Predictors of alcohol problems in college women: The role of depressive symptoms, disordered eating, and family history of alcoholism

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A R T I C L E   I N F O

Keywords:
Alcohol use
College women
Eating behaviors
Depressive symptoms
Family history

A B S T R A C T

Disordered eating and depressive symptoms are established correlates of alcohol use in college women. Family history of alcoholism (FHA) is also related to problematic alcohol use, but there have been limited studies of how it relates to other established cofactors in women. Predictive associations between disordered eating (i.e., overall levels as well as binge eating), depressive symptoms, and alcohol problems were examined in a sample of 295 female twins. The direct and moderating effects of FHA on the relationships between alcohol problems, disordered eating, and depressive symptoms were investigated. Using hierarchical linear modeling depressive symptoms, but not disordered eating or FHA, significantly predicted alcohol problems. However, there was a significant interaction between disordered eating and FHA; disordered eating was associated with alcohol problems in those with a positive FHA. The implications for high-risk subgroups of college women are discussed.

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1. Introduction

Among college students frequent and excessive alcohol use is normative (Kuo et al., 2002; Wechsler, Dowdall, Davenport, & Castillo, 1995). The majority of college students will adopt more moderate patterns of alcohol use later in the lifespan (Bartholow, Sher, & Krull, 2003; Jackson, Sher, Gotham, & Wood, 2001); however, there is a high-risk subgroup for which alcohol problems during adolescence and young adulthood are predictive of future alcohol abuse (Bennett, McCrady, Johnson, & Pandina, 1999; Warner, White, & Johnson, 2007). Despite the presence of this high-risk group, a large number of studies focus on the frequency and amount of alcohol use (Carey, 1995; O'Malley & Johnston, 2002), including heavy episodic use in college students and its related consequences (McCabe, 2002; Wechsler & Kuo, 2003), rather than indicators of problematic (i.e., hazardous and high-risk) alcohol use. Studies of college women, in particular, have tended to focus on patterns and correlates of use (Anderson, Martens, & Cimini, 2005; LaBrie et al., 2008; Piran & Robinson, 2005). The present study will address these gaps in the literature by examining predictors of problematic alcohol use in college women.

While there are many individual difference and social predictors of alcohol problems, we were most interested in examining gender-relevant dimensions; therefore, the current study focused specifically on the co-factors of depressive symptoms and disordered eating, symptoms that are more commonly found in women than men. Indeed, there is strong evidence that depression and bulimic symptoms are linked to problematic alcohol use in women. However, many female college students exhibit depressed or disordered eating symptoms but do not develop alcohol problems. Thus, there likely are other factors that interact with these symptoms to increase risk for alcohol problems in women. Family history of alcoholism (FHA) has been associated with alcohol problems in college students (Capone & Wood, 2008) and is a prime candidate to examine in the relationship between these dimensions.

Depression (Beck, Thomsb, Mahoney, Fingar, 1995; Nolen-Hoeksema, 2004) and disordered eating (Anderson, Martens, & Cimini, 2005; Corcos et al., 2001) are associated with alcohol problems in women. Both prospective and cross-sectional studies have supported the relationship between depressive symptoms and higher rates of alcohol use as well as related problems. For example, in a prospective community-based study, depressive disorders and symptoms were predictive of higher rates of heavy drinking in women (Dixit & Crum, 2000). Among alcoholic women, depression and emotional distress are robust correlates of drinking behaviors (see Nolen-Hoeksema, 2004). In a survey of college women, depressive symptoms were found to significantly correlate with alcohol problems, even after controlling for the frequency of alcohol use (Harrell & Karim, 2008).

Disordered eating also has been linked to addictive behaviors, including alcohol problems (Davis & Claridge, 1998). A cross-sectional study of first-year college students revealed that dieting and binge eating were associated with more frequent and problematic alcohol use (Krahn, Kurth, Gomberg, & Drewnowski, 2005). In particular, bulimia nervosa, binge eating, and purging are more strongly related to alcohol use disorders than restrictive eating behaviors (Anderson et al., 2005; Bushnell et al., 1994; Corcos et al., 2001; Lilienfeld et al., 1997; Wiederman & Pryor, 1996b). For example, in an online study of college...
students with eating disorders, bulimia nervosa was associated with more negative consequences related to alcohol use (Dunn, Larimer, & Neighbors, 2002).

