

Alcohol-Induced Amnesia and Personalized Drinking Feedback: Blackouts Predict Intervention Response

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Alcohol-induced amnesia (“blackout”) is a reliable predictor of alcohol-related harm. Given its association with other negative consequences, experience of alcohol-induced amnesia may serve as a teachable moment, after which individuals are more

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likely to respond to intervention. To test this hypothesis, alcohol-induced amnesia was evaluated as a moderator of brief intervention effect on (a) alcohol-related consequences and (b) the proposed intervention mediators, protective behavioral strategies and peak blood alcohol concentration (BAC). Baseline alcohol risk measured using the Alcohol Use Disorders Identification Test (AUDIT) was also evaluated as a moderator to rule out the possibility that amnesia is simply an indicator of more general alcohol risk. College students ($N = 198$) reporting alcohol use in a typical week completed assessments at baseline and 1-month follow-up as part of a larger intervention trial. Participants were randomized to assessment only (AO; $n = 58$) or personalized feedback intervention (PFI; $n = 140$). Hierarchical regression was used to examine direct and indirect intervention effects. A significant group-by-amnesia interaction revealed that only PFI participants who had experienced alcohol-induced amnesia in the past month reported decreases in alcohol consequences at 1-month follow-up. The PFI reduced alcohol-related consequences indirectly through changes in peak BAC, but only

among those who had experienced amnesia at baseline. In contrast, baseline alcohol risk (AUDIT) did not moderate intervention effects, and use of protective behavioral strategies did not statistically mediate intervention effects. Findings suggest that loss of memory for drinking events is a unique determinant of young adult response to brief alcohol intervention. Normative feedback interventions may be particularly effective for individuals who have experienced alcohol-induced amnesia in the past 30 days.

Keywords: blackout; moderator; normative feedback; college students; young adults

UP TO 66% OF COLLEGE STUDENTS report losing memory of events that occur while they are intoxicated (Marino & Fromme, 2015). This experience of alcohol-induced amnesia is one of the most commonly reported alcohol-related consequences among young adults who drink (Barnett et al., 2014; Chartier, Hesselbrock, & Hesselbrock, 2011; Hallett et al., 2013) and has been associated with outcomes from social regret to sexual assault (Mundt, Zakletskaia, Brown, & Fleming, 2012; Valenstein-Mah, Larimer, Zoellner, & Kaysen, 2015; Wilhite & Fromme, 2015). On large university campuses, the emergency medical care costs associated with intoxication that led to alcohol-induced amnesia are estimated to range from \$469,000 to \$546,000 per year (Mundt & Zakletskaia, 2012). Thus, increased understanding of alcohol-induced amnesia may inform both young adult health outcomes and community health care costs.

Alcohol-induced amnesia (sometimes referred to as a “blackout”) is a period of anterograde amnesia in which individuals cannot remember what happened while they were drinking, though immediate sensory and short-term memories and previously consolidated long-term memories remain intact (White, 2003). This type of amnesia was once considered a hallmark of alcohol use disorder (Jellinek, 1946); however, research revealed that alcohol-induced amnesia is also common among nondependent drinkers (White, Jamieson-Drake, & Swartzwelder, 2002). The prevalence of alcohol-induced amnesia among social drinkers has decreased the degree of concern evoked by a single “blackout” episode (White et al., 2002); yet the experience of alcohol-induced amnesia inherently involves some alcohol-related risk. First, the inability to retain memories for more than a few minutes may limit individuals’ awareness of the current environment and potential danger, placing them at risk for negative outcomes. Second, because high levels of intoxication are typically required to produce memory impairments (Tokuda, Izumi, & Zorumski, 2011), other negative consequences are likely to

occur. Moreover, rapid rise in blood alcohol concentration (BAC) may be more predictive of alcohol-induced amnesia than drinking quantity alone (Wetherill & Fromme, 2016; White, 2003). Thus, individuals who experience alcohol-induced amnesia are not only at increased risk for consequences, but may also have limited awareness of or ability to moderate BAC through use of protective behavioral strategies (e.g., avoiding shots, drinking water).

Given its association with other negative consequences, experience of alcohol-induced amnesia may serve as a teachable moment, after which individuals are more likely to respond to alcohol intervention. A number of researchers have suggested that “blackout” screening questions could be used to identify individuals at risk for alcohol-related consequences who, ostensibly, may benefit from intervention (Hingson, Zha, Simons-Morton, & White, 2016; Merrill et al., 2016; Mundt et al., 2012). Wilhite and Fromme (2015) took these recommendations a step further, suggesting that interventions emphasizing use of protective behavioral strategies may reduce alcohol problems among individuals with a history of alcohol-induced amnesia because use of these strategies may prevent rapid rise in BAC. However, studies to date have not examined whether drinkers with a history of alcohol-induced amnesia respond differently to intervention or the role that use of protective behavioral strategies and peak BAC may play in those outcomes.

Data from a randomized controlled trial (RCT) evaluating a brief computerized intervention for heavy alcohol use was re-analyzed to determine if and how recent experience of alcohol-induced amnesia moderates intervention efficacy. First, it was hypothesized that intervention participants reporting alcohol-induced amnesia in the past month would report greater decreases in alcohol-related consequences than those who did not experience alcohol-induced amnesia (i.e., alcohol-induced amnesia would moderate the direct intervention effect on alcohol-related consequences). Second, it was hypothesized that intervention participants reporting alcohol-induced amnesia in the past month would report greater increases in use of protective behavioral strategies and greater decreases in peak BAC, which would account for improvements in alcohol-related consequences (i.e., alcohol-induced amnesia would moderate the indirect intervention effect on consequences through protective behavioral strategies or peak BAC, consistent with a moderated mediation model). To determine the specificity of alcohol-induced amnesia, as opposed to general risk for alcohol problems, in identifying individuals who may benefit from brief interventions, individuals’ scores on the Alcohol Use Disorders Identification Test (Saunders, Aasland, Babor, de la

Fuente, & Grant, 1993) were also examined as a moderator of intervention efficacy.

