A Mediation Model of Racial Discrimination and Alcohol-Related Problems Among African American College Students

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ABSTRACT. Objective: Racial discrimination has been identified as an important predictor of alcohol-related outcomes for African Americans. The goal of the current study was to extend previously found links between lifetime discrimination, alcohol use, and alcohol problems as well as to elucidate the affective mechanisms underlying these associations, as moderated by gender. **Method:** A multiple-groups structural equation model was computed using survey data collected from 619 students from a historically Black college/university. **Results:** The final model provided excellent fit to the data, explaining 6% of the variance in alcohol consumption and 37% of the variance in alcohol problems. Dis-

crimination was a significant predictor of alcohol-related problems but not, by and large, level of use. For men, anger—but not discrimination-specific anger—was a significant partial mediator of the link between discrimination and both alcohol use and alcohol problems. Depression partially mediated the link between discrimination and alcohol problems for both men and women. **Conclusions:** The results suggest that, for African Americans whose drinking leads to drinking-related problems, discrimination and poor affective self-regulation are highly relevant and predictive factors, especially for men. (*J. Stud. Alcohol Drugs*, 75, 228–234, 2014)

AFRICAN AMERICANS, compared with European Americans, show a later onset of alcohol use and lower drinking levels until approximately age 30 (Gibbons et al., in press). Despite these indications that African Americans should exhibit fewer alcohol-related problems than European Americans, they are actually at higher risk for alcohol-related consequences, dependence symptoms, and alcohol-related morbidity and mortality at comparable levels of use (Chartier and Caetano, 2010; Mulia et al., 2009). One explanation for this racial disparity may be the influence of perceived racial discrimination (Pascoe and Richman, 2009). Discrimination, which is experienced chronically and frequently by African Americans of all ages (Seaton et al., 2008; Williams et al., 2012) and is profoundly stressful in both physiological and subjective terms (Ong et al., 2009; Richman et al., 2010), has been identified as an important

predictor of alcohol use, problems, and dependence among African Americans (Gibbons et al., 2010; Mulia et al., 2008; Richman et al., 2013; Terrell et al., 2006). Despite the disproportionately negative impact of alcohol consumption on African Americans, they remain an understudied population, especially with regard to college student drinking.

College students typically consume heavy amounts of alcohol and are among those at the highest risk for alcohol-related problems (Hingson et al., 2005; O'Malley and Johnston, 2002). Although African American students drink relatively less than their European American peers, excessive use and problems are still prevalent among this group (Meilman et al., 1995; Siebert et al., 2003). Furthermore, African American students report frequent experiences with discrimination (Chao et al., 2012; Swim et al., 2003). The goal of the current study, therefore, was to examine relations between lifetime discrimination, alcohol use, and drinking problems among African Americans attending a historically Black college/university (HBCU). Furthermore, we aimed to elucidate the affective mechanisms underlying these associations, specifically as pertaining to depression and anger.

Mediating role of negative affect

Many psychosocial theories of alcohol use note negative affect as a primary motivation for drinking (e.g., Cooper et al., 1995). Discrimination has been shown to elicit both internalizing and externalizing emotional reactions (e.g.,

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Ong et al., 2009; Williams et al., 2012), each of which may have differing effects on alcohol use and related problems. For internalizing reactions, discrimination predicts increased depressive symptoms in both adults and adolescents (e.g., Brody et al., 2006; Klonoff et al., 1999; Ong et al., 2009; Seaton et al., 2008). Moreover, depression is associated with alcohol problems and, to a lesser degree, with level of use (Cooper et al., 1995; Patock-Peckham and Morgan-Lopez, 2007; Peirce et al., 1994). These associations may be especially pronounced for women (Belle and Doucet, 2003; Schulz et al., 2006), who are at higher risk for depression than men (Hankin and Abramson, 2001) and whose drinking has been previously linked to depressive symptoms resulting from early-life stress (e.g., Chan et al., 2013).

For externalizing symptoms, anger has been associated with risk taking, such as increased alcohol use, more strongly than have internalizing emotions such as depression (Curry and Youngblade, 2006; Gibbons et al., 2010). Furthermore, the relation between discrimination and drinking, at least as mediated by anger, may be more pronounced among African American men (Brodish et al., 2011), because they are more likely than African American women to report anger in response to discrimination (Chao et al., 2012). A heretofore unresolved issue is whether the effects of discrimination on substance use are attributable to anger directly in response to discrimination (Terrell et al., 2006) or to more generalized anger (Gibbons et al., 2010). The latter explanation is supported by the finding that recurrent discrimination may erode African Americans' self-regulatory abilities, leading to increased anger and, subsequently, more substance use (Gibbons et al., 2012a). Furthermore, African Americans who have experienced high levels of discrimination have been shown to be more reactive to stressful life events (Richman et al., 2010; cf. Ong et al., 2009). To our knowledge, the current study is the first to test whether relations between discrimination and alcohol use and problems are mediated by generalized anger or discrimination-specific anger.

