et al. (2012) found that depressive symptoms were associated with subsequent heavy drinking patterns in a cohort of HIV-infected women, however, baseline heavy drinking rates were relatively low in this population (7%). Given the impact of depressive symptoms on risk factors for higher alcohol involvement (Holahan et al., 2004; McKellar et al., 2008; Ralston and Palfai, 2012; Swendsen and Merikangas, 2000), there is reason to believe that higher levels of depressive symptoms may be linked with increased alcohol consumption over time. Developing a better understanding the impact of depression on alcohol use among HIV-infected men and women in populations with a high prevalence of heavy drinking is an important and understudied issue.

Alcohol use is of particular concern for the HIV epidemic in Russia. Recent estimates suggest that approximately 1 million individuals are living with HIV in Russia with over 100,000 new cases per year (UNAIDS/WHO, 2009). In contrast to major modes of transmission through same sex activity, those in Russia continue to show high rates of intravenous drug use (IDU) transmission and transmission to opposite sex partners (Cohen, 2010). Alcohol use represents a central risk factor for unprotected sexual behavior among those living with HIV (Krupitsky et al., 2005; Shuper et al., 2009). Consistent with the high rates of hazardous/harmful alcohol use in the general population, Russian HIV-infected patients commonly exhibit patterns of heavy alcohol consumption, with estimates of lifetime alcohol dependence exceeding 50% (Krupitsky et al., 2005).

Despite the high rates of depression among Russian HIV-infected patients (e.g., Amirkhanian et al., 2011), there is relatively little known from prospective studies about whether depression influences alcohol use patterns among patients living with HIV. The high prevalence of alcohol use among Russian HIV-infected patients provides an opportunity to gain an understanding of how depression influences alcohol use over time among a population that exhibits high levels of alcohol consumption.

Efforts to study depression symptom severity among those with HIV across cultures have typically relied on translated versions of instruments developed in the US. Although there are a number of measures that have been used to assess depressive symptoms (e.g., CES-D, BDI) that have been used in Russian studies that have examined HIV-risk (e.g., Amirkhanian et al., 2011; Krupitsky et al., 2013), the Beck Depression Inventory-II (Beck et al., 1996) represents one of the few measures that is directly based on Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) symptom criteria. This 21-item self-report measure represents an important revision of previous versions of the BDI as it has changed the time frame (i.e., 2 weeks) and the items to be more consistent with current conceptualizations of depression. Previous research has suggested that the BDI-II has good psychometric characteristics as a measure of depressive symptom severity across a number of cultures (e.g., Campos and Goncalves, 2011; Ghassemzadeh et al., 2005). Similarly, studies of a range of medical patient populations have shown that the BDI-II may be a reliable and valid measure of depressive symptoms for a variety of medical populations (e.g., Aiken et al., 1999; Arnau et al., 2001; Grothe et al., 2005; Viljoen et al., 2003) but has been used sparingly in international studies of HIV patients and, to our knowledge, has not been utilized among Russian HIV patients. The two highly correlated somatic and cognitive factors of the BDI-II identified in previous work (Beck et al., 1996; Steer et al., 1999) also provides investigators the capacity to specifically assess cognitive symptoms of depression as an alternative index of depressive symptoms that does not include items that potentially overlap with the somatic symptoms of HIV (Kalichman et al., 1995; Safren et al., 2012).

The main objective of this study was to prospectively examine the influence of depressive symptoms on subsequent alcohol use behavior (i.e., number of drinks per day and number of heavy drinking days) among HIV-infected heavy drinking patients. Heavy drinking is defined by National Institute for Alcohol Abuse and Alcoholism (NIAAA) guidelines of 14 total drinks per week or single occasion drinking of 5 drinks for men and 7 drinks per