Material and Methods

PARTICIPANTS AND PROCEDURE

College students were recruited from a large, 4-year, land grant university as part of an RCT examining the efficacy of three computerized personalized feedback interventions (PFIs) for heavy alcohol use. Details regarding study design and intervention content have been published elsewhere (Miller, Leavens, Meier, Lombardi, & Leffingwell, 2016). Briefly, students reporting alcohol use in a typical week were recruited primarily from a research participant pool to complete an online assessment of health behaviors. After providing informed consent, participants ($N = 212$) completed baseline measures online from remote locations. They were randomly assigned to receive (a) feedback comparing their typical drinking quantities and frequencies to those of same-sex students on campus (normative feedback; $n = 51$); (b) normative feedback in addition to feedback regarding practical costs of drinking and participant use of protective behavioral strategies ($n = 51$); (c) normative feedback in addition to feedback of their choice ($n = 51$); or (d) assessment only ($n = 59$). Participants in the PFI groups received their feedback via email within 48 hours of the baseline assessment and provided a read receipt to verify that they had read their feedback. One month after baseline, participants completed the follow-up assessment online from remote locations. All procedures were approved by the university's institutional review board, and a Certificate of Confidentiality was obtained to protect the confidentiality and integrity of data.

Of the 212 participants in the parent trial, 14 were excluded for missing data, resulting in a data analytic sample of 198 participants. Participants who were included versus excluded from analyses did not differ significantly at baseline in age, gender, drinks per week, experience of alcohol-induced amnesia, peak BAC, use of protective behavioral strategies, or experience of alcohol-related consequences. Because the three interventions piloted in the parent trial did not demonstrate differential efficacy in reducing alcohol use or consequences (Miller et al., 2016), intervention participants were collapsed into a single personalized feedback intervention group (PFI $n = 140$) and compared to the assessment only control group (AO $n = 58$).

MEASURES

Covariates

Participants reported their age, gender, weight, and race/ethnicity at baseline. The Daily Drinking Questionnaire (Collins, Parks, & Marlatt, 1985)

was included as a face valid measure of alcohol use at baseline. Using a 7-day grid, participants indicated how many drinks they had consumed on each day of a typical week in the past 30 days. Participants were provided with standard drink definitions (i.e., 12 oz beer, 8–9 oz malt liquor, 5 oz table wine, 1.5 oz distilled spirits) but were not asked to indicate the type of alcohol consumed. Responses were summed to calculate the number of drinks consumed in a typical week.

Primary Outcome

The Brief Young Adult Alcohol Consequences Questionnaire (BYAACQ) is a 24-item measure of alcohol-related consequences that has been validated for use in college student samples (Kahler, Hustad, Barnett, Strong, & Borsari, 2008; Kahler, Strong, & Read, 2005). At baseline and follow-up, participants indicated (*yes/no*) if they had experienced outcomes such as “taking foolish risks” or “passing out” as a result of drinking in the past 30 days. Because alcohol-induced amnesia was examined as a predictor of intervention outcome, the “blackout” item was removed from the BYAACQ at baseline and follow-up. Thus, possible scores ranged from 0 to 23. Internal consistency was high ($\alpha = .81$).

Moderators

The BYAACQ item, “In the past month, I have not been able to remember large stretches of time while drinking,” was used as a single-item measure of alcohol-induced amnesia in the past 30 days. The reliability and validity of this single item have not been studied; however, no psychometrically validated measure of alcohol-induced amnesia currently exists (Wetherill & Fromme, 2016).

The 10-item Alcohol Use Disorders Identification Test (AUDIT) has demonstrated validity in discriminating between individuals who engage in dangerous and nondangerous alcohol consumption across six nations (Saunders et al., 1993). Among college students, a cutoff score of 8 or more demonstrates sensitivity of 82% and specificity of 78% in identifying current high-risk drinking (Kokotailo et al., 2004). Three items assess the quantity and frequency of alcohol use in the past year, and seven items assess symptoms indicative of hazardous drinking (e.g., feeling guilt or remorse after drinking) on a scale from 0 (*never*) to 4 (*daily or almost daily*). Responses were summed, and individuals scoring 8 or more were categorized as screening positive for “alcohol risk.” A cutoff score, rather than the continuous measure, was used to be consistent with the dichotomous nature of the alcohol-induced amnesia item and to model the way in which this screening measure may be used in

primary care or treatment settings. Because one of the AUDIT items assesses alcohol-induced amnesia, analyses were run both including and excluding the “blackout” item from the measure. The pattern of outcomes was the same; thus, all items were retained in analyses to maintain the integrity of the cutoff score and, again, to represent the AUDIT as it would be used in clinic settings.

Mediators

The Protective Behavioral Strategies Scale (Martens et al., 2005; Martens, Pedersen, LaBrie, Ferrier-Auerbach, & Cimini, 2007) is a 15-item measure of behaviors aimed to reduce alcohol-related consequences while drinking (e.g., drinking slowly, avoiding drinking games). Participants indicated how frequently they had engaged in each behavior in the past 30 days on a scale from 1 (*never*) to 6 (*always*). This measure has demonstrated strong reliability and external validity (Martens et al., 2005, Martens et al., 2007). Internal consistency in this sample was good ($\alpha = .82$).

To assess peak BAC, participants indicated the number of drinks consumed and number of hours over which they were consumed on their heaviest drinking episode in the past 30 days. Widmark's (1932) formula, which takes into account volume

and duration of alcohol consumption as well as participant sex and weight, was used to calculate estimated peak BAC.

DATA SCREENING AND ANALYSIS PLAN

Data were screened for missing values, normality, baseline differences between groups (see Table 1), and multicollinearity (see Table 2) prior to analysis. Fourteen participants from the parent trial were excluded from analyses because they were missing more than 30% of data on the BYAACQ ($n = 10$) or AUDIT ($n = 4$). Skewness and kurtosis estimates for predictor and outcome variables were within the acceptable range (Tabachnick & Fidell, 2007). Intervention and control participants differed significantly in gender, alcohol-induced amnesia, and alcohol-related consequences at baseline; while amnesia/no amnesia groups differed in age and drinking variables (see Table 1). To account for these baseline group differences, all models controlled for age, gender, history of amnesia, and drinking variables at baseline. Linear regression was used to test for multicollinearity among the variables depicted in Table 2. Diagnostic statistics indicated moderate to high levels of tolerance (0.5–0.9) and low variance inflation (1.1–2.1) among variables, indicating that a substantial amount of variance in

Table 1
Group Differences in Demographic, Predictor, and Outcome Variables at Baseline ($N = 198$)

