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Alcohol-induced blackouts and maternal family history of problematic alcohol use

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Abstract

Background—Consequences of heavy drinking include alcohol-induced blackouts, which are periods of amnesia for all or part of a drinking event. One risk factor for blackouts is family history of problematic alcohol use (FH+); however, research rarely distinguishes maternal from paternal FH+. The objective of this study was to examine whether maternal or paternal FH+ better predicts likelihood of experiencing blackouts than a general measure of overall FH+, and whether gender moderates this association.

Method—Participants (N = 1,164; 65.4% Female) were first-time college freshman (age range = 17–19) who participated in a 6-year, 10-assessment, longitudinal study in the United States. Alcohol-induced blackouts, the dependent measure, were dichotomized (yes/no) based on endorsement of memory problems after drinking using a single item during Years 4–6. FH+, captured at baseline, was coded if participants self-reported that their mother, father, or any of their four grandparents were a possible or definite problem drinker.

Results—Overall, 773 (66.4%) participants reported experiencing blackouts during Years 4–6. Women were more likely to report blackouts than men; however, compared with women with a maternal FH+, men with a maternal FH+ were more than twice as likely to report blackouts.

Discussion—Men appear to be more susceptible than women to the effects of a maternal FH+. Genetic and environmental explanations for this finding are discussed. In sum, these findings are an important step towards understanding a significant yet understudied negative consequence of heavy alcohol use.

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Contributors

Elise N. Marino, B.A. provided the conceptualization of this project, conducted the analyses, and wrote the most substantial portions of the manuscript.

Kim Fromme, Ph.D. designed the original longitudinal study, obtained funding for the research, was responsible for collection of all data used in this report, and contributed to the writing of the manuscript.

Both authors collaborated on the interpretation of the findings.

Conflict of interest

All authors report no conflict of interest.

Keywords

Alcohol; Blackout; Memory; Family history; Gender

1. Introduction

Heavy drinking, which is especially prevalent among college students (Substance Abuse and Mental Health Services Administration, 2013) is associated with behavioral risk-taking and an increased likelihood of developing alcohol use disorders (White & Hingson, 2014). Another consequence of heavy drinking is blackouts, which are periods of amnesia for all or part of a drinking event (Hartzler & Fromme, 2003b; Wetherill & Fromme, 2011; White, 2003).

Blackouts do not involve loss of consciousness, but rather reflect a failure to form long-term memories when information is not transferred from short-term memory (White, 2003). Further, individuals with a history of blackouts, compared with those without such history, show impaired episodic or contextual memory when intoxicated (Hartzler & Fromme, 2003b; Wetherill & Fromme, 2011). Specifically, they have difficulties recalling spatio-temporal and social context of events and show different patterns of brain activation (Wetherill, Schnyer, & Fromme, 2012).

Generally, blackouts occur with excessive alcohol intake and at higher blood alcohol concentrations (BAC; Jennison & Johnson, 1994; White, Signer, Kraus, & Swartzwelder, 2004; White, 2003), although blackouts have been reported at BACs as low as .07g% (Hartzler & Fromme, 2003a). Importantly, only about 50% of drinkers experience blackouts (White, Jamieson-Drake, & Swartzwelder, 2002), suggesting differential susceptibility to alcohol's effects on memory processes and potentially neurological differences between those who do and do not report blackouts.

Whereas the extant literature is somewhat limited, several individual risk factors have been identified. Being female (White et al., 2002), Caucasian (Jennison & Johnson, 1994), having an early onset of drinking (Jennison & Johnson, 1994; White et al., 2002), and a family history of problematic alcohol use (Jennison & Johnson, 1994; LaBrie, Hummer, Kenney, Lac, & Pedersen, 2011) increase one's likelihood of experiencing blackouts. In fact, blackouts share some of the same individual risk factors as alcohol dependence, such as early onset of drinking (Dawson, Goldstein, Chou, Ruan, & Grant, 2008) and family history of alcohol use disorders (Sørensen et al., 2011). Just as there is thought to be a common genetic vulnerability towards developing alcohol use disorders (Kendler, Aggen, Prescott, Crabbe, & Neale, 2012), other consequences of heavy drinking, such as blackouts, may also be heritable. Indeed, twin studies support a genetic vulnerability for blackouts (Nelson et al., 2004). Thus, a genetic vulnerability may explain, in part, why not everyone who drinks heavily will experience blackouts.