week or single occasion drinking of 4 drinks for women. Although they appear to be associated with different patterns of risk behaviors among HIV patients, both heavy episodic drinking and quantity of alcohol consumption are associated with important HIV outcomes (Grodensky et al., 2012; Shuper et al., 2009). We hypothesized that those with more severe depressive symptoms on the BDI-II would consume more drinks per day and exhibit more frequent heavy drinking episodes. In addition, we explored the related question of whether those with clinically significant elevated depressive symptoms (BDI-II 14) would show greater alcohol use than those without elevated depressive symptoms. Given the debate about whether the somatic items on depression measures represent symptoms of depression for medical patients or are confounded with the somatic components of medical conditions (Aiken et al., 1999; Arnau et al., 2001; Clark et al., 1998), some investigators have suggested that cognitive symptoms may provide a more precise method of assessing depressive symptoms among those with HIV (Kalichman et al., 1995; Safren et al., 2012). Thus, in secondary analyses, we also examined whether higher scores on the Cognitive subscale of the BDI-II were associated with alcohol use among this HIV-infected sample. Finally, as there is some evidence that the association between depressive symptoms and alcohol use may be stronger among men in certain HIV-infected populations (e.g., Longmire-Avital et al., 2012), exploratory analyses were conducted to assess whether the influence of depressive symptoms on indices of alcohol use was moderated by gender.

2. Methods

2.1. Participant selection and recruitment

We examined Russian HIV-infected heavy drinkers enrolled in the HERMITAGE study, a randomized controlled trial (RCT) of a secondary HIV prevention intervention. The study was approved by Institutional Review Boards of Boston University and Pavlov State Medical University. Eligibility criteria included the following: documented HIV-infection, reported anal or vaginal sex without a condom in the past 6 months, 18–70 years old, and heavy drinking in the past 6 months as previously defined. Participants ($n = 700$) were recruited from four inpatient and outpatient HIV and narcology (i.e., addiction treatment) care sites in St. Petersburg, Russia. Recruitment was conducted within the hospital settings. Research associates (RAs) approached patients, assessed eligibility, and offered participation in the study. Those identified by staff as experiencing significant cognitive impairment or acute illness that might preclude participation were not excluded. Of the 921 subjects who were screened, 79% ($n = 732$) were eligible and 76% ($n = 700$) completed the baseline assessment.

2.2. Measures

2.2.1. Alcohol outcomes—The two key alcohol outcomes from the previous 30-days were (1) number of heavy drinking days and (2) number of drinks per day. We assessed alcohol consumption using the interview-based 30-day Time-Line Follow Back (TLFB) (Sobell and Sobell, 1992), an extensively validated instrument measuring quantity, frequency, and types of standardized drinks consumed in the previous 30 days. Alcohol use was assessed at baseline, 6-month, and 12-months.

2.2.2. Depression—The main independent variable was depressive symptoms assessed by the official Russian translation of the Beck Depression Inventory-II (Beck, 2007). The BDI-II consists of a set of 21 symptom questions that are scored 0–3 based on severity. Patients use the past two weeks as the time frame for their responses. The BDI-II is used to indicate those with elevated depressive symptoms (BDI-II ≥ 14) and to identify those with different levels of depression severity (mild = 14–19, moderate = 20–29, severe = 30–63 (Beck et al., 1996). The particular advantage of the BDI-II is that it provides a specific measure of depressive symptoms rather than general distress and it is anchored to DSM-IV diagnostic criteria for major depression. The BDI-II has been used to assess depression among medical patients (Arnau et al., 2001; Viljoen et al., 2003), including those with HIV (Lipps et al., 2010). Previous work has identified two highly correlated factors, cognitive/affective and somatic (Beck et al., 1996; Steer et al., 1999) that make up a single higher-order factor. To examine whether the psychometric characteristics of the Russian BDI-II (Beck et al., 1997) findings in this HIV sample was consistent with the factor solution described by Beck et al. (1996); i.e., two correlated factors, cognitive/affective and somatic, that comprise a higher-order factor), we conducted a preliminary confirmatory factor analysis. Goodness-of-fit appeared adequate based on three fit indices: Comparative Fit Index (CFI) = 0.92, Root Mean Square Error of Approximation (RSMEA) = 0.05, and Standardized Root Mean Square Residual (SRMSR) = 0.04, but not on the chi-square statistic ($\chi^2 = 548.71, p < .01$) which may be a result of the larger sample size (Brown, 2006). Cronbach's alpha for the Total BDI-II in the current sample was .90, while the Cognitive (alpha = .81) and Somatic (alpha = .83) subscales also showed adequate internal consistency. As expected, these subscales were highly correlated ($r = .71$).

2.2.3. Covariates—A series of additional instruments were administered to patients at baseline that included study site and measures of age, gender, alcohol use, and injection drug use in the past 6 months. These variables, along with randomization group and time, served as covariates to control for potential confounds in the prospective analysis between depressive symptoms and alcohol use variables.

