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The role of hazardous drinking reductions in predicting depression and anxiety symptom improvement among psychiatry patients: A longitudinal study

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Abstract

Background—Co-occurrence of depression, anxiety, and hazardous drinking is high in clinical samples. Hazardous drinking can worsen depression and anxiety symptoms (and *vice versa*), yet less is known about whether reductions in hazardous drinking improve symptom outcomes.

Methods—Three hundred and seven psychiatry outpatients were interviewed (baseline, 3-, 6-months) for hazardous drinking (drinking over recommended daily limits), depression (PHQ-9), and anxiety (GAD-7) as part of a hazardous drinking intervention trial. Longitudinal growth models tested associations between hazardous drinking and symptoms (and reciprocal effects between symptoms and hazardous drinking), adjusting for treatment effects.

Results—At baseline, participants had moderate anxiety ($M=10.81$; $SD=10.82$) and depressive symptoms ($M=13.91$; $SD=5.58$); 60.0% consumed alcohol at hazardous drinking levels. Over 6-months, participants' anxiety ($B=-3.03$, $p<.001$) and depressive symptoms ($B=-5.39$, $p<.001$) improved. Continued hazardous drinking led to slower anxiety ($B=0.09$, $p=.005$) and depressive symptom ($B=0.10$, $p=.004$) improvement; reductions in hazardous drinking led to faster anxiety ($B=-0.09$, $p=.010$) and depressive ($B=-0.10$, $p=.015$) symptom improvement. Neither anxiety ($B=0.07$, $p=.066$) nor depressive ($B=0.05$, $p=.071$) symptoms were associated with hazardous drinking outcomes.

Limitations—Participants were psychiatry outpatients, limiting generalizability.

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Contributors

Drs. Bahorik and Satre formulated the research questions and constructed the study design. Dr. Bahorik wrote the first draft of the manuscript and all authors provided critical revisions. All authors contributed to and approved the final manuscript.

Conflict of interest

None.

Conclusions—Reducing hazardous drinking can improve depression and anxiety symptoms but continued hazardous use slows recovery for psychiatry patients. Hazardous drinking-focused interventions may be helpful in promoting symptom improvement in clinical populations.

Keywords

Depression; Alcohol; Hazardous drinking; Anxiety

1. Introduction

Co-occurrence of depression, anxiety, and hazardous drinking (drinking over recommended daily limits) is prevalent in the general population (Grant et al., 2004), and even more common among psychiatry patients (Sullivan et al., 2005). Hazardous drinking is problematic because it can impede treatment, raise the risk of escalation for alcohol problems, and lead to poor mental health outcomes (Kuo et al., 2006; Mills et al., 2009). To date, considerable work has examined relations between hazardous drinking, anxiety, and depression, and document findings important to symptom recovery. Hazardous drinking can interfere with psychosocial functioning and precipitates stressful interpersonal problems (Kung, 2000), which can in turn worsen mental health outcomes (Bell et al., 2015). Clinical investigations also report hazardous drinking as a risk factor for depression (Boschloo et al., 2012), and that continued alcohol consumption worsens anxiety and depressive symptoms (Schuckit and Hesselbrook, 1994). One study found patients treated for alcohol use disorder showed improvement (decreases) in symptoms following abstinence (Brown and Schuckit, 1988), indicating a potential association between reductions in alcohol consumption to symptom improvement. Such findings suggest that while hazardous drinking exacerbates symptoms, reductions in hazardous drinking may contribute to symptom improvement.

Studies report that anxiety and depressive symptoms can lead to higher alcohol intake, potentially consumed to reduce psychological distress or ‘self-medicate’ (Conner et al., 2009). Supporting the ‘self-medication’ model, a recent general population study tested bi-directional effects (alcohol consumption drives changes in general mental health and *vice versa*) of relations between mental health and heavy drinking, and found that mental health was the leading indicator of change (Bell et al., 2015). Whether similar findings would emerge in a clinical sample of psychiatry patients is largely unknown.