Current study

We examined associations among lifetime discrimination, alcohol use, and alcohol-related problems in a sample of African Americans from an HBCU. This study adds to current knowledge by examining whether the associations between discrimination and alcohol use and problems are mediated by generalized or discrimination-specific anger; we expected that, concurrent with past research, generalized anger would mediate these effects (Gibbons et al., 2010). Furthermore, we included alcohol-related problems as a distal outcome, which allowed for testing of a differential mediation hypothesis suggested by earlier research in that depression would predict alcohol problems but not level of use, whereas anger would predict problems both directly and indirectly through consumption (Cooper et al., 1995). Furthermore, prior evi-

dence suggests that the effects of discrimination on drinking and/or drinking problems would be mediated through depression for women and through generalized anger for men (Brodish et al., 2011; Schulz et al., 2006). Last, to differentiate the effects of racial discrimination from other sources of early-life stress, we accounted for experiences of traumatic life events (Ford et al., 2000) and childhood exposure to a risky family (i.e., one typified by conflict, non-nurturing interactions, and/or neglect; Taylor et al., 2004), both of which have been previously linked to problem drinking, depression, and anger (e.g., Aseltine et al., 2000; Chan et al., 2013; Sher et al., 2005).

Method

Participants

All procedures for this study were approved by the institutional review boards at both the study site and the corresponding authors' institution. The initial sample consisted of 741 undergraduates from an HBCU. Notably, HBCUs are typically quite diverse with regard to academic selectivity, level of local populace enrollment, and financial background (Freeman and Thomas, 2002), and African Americans' drinking rates do not significantly differ between HBCUs and other institutions (Meilman et al., 1995). Students were excluded from analyses if they met one or more of the following criteria: they self-identified as other than Black/African American race, African ancestry, or mixed race including African ancestry ($n = 12$); they reported being younger than 18 years of age in the baseline survey ($n = 1$); or they had ever sought treatment for alcohol issues ($n = 8$). Participants also were excluded from the final model if they were missing any items for the discrimination scale ($n = 88$), three or more of the traumatic events scale items ($n = 20$), or an alcohol use score ($n = 28$). These criteria resulted in a final sample for analysis of 619 participants.

Measures

Frequency of discrimination was measured with the Schedule of Racist Events (SRE; Landrine and Klonoff, 1996). This scale has been well validated (Klonoff and Landrine, 1999) with high scores having been previously associated with alcohol use, as well as with anger and depression (Gibbons et al., 2010; Klonoff et al., 1999). Respondents were asked to indicate how frequently they had encountered 14 different discrimination-related experiences in their lifetime, using a 5-point scale from *never* to *all of the time* ($\alpha = .92$; the two highest scale points were collapsed because of the relative rarity of endorsement for the most extreme category). Where appropriate, statements ended with "because you are Black" to ensure that participants' reported experiences were perceived to be related to race.

Discrimination-specific anger was measured with three additional items from the SRE, using the same response scale. Although the SRE has traditionally been treated as unidimensional (Klonoff and Landrine, 1999), qualitative review of the measures suggested that three items assessed anger in response to discrimination rather than frequency of experiences. This distinction was supported empirically by a confirmatory factor analysis (CFA), which was tested using a robust weighted least squares estimator with diagonal weight matrix (WLSMV) in *Mplus 7* (Muthén and Muthén, 2012) and indicated significant improvement to model fit with the two factor model, $\chi^2(1) = 52.0, p < .0001$. In addition, the discrimination-specific anger item loadings increased from .65–.76 in the one-factor model to .71–.84 in the two-factor model, supporting the separation of the SRE into two separate constructs. The correlation between a composite of the three anger-related items ($\alpha = .73$) with the rest of the SRE was .70, indicating a strong association, but not collinearity.

Exposure to a risky family was measured using the Risky Family Questionnaire (Taylor et al., 2004). Respondents were asked about the frequency of both positive (3 items) and negative (10 items) family behaviors they experienced between ages 5 and 15 using a 5-point scale from *not at all* to *very often*. Importantly, inclusion of this scale controlled for family history of problematic alcohol or other drug use in the model. Positive items were reverse-scored, and all items were summed ($\alpha = .86$). Traumatic life events were measured with the Traumatic Events Screening Inventory–Adult (Ford et al., 2000), which produced a lifetime count of 18 different events (e.g., loss of a loved one, physical/sexual/emotional victimization).