Although not studied extensively for blackouts, a family history of problematic alcohol use (FH+) is a risk factor for other alcohol-related consequences. For instance, FH+ individuals were more likely to develop alcohol dependence (Sørensen et al., 2011), which speaks to a

genetic predisposition for consequences in FH+ individuals. Indeed, FH+ men and women drank more and experienced more negative consequences from their alcohol use compared with FH-individuals of the same gender (LaBrie, Migliuri, Kenney, & Lac, 2010).

When comparing across genders, FH+ differentially affects men and women. For example, FH+ was related to negative consequences resulting from alcohol use only for men (Larimer, Anderson, Baer, & Marlatt, 2000). As such, FH+ may be a more powerful risk factor for negative consequences, including blackouts, in men compared with women. This would suggest that men may be more vulnerable than women to the same amount of genetic risk.

To date, few studies have distinguished between maternal and paternal FH+; although, this distinction may be informative for understanding differential risk for negative consequences of heavy drinking. For instance, maternal FH+ in men predicted increased lifetime alcohol use, heavy drinking, drinking problems, and drinking to cope compared with women with a maternal FH+ (Cooper, Peirce, & Tidwell, 1995). Because the majority of the primary caretakers for children continues to be women (Shackelford, Weekes-Shackelford, & Schmitt, 2005), it may be that an alcoholic mother creates more distress than an alcoholic father within the household, and this distress may contribute to her male children exhibiting externalizing behaviors, such as problems with substance abuse, whereas her female children may respond with greater internalizing behaviors, such as anxiety and depression (Cooper et al., 1995). One study, however, failed to find a significant association between maternal and paternal FH+ and problematic alcohol use (Chermack, Wryobeck, Walton, & Blow, 2006). Given the contradictory findings regarding maternal and paternal FH+, further research is needed, especially for understudied consequences of heavy drinking, such as blackouts.

The current study was designed to investigate the association between maternal and paternal FH+ and blackouts. Consistent with previous studies (Jennison & Johnson, 1994; LaBrie et al., 2011), we hypothesized that having a FH+ would predict greater likelihood of experiencing blackouts. To expand this research, however, we also separately analyzed the effects of maternal and paternal FH+ on the occurrence of blackouts. Further, we hypothesized that gender would moderate the association between FH+ and blackouts, such that men who have a maternal FH+ would show a greater likelihood of experiencing blackouts than women or those with a paternal FH+. To our knowledge, no study has examined the differential effects of maternal or paternal FH+ on blackouts.

2. Method

2.1 Participants

All participants were first-time college freshmen who were recruited the summer before they matriculated into a large, state university in the Southwestern United States. These data are a result of their participation in a 6-year, longitudinal study examining alcohol use and behavioral risks during the transition from high school through college. Initially, incoming freshman were invited to participate ($N = 6,391$), and a subset agreed to participate and met the additional inclusion criterion of being unmarried ($n = 4,832$). Of these, 3,046

participants were randomized to be assessed longitudinally, whereas others were assigned to conditions not relevant to the current analyses. Among those assigned to be assessed longitudinally, those who provided informed consent and completed the baseline survey comprised the longitudinal sample ($n = 2,245$).

Participants completed online assessments 10 times over the 6 years: high school (baseline), twice a year during Years 1–3, and then once a year during Years 4–6. All participants included in these analyses must have completed assessments for at least baseline and Year 6 ($N = 1,164$; 65.4% Female), but some may have also completed assessments at Years 4 and 5. Average age at Year 6 was 23.8 years ($SD = 0.4$), 701 (60.2%) participants were Caucasian, 259 (22.3%) were Asian, 46 (4.0%) were Black, and 158 (13.5%) reported other races.