Studies have identified several ways alcohol intake potentially influences symptoms (and *vice versa*) in clinical samples, yet few have examined whether reduction in hazardous drinking contributes to improvement in anxiety and depression symptoms over time. Evidence to this in a clinical sample would signal a need for symptom stabilization efforts on alcohol screening and intervention when needed, which may accelerate recovery. As a primary study aim we examined associations between hazardous drinking and consumption reduction on anxiety and depression symptoms over 6-months in 307 psychiatric outpatients. Given the potential for bidirectional effects, we also explored associations between anxiety, depression, and hazardous drinking outcomes. Such findings would indicate that symptoms worsen hazardous drinking outcomes; and thus slow the rate of reduction in drinking days over time.

2. Method

2.1. Participants

The 307 participants were selected from a randomized controlled trial of motivational interviewing (MI) in alcohol/drug use treatment for depression (Satre et al., 2016). Patients were recruited from: Kaiser Permanente Southern Alameda Medical Center Department of Psychiatry in Union City and Fremont, California. Inclusion criteria required: age ≥ 18 ; ≥ 5 Patient Health Questionnaire (PHQ-9) (Kroenke et al., 2001); absence of mania/psychosis; drug use (illicit/non-prescribed use) or hazardous drinking $\geq 3/\geq 4$ drinks/day for women/men within 30 days. These participants were recruited at more conservative hazardous drinking levels than used in general population studies, as psychiatry patients often have medication interactions with alcohol; data were collected at the more widely used $\geq 4/\geq 5$ drinks/day for women/men (Satre et al., 2016).

These 307 patients comprised our study sample, and we examined hazardous drinking patterns and hazardous drinking reductions on symptom recovery at the more widely used $\geq 4/\geq 5$ drinks/day levels for women/men, adjusting for treatment effects. We also explored the effect of symptoms on hazardous drinking outcomes.

2.2. Measures

2.2.1. Hazardous drinking and hazardous drinking reductions—Consistent with NIAAA guidelines (NIAAA, 2005), hazardous drinking cut-offs were defined as $\geq 4/\geq 5$ drinks in a day for women/men. A single-item hazardous drinking screener determined patients' number of hazardous drinking days in the month before study interviews (baseline, 3-, 6-months); higher values indicated more drinking days at the hazardous drinking level. This screener has been validated as a predictor of substance use disorder risk (Dawson, 2010). Using original values of hazardous drinking days determined at study interviews, a time-varying reduction in hazardous drinking (Δ – hazardous drinking) variable was computed by subtracting hazardous drinking levels in subsequent study periods from levels in prior periods (hazardous drinking at 3 months – hazardous drinking at baseline = Δ in hazardous drinking at three months, *etc.*).

2.2.2. Symptoms—PHQ-9 measured depression 2 weeks prior to each interview; higher scores indicate greater depression (9-items, range 0–27; score >5 = at least mild depression) (Kroenke et al., 2001). Generalized Anxiety Disorder (GAD-7) scale measured anxiety in the 2 weeks prior to each interview; higher scores indicate greater anxiety (7-items, range 0–21; score >5 = at least mild anxiety) (Spitzer et al., 2006).

2.3. Procedures

Data were collected for a randomized controlled trial of MI in alcohol/drug use treatment for depression. The methods and outcomes of the parent study have been reported (Satre et al., 2016). Eligibility was determined by baseline alcohol/drug use and PHQ-9 score. Demographics were collected at baseline and patients were randomized (MI: 45-min in-person session followed by two 15-min booster sessions; Control: 2-page brochure on substance use risks). Patients were offered \$50 gift cards for completing the interviews. Of

the 307 participants, 296 (96%) completed the 3-month telephone follow up, 302 (98%) completed the 6-month follow up. The University of California, San Francisco (UCSF) Committee on Human Subjects and the Kaiser Permanente Institutional Review Board approved the procedures. Patients were provided with written informed consent prior to participation.