2.3. Procedures

The project was presented to participants as a multi-session health intervention study. Participants were asked to participate in a five-session intervention that included both individual and group based components. Assessments were primarily conducted by a trained research associate in a face-to-face interview format though some questionnaires (e.g., BDI-II) were completed using self-reported paper format. Follow-up outcome assessments occurred at 6 ($n = 523$; 75%) and 12 months ($n = 492$; 70%). Participants were randomized to an intervention or control condition. The intervention condition was a secondary HIV-prevention intervention based on Healthy Relationships Intervention (Kalichman et al., 2001). A key modification was the inclusion of two individual sessions designed to address HIV risk behavior, IDU, and alcohol use. The individual sessions integrated sexual risk reduction counseling with motivational interviewing strategies to reduce risky alcohol and drug use. This aspect of the intervention focused on increasing motivation to change and self-efficacy. Drug and alcohol use were addressed in one of the group sessions as barriers to HIV disclosure. Harm reduction skills related to IDU were also presented in the group

sessions. Participants in the Attention Control condition completed a five-session intervention (two individual sessions) that provided psychoeducation about social support, nutrition, and stress management. Additional details of methods for the RCT have been presented elsewhere (Samet et al., in preparation).

2.4. Statistical analyses

We used descriptive statistics to characterize the study sample at enrollment. The primary analyses incorporated both the 6 and 12-month outcomes into a single model using longitudinal generalized estimating equations (GEE) regression methods. Depressive symptoms were modeled using BDI-II depression severity levels (primary analyses) and screening threshold scores (BDI-II > 14; secondary analyses). Separate multivariable GEE overdispersed Poisson regression models were fit to assess the associations between depressive symptoms and each of the two alcohol-related outcomes (e.g., number of heavy drinking days; number of drinks per day). The analyses were lagged such that depressive symptoms at a given timepoint were used to predict alcohol outcomes at the subsequent follow-up 6 months later. The outcomes were modeled as count data and the Pearson chi-square correction was used to account for overdispersion in the data. The longitudinal models account for the correlation from using repeated observations for the same subject. GEE Poisson models were fit using an independence working correlation matrix and a log link. Results are reported using the empirical standard errors. Depressive symptoms and injection drug use were modeled as time-varying variables.

Incidence rate ratios (IRR) presented from the Poisson regression models represent the relative increase in the rate of outcomes for each depressive symptom category (mild, moderate, severe) compared to the “no elevated depressive symptoms” group. The primary set of analyses examined how depressive symptoms were associated with alcohol use over time based on established levels of severity on the BDI-II (Beck et al., 1996). Secondary analyses conducted based on screening threshold scores for substantial depressive symptoms at study entry (BDI-II ≥ 14) allowed us to examine whether those identified as “depressed” through screening with the BDI-II would show increased alcohol involvement over time. Additional analyses explored the influence of depressive symptom severity on alcohol use by modeling quartiles of the Cognitive subscale of the BDI-II in order to avoid an assumption of linearity with the lowest quartile serving as the comparison group. In each of the regression models, potential confounding factors that have been previously shown to be related to depressive symptoms and alcohol use (e.g., age, gender, injection drug use) were included as covariates (Jones-Webb et al., 1996; Kertesz et al., 2003; Kim et al., 2003) along with baseline value of the outcome, randomization group, recruitment site, and time. Exploratory analyses examined gender as a potential effect modifier by testing the interaction between the depressive symptoms variable and gender. If the *p*-value for interaction was significant at an alpha level of 0.15, subsequent analyses would be performed stratified by gender. In order to minimize multiple comparisons, pairwise testing was not performed unless the global *p*-value for depressive symptom severity was significant at an alpha level of 0.05. Analyses were performed using SAS software (version 9.2 SAS Institute, Cary, NC).