While some studies have investigated both disordered eating and depressive symptoms as related to women's drinking behaviors, there has been insufficient investigation of possible third factors that may be related to drinking behaviors in college women. In particular, FHA has received limited attention in relationship to established co-factors in women, even though familial alcoholism is strongly related to alcohol use disorders. For example, FHA has been associated with early onset alcoholism and other alcohol use disorders (Dawson, 2000). In a recent cross-sectional analysis of the influence of FHA in a majority female sample, there was an association between alcohol problems and FHA (Capone & Wood, 2008). Nonetheless, there have generally been fewer studies of family history in college women, and these studies have not always found associations between alcohol use behaviors and parental history of alcoholism (Bogart, Yeatman, Sirridge, & Geer, 1995). These inconsistent results highlight the need for additional studies of FHA on college women. In addition, there is a need for studies examining interactions between family history and other risk factors. It may be that a positive FHA only increases risk in women with other indicators of risk (i.e., depressive symptoms, disordered eating).

The current study focuses on this possibility by examining depressive symptoms, disordered eating, and FHA as predictors of alcohol-problems in college women. Importantly, we tested the direct and moderating effects of FHA on associations between depressive symptoms, disordered eating, and alcohol problems. We hypothesized that FHA, depressive symptoms and disordered eating would all be independently associated with alcohol problems. We also predicted that FHA would moderate the relationship between depressive symptoms and disordered eating as predictors of alcohol problems, such that among women with a FHA, there would be a significant association between disordered eating and depressive symptoms and alcohol problems. Further, because binge eating exhibits specific links with alcohol problems, we examined binge eating in addition to an overall measure of disordered eating.

2. Method

2.1. Participants and procedure

Participants were drawn from the Michigan State University Twin Registry (Klump & Burt, 2006), which is a twin registry with a lifespan perspective focused on understanding risk factors for internalizing and externalizing disorders. The initial sample included 295 same-sex female twins (mean age = 20.93; SD = 2.46). Six participants did not complete the alcohol problems assessment and thus, were excluded from analyses. One set of triplets was also excluded from analyses, because the standard dyadic data design was utilized, where one person is linked to only one other person (Kenny, Kashy, & Cook, 2006). The final sample size therefore included 286 female twins (158 monozygotic [MZ] and 128 dizygotic [DZ]; 97% of initial sample; mean age = 20.92 years; SD = 2.47). Zygosity of the twins was determined using a physical similarity questionnaire shown through previous research to be over 95% accurate (Plomin, DeFries, & McClearn, 1990). Participants identified as Caucasian (83%), African American (11%), Hispanic (2%), Asian American (1%), or “other” (3%) ethnicity. The ethnic distribution of participants in this study is broadly representative of the ethnic breakdown of the surrounding area (Culbert, Breedlove, Burt, & Klump, 2008).

Participants were recruited using a number of strategies (see Klump & Burt, 2006 for detailed recruitment information). Participants were recruited through posted flyers, newspaper advertisements, and a twin research e-mail listserv. In the later stages of recruitment, twins were identified using birth records through the Michigan Department of Community Health (MDCH) and the Michigan Bureau of Integration, Information, and Planning Services (MBIIP). Assessments were administered either in the laboratory via computerized assessments or through the mail. Twins were not asked whether or not they were currently living with their parents during the time of the assessment. However, based on comparisons between permanent home addresses and current addresses, we were able to determine that the majority (i.e., over 55%) of twins were not living at home during the time of the assessment.

2.2. Measures

2.2.1. Alcohol problems and history

2.2.1.1. Alcohol problems. The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, De La Fuente, & Grant, 1993) is a 10-item self-report measure used to assess harmful and hazardous amounts of alcohol use. The items assess alcohol consumption (i.e., “how often do you have a drink containing alcohol”), drinking behavior (i.e., “how often during the last year have you found that you were not able to stop drinking once you had started”), adverse reactions (i.e., “how often during the last year have you had a feeling of guilt or remorse after drinking”), and problems related to alcohol use (i.e., “have you or someone else been hurt as a result of your drinking”). Items are rated on a Likert scale for questions related to alcohol consumption, drinking behavior, and adverse reactions. A scale ranging from 0 to 2 is used for items assessing alcohol related problems. Total scores range from 0 (non-hazardous consumption) to 40 (the highest severity of hazardous consumption). A score of 8 or higher indicates a strong likelihood that the person is engaging in harmful alcohol consumption (Saunders et al., 1993).