2.2. Measures

2.2.1 Demographics—Participants reported demographic information, including gender and race, which were used in all of our analyses.

2.2.2 Alcohol-induced blackouts—A single item asked respondents to report the frequency with which they “had difficulty remembering things you said or did or events that happened while you were drinking.” Responses were coded on a 5-point Likert-type scale, where 1 = never, 2 = some of the time, 3 = half of the time, 4 = most of the time, and 5 = always. Blackouts were dichotomized (yes/no) with a response of 2 or greater at Years 4–6 being coded as positive. We aggregated blackouts across these three years in order to more broadly capture the prevalence of blackouts because assessments only asked about the previous three months.

2.2.3 Family history of problematic alcohol use—FH+ was assessed at baseline using the Family Tree Questionnaire (Mann, Sobell, Sobell, & Pavan, 1985), a self-report measure that asks about the alcohol use of mother, father, grandparents, and siblings. Response options were: never drank, social drinker, possible problem drinker, and definite problem drinker. Respondents were coded as FH+ if they reported a mother, father, or any of their four biological grandparents as a possible or definite problem drinker (Vaughan, Corbin, & Fromme, 2009). Overall FH+ was calculated using mother, father, and all four grandparents. Maternal FH+ was calculated using mother, maternal grandmother, and maternal grandfather. Paternal FH+ was calculated using father, paternal grandmother, and paternal grandfather.

2.2.4 Alcohol use—The Daily Drinking Questionnaire (DDQ; Collins, Parks, & Marlatt, 1985) measured participants’ alcohol use in terms of quantity and frequency of typical drinking. Using an open-ended response format, participants indicated the number of standard drinks they consumed on each day in a typical week during the past 3 months. Frequency was calculated as the average number of drinking days, and quantity was calculated as the average number of drinks per drinking day. Number of drinks was calculated based on standard drinks, where a standard drink was defined as 1.5 fluid ounces of liquor, 12 fluid ounces of beer, or five fluid ounces of wine.

2.3 Procedure

All procedures were approved by the university's Institutional Review Board, and all participants provided informed consent. Initially, all members of the entering class of 2004 were approached in person during orientation or by postal mail during the summer before classes began. If they were never married and completed the baseline survey, they were eligible for randomization into the study. Participants were emailed a link to the surveys at ten time points: summer before college; fall and spring semesters of freshman, sophomore, and junior years; and fall semester of senior year and the following two years. Further detail regarding recruitment and email methodology are published elsewhere (Hatzenbuehler, Corbin, & Fromme, 2008). Participants were compensated \$30 for baseline, \$20 for fall surveys during Years 1–3, \$25 for spring surveys during Years 1–3, and \$40 each for Years 4–6.

2.4 Statistical analysis

Analyses were performed using SPSS Version 18. First, in order to characterize those who experienced blackouts, we examined the bivariate relations among demographics by blackout status. Bivariate comparisons were computed using χ^2 tests for categorical variables and two-tailed *t*-tests for continuous variables. Next, we calculated the gender distribution of those reporting an overall, maternal, and paternal FH+ using χ^2 tests to ensure that there were no significant gender differences in rates of FH+ that might bias our moderation analyses.

Using three logistic regression models, we examined whether overall, maternal, and paternal FH+ predicted likelihood of experiencing blackouts. For our moderation analyses, we then examined whether there was a significant interaction between gender and overall, maternal, or paternal FH+. The dichotomous outcome measure was any self-reported blackouts across Years 4–6. Because blackouts occur most often after excessive drinking (Jennison & Johnson, 1994) and at higher BACs (White, 2003), we controlled for past 3 month quantity and frequency of drinking. Additionally, due to evidence of differential pathways to alcohol dependence by race (Akins, Smith, & Mosher, 2010; Alvanzo et al., 2011), we controlled for race.

3. Results

Overall, 773 (66.4%) participants reported blackouts, and 441 (37.9%) reported an overall FH+. Additionally, 240 (20.6%) reported a maternal FH+, and 315 (27.1%) reported a paternal FH+. The average number of drinking days in a typical week was 1.4 (*SD* = 1.4), and the average number of drinks per drinking day was 2.5 (*SD* = 2.7).