Variables	Total Sample ($N = 198$)	PFI ($n = 140$)	AO ($n = 58$)	χ^2 / t (df)	Amnesia ($N = 87$)	No Amnesia ($N = 111$)	χ^2 / t (df)
Age in years (<i>SD</i>)	19.9 (2.4)	20.1 (2.7)	19.6 (1.8)	-1.29 (196)	19.5 (1.6)	20.3 (2.9)	2.14 (196)*
Female (%)	118 (60%)	77 (55%)	41 (71%)	4.19 (1)*	55 (63%)	63 (57%)	0.85 (1)
Race (white vs. non-white)	—	—	—	0.14 (1)	—	—	0.22 (1)
Caucasian (%)	174 (88%)	122 (87%)	52 (90%)	—	77 (89%)	97 (87%)	—
Hispanic or Latino (%)	6 (3%)	4 (3%)	2 (3%)	—	3 (3%)	3 (3%)	—
African American (%)	4 (2%)	4 (3%)	0 (0%)	—	2 (2%)	2 (2%)	—
Asian American (%)	2 (1%)	1 (1%)	1 (2%)	—	0 (0%)	2 (2%)	—
American Indian / Alaskan Native (%)	6 (3%)	6 (4%)	0 (0%)	—	2 (2%)	4 (4%)	—
Bi/Multiracial (%)	5 (3%)	2 (1%)	3 (5%)	—	2 (2%)	3 (3%)	—
Not reported (%)	1 (1%)	1 (1%)	0 (0%)	—	1 (1%)	0 (0%)	—
Age of first drink (<i>SD</i>)	15.6 (2.3)	15.7 (2.2)	15.4 (2.4)	-0.71 (195)	15.2 (2.3)	15.9 (2.2)	2.38 (195)*
Drinks per week (<i>SD</i>)	16.4 (11.5)	17.0 (12.8)	15.0 (7.7)	-1.33 (171 [‡])	20.0 (13.4)	13.6 (8.9)	-3.81 (142 [‡])*
Drinking days per week (<i>SD</i>)	2.8 (1.1)	2.9 (1.1)	2.7 (1.0)	-1.16 (196)	3.0 (1.2)	2.7 (1.0)	-2.22 (165 [‡])*
Drinks per drinking day (<i>SD</i>)	5.9 (3.6)	6.0 (3.8)	5.7 (2.9)	-0.48 (140 [‡])	6.8 (4.2)	5.2 (2.8)	-3.02 (141 [‡])***
Amnesia (%)	87 (44%)	54 (39%)	33 (57%)	5.59 (1)*	87 (100%)	0 (0%)	—
AUDIT score (<i>SD</i>) ¹	10.4 (4.6)	10.1 (4.5)	11.2 (4.9)	1.56 (196)	12.8 (4.6)	8.5 (3.7)	-7.14 (163 [‡])*
AUDIT ≥ 8 (%)	154 (78%)	107 (76%)	47 (81%)	0.50 (1)	81 (93%)	73 (66%)	21.09 (1)***
Protective strategies (<i>SD</i>)	50.5 (12.3)	51.0 (12.3)	49.3 (12.4)	-0.86 (193)	48.1 (11.8)	52.3 (12.4)	2.41 (193)*
Peak BAC (<i>SD</i>)	.21 (.10)	.21 (.11)	.20 (.09)	-0.46 (196)	.25 (.10)	.18 (.10)	-4.91 (196)***
Alcohol consequences (<i>SD</i>) ¹	6.0 (4.2)	5.4 (4.0)	7.3 (4.4)	2.86 (196)**	8.3 (4.0)	4.1 (3.3)	-8.11 (196)***

Note. * $p < .05$, ** $p < .01$, *** $p < .001$. ¹Blackouts item was removed from total score. [‡]Equal variances not assumed. AO = assessment only. AUDIT = Alcohol Use Disorders Identification Test. BAC = blood alcohol concentration. Consequences = alcohol-related consequences. PFI = personalized feedback intervention. All outcome models controlled for between-group differences in age, gender, drinks per week, AUDIT, and levels of outcome variables at baseline.

Table 2
Zero-Order Correlations Among Study Variables ($N = 198$)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Age	–										
2. Female (vs. male)	-0.17*	–									
3. BL drinks	0.002	-0.42***	–								
4. BL consequences	-0.13	0.14	0.23**	–							
5. BL peak BAC	-0.16*	-0.02	0.54***	0.32***	–						
6. BL strategies	0.09	0.10	-0.38***	-0.24**	-0.34***	–					
7. PFI (vs. AO)	0.09	-0.15*	0.08	-0.20**	0.03	0.06	–				
8. AUDIT ≥ 8	-0.14	-0.14*	0.40***	0.47***	0.35***	-0.43***	-0.05	–			
9. Amnesia	-0.15*	0.07	0.28***	0.50***	0.33***	-0.17*	-0.17*	0.33***	–		
10. 1MO peak BAC	-0.14*	-0.02	0.39***	0.26***	0.38***	-0.34***	-0.16*	0.32***	0.21**	–	
11. 1MO strategies	0.09	0.15*	-0.22**	-0.25**	-0.10	0.56***	0.02	-0.31***	-0.17*	-0.26***	–
12. 1MO consequences	-0.07	0.02	0.22**	0.60***	0.21**	-0.33***	-0.31***	0.33***	0.23**	0.44***	-0.22**

Note. * $p < .05$. 1MO = 1 month. Amnesia = alcohol-induced amnesia in the past 30 days. AO = assessment only. AUDIT = Alcohol Use Disorders Identification Test. BAC = blood alcohol concentration. BL = baseline. Consequences = alcohol-related consequences. PFI = personalized feedback intervention. Strategies = protective behavioral strategies. Diagnostic tests for multicollinearity indicated moderate to high levels of tolerance (0.5-0.9) and low variance inflation (1.1-2.1) among variables.

each predictor was unaccounted for by the other predictors.

Primary outcome analyses were conducted in SAS 9.4. Hierarchical multiple regression was used to examine (a) alcohol-induced amnesia and (b) AUDIT score as moderators of intervention effect on alcohol-related consequences. Age, gender, baseline drinks per week, and baseline alcohol-related consequences were included in the model as covariates. In Step 1 of the model, covariates, group (PFI vs AO), baseline alcohol-induced amnesia (yes vs no), and baseline AUDIT score (yes vs no ≥ 8) were examined as predictors of alcohol-related consequences at 1-month follow-up. In Step 2, interactions evaluating alcohol-induced amnesia (Group X Amnesia) and alcohol risk (Group X AUDIT) as moderators of intervention effect were included in the model. Follow-up tests of simple slopes were conducted to determine the significance of the association between group and alcohol-related consequences at high and low levels of the moderator (Aiken & West, 1991; Cohen, Cohen,

West, & Aiken, 2003). High and low values of each moderator were specified as a positive versus negative alcohol-induced amnesia or AUDIT screen, respectively. Predictor variables were mean centered to aid in interpretation of outcomes.