In a study of nearly 1000 male and female undergraduates, the AUDIT displayed internal consistency reliability of .80 (Fleming, Barry, & MacDonald, 1991). Discriminant validity was established by comparing male and female non-drinkers to alcoholics, with 0.5% of the non-drinkers and 99% of the alcoholics scoring over 8 on the AUDIT (Saunders et al., 1993). The AUDIT scale exhibited good internal consistency in our sample of women (α=.82); the mean AUDIT score for our sample was 4.48 with 22% (n=62) participants reporting alcohol use in the problematic range (i.e., AUDIT scores >8).

2.2.1.2. Family history of alcoholism. Family history of alcoholism (FHA) was assessed using a Family Background Form developed by MSUTR researchers. This self-report form lists 19 psychiatric disorders (e.g., alcoholism, schizophrenia) or other problem behaviors (e.g., delinquency or crime) and asks participants to indicate whether they have a family member with the illness and what relationship that person has with the respondent (e.g., aunt). If the respondent acknowledges a family member has a listed illness (e.g., alcoholism), they are asked to report the gender, whether the relative was a paternal or maternal relation, and whether or not they were hospitalized or received other treatments. Because there is some evidence of stronger effects when family history is defined based on a history of treatment (Baer, 2002) FHA was coded as “present” if participants reported at least one first degree relative as having been hospitalized or received treatment for the illness; family history was coded as “absent” if participants denied a history of treatment (or hospitalization) for alcoholism in their family. If only one twin in a pair indicated a family history of treatment for alcoholism, the pair was coded as family history positive. Thirty-three women in this sample (12%) reported having a first degree relative who had been treated for alcoholism.
Table 1

Pearson correlations between alcohol problems and predictors

<table>
<thead>
<tr>
<th>Subscale</th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Alcohol problems</td>
<td>1.00</td>
<td>.23⁎⁎</td>
<td>.30⁎⁎</td>
<td>.19⁎</td>
<td>.08a</td>
</tr>
<tr>
<td>2. Depression</td>
<td>–</td>
<td>1.00</td>
<td>.53⁎⁎</td>
<td>.49⁎</td>
<td>.13a</td>
</tr>
<tr>
<td>3. Disordered eating</td>
<td>–</td>
<td>–</td>
<td>1.00</td>
<td>.72⁎⁎</td>
<td>.05a</td>
</tr>
<tr>
<td>4. Binge eating</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>1.00</td>
<td>.00a</td>
</tr>
<tr>
<td>5. Family history of alcoholism</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>1.00a</td>
</tr>
</tbody>
</table>

Note. N=271–286; sample sizes vary by subscale due to missing data.

⁎⁎Point-biserial correlation.
⁎ p<.05, ⁎⁎ p<.01.

2.2.2. Disordered eating

2.2.2.1. Minnesota Eating Behavior Survey. The Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005) is a 30-item true/false self-report questionnaire that assesses disordered eating attitudes and behaviors. The Total Score on the MEBS is an overall measure of disordered eating which taps the following constructs: Body Dissatisfaction (i.e., dissatisfaction with one’s size or shape), Weight Preoccupation (i.e., preoccupation with dieting, thinness, and weight), Binge Eating (i.e., thoughts about overeating or the tendency to binge eat) and Compensatory Behavior (i.e., the use of compensatory behaviors such as self-induced vomiting or diuretics for weight loss).

The MEBS Total Score and Binge Eating subscale were utilized in the analyses. Both scales showed moderate-to-high internal consistency in young adult samples, (α=.89 and .68, respectively; von Ranson et al., 2005). The MEBS Total Score and Binge Eating subscale also demonstrated sufficient discriminant and concurrent validity (von Ranson et al., 2005). A clinical cutoff for the MEBS Total Score has not been established; however, women in this sample exhibited a range of disordered eating symptoms (range=0–29; M=8.43; SD=6.43). Additionally, 18 women (6%) indicated significant levels of disordered eating with MEBS Total Scores of 20 or higher. The mean score for the seven-item Binge Eating subscale for our sample was 1.56 (SD=1.77) with a range of 0–7. Further, 27 women (9%) scored a 5 or higher on the Binge Eating subscale suggesting significant levels of binge eating pathology among some women in the sample.