3. Results

3.1. Descriptive statistics

A large proportion (64%) of the sample reported elevated symptoms of depression (BDI-II 14) at baseline with 22%, 25%, and 17% of the sample meeting criteria for mild, moderate and severe depressive symptoms, respectively. Table 1 presents baseline characteristics of participants by depression severity level. Patients in the study were predominantly male (59.3%) and exhibited high levels of alcohol involvement and depressive symptoms. The mean number of heavy drinking days in the past month was 6.5 ($SD = 8.6$) and the mean number of drinks per day was 3.0 ($SD = 4.7$). Those who completed the study reported fewer depressive symptoms (61% vs. 71% with depressive symptoms, $p = .004$), fewer average drinks per day (2.7 vs. 3.7, $p = .006$) and fewer heavy drinking days (6.0 vs. 7.4, $p = .03$) at baseline. The 700 subjects provided 1015 observations across the 6 and 12-month follow-up timepoints. As shown in Table 2, depressive symptom severity was lower among this cohort at the 6-month and 12-month outcome points compared to baseline. Similarly, alcohol consumption rates were lower at the 6-month and 12-month timepoints compared to baseline.

3.2. Association between BDI-II scores and alcohol consumption

3.2.1. Depressive symptoms severity and alcohol outcomes—In order to characterize the association between depressive symptoms and alcohol use, we examined whether severity of depressive symptoms was associated with more drinking over time as measured by drinks per day and heavy drinking days. Covariate adjusted analyses showed a significant effect of depressive symptom severity on drinks per day (global $p = .03$). Incidence rate ratios for the association between each level of depressive symptom severity and each of the alcohol outcomes are presented in Table 3. Compared to those with no depressive symptoms, those with mild [IRR = 1.55, (95% CI: 1.14, 2.09), $p = .01$] depressive symptoms reported significantly more drinks per day; however those with moderate [IRR = 1.14 (95% CI: 0.83, 1.56), $p = .41$] and severe [IRR = 1.48 (95% CI: 0.93, 2.34), $p = .10$] depressive symptoms appeared to report more drinks per day but the results were not statistically significant. Thus, these results did not suggest a linear relationship between depressive symptoms and the amount of drinking per day. Adjusted analyses on the influence of depressive symptom severity levels on the other primary outcome, heavy drinking days, was not statistically significant (global $p = .19$), although the observed associations for mild, moderate, and severe BDI-II levels were similar to the analysis on drinks per day. Exploratory analyses conducted without intervention group or IDU status as covariates had similar results. In addition, we did not find evidence that intervention or IDU moderated the influence of depression on alcohol outcomes.

3.2.2. Elevated depressive symptoms and alcohol outcomes—To examine the predictive value of clinical screening thresholds, we examined the association between clinical screening cutoff scores (BDI-II 14) and alcohol use. These adjusted analyses, which collapse across depressive symptom severity categories, showed a significant association between depressive symptoms and drinks per day only. Those who experienced elevated depressive symptoms showed an approximately 1.37 fold increase in drinks per day

(95% CI: 1.05, 1.77), $p = .02$]. Consistent with the depression severity analyses, those who exceeded screening thresholds on the BDI-II (14) did not, show significantly more heavy drinking days at follow-up, although the magnitude of effect was clinically important [IRR = 1.34 (95% CI: 0.98, 1.83), $p = .07$].

3.2.3. BDI-II cognitive subscale severity and alcohol outcomes—Given the overlap between HIV symptoms and symptoms of depression, there has been some debate in the literature about how best to assess depression in this population (Kalichman et al., 1995). Some have suggested that cognitive symptoms may more accurately represent depression as they are less likely to be confounded with HIV symptoms (Safren et al., 2012). Consequently, we examined whether level of depressive symptom severity as measured by the BDI-II Cognitive subscale was associated with greater alcohol consumption. Cognitive symptom severity levels were defined based on quartiles to avoid assumptions of linearity and because there are no clinically defined thresholds for the subscale. IRRs for the association between cognitive depressive symptom severity and each of the alcohol outcomes are presented in Table 4. Consistent with analyses on BDI-II total symptom severity, adjusted analysis on drinks per day showed a significant overall effect of cognitive symptom severity (global $p = .01$). Interestingly, only those in the 3rd quartile [IRR = 1.76 (95%CI: 1.26, 2.44), $p < .01$] showed significantly greater drinking at the subsequent timepoint compared to those with no/mild (1st quartile) cognitive symptoms of depression. Analysis of the association between cognitive symptom severity and subsequent heavy drinking days was not significant (global $p = .13$).