First, we analyzed differences among those who did and did not report blackouts. As shown in Table 1, bivariate comparisons indicated that those who reported blackouts were significantly more likely to be Caucasian (*p* = .003), drink more frequently (*p* < .001), drink in greater quantities (*p* < .001), have an overall FH+ (*p* = .02), and have a paternal FH+ (*p* = .02). The association between maternal FH+ and blackouts was not significant in a bivariate comparison.

We then examined whether there were gender differences in our three FH+ variables, and found no significant differences by gender for any of the FH+ variables, all p 's > .05 (Table 2). Thus, we concluded that there would be no significant gender bias for our moderation analyses in the subsequent multivariate models.

Next, we performed three separate logistic regressions for each FH+ predictor (Table 3). In all three models, greater quantity (adjusted odds ratio (aOR) = 1.64, 95% confidence interval (CI) [1.47, 1.83]) and greater frequency (aOR = 1.36, 95% CI [1.18, 1.56]) of drinking were significantly associated with blackouts. Further, a significant main effect for gender (aOR = 1.42, 95% CI [1.03, 1.96]) indicated that compared with men, women were significantly more likely to report blackouts. We then examined whether FH+ predicted the likelihood of blackouts, and results indicated that no FH+ variable significantly predicted blackouts. Having a maternal FH+, however, trended toward a greater likelihood of reporting blackouts ($p = .064$).

Despite a lack of main effects of FH+ on the likelihood of blackouts, we proceeded with our a priori hypothesis that the effect of FH+ would be moderated by gender. After controlling for race and quantity and frequency of drinking, the interaction among maternal FH+ and gender was significant (aOR = 2.24, 95% CI [1.01, 4.96]), indicating that men but not women with a maternal FH+ were significantly more likely to report blackouts. Conversely, neither overall FH+ by gender nor paternal FH+ by gender significantly predicted likelihood of blackouts.

4. Discussion

Based on evidence that genetic factors contribute to alcohol dependence (Kendler et al., 2012) and blackouts (Nelson et al., 2004), we examined the association among these two alcohol-related consequences. Among our sample of first-time university students, 37% had an overall FH+, and 66% reported blackouts. We further examined the independent effects of maternal and paternal FH+ on the likelihood of blackouts. Consistent with previous research (Jennison & Johnson, 1994; LaBrie et al., 2011; White et al., 2004; White, 2003), analyses indicated that those who reported blackouts were more likely to be female, Caucasian, frequent, heavy drinkers who reported an overall FH+, and a paternal FH+. Once we controlled for race and typical drinking in the multivariate models, however, neither overall nor paternal FH+ had a main effect on the experience of blackouts. Despite this, our hypothesis-driven moderation analyses revealed that gender interacted with FH+, such that men with a maternal FH+ were more than twice as likely to report blackouts compared with women with a maternal FH+. Genetic and environmental factors may help explain this finding.

Consistent with our findings, early studies suggested that the genetic heritability of alcohol use disorders was greater in men than women (McGue, Pickens, & Sviki, 1992). Recent research, however, indicates that the genetic heritability of alcohol dependence and drinking is equal across genders (Beek, Moor, Geels, Willemsen, & Boomsma, 2014; Heath et al., 1997; Hicks, Krueger, Iacono, McGue, & Patrick, 2004). In other words, there appears to be equal genetic susceptibility towards alcohol dependence and alcohol consumption in men

and women, which is in line with a diminishing gender gap in prevalence rates of alcohol use and related consequences (Keyes, Li, & Hasin, 2011). Whereas this indicates that there is equal genetic heritability of alcohol-related problems among men and women, our findings suggest that men may be more vulnerable than women to the same amount of genetic risk.