In the case of significant moderation, exploratory analyses were conducted, examining alcohol-induced amnesia as a moderator of the indirect intervention effect on alcohol-related consequences through (a) protective behavioral strategies or (b) peak BAC. These variables were modeled separately as mediators of intervention effect. Analyses were conducted using PROCESS Model 7 for moderated mediation, as depicted in Figure 1 (Hayes, 2013). Although the limitation of only two assessment time points does not allow for true tests of mediation (accounting for temporal precedence of mediator and outcome variables), these analyses were conducted to quantify indirect intervention effects, in hopes of informing future research. The PROCESS program utilizes standard errors and bootstrap confidence intervals

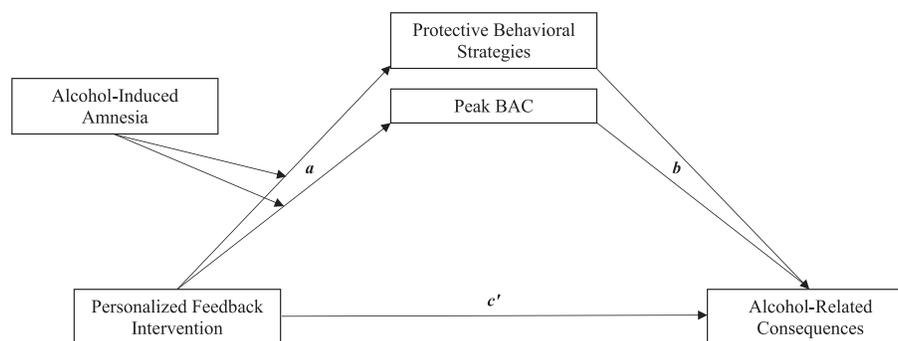


FIGURE 1 Conceptual model for moderated mediation analyses

(CIs) to estimate indirect effects. The macro computes the indirect path from *a* to *b* using the *ab* product term approach, with bootstrapped 95% asymmetric CIs (Hayes, 2013; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). This method of analysis was chosen because bootstrapping limits assumptions and Type I error while increasing power to detect effects in small samples (Fritz, Taylor, & MacKinnon, 2012; Hayes & Scharkow, 2013). Although modeled simultaneously, the macro provides information to estimate (a) if alcohol-induced amnesia moderated intervention effect on protective behavioral strategies or peak BAC, (b) if improvement in protective behavioral strategies or peak BAC statistically mediated intervention effect on alcohol-related consequences, and (c) if alcohol-induced amnesia moderated this statistically mediated effect.

Results

DESCRIPTIVE STATISTICS

Participant characteristics are depicted in Table 1, and Pearson correlation coefficients for study variables are presented in Table 2. Participants were 4-year college students ($N = 198$, 60% women) reporting alcohol use in a typical week. Of the 87 participants (44%) who reported alcohol-induced amnesia in the past month, 93% indicated harmful or hazardous drinking on the AUDIT (cutoff ≥ 8). Conversely, of the 154 participants (78%) who indicated harmful or hazardous drinking on the AUDIT, 53% reported alcohol-induced amnesia in the past month.

MODERATED EFFECTS

Direct Intervention Effects

Main effects and interaction terms are presented in Table 3, and descriptive statistics for each outcome are depicted in Table 4. Consistent with hypotheses, the interaction between group and alcohol-induced amnesia was a significant predictor of alcohol-related consequences at 1-month follow-up ($B = -3.33$, $SE = 1.28$, $p = .01$). The interaction between group and alcohol risk measured using the AUDIT was not significant ($B = -0.67$, $SE = 1.58$, $p = .67$).¹ Tests of simple slopes indicated that individuals who experienced alcohol-induced amnesia in the past month demonstrated a decrease in alcohol-related consequences as a result of the intervention ($B = -3.54$, $SE = 1.69$, $p = .03$), while those who denied past-month alcohol-induced amnesia did not ($B = -0.21$, $SE = 1.35$, $p = .88$; see Figure 2).

¹ The significance of the Group X Amnesia interaction and nonsignificance of Group X AUDIT interaction did not change when examining full-scale continuous AUDIT scores or dichotomous AUDIT scores based on a cutoff of 15.

Table 3

Main Effects and Interaction Terms in the Prediction of Alcohol-Related Consequences at 1-Month Follow-Up ($N = 198$)

	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>
<i>Step 1: Main Effects</i>				
Intercept	1.21	2.60	0.47	.64
Age	0.02	0.11	0.19	.85
Female (vs. male)	-0.25	0.64	-0.39	.70
BL drinks per week	0.05	0.03	1.58	.11
BL consequences	0.68	0.08	8.16	< .001
PFI (vs. AO)	-2.47	0.62	-3.99	< .001
AUDIT ≥ 8	0.62	0.79	0.78	.44
Amnesia	-1.42	0.65	-2.17	.03
<i>Step 2: Interaction</i>				
Group \times AUDIT	-0.67	1.58	-0.42	.67
Group \times Amnesia	-3.33	1.28	-2.60	.01

Note. Amnesia = alcohol-induced amnesia in the past 30 days. AO = assessment only. AUDIT = Alcohol Use Disorders Identification Test. PFI = personalized feedback intervention.

Indirect Intervention Effects

A moderated mediation model was conducted to determine whether alcohol-induced amnesia moderated an indirect intervention effect on alcohol-related consequences through protective behavioral strategies. Group did not have a significant effect on use of protective behavioral strategies at one-month follow-up ($a = -0.75$, $SE = 2.97$, $p = .80$), and past-month alcohol-induced amnesia did not moderate this effect ($B = 0.09$, $SE = 4.07$, $p = .98$). Inclusion in the PFI group was associated with fewer alcohol-related consequences at 1-month follow-up ($c' = -2.34$, $SE = 0.63$, $p < .001$), but increased use of protective behavioral strategies at 1-month follow-up was not ($b = 0.01$, $SE = 0.02$, $p = .62$). There was no indirect group effect on alcohol-related consequences through use of protective behavioral strategies among those who did ($ab = -0.01$, $SE = 0.10$, 95% CI = -0.29, 0.13) or did not ($ab = -0.01$, $SE = 0.08$, 95% CI = -0.27, 0.12) experience alcohol-related amnesia at baseline.