3.2.4. Stratified analyses by gender—Given that a number of studies have identified gender as a moderator of depression effects on alcohol use outcomes, each set of analyses was conducted with gender as an interaction term. None of these analyses suggested that the effects of depressive symptoms were significantly different by gender (all p -values > 0.15).

4. Discussion

The current study examined whether depressive symptoms predicted subsequent alcohol use among Russian HIV-infected heavy drinkers in a prospective study that examined drinking outcomes over a 12-month period. Although previous work has examined the association between depressive symptoms and alcohol use among HIV-infected patients (e.g., Ghebremichael et al., 2009; Sullivan et al., 2008), there has been little research that has examined whether depressive symptoms are associated with subsequent alcohol use in prospective studies among populations who engage in heavy drinking. Moreover, few studies have explored the influence of depression using measures that reflect current conceptualizations of depressive symptomatology.

Using the BDI-II (Beck et al., 1996), the current study provides support for the view that depressive symptoms are associated with higher levels of alcohol consumption among Russian HIV-infected heavy drinkers. Analyses using the BDI-II threshold for elevated depressive symptoms, clinical severity categories, and the cognitive subscale provide a detailed characterization of the relation between depressive symptoms and specific drinking outcomes that pose risks for HIV-infected patients.

There was a significant effect of BDI-II clinical severity level on number of subsequent drinks per day but not on heavy drinking days in this sample. Examination of specific BDI-II severity levels using the full measure suggested that mild depressive symptom severity may be significantly different than the non-depressed category. Additional analyses supported the view that BDI-II depression screening threshold (BDI = 14) may be at risk for greater alcohol consumption even when controlling for known predictors of alcohol use (e.g., injection drug use). To address the issue that has been raised in previous work that HIV disease symptoms may confound measurement of depression in HIV patients, we examined quartiles of the Cognitive subscale and found that severity of depressive symptoms significantly predicted drinks per day. However, analyses of cognitive symptoms suggested that moderate levels of cognitive depressive symptoms were most strongly associated with drinking, consistent with previous suggestions (e.g., Simoni et al., 2011) that depressive symptoms may show a curvilinear relation with certain risk behaviors with the impact of depressive symptoms being diminished at the severe end of the spectrum. Taken together, these analyses on severity do not provide evidence for a clear linear relationship between depressive symptoms severity and drinks per day.

Although the analysis on the influence of depressive symptoms severity on number of heavy drinking days was not statistically significant, the pattern of results (adjusted IRRs) was largely consistent with the analysis on drinks per day. The absence of a significant overall effect for depressive symptoms severity level on this outcome may in part be due to the notably lower frequency of heavy drinking days observed in this sample at the 6 and 12-month assessments. Mean heavy drinking days in the past month was 6.5 ($SD = 8.6$) at baseline, while it was 2.1 ($SD = 5.0$) and 2.4 ($SD = 5.3$) at 6 and 12-month outcomes, respectively. This difference in heavy drinking may be influenced by a variety of factors such as engagement in treatment, increasing severity of HIV illness, and differential attrition among heavier drinkers.

The evidence for the influence of depressive symptoms on drinking appears to be strongest for amount of alcohol consumed per day rather than the number of heavy drinking days. Although episodic heavy drinking represents a clear risk to the health of an individual, even moderate levels of alcohol consumption may present behavioral risks to HIV-infected patients (Grodensky et al., 2012), such as unprotected sexual behavior (Shuper et al., 2009), especially for those who tend to consume alcohol in contexts related to sexual activity (Fisher et al., 2007, 2010). However, information about what constitutes a “safe” level of drinking for an HIV-infected patient remains to be specified and is likely determined by important patient individual differences (e.g., CD4 count, behavioral risks, treatment adherence).

Finally, exploratory analyses did not suggest an interaction between gender and depressive symptoms. Although depression tends to be more frequent among women, the predictive value of depressive symptoms on alcohol use has shown mixed effects for different medical populations. It does not appear that gender significantly moderates the impact of depressive symptoms as a predictor of alcohol use and interaction effects may be population specific.