2.4. Data analysis

We examined: (1) longitudinal associations of participants' symptoms with time; (2) associations between hazardous drinking, reduction in hazardous drinking and symptoms; and (3) longitudinal associations between symptoms and hazardous drinking. As we are interested in examining change over time for symptom and hazardous drinking outcomes and then identifying differences in rates of change (Raudenbush and Bryk, 2009), we constructed a series of mixed-effects growth models employing restricted maximum likelihood estimation for estimating the parameters of continuous outcomes. This approach to longitudinal data analysis is a form of hierarchical linear modeling for repeated measures data where multiple measurement occasions are nested within persons (Raudenbush and Bryk, 2009). We first constructed unconditional models predicting depression/anxiety measures from time (coded: 0=baseline; 1=3-months; 2=6-months) to examine the overall trajectory of participants' symptom levels over the follow-up. Subsequently, conditional growth models were constructed predicting symptom outcomes from time and time-varying hazardous drinking. To examine reduction in hazardous drinking on symptom outcomes, we computed growth models predicting symptom outcomes from time and time-varying Δ -hazardous drinking. Finally, to examine associations between symptoms and hazardous drinking outcome, conditional growth models were constructed predicting hazardous drinking from time and time-varying symptoms. Hazardous drinking was examined across all interviews as time-varying predictor and outcome. Conditional growth models included age, gender, MI as time-invariant covariates and psychiatry visits (medication management and/or psychotherapy visits) as a time-varying covariate. Rather than discard partial completers (~4.0% of the sample), the expectation maximization approach was used to handle missing data at the time of analysis. All variables were kept in their original distributions. Analyses were carried out in R version 2.14.2 (R Development Core Team, 2015) and HLM7 (Raudenbush et al., 2005). Statistical significance was defined at $p < .05$.

3. Results

3.1. Baseline characteristics, levels of hazardous drinking, and symptoms

Seventy percent were women and the average age was 37 ($SD=13.18$). Sixty percent reported hazardous drinking during baseline interviews and displayed a median of 1 (IQR: 0–4) hazardous drinking days in the prior month. Participants had moderate levels of anxiety ($M=10.81$; $SD=5.16$) and depression ($M=13.85$; $SD=5.58$). Further information on participants' characteristics can be found in supplementary Table S1.

3.2. Longitudinal associations between hazardous drinking, reduction in hazardous drinking, and symptoms

After finding relatively high anxiety and depressive symptom levels among the sample at baseline, we examined the longitudinal trajectories of these symptoms. Unconditional growth model results showed participants' anxiety ($B=-3.03$, $p<.001$) and depressive symptoms ($B=-5.39$, $p<.001$) improved (Table 1; Fig. 1). Hazardous drinking was associated with slower anxiety ($B=0.09$, $p=.005$) and depressive symptom ($B=0.10$, $p=.004$) improvement, whereas reductions in hazardous drinking was associated with faster improvement in anxiety ($B=-0.09$, $p=.010$) and depressive ($B=-0.10$, $p=.015$) symptoms. Neither the average effect of anxiety ($B=0.07$, $p=.066$) nor depressive ($B=0.05$, $p=.071$) symptoms were associated with hazardous drinking outcomes (not shown).

Post-hoc moderator analyses employing mixed-effects growth models explored if the described associations between hazardous drinking and symptoms varied in magnitude by the quantity of drinks patients consumed per occasion. Results showed no significant interactions between hazardous drinking and drinking quantity on anxiety ($B=0.01$, $p=.972$) or depression ($B=0.01$, $p=.489$). Similarly, we found no significant interactions between hazardous drinking reduction and drinking quantity reduction on anxiety ($B=-0.02$, $p=.111$) or depressive ($B=-0.03$, $p=.264$) symptoms (not shown).

4. Discussion

Studies have found that hazardous drinking leads to worse symptoms and *vice versa*, yet few conducted with clinical samples have investigated the association of reduction in hazardous consumption and symptoms over time. Therefore, we studied 307 psychiatric outpatients on their hazardous drinking patterns and reductions in hazardous consumption on anxiety and depression symptom recovery; and also explored the effect of symptoms on hazardous drinking outcomes over 6-months.

Results showed patients' symptoms were the highest at baseline, and then continued to improve (on average, symptoms decreased in severity) over time. Patients who consumed alcohol at hazardous levels experienced slower symptom recovery; those reducing hazardous drinking experienced faster symptom improvement. We found no evidence of a longitudinal relationship between symptoms and hazardous drinking outcomes.