2.2.3. Depressive symptoms

2.2.3.1. Beck Depression Inventory II. The Beck Depression Inventory II (BDI–II; Beck, Steer, & Brown, 1996) was used to assess affective, cognitive, and somatic depressive symptoms. Within non-psychiatric patient populations and undergraduate populations, concurrent validity with observer rating scales had a mean correlation of .65 (Richter et al., 1998). The BDI–II has been shown to have high internal consistency among undergraduate (α=.93) and outpatient (α=.92) populations (Beck et al., 1996). Internal consistency for the BDI–II was high in this sample of women (α=.92) and the mean BDI–II score was 8.90 (SD=8.56). Clinical cutoffs for the BDI–II have been previously established with 72 women (25%) scoring in the dysphoric or depressed range (i.e., score of 13 or higher; Dozois, Dobson, & Ahnberg, 1998).

1 The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmsted, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from the Psychological Assessment Resources, Inc.

2.3. Analysis

2.3.1. Data preparation

In order for interactions to be more easily interpreted (Kenny et al., 2006), all predictor variables were centered on the grand mean, and FHA was coded as 1 for “present” and 0 for “absent.” Square root transformations were performed for the AUDIT, BDI–II, and Binge Eating variables due to moderate positive skew.

2.3.2. Analysis plan

Pearson correlations were computed to examine initial associations between the independent (e.g., disordered eating) and outcome (i.e., alcohol problems) variables. In order to control for the non-independence of twin data, hierarchical linear modeling (HLM) was used to examine predictive relationships between disordered eating (total score or binge eating score), depressive symptoms, FHA, and alcohol use (Kenny et al., 2006). In the current study, there were two levels of data. The lower level represented the individual. The upper level was the twin pair within which each individual was nested.

The final HLM models included the independent variables of depression–neuroticism, disordered eating (total score in one model and binge eating score in the other), and FHA as predictors of alcohol problems. All main effects were examined, as well as the interaction of FHA and the other predictor variables. These interactions were examined to determine whether depressive symptoms and disordered eating (or binge eating) were more predictive of alcohol problems in women with a FHA.

3. Results

3.1. Preliminary analyses

Pearson and point-biserial correlations between depressive symptoms, disordered eating, binge eating, FHA, and alcohol problems are presented in Table 1. With the exception of FHA, all of the independent variables were significantly and positively correlated with alcohol problems.

3.2. Hierarchical Linear Modeling analysis

HLM results for both models examining disordered eating and binge eating are presented in Table 2. The correlations between the disordered eating variables and depression were moderate.

Table 2

Results from hierarchical linear models examining the influence of depression, family history of alcoholism, and disordered eating or binge eating on alcohol problems in women

<table>
<thead>
<tr>
<th>Model</th>
<th>B(SE)</th>
<th>T</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Null model</td>
<td>2.15 (.07)</td>
<td>30.28</td>
<td>142</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Intercept</td>
<td>2.13 (.07)</td>
<td>28.56</td>
<td>129</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Depression</td>
<td>.16 (.05)</td>
<td>3.23</td>
<td>232</td>
<td>.01</td>
</tr>
<tr>
<td>Disordered eating</td>
<td>.01 (.01)</td>
<td>1.29</td>
<td>217</td>
<td>.20</td>
</tr>
<tr>
<td>Family Hx of alcoholism</td>
<td>.19 (.22)</td>
<td>.86</td>
<td>144</td>
<td>.39</td>
</tr>
<tr>
<td>Depression * Fam Hx Alc</td>
<td>-.18 (.16)</td>
<td>-.11</td>
<td>260</td>
<td>.24</td>
</tr>
<tr>
<td>Dis. Eating * Fam Hx Alc</td>
<td>.06 (.03)</td>
<td>.23</td>
<td>240</td>
<td>.03</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Null model</td>
<td>2.15 (.07)</td>
<td>30.28</td>
<td>142</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Intercept</td>
<td>2.13 (.08)</td>
<td>27.77</td>
<td>131</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Depression</td>
<td>.19 (.05)</td>
<td>3.88</td>
<td>236</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Binge eating</td>
<td>-.02 (.11)</td>
<td>-.22</td>
<td>191</td>
<td>.83</td>
</tr>
<tr>
<td>Family Hx of alcoholism</td>
<td>.18 (.23)</td>
<td>.76</td>
<td>146</td>
<td>.45</td>
</tr>
<tr>
<td>Depression * Fam Hx Alc</td>
<td>-.06 (.15)</td>
<td>-.43</td>
<td>254</td>
<td>.67</td>
</tr>
<tr>
<td>Binge Eating * Fam Hx Alc</td>
<td>.32 (.32)</td>
<td>.98</td>
<td>224</td>
<td>.33</td>
</tr>
</tbody>
</table>