A second moderated mediation model was conducted to determine whether alcohol-induced amnesia moderated an indirect intervention effect on alcohol-related consequences through peak BAC. Similar to the model for protective behavioral strategies, group did not have a significant effect on peak BAC at 1-month follow-up ($a = -0.01$, $SE = 0.02$, $p = .73$), and past-month alcohol-induced amnesia only marginally moderated this effect ($B = -0.05$, $SE = 0.03$, $p = .05$). Inclusion in the PFI group ($c' = -1.80$, $SE = 0.60$, $p < .01$) and decreases in peak BAC ($b = 15.72$, $SE = 3.07$, $p < .001$) were associated with fewer alcohol-related consequences at 1-month follow-up. There was an indirect group

Table 4
Descriptive Statistics for Outcomes Among Men and Women in the Intervention ($n = 140$) and Control ($n = 58$) Conditions

Consequences ¹	Total ($N = 198$)				Men ($n = 80$)				Women ($n = 118$)			
	n	BL	1mo	Δ	n	BL	1mo	Δ	n	BL	1mo	Δ
Amnesia/PFI	54	7.9 (4.3)	4.0 (4.4)	-3.9	21	7.7 (4.4)	4.6 (4.8)	-3.1	33	8.1 (4.3)	3.6 (4.2)	-4.5
No Amnesia/PFI	86	3.9 (2.8)	3.2 (3.8)	-0.7	42	3.3 (2.6)	3.2 (3.8)	-0.1	44	4.4 (2.9)	3.1 (3.9)	-1.3
Amnesia/AO	33	8.9 (3.4)	8.6 (5.6)	-0.3	11	7.7 (3.0)	9.4 (6.2)	+1.7	22	9.6 (3.5)	9.4 (6.2)	-0.2
No Amnesia/AO	25	5.0 (4.6)	4.5 (5.6)	-0.5	6	6.2 (6.0)	6.0 (8.0)	-0.2	19	4.7 (4.1)	4.0 (4.8)	-0.7
Peak BAC												
Amnesia/PFI	54	.25 (.10)	.17 (.11)	-0.08	21	.29 (.10)	.21 (.11)	-0.08	33	.23 (.10)	.14 (.10)	-0.09
Amnesia/AO	33	.24 (.09)	.22 (.09)	-0.02	11	.22 (.19)	.19 (.11)	-0.03	22	.25 (.07)	.23 (.08)	-0.02
No Amnesia/PFI	86	.19 (.10)	.14 (.09)	-0.05	42	.18 (.10)	.14 (.10)	-0.04	44	.20 (.10)	.15 (.09)	-0.05
No Amnesia/AO	25	.16 (.08)	.15 (.06)	-0.01	6	.17 (.05)	.16 (.05)	-0.01	19	.15 (.09)	.15 (.06)	.00
Strategies												
Amnesia/PFI	51	49.3 (12.8)	51.6 (15.6)	+2.3	20	45.5 (14.8)	46.8 (12.4)	+1.3	31	51.7 (10.9)	54.7 (16.8)	+3.0
Amnesia/AO	32	46.4 (10.4)	49.9 (13.2)	+3.5	11	49.0 (8.4)	52.7 (12.2)	+3.7	21	45.1 (11.3)	48.4 (13.8)	+3.3
No Amnesia/PFI	81	53.2 (14.4)	57.4 (16.0)	+4.2	40	51.5 (11.5)	54.2 (13.3)	+2.7	41	53.1 (12.5)	57.3 (15.1)	+4.2
No Amnesia/AO	23	52.3 (12.0)	55.7 (14.2)	+3.4	5	42.6 (15.0)	37.6 (13.8)	-5.0	18	56.1 (13.2)	62.9 (11.8)	+6.8

Note. Δ = mean change, not accounting for baseline group differences or other variables in the model. ¹Blackouts item removed from total score. Amnesia = alcohol-induced amnesia. AO = assessment only. BAC = blood alcohol concentration. Consequences = alcohol-related consequences. PFI = personalized feedback intervention. Strategies = protective behavioral strategies.

effect on alcohol-related consequences through peak BAC among those who did experience alcohol-related amnesia at baseline ($ab = -0.95$, $SE = 0.38$, 95% CI = -1.85, -0.30). This indirect group effect was not significant among those who denied alcohol-induced amnesia at baseline ($ab = -0.11$, $SE = 0.24$, 95% CI = -0.60, 0.36). In other words, only among those with a recent history of alcohol-induced amnesia, participation in the PFI was associated with decreased peak BAC, which in turn was associated with fewer alcohol-related consequences.

POST-HOC ANALYSES

Five additional models were conducted to determine the specificity of alcohol-induced amnesia as a

moderator of intervention effect in comparison to other alcohol-related consequences. For each of these models, the moderating item was removed from the total BYAACQ score in order to avoid confounds between predictor and outcome variables (e.g., in models examining “driven a car” as a moderator, the “driven a car” item was removed from the total BYAACQ score at baseline and follow-up). Because running 24 separate models (one for each item of the BYAACQ) would inflate Type I error, the three consequences rated as “most negative” among college students according to Merrill et al. (2013) were examined: “I have driven a car when I knew I had had too much to drive safely” ($B = -1.19$, $SE = 1.71$, $p = .49$); “my drinking has created problems between myself and my boyfriend/

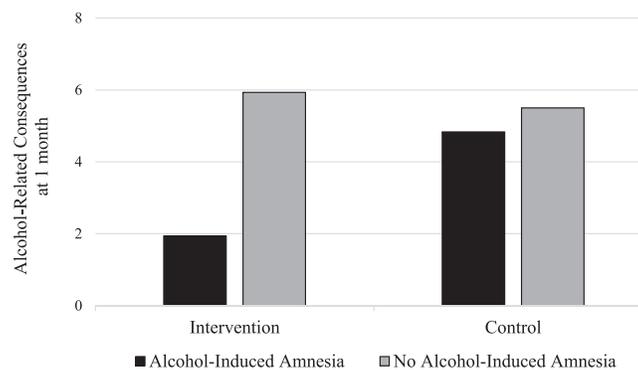


FIGURE 2 Group by alcohol-induced amnesia interaction on alcohol-related consequences at one month, adjusted for other variables in the model

girlfriend/spouse or parent” ($B = -1.29, SE = 1.76, p = .46$); and “the quality of my work or school work has suffered because of my drinking” ($B = -1.27, SE = 1.74, p = .47$). None of these items significantly moderated intervention effect on alcohol-related consequences. However, only 11% to 14% of participants endorsed these consequences.