In line with an epigenetics framework, our findings indicate that the genetic predisposition towards experiencing alcohol-induced blackouts inherited from the mother is expressed when men grow up in risky environments that set them on a trajectory of heavy, episodic drinking. One potential mechanism for this may be alterations in gamma aminio-butyric acid (GABA). For instance, having low levels of GABA is associated with a lower sedative response to alcohol, which may lead to continued heavy drinking, and then blackouts (Rose & Grant, 2010). Further, heavy drinkers have lower levels of GABA in the anterior cingulate cortex, and this is associated with experiencing more alcohol-related consequences (Silveri et al., 2014). Thus, the genetic risk may be expressed when there are low levels of GABA, leading to heavy drinking and an increased likelihood of experiencing alcohol-related consequences, such as blackouts.

Specifically, our results indicate that the genetic risk for blackouts is transferred from mother to son. This pattern of stronger genetic transmission by mothers is also evident in several psychological disorders. For instance, in Alzheimer's disease (Mosconi et al., 2010) and bipolar disorder (McMahon, Stine, Meyers, Simpson, & DePaulo, 1995), children with an affected mother are at greater risk of developing the disorder compared with children with an affected father. In addition, consistent with our findings, the sons of mothers with schizophrenia were twice as likely to develop psychosis compared with daughters (Goldstein et al., 2011). All three disorders have a strong genetic component (Gatz et al., 2006; Lichtenstein et al., 2009; Wray & Gottesman, 2012), and evidence indicates that an affected mother, as opposed to an affected father, confers greater risk for development of the disorder in the child. Moreover, with evidence that women need higher genetic risk to reach a threshold for neurodevelopmental disorders than men (Jacquemont et al., 2014), we might expect stronger genetic transmission from mothers to sons. Our results are consistent with the idea that a maternal FH+ produces sufficient genetic risk to push men, but not women, over the threshold for experiencing the neurocognitive phenomenon of blackouts.

In conjunction with a genetic predisposition, a maternal FH+ may produce a high-risk environment for the initiation of problematic drinking. For instance, if the mother is dependent on alcohol, and especially if she is the primary caretaker, the children have ample opportunities to witness her modeling of maladaptive drinking. Additionally, there may be lower parental monitoring, contributing to a chaotic environment that creates stress in the children. As a consequence, men may be more likely to abuse alcohol as a method of coping with the stress of having an alcoholic mother, a theory that has been previously proposed (Cooper et al., 1995). That is, their genetic vulnerability towards heavy drinking, as a result of having a maternal FH+, may lead men to use alcohol as a means of coping with a stressful family environment.

Further, a mother who is dependent on alcohol and/or who grew up with alcoholic parents may have more permissive views about alcohol use, especially for her son. These views can promote an environment whereby drinking may be perceived as acceptable and alcohol may be readily available. This type of environment is problematic as early initiation of alcohol use is largely determined by social/environmental factors, such as family and peers (Dick, 2011). Moreover, early onset of drinking is one predictor of blackouts (Jennison & Johnson, 1994; White et al., 2002). As such, environments that provide access to alcohol and condone drinking, especially for boys, can lead to early alcohol use, which may start a pattern of heavy drinking that can carry forward into adulthood. An established pattern of heavy drinking then leads to blackouts, especially in genetically vulnerable men.

Given this, it is likely that the genetic predisposition passed from the mother is working in parallel with the risky environment she may create to produce a greater likelihood that her son will experience blackouts. Because a maternal FH+, specifically for men, is associated with heavy alcohol use, alcohol-related negative consequences (Cooper et al., 1995), and now blackouts, genetic vulnerability coupled with a risky environment likely drives this association. Indeed, research indicates that genetics have a greater influence on alcohol use when there is an environment with low parental monitoring or high alcohol availability (Kendler, Gardner, & Dick, 2011). Ultimately, because evidence clearly demonstrates that heavy drinking leads to blackouts, those genetically vulnerable men who grew up in an environment that allowed or encouraged alcohol use have an increased likelihood of later experiencing blackouts.

Given these findings, targeted prevention programs might focus on men with a maternal FH+ as they are at high risk for blackouts. Because men with a maternal FH+ could possibly be identified at a young age, these interventions might begin with children as early as elementary school age. Consequently our findings may inform prevention programs that educate families about alcohol and its negative consequences, such as the Strengthening Families Program (Spath, Reyes, Redmond, & Shin, 1999) and Raising Healthy Children Project (Brown, Catalano, Fleming, Haggerty, & Abbott, 2005).