Note. Fam Hx Alc: Family history of alcoholism; Dis. eating: Disordered eating. Values from the hierarchical linear models can be interpreted as unstandardized regression coefficients. Total variance explained (i.e., R²) by the HLM models was determined using pseudo R² (Kenny et al., 2006). Significant findings are indicated in bold emphasis.
Collinearity statistics showed that tolerance values were higher than 0.7, indicating that all of the variables were unique in their contribution to the models.

In both models, there was a significant main effect of depressive symptoms on alcohol problems, with depression being associated with higher levels of alcohol problems. There were no significant main effects of disordered eating (either overall disordered eating or binge eating) or FHA in either model. However, there was a significant interaction between FHA and disordered eating in the model for overall disordered eating. Fig. 1 illustrates this interaction showing that higher levels of disordered eating were associated with more alcohol problems for women with a positive FHA.

4. Discussion

This study contributes to the growing literature on predictors of hazardous drinking patterns among female college students and further unpacks the relevance of depressive symptoms, disordered eating, and family history. In both of the multilevel models, depressive symptoms demonstrated a significant main effect. There was also a significant interaction between disordered eating and FHA as predictive of alcohol problems, suggesting that disordered eating is significantly related to alcohol problems among young women with a positive FHA. There were no main effects for overall disordered eating, binge eating, or FHA. Overall, these findings underscore the relevance of affect in college women’s alcohol problems. These findings also offer further evidence that a FHA increases the positive association between disordered eating and alcohol problems.

Consistent with past cross-sectional studies of college women (Harrell & Karim, 2008), our findings confirm the importance of depressive symptoms as related to problematic alcohol use among college women. Although the direction of association remains unclear, previous research suggests that alcohol-problems are related to maladaptive efforts to self-medicate depressive symptoms (Cooper, Fronc, Russell, & Mudar, 1995; Kuntsche, Knibbe, Gmel, & Engels, 2006) and regulate negative emotions (Kassel, Jackson, & Unrod, 2000). Cross-sectional analysis has indicated that a pathway from negative affect to maladaptive coping is associated with increased alcohol problems (Read, Wood, Kahler, Madow, & Palfai, 2003). Alternatively, the negative consequences of alcohol misuse may result in increased depressive symptoms which then reinforce continued problematic alcohol use. Additional research is needed in order to further examine these mechanisms.

With respect to our findings of an interaction between FHA and disordered eating, young women who report a positive FHA as well as disordered eating may represent a particular high-risk subgroup. There is evidence of both genetic (Agrawal et al., 2008) and familial factors (Holderness et al., 1994) that influence the relationship between disordered eating and alcohol problems in young women. Young women with a positive FHA may also be higher on individual difference dimensions associated with disordered eating and alcoholism, such as impulsivity (Wiederman & Pryor, 1996a).

Beyond the college setting, these findings may also have implications for clinical settings. In clinical samples of women with eating disorders, there may be a higher proportion of individuals with a positive family history than in community samples. Therefore, the strength of the relationship between disordered eating and alcohol problems may be stronger when examining clinical or more highly symptomatic subgroups.

Counter to our hypothesis, we did not find significant main effects for disordered eating or binge eating in the multilevel analysis. These findings are inconsistent with the established link between disordered eating and alcohol use among young women (Bulik et al., 2004; Holderness, Brooks-Gunn, & Warren, 1994; Wiederman & Pryor, 1996a). However, it may be the case that our use of a purely community sample limited our ability to detect significant effects. Some past studies supporting co-morbidity between disordered eating and alcohol use focused on the severity of symptoms as related to alcohol use (Anderson et al., 2005; Kranb et al., 2005; Piran & Robinson, 2005). Other studies have examined prevalence rates of alcohol abuse and dependence among women with patterns of disordered eating and other psychiatric symptoms (Dansky, Brewerton, & Kilpatrick, 2000). Past studies of non-clinical samples have found links between eating behaviors and affect; therefore, sampling difference would not fully account for this discrepancy between our findings and past studies. Future research should further determine factors affecting the strength of the relationship between disordered eating behaviors and alcohol problems among young adult women.