To ensure that the nonsignificance of these models was not due to floor effects, a random number generator (random.org; numbers 1–24 for each item of the BYAACQ) was used to identify two additional moderators that were more common in this sample. The first two numbers that corresponded to items endorsed by $\geq 20\%$ of the sample were 23 and 2, which correspond to the BYAACQ items, “My physical appearance has been harmed by my drinking” (endorsed by 25%) and, “I have had a hangover the morning after I was drinking” (endorsed by 64%). Neither “physical appearance” ($B = -1.25, SE = 1.76, p = .48$) nor “hangover” ($B = -1.23, SE = 1.72, p = .47$) moderated intervention effect on alcohol-related consequences.

Discussion

Research in the last 50 years has examined the mechanisms and consequences of alcohol-induced amnesia (Wetherill & Fromme, 2016). However, the impact of this form of memory loss on future behavior and its implications for interventions have been understudied. This is the first study to examine alcohol-induced amnesia as a moderator of intervention effect on alcohol use outcomes. Findings suggest that personalized feedback interventions are associated with decreases in alcohol-related consequences (e.g., feeling sick, saying or doing embarrassing things) among individuals who have experienced alcohol-induced amnesia in the past month, due in part to intervention effect on peak BAC among these individuals. This moderation effect was observed independent of alcohol risk measured using the AUDIT, and this more general measure of hazardous drinking did not produce the same moderating effect on intervention outcomes. Thus, alcohol-induced amnesia seems to be a unique predictor of young adults’ response to personalized feedback interventions and may serve as an indicator of individuals for whom personalized feedback would be an effective intervention.

Individuals who had experienced alcohol-induced amnesia in the past 30 days reported greater decreases in alcohol-related consequences in response to personalized feedback than those who had not experienced alcohol-induced amnesia. The fact that more general alcohol risk did not produce a similar moderation effect suggests that there is something unique about alcohol-induced amnesia—beyond

heavy drinking—that makes these individuals particularly responsive to personalized alcohol feedback. We speculate that, as a result of memory loss, individuals who experience alcohol-induced amnesia feel less control over their behavior while drinking, and additional feedback indicating that they are consuming more alcohol than their peers is enough to prompt behavior change. This would be consistent with the Health Belief Model (Strecher, Champion, & Rosenstock, 1997), which posits that behavior change occurs in part as a function of perceived threat and cues to action. In this case, alcohol-induced amnesia may serve as a threat (susceptibility to memory loss and questionable control over behavior while “blacked out”), and personalized feedback may serve as a cue to change behavior. While it is possible that other negatively valenced consequences also serve as “threats” that prime individuals for behavior change, the other consequences examined in this study did not moderate intervention effect. The nonsignificance of some of these findings may be due to low prevalence rates; thus, future research examining these moderating effects in adequately powered samples is encouraged. However, the authors believe that alcohol-induced amnesia is a consequence of special concern because it is prevalent among young adults, it is strongly associated with other negative drinking outcomes, and “blackout” items are already included on widely used clinical screening tools (such as the AUDIT). Thus, screening for individuals who report a recent history of alcohol-induced amnesia may help identify individuals who will respond to intervention.

While the pronounced benefit of personalized feedback interventions for individuals who have recently experienced alcohol-induced amnesia is promising, this differential intervention effect was not attributable to increased use of protective behavioral strategies. This finding is especially curious, since intervention participants who had experienced alcohol-induced amnesia did report decreases in peak BAC, which implies that participants were moderating alcohol intake. It is possible that this lack of effect was due in part to measurement error in the protective behavioral strategies scale. Specifically, the protective behavioral strategies feedback provided to intervention participants in this study included a number of strategies that were not assessed in the outcome measure (e.g., eat before/during drinking, start drinking later in the day, avoid mixing alcohol and other drugs, choose drinks that are a lower proof, play drinking games with water). Thus, it is possible that intervention participants increased these behaviors as a result of the intervention and these changes were not captured in the protective behavioral strategies outcome measure. This

hypothesis is supported by the fact that research published after the initiation of this trial added five items to the scale to improve its content validity (Treloar, Martens, & McCarthy, 2015).

CLINICAL IMPLICATIONS

Nearly half of the participants in this sample reported losing memory of events that occurred while they were drinking in the past 30 days. This is consistent with previous studies, which typically report college prevalence rates around 50% (Wetherill & Fromme, 2016). The widespread occurrence of alcohol-induced amnesia among college students is concerning, as loss of memory indicates neurocognitive impairment and a level of alcohol intoxication that elevates risk of other negative outcomes (Hingson et al., 2016). Collectively, findings suggest that screening for alcohol-induced amnesia may help identify individuals who would benefit from brief personalized feedback comparing their alcohol use to that of their peers. Focus on amnesia—as opposed to drinking quantity—may increase the specificity of current screening and intervention efforts because a number of young adults who screen positive for heavy drinking may not experience alcohol problems and/or respond to intervention. As an example, 78% of participants screened positive for harmful or hazardous drinking on the AUDIT, but only 53% of those reported alcohol-induced amnesia in the past month. Results of this study suggest that providers should focus intervention efforts on the subset who endorsed amnesia. Thus, health care providers are encouraged to ask young adults about their experience of alcohol-induced amnesia in the past 30 days when screening for heavy alcohol use in clinic settings and to capitalize on this opportunity by providing an evidence-based intervention, if appropriate. Notably, only 51 (36%) of intervention participants in this trial received feedback on protective behavioral strategies; thus, interventions may not need to include feedback on protective behavioral strategies to have this beneficial effect.

LIMITATIONS

The randomized clinical trial design of this study was ideal for examination of differential PFI efficacy among a subset of drinkers who may be at unique risk for alcohol-related consequences. However, study design was limited in several ways. First, participants in the intervention group received one of three different feedback profiles (normative feedback, normative feedback in addition to feedback on practical costs and protective behavioral strategies, or normative feedback in addition to feedback of their choice), but were collapsed into one group for the present study. It is possible that there were

differential mediation or moderation effects as a result of the type of feedback received; however, outcomes at 1-month follow-up did not differ as a function of feedback content (Miller et al., 2016). Second, mediator and outcome variables were assessed at the same time point, which prevents us from stating with certainty that decreases in peak BAC preceded change in alcohol-related consequences. Future research establishing temporal precedence and causal associations between these variables is encouraged. Finally, data were collected via self-report, which is prone to inconsistencies with objective measures of alcohol (Kraus et al., 2005). However, biomarkers of alcohol use and self-reported drinking outcomes are strongly correlated ($r = 0.76$; Leffingwell et al., 2013). Moreover, primary outcomes were change in alcohol use (as opposed to absolute alcohol use values), in which case outcome estimates may be accurate if under-/overestimations of alcohol use are consistent within individuals over time.