4.1. Clinical implications

These findings have clinical implications. Our results confirm that hazardous drinking is a prevalent and symptom-destabilizing problem for psychiatry patients, and also indicate that reductions in hazardous consumption can help improve symptoms. It can be inferred that delivering evidence-based brief alcohol interventions (Baker et al., 2012; Satre et al., 2016) to psychiatry patients who consume alcohol at hazardous levels (and introducing alcohol screening measures to ensure hazardous drinking is detected) would have a beneficial effect in terms of accelerating symptom improvement beyond usual psychiatric care alone. Our findings may inform the development of interventions to reduce the health impact of co-occurring hazardous drinking, anxiety, and depression.

This work also provides further support that on-going efforts to improve education for psychiatry patients around alcohol use and hazardous drinking are important (Edlund et al., 2012). Educating patients with hazardous drinking, anxiety, and depression that cutting back on drinking may be useful to help alleviate symptom distress. Perhaps if patients with these co-occurring conditions were aware that ongoing psychiatric distress could be linked to alcohol consumption levels, they would be more likely to consider treatment options to reduce drinking.

4.2. Study limitations

Limitations should be noted. Because our sample was selected from an outpatient psychiatry setting, findings are not directly comparable to recent general population studies that have examined relationships between sub-syndromal symptoms and alcohol use (not problem level consumption) (Bell et al., 2015). Participants were in a randomized controlled trial of MI and had PHQ-9 depression ≥ 5 ; potentially further limiting generalizability. We adjusted for treatment effects and psychiatry visits, yet patients could have been participating in treatments we did not account for analytically; and thus, cause-and-effect determinations cannot be made. Hazardous drinking was skewed and may have biased the findings; however, robust methods were used and provide more sensitive tests under these conditions (Raudenbush and Bryk, 2009). We did not have a large enough sample to examine risky substance use other than alcohol. We used standardized measures and general population hazardous drinking levels; yet, further research will be needed to test effects in more generalizable samples to the recent literature on co-occurring hazardous drinking, anxiety, and depression.

5. Conclusions

We found that reducing hazardous drinking can improve symptoms but continued alcohol consumption at hazardous levels slows symptom recovery for psychiatry patients. Our findings suggest that it would be useful for psychiatry service providers to deliver alcohol screening and hazardous-drinking focused interventions when needed to patients with co-occurring anxiety, depression, and hazardous drinking.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jad.2016.07.039>.