FHA did not moderate the relationship between depressive symptoms and alcohol problems among college women. In this sample, negative affect was associated with alcohol problems independent of family or genetic risks. This further underscores the relevance of depressive symptoms in patterns of alcohol problems among young women (Harrell & Karim, 2008). Future research using a more sensitive methodology, examining parent data for example, may yield findings that support the influence of FHA on depressive symptoms in alcohol problems.

The interaction between binge eating and FHA was also not significant. Although there was a small difference in the reliability of the binge eating scale (i.e., .76) compared to the disordered eating total score (i.e., .88), this finding may, in fact, reflect the difficulty of assessing binge eating using self-report questionnaires. Such assessments can result in false positives (Fairburn & Belin, 1994). The overall disordered eating score includes assessments of body dissatisfaction, weight preoccupation and purging behaviors; this indicates that a more comprehensive assessment of eating behaviors should be examined in relationship to FHA in women. Our findings point to the fact that among women with a positive FHA, there is a link between disordered eating and alcoholism. This finding offers support for the broader perspective that disordered eating is closely related to other addictive behaviors (Davis & Claridge, 1998) largely through shared genetic and familial risk factors.

FHA was not directly related to alcohol problems in this sample. Participants were not over-sampled based on FHA, which may explain the lack of finding. However, the interaction between FHA and disordered eating may offer further evidence that a FHA increases the positive association between disordered eating and alcohol problems for women with a positive FHA.

Fig. 1. Graph of interaction of family history of alcoholism and eating behaviors predicting alcohol problems.
these non-significant findings (Baer, 2002; Sher, Wiltzer, Wood, & Brent, 1991). We also used a conservative measure of FHA and coded only participants who reported that a first-degree relative had received treatment for alcoholism as family history positive. This may explain why we did not find a direct association between FHA and alcohol problems in our sample. In many cases individuals do not receive treatment for their alcohol use disorders. However, we felt that this conservative measure best provided confirmation that the relative had a clinical alcohol use disorder. Furthermore, we used the family history method, which involved probands’ reports on family members rather than a family study method, which would have involved interviews of each family member. This may have resulted in underreporting of FHA by the participants due to their lack of knowledge of the incidents of alcoholism in the family.

4.1. Limitations and future directions

There are some limitations to this study. The sample was relatively homogeneous in terms of ethnicity. Given persistent findings of racial/ethnic differences in patterns of college drinking (O’Malley & Johnston, 2002), the findings may not be generalizable to a multi-ethnic sample. These analyses were also cross-sectional, so we are not able to speak to temporal relationships or causal factors that might explain these relationships. Future studies should further examine the directions of these relationships prospectively.

Our full models including disordered eating and binge eating only explained 7% and 3%, respectively, of the variance in alcohol problems. Although this is small, it is consistent with the variance accounted for in past studies examining co-factors of addictive behaviors in college samples (Benjamin & Wulfert, 2005; VanVoorst & Quirk, 2003). It was beyond the scope of the current analyses, however, future studies should further examine individual difference and personality dimensions related to alcohol use such as impulsivity and sensation seeking (Kuntsche, et al., 2006).

It is also important to note that although our sample consisted of twins, our model controlled for the interrelatedness. Future studies should examine the genetic mediation of these effects by examining zygosity in the relationship between FHA and alcohol problems among young adults.

Although our sample consisted of college women, our findings have broader implications. More research on clinical samples of female adolescents and adults is necessary to determine the relevance of family history in the relationship between disordered eating and alcohol problems. It may be the case that the high-risk subgroup is particularly resistant to treatment for both alcohol use and eating disorders. In both clinical and community samples, depression has been associated with alcohol problems among women. These results highlight the fact that interventions addressing affect regulation that have been applied to clinical and community samples may be relevant for college women as well. Future studies should use more direct and comprehensive assessments of family history to examine this possibility to replicate and extend our results.

Acknowledgements

The authors wish to thank the staff of the Michigan State University Twin Registry for their work on data collection and preparation. We also thank Dawn M. Kepler for her editorial assistance on this manuscript.

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