Two additional limitations were not expected a priori. First, the protective behavioral strategies scale utilized in the current study may have had limited content validity, as it has been updated since completion of study enrollment (Treloar et al., 2015). In addition, a higher percentage of women were included in the control than intervention group. While gender was not significantly associated with endorsement of amnesia (the primary moderator) or alcohol-related consequences (the primary dependent variable), gender differences in rates of alcohol metabolism and, potentially, neurobiological vulnerability to alcohol may lead to differential rates of alcohol-induced amnesia among women and men (Mumenthaler, Taylor, O'Hara, & Yesavage, 1999; Sharrett-Field, Butler, Reynolds, Berry, & Prendergast, 2013). While some research has failed to find gender differences in rates of alcohol-induced amnesia (Marino & Fromme, 2015; Merrill et al., 2016; Mundt et al., 2012), other research has found gender differences (Schuckit, Smith, Goncalves, & Anthenelli, 2016). Gender was included as a covariate in the models detailed in this paper; however, future studies examining gender differences in the association between alcohol-induced amnesia and consequences are encouraged.

CONCLUSIONS

Personalized alcohol feedback is associated with decreases in alcohol problems among college students, likely due to its influence on peak BAC; however, this effect is only significant among individuals who have experienced alcohol-induced amnesia in the past 30 days. Alcohol-induced amnesia—but not alcohol risk measured using the AUDIT—moderated intervention effect on alcohol-related consequences.

Thus, there is something unique about loss of memory for drinking events that enhances response to the personalized feedback interventions. Given the prevalence of alcohol-induced amnesia among college students and its association with other negative outcomes, personalized feedback interventions are not only appropriate but also may be particularly effective for individuals who have experienced alcohol-induced amnesia in the past 30 days.

Glossary

Alcohol-induced amnesia A period of anterograde amnesia in which individuals cannot remember what happened while they were drinking, though immediate sensory and short-term memories and previously consolidated long-term memories remain intact. Also referred to as alcohol-induced “blackout.”

Alcohol risk Risk for harmful or hazardous drinking, measured using the Alcohol Use Disorders Identification Test (AUDIT).

Personalized feedback intervention A single-session intervention for heavy alcohol use that incorporates feedback on an individual’s drinking behavior (e.g., how much or how often one drinks in comparison to peers, how many calories one consumes in alcohol, how often one uses a particular protective behavioral strategy).

Protective behavioral strategies Behaviors aimed to reduce the consequences associated with heavy alcohol use (e.g., drinking water between drinks, avoiding drinking games).

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

References

- Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks, CA: Sage Publications.
- Barnett, N. P., Clerkin, E. M., Wood, M., Monti, P. M., Tevyaw, T. O. L., Corriveau, D., . . . Kahler, C. W. (2014). Description and predictors of positive and negative alcohol-related consequences in the first year of college. *Journal of Studies on Alcohol and Drugs*, 75(1), 103–114.
- Chartier, K. G., Hesselbrock, M. N., & Hesselbrock, V. M. (2011). Alcohol problems in young adults transitioning from adolescence to adulthood: The association with race and gender. *Addictive Behaviors*, 36, 167–174. <https://doi.org/10.1016/j.addbeh.2010.10.007>
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences (3rd ed.)*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Collins, R. L., Parks, G. A., & Marlatt, G. A. (1985). Social determinants of alcohol consumption: The effects of social interaction and model status on the self-administration of alcohol. *Journal of Consulting and Clinical Psychology*, 53, 189–200. <https://doi.org/10.1037/0022-006X.53.2.189>
- Fritz, M. S., Taylor, A. B., & MacKinnon, D. P. (2012). Explanation of two anomalous results in statistical mediation analysis. *Multivariate Behavioral Research*, 47, 61–87. <https://doi.org/10.1080/00273171.2012.640596>
- Hallett, J., Howat, P., McManus, A., Meng, R., Maycock, B., & Kypril, K. (2013). Academic and personal problems among Australian university students who drink at hazardous levels: web-based survey. *Health Promotion Journal of Australia*, 24, 170–177.
- Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York, NY: Guilford Press.
- Hayes, A. F., & Scharkow, M. (2013). The relative trustworthiness of inferential tests of the indirect effect in statistical mediation analysis: Does method really matter? *Psychological Science*, 24, 1918–1927. <https://doi.org/10.1177/0956797613480187>
- Hingson, R. W., Zha, W., Simons-Morton, B., & White, A. (2016). Alcohol-induced blackouts as predictors of other drinking related harms among emerging young adults. *Alcoholism: Clinical and Experimental Research*, 40, 776–784. <https://doi.org/10.1111/acer.13010>
- Jellinek, E. M. (1946). Phases in the drinking history of alcoholics: Analysis of a survey conducted by the official organ of Alcoholics Anonymous. *Quarterly Journal of Studies on Alcohol*, 7, 1–88. <https://doi.org/10.15288/QJSA.1946.7.1>
- Kahler, C. W., Hustad, J., Barnett, N. P., Strong, D. R., & Borsari, B. (2008). Validation of the 30-day version of the Brief Young Adult Alcohol Consequences Questionnaire for use in longitudinal studies. *Journal of Studies on Alcohol and Drugs*, 69, 611–615. <https://doi.org/10.15288/jsad.2008.69.611>
- Kahler, C. W., Strong, D. R., & Read, J. P. (2005). Toward efficient and comprehensive measurement of the alcohol problems continuum in college students: The Brief Young Adult Alcohol Consequences Questionnaire. *Alcoholism: Clinical and Experimental Research*, 29, 1180–1189. <https://doi.org/10.1097/01.alc.0000171940.95813.a5>
- Kokotailo, P. K., Judith, E., Gangnon, R., Brown, D. D., Mundt, M. P., & Fleming, M. (2004). Validity of the Alcohol Use Disorders Identification Test in college students. *Alcoholism: Clinical and Experimental Research*, 28, 914–920. <https://doi.org/10.1097/01.ALC.0000128239.87611.F5>
- Kraus, C. L., Salazar, N. C., Mitchell, J. R., Florin, W. D., Guenther, B., Brady, D., Swartzwelder, S. H., & White, A. M. (2005). Inconsistencies between actual and estimated blood alcohol concentrations in a field study of college students: Do students really know how much they drink? *Alcoholism: Clinical and Experimental Research*, 29, 1672–1676. <https://doi.org/10.1097/01.alc.0000179205.24180.4a>
- Leffingwell, T. R., Cooney, N. J., Murphy, J. G., Luczak, S., Rosen, G., Dougherty, D. M., & Barnett, N. P. (2013). Continuous objective monitoring of alcohol use: Twenty-first century measurement using transdermal sensors. *Alcoholism: Clinical and Experimental Research*, 37(1), 16–22. <https://doi.org/10.1111/j.1530-0277.2012.01869.x>
- MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods*, 7, 83–104.
- Marino, E. N., & Fromme, K. (2015). Alcohol-induced blackouts and maternal family history of problematic