It is important to consider certain features of this study when interpreting these findings. First, and most importantly, these findings must be considered within the Russian HIV population and caution must be taken to generalize these results to other populations. For example, in contrast to HIV population studies in the US which has a large proportion of MSM, in the current sample less than 4% of the patients identified themselves as MSM. The epidemiological factors that have influenced the growth of the HIV epidemic in Russia continues to be injection drug use and heterosexual contact with injection drug users (Mills et al., 2013). Moreover, alcohol use is particularly high among Russian HIV samples, as it presents an important risk for the general Russian population, particularly among men (Leon et al., 2009). A second consideration is the fact that the sample consisted mostly of individuals who were in treatment for HIV and/or addiction who participated in an HIV prevention intervention trial. Whether those who volunteer for such studies represent the broader HIV-infected Russian population is not known. Third, although we did adjust for randomization group in regression analyses, participants were exposed to risk reduction interventions offered as part of the study in both conditions which may have influenced the associations between variables examined here. As noted above, the results do not change with the inclusion or exclusion of this variable, however, one cannot discount the potential influence of trial-related procedures on outcomes. Finally, this was a challenging sample to engage with less than 80% of the sample completing follow-up procedures. Those who completed the study tended to be lower on depression and alcohol variables. It is unclear how this may have impacted the study findings but must be considered in the interpretation and generalizability of the results.

Despite these limitations, there were a number of strengths in this work including the use of current assessment instruments, relatively large sample size, broad inclusion criteria, and the use of lagged analyses to examine the influence of depressive symptoms over time. In particular, the study used a measure of depressive symptoms that corresponds to more recent perspectives on clinical depression in DSM-IV. As such, this measure provides both the content and time-frame that is consistent with depression. Moreover, this study provided initial evidence of the psychometric characteristics of the Russian version of the BDI-II. Despite the advantages of using a specific measure of depressive symptoms severity that is linked to DSM-IV conceptions of depression, the factor structure of the BDI-II among Russian HIV patients has not been examined. Results from the confirmatory factor analysis and the subscale reliabilities do provide preliminary evidence that its psychometric characteristics are consistent with previous work. These analyses do not address the controversy about whether somatic symptoms should be considered to be part of the depressive syndrome among HIV patients or are confounded with medical symptoms of HIV (e.g., Kalichman et al., 1995). However, the advantage of the BDI-II is that the two intercorrelated factors also provide an opportunity to explore cognitive symptoms of depression specifically to reduce potential concerns about confounds between symptoms of HIV and depression.

In sum, these findings provide potentially important clinical information about how depressive symptoms may be linked with subsequent drinking among this population. It suggests that when assessed with a measure specific to depressive symptoms (i.e., BDI-II), HIV-infected heavy drinkers with elevated depressive symptoms are at greater risk for

alcohol consumption over time. Examination of clinical severity and the cognitive symptom subscales, in this prospective study provides initial information about how depressive symptoms may be associated with drinking among HIV-infected heavy drinkers. Given the high prevalence of elevated depressive symptoms in this sample and the impact of heavy drinking on HIV disease progression and health more generally, clinicians should attempt to address depressive symptoms directly in prevention and treatment efforts.

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Table 1
Baseline characteristics of HIV-infected Russian heavy drinkers by BDI-II^a depression severity level (*n* = 700).

Variable	Overall	None	Mild	Moderate	Severe	<i>p</i> -Value
Age, mean (SD)	30.1 (5.2)	30.0 (5.2)	29.7 (4.2)	29.8 (4.9)	31.3 (6.5)	.03
Gender						
Female, no. (%)	285 (40.7%)	75 (30.1%)	61 (38.9%)	77 (44.5%)	72 (59.5%)	<.001
Male, no. (%)	415 (59.3%)	174 (69.9%)	96 (61.1%)	96 (55.5%)	49 (40.5%)	
IDU ^b no. (%)	294 (42.0%)	65 (26.1%)	74 (47.1%)	88 (50.9%)	67 (55.4%)	<.001
Drinks Per day ^c	3.0 (4.7)	2.7 (3.9)	3.0 (3.0)	3.0 (4.9)	3.8 (7.2)	
Heavy drinking days ^d	6.5 (8.6)	5.9 (8.0)	7.1 (9.0)	6.6 (8.5)	6.8 (9.3)	.53

^aBDI-II: Beck Depression Inventory-Second Edition.

^bIDU: injection drug use in Past 30 days.

^{c,d}Drinks per day and heavy drinking days from the 30-day time line follow-back.

Table 2

Depressive symptom severity and alcohol use among HIV-infected Russian heavy drinkers at each timepoint.