Some caveats of our study should be mentioned. Assessments were self-report and retrospective as participants were asked to report on their blackouts and perception of family's drinking patterns. In addition, the majority of the study sample was Caucasian; although, it reflected the heterogeneity similar to other academic institutions in the geographical area. Participants also began the study as university freshmen, so findings may not generalize to other populations. Further, while it is challenging to study blackouts, alcohol self-reports are reliable and valid (Del Boca & Darkes, 2003), and blackouts are an accepted phenomena (e.g., Rose & Grant, 2010).

Despite limitations, findings come from a prospective study that spanned 6 years with a relatively large and ethnically diverse sample, which provided an opportunity to examine drinking patterns and blackouts during the transition into adulthood. Our findings extend the extant literature by demonstrating that a maternal FH+ is a risk factor for blackouts in men. Whereas replication of our findings is needed, they are an important step towards

understanding a significant yet understudied consequence of heavy drinking that is especially salient among young adults.

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Highlights

- 66.4% of university students reported blackouts
- Controlling for drinking, women had a greater likelihood of experiencing blackouts
- Men with a maternal family history of alcohol problems were more likely to black out

Table 1

Bivariate analyses of demographic and clinical characteristics by blackout status

Participant Characteristics (N = 1,164)	Blackout Yes (n = 773)	Blackout No (n = 391)	p value
Demographics			
Female	499 (64.6%)	262 (67.0%)	.406
Caucasian	489 (63.3%)	212 (54.2%)	.003
Age	23.8 (0.4)	23.7 (0.4)	.118
Alcohol use (past 3 months)			
Quantity of alcohol use (drinks/drinking day)	3.1 (3.1)	1.4 (1.4)	< .001
Frequency of alcohol use (drinking days/week)	1.7 (1.5)	0.8 (1.1)	< .001
Family history of problematic alcohol use (FH+)			
Overall FH+	311 (40.2%)	130 (33.2%)	.020
Maternal FH+	170 (22.0%)	70 (17.9%)	.103
Paternal FH+	227 (29.4%)	88 (22.5%)	.013

Notes: Mean (SD) or n (%).

Table 2

Bivariate analyses of FH+ by gender

Participant Characteristics (N = 1,164)	Female (n = 761)	Male (n = 403)	<i>p</i> value
Family history of problematic alcohol use (FH+)			
Overall FH+	302 (39.7%)	139 (34.5%)	.082
Maternal FH+	164 (21.6%)	76 (18.9%)	.280
Paternal FH+	217 (28.5%)	98 (24.3%)	.126

Notes: n (%).

Table 3

Logistic regression coefficients for overall FH+, paternal FH+, and maternal FH+ on blackouts

Overall FH+	
Race	0.92 [0.69, 1.23]
Quantity of drinking	1.64 [1.47, 1.83] [≠]
Frequency of drinking	1.35 [1.18, 1.55] [≠]
Gender	1.37 [0.96, 1.95]
Overall FH+	1.49 [0.89, 2.51]
Gender x Overall FH+	0.73 [0.39, 1.36]
Paternal FH+	
Race	0.93 [0.70, 1.24]
Quantity of drinking	1.65 [1.48, 1.84] [≠]
Frequency of drinking	1.34 [1.17, 1.54] [≠]
Gender	1.17 [0.84, 1.63]
Paternal FH+	1.11 [0.63, 1.97]
Gender x Paternal FH+	1.23 [0.62, 2.45]
Maternal FH+	
Race	0.92 [0.69, 1.22]
Quantity of drinking	1.64 [1.47, 1.83] [≠]
Frequency of drinking	1.36 [1.18, 1.56] [≠]
Gender	1.42 [1.03, 1.96] [*]
Maternal FH+	1.92 [0.96, 3.81]
Gender x Maternal FH+	0.45 [0.20, 0.99] [*]

Notes:

^{*} $p < .05$,[≠] $p < .001$; adjusted odds ratios [95% confidence intervals].