- alcohol use. *Addictive Behaviors*, 45, 201–206. <https://doi.org/10.1016/j.addbeh.2015.01.043>
- Martens, M. P., Ferrier, A. G., Sheehy, M. J., Corbett, K., Anderson, D. A., & Simmons, A. (2005). Development of the Protective Behavioral Strategies Survey. *Journal of Studies on Alcohol*, 66, 698–705.
- Martens, M. P., Pedersen, E. R., LaBrie, J. W., Ferrier-Auerbach, A. G., & Cimini, M. D. (2007). Measuring alcohol-related protective behavioral strategies among college students: Further examination of the Protective Behavioral Strategies Scale. *Psychology of Addictive Behaviors*, 21(307-315). <https://doi.org/10.1037/0893-164X.21.3.307>
- Merrill, J. E., Read, J. P., & Barnett, N. P. (2013). The way one thinks affects the way one drinks: Subjective evaluations of alcohol consequences predict subsequent changes in drinking behavior. *Psychology of Addictive Behaviors*, 27, 42–51. <https://doi.org/10.1037/a0029898>
- Merrill, J. E., Treloar, H., Fernandez, A., Monnig, M. A., Jackson, K. M., & Barnett, N. P. (2016). Latent growth classes for alcohol-related blackouts among college student drinkers. *Psychology of Addictive Behaviors*, 30, 827–837.
- Miller, M. B., Leavens, E. L., Meier, E., Lombardi, N., & Leffingwell, T. R. (2016). Enhancing the efficacy of computerized feedback interventions for college alcohol misuse: An exploratory randomized trial. *Journal of Consulting and Clinical Psychology*, 84, 122–133. <https://doi.org/10.1037/ccp0000066>
- Mumenthaler, M. S., Taylor, J. L., O'Hara, R., & Yesavage, J. A. (1999). Gender differences in moderate drinking effects. *Alcohol Research & Health*, 23, 55–64.
- Mundt, M. P., & Zakletskaia, L. I. (2012). Prevention for college students who suffer alcohol-induced blackouts could deter high-cost emergency department visits. *Health Affairs*, 31, 863–870. <https://doi.org/10.1377/hlthaff.2010.1140>
- Mundt, M. P., Zakletskaia, L. I., Brown, D. D., & Fleming, M. F. (2012). Alcohol-induced memory blackouts as an indicator of injury risk among college drinkers. *Injury Prevention*, 18, 44–49. <https://doi.org/10.1136/ip.2011.031724>
- Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. II. *Addiction*, 88, 791–804. <https://doi.org/10.1111/j.1360-0443.1993.tb02093.x>
- Schuckit, M. A., Smith, T. L., Goncalves, P. D., Anthenelli, R. (2016). Alcohol-related blackouts across 55 weeks of college: Effects of European-American ethnicity, female sex, and low level of response to alcohol. *Drug and Alcohol Dependence*, 169, 163–170.
- Sharrett-Field, L., Butler, T. R., Reynolds, A. R., Berry, J. N., & Prendergast, M. A. (2013). Sex differences in neuroadaptation to alcohol and withdrawal neurotoxicity. *Pflugers Archiv: European Journal of Physiology*, 465, 643–654. <https://doi.org/10.1007/s00424-013-1266-4>.
- Strecher, V. J., Champion, V. L., & Rosenstock, I. M. (1997). The health belief model and health behavior. In D. S. Gochman & D. S. Gochman (Eds.), *Handbook of Health Behavior Research 1: Personal and Social Determinants* (pp. 71–91). New York, NY: Plenum Press.
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using Multivariate Statistics* (5th ed.). New York, NY: Haper and Row.
- Tokuda, K., Izumi, Y., & Zorumski, C. F. (2011). Ethanol enhances neurosteroidogenesis in hippocampal pyramidal neurons by paradoxical NMDA receptor activation. *The Journal of Neuroscience*, 31, 9905–9909. <https://doi.org/10.1523/JNEUROSCI.1660-11.2011>
- Treloar, H., Martens, M. P., & McCarthy, D. M. (2015). The Protective Behavioral Strategies Scale-20: Improved content validity of the serious harm reduction subscale. *Psychological Assessment*, 27, 340–346. <https://doi.org/10.1037/pas0000071>
- Valenstein-Mah, H., Larimer, M., Zoellner, L., & Kaysen, D. (2015). Blackout drinking predicts sexual revictimization in a college sample of binge-drinking women. *Journal of Traumatic Stress*, 28, 484–488. <https://doi.org/10.1002/jts.22042>
- Wetherill, R. R., & Fromme, K. (2016). Alcohol-induced blackouts: A review of recent clinical research with practical implications and recommendations for future studies. *Alcoholism: Clinical and Experimental Research*, 40, 922–935. <https://doi.org/10.1111/acer.13051>
- White, A. M. (2003). What happened? Alcohol, memory blackouts, and the brain. *Alcohol Research & Health*, 27, 186–196.
- White, A. M., Jamieson-Drake, D. W., & Swartzwelder, H. S. (2002). Prevalence and correlates of alcohol-induced blackouts among college students: Results of an e-mail survey. *Journal of American College Health*, 51, 117–119. <https://doi.org/10.1080/07448480209596339>
- Widmark, E. M. P. (1932). Die theoretischen Grundlagen and die praktische Verwendbarkeit der gerichtlich-medizinischen Alkoholbestimmung. *Journal of the American Medical Association*, 98, 1834. <https://doi.org/10.1001/jama.1932.02730470056035>
- Wilhite, E. R., & Fromme, K. (2015). Alcohol-induced blackouts and other negative outcomes during the transition out of college. *Journal of Studies on Alcohol and Drugs*, 76, 516–524. <https://doi.org/10.15288/jsad.2015.76.516>

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