Variable	Baseline	6-Month	12-Month
<i>N</i> (%)	700 (100%)	523 (75%)	492 (70%)
Depressive symptoms			
BDI-II ^a severity no. (%)			
None (<14)	249 (35.6%)	267 (51.1%)	253 (51.4%)
Mild (14–19)	157 (22.4%)	81 (15.5%)	83 (16.9%)
Moderate (20–28)	173 (24.7%)	98 (18.7%)	84 (17.1%)
Severe (29+)	121 (17.3%)	77 (14.7%)	72 (14.6%)
Alcohol consumption ^b			
Median drinks per day (IQR) ^c	2.0 (1.3)	0.0 (0.1)	0.0 (0.2)
Mean drinks per day (SD)	3.0 (4.7)	0.9 (1.7)	1.2 (2.7)
Median heavy drinking days (IQR)	2.5 (1.9)	0.0 (0.2)	0.0 (0.3)
Mean heavy drinking days (SD)	6.5 (8.6)	2.1 (5.0)	2.4 (5.3)

^aBDI-II: Beck Depression Inventory-Second Edition.

^bDrinks per day and heavy drinking days from the 30-day time line follow-back.

^cIQR: interquartile range (25th and 75th percentiles).

Table 3

The association between BDI-II depressive symptom severity and subsequent alcohol outcomes among HIV-infected Russian drinkers ($n = 700$).

	<u>Unadjusted</u>		<u>Adjusted^d</u>	
	IRR ^a (CI) ^b	<i>p</i>	IRR ^a (CI) ^b	<i>p</i>
Drinks per day				
Depressive symptom severity		.06 ^c		.03 ^c
None	–		–	
Mild	1.51 (1.12, 2.04)		1.55 (1.14, 2.09)	.01
Moderate	1.15 (0.84, 1.59)		1.14 (0.83, 1.56)	.41
Severe	1.53 (0.92, 2.53)		1.48 (0.93, 2.34)	.10
Heavy drinking days				
Depressive symptom severity		.14 ^c		.19 ^c
None	–		–	
Mild	1.58 (1.05, 2.38)		1.49 (1.01, 2.19)	
Moderate	1.26 (0.85, 1.87)		1.16 (0.79, 1.72)	
Severe	1.47 (0.95, 2.27)		1.39 (0.88, 2.19)	

^a IRR: incidence rate ratio.

^b CI: 95% confidence interval.

^c Represents global *p*-value, pairwise comparison reported only if global *p*-value α 0.05.

^d Longitudinal Poisson regression models adjusting for baseline value of alcohol use variable, gender, age, injection drug use, study site, randomization group, and time.

Models reflect associations over time between depressive symptoms and alcohol outcomes at 6 and 12 months.

Table 4

The association between cognitive symptom severity (BDI-II cognitive subscale) and alcohol outcomes among HIV-infected Russian drinkers ($n = 700$).

	<u>Unadjusted</u>		<u>Adjusted^d</u>	
	IRR^a (CI)^b	<i>p</i>	IRR^a (CI)^b	<i>p</i>
Drinks per day				
Cognitive symptom severity		.02 ^c		.01 ^c
Second quartile	1.17 (0.83, 1.64)	.37	1.23 (0.89, 1.71)	.21
Third quartile	1.69 (1.18, 2.41)	<.01	1.76 (1.26, 2.44)	<.01
Fourth quartile	1.55 (0.99, 2.44)	.06	1.40 (0.89, 2.21)	.15
Heavy drinking days				
Cognitive symptom severity		.05 ^c		.13 ^c
Second quartile	0.98 (0.62, 1.54)		0.99 (0.66, 1.49)	
Third quartile	1.67 (1.04, 2.67)		1.44 (0.97, 2.14)	
Fourth quartile	1.35 (0.83, 2.19)		1.07 (0.66, 1.76)	

^aIRR: incidence rate ratio.

^bCI: 95% confidence interval.

^cRepresents global p -value, pairwise comparison reported only if global p -value $\alpha = 0.05$.

^dLongitudinal Poisson regression models adjusting for baseline value of the alcohol use variable, gender, age, injection drug use, study site, randomization group, and time.

Models reflect associations over time between depressive symptoms and alcohol outcomes at 6 and 12 months.