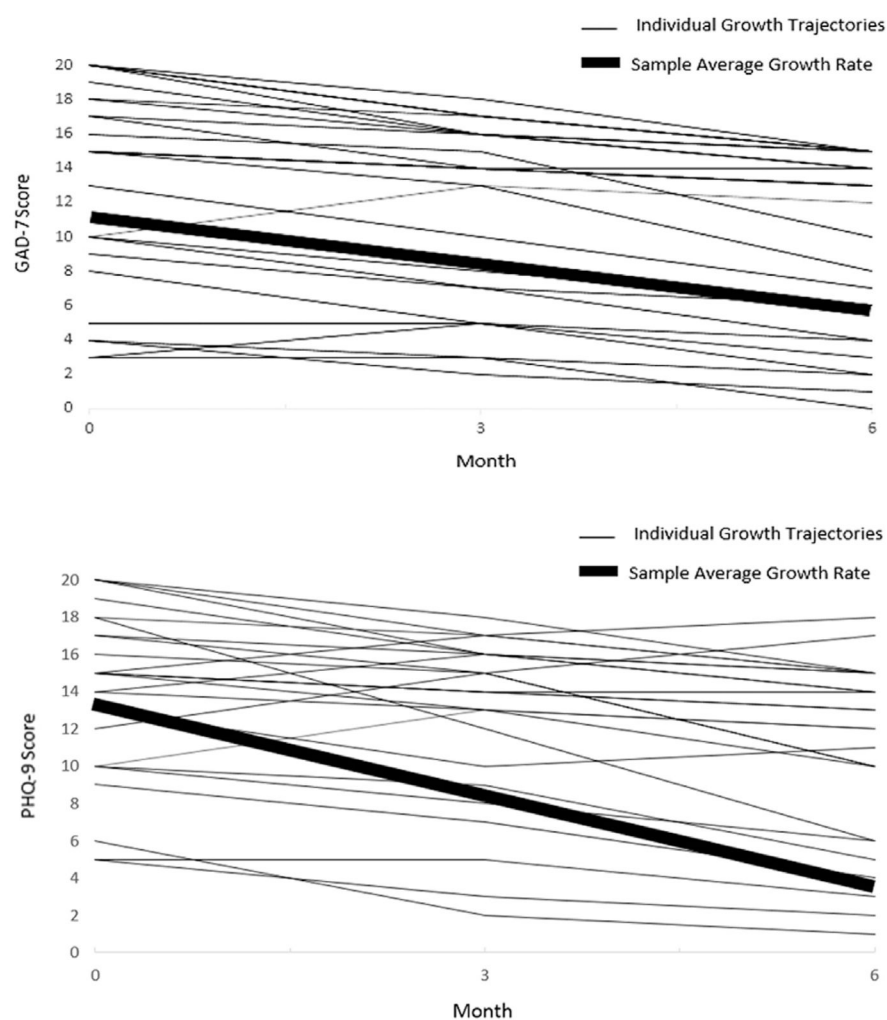


Fig. 1. Unconditional growth models showing the average improvement in symptoms for the sample (N=307) over 6-months, derived from individual growth trajectories (n=25 displayed) of participant's symptoms scores over the study duration.

Table 1

Longitudinal predictors of anxiety and depression symptoms for substance misusing psychiatry patients.

Variable	Depression ^a			Anxiety ^b				
	B	SE	t	p	B	SE	t	p
Unconditional growth model								
Time	-5.39	0.34	-15.70	<.001	-3.03	0.27	-10.92	<.001
Δ – Hazardous drinking conditional growth model ^c								
Age	-0.01	0.02	-0.43	.666	-0.00	0.02	0.00	.998
Female	2.20	0.68	3.20	<.001	1.15	0.65	1.76	.079
MI treatment	-0.27	0.63	-0.43	.663	-0.04	0.59	-0.06	.945
Time	-4.23	1.79	-2.35	.019	-2.92	1.50	-1.94	.053
Time×age	0.02	0.02	1.02	.305	-0.02	0.02	-1.16	.243
Time×female	-1.03	0.74	-1.39	.164	0.07	0.62	0.12	.903
Time × MI treatment	0.47	0.70	0.67	.501	-0.08	0.55	-0.15	.881
Psychiatry visits ^d	0.17	0.08	2.13	.033	0.06	0.07	0.91	.361
Δ – Hazardous drinking ^e	-0.10	0.04	-2.44	.015	-0.09	0.03	-2.56	.010
Hazardous drinking conditional growth model ^c								
Age	0.01	0.60	0.23	.816	-0.00	0.02	-2.22	.026
Female	2.43	0.66	3.67	<.001	1.27	0.63	-0.23	.810
MI treatment	0.26	0.60	0.43	.663	0.26	0.57	0.45	.648
Time	-1.88	0.60	-3.10	.002	-1.24	0.57	-2.22	<.001
Time×age	-0.01	0.01	-1.01	.986	-0.01	0.02	-0.22	.824
Time×female	-1.00	0.01	-2.67	<.001	-0.00	0.01	-0.12	.903
Time×MI treatment	-0.40	0.33	1.18	.237	-0.08	0.55	-0.50	.615
Psychiatry visits ^d	-0.07	0.05	-1.30	.193	0.03	0.04	-0.71	.475
Hazardous drinking ^f	0.10	0.03	2.84	.004	0.09	0.03	2.79	.005

B=beta coefficient; SE=standard error; t=t-ratio; p=p-values <.05 are presented in boldface; MI Treatment=motivation interviewing treatment condition.

^a PHQ-9 total score; higher scores indicate greater depression severity.

^b GAD-7 total score; higher scores indicate greater anxiety severity.

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C_e Conditional growth models were fit using restricted maximum likelihood estimation.
 d_p Psychiatry visits=time-varying covariate estimating the number of psychiatry visits prior to each interview.
 e Hazardous drinking=time-varying independent variable estimating average reductions in patient hazardous drinking ($\geq 4/\geq 5$ drinks in a day for women/men) days.
 f_j Hazardous drinking=time-varying independent variable estimating patient hazardous drinking ($\geq 4/\geq 5$ drinks in a day for women/men) days.