Depressive symptoms were measured with the short form of the Beck Depression Inventory (BDI; Beck and Beck, 1972). Participants responded to 13 statements regarding how depressed they had felt in the past week, using a 4-point scale ($\alpha = .86$; the two highest scale points were collapsed because of the rarity of endorsement for the most extreme category). Generalized anger was measured with the three items from the anger subscale (Bryant and Smith, 2001) of the Buss–Perry Aggression Questionnaire–Short Form (Buss and Perry, 1992). Participants responded using a 5-point scale from *extremely uncharacteristic of me* to *extremely characteristic of me* ($\alpha = .73$).

Alcohol use comprised a composite score accounting for both frequency and quantity of drinking. Participants indicated the average number of drinks they had consumed for each day of the week over the past 3 months. The seven daily drink count estimates were averaged and then multiplied by 30, thereby creating an alcohol use variable approximating the number of drinks consumed in a typical month. To mitigate the potential influence of outliers, values of more than four drinks per day were fixed at the four drinks per day value of 120 ($n = 11$).

Alcohol problems were measured with the Brief Young Adult Alcohol Consequences Questionnaire (Kahler et al., 2005). Participants completed 24 items assessing the frequency of eight different alcohol-related problem domains in the past year: academic/occupational, blackouts, impaired control, physiologic dependence, risky behaviors, self-care, self-perception, and social/interpersonal. Responses were made using a scale of *never*, *one to two times*, *three to five times*, and *more than five times*. All multi-item subscales showed acceptable reliability (α 's $> .75$), with the exception of social/interpersonal ($\alpha = .54$).

Procedure

Undergraduates from an HBCU in the mid-Atlantic United States were recruited from 2008 to 2011 via flyers, campus newspaper advertisements, emails, and face-to-face interactions to participate in a study examining relations between genes, daily experiences, and health. Interested students attended an introductory session with up to four other students, at which time they gave informed consent; no student declined to provide consent at this session. At this time, participants also provided salivary DNA and were recruited to participate in a 30-day diary study (see Kranzler et al., 2012; O'Hara et al., in press). Participants were then given login information for a secure website to complete the baseline survey, for which participants were compensated \$20 and from which all measures relevant to the current analyses were derived.

Analysis plan

To test a theoretical model that included multiple predictors, mediators, and outcomes, as well as latent factors, we computed a structural equation model using *Mplus 7* (Muthén and Muthén, 2012). Discrimination was treated as a latent exogenous factor with categorical indicators, whereas the exogenous predictors for risky family and traumatic life events were treated as manifest. All three affective mediators were treated as latent constructs, with generalized anger and discrimination-specific anger both specified using categorical indicators. Alcohol use was treated as a manifest construct and was rescaled by dividing by 10 to keep it in the same scale as the other constructs, thereby facilitating model convergence. For alcohol-related problems, eight indicators were generated from the means of each subscale, and these “parceled” indicators were used to create a latent factor (Coffman and MacCallum, 2005).

Individual CFAs were computed to validate each latent factor. Next, a measurement model was tested to affirm that these factors were associated as expected and model fit was acceptable. A fully saturated structural equation model was then computed based on the hypothesized model. The traumatic-life-events variable was trimmed because it had

no significant direct or indirect effects on the outcomes of interest after we accounted for lifetime discrimination and risky-family background. Next, each path was individually allowed to vary by gender; a path was permitted to remain “free” (i.e., no equality constraint across genders) if the moderated path significantly improved model fit, as indicated by a chi-square difference test. Finally, all paths that were nonsignificant for both men and women were sequentially trimmed.

Results

Descriptive statistics

Of the total sample ($N = 619$), 53% were women, and individuals ranged in age from 18 to 27 years old ($M = 20.0$, $SD = 1.6$). Students averaged 39 reported drinks in a typical month ($SD = 27$) and reported an average drinking problem score of 1.6 ($SD = 0.4$). Substantial levels of lifetime discrimination were reported by participants: 80% ($n = 496$) indicated that one or more discriminatory events occurred at least sometimes, with 25% ($n = 152$) endorsing more than half of the items, a finding that is comparable to rates found in other minority samples (e.g., Seaton et al., 2008). Per recommended cutoffs (Beck and Beck, 1972), approximately two thirds of the sample had absent to minimal levels

of depression (BDI score < 5 ; $n = 418$); 15% were mildly distressed (BDI score = 5–7; $n = 93$); 14% were moderately distressed (BDI score = 8–15; $n = 89$); and 3% were severely distressed (BDI score ≥ 16 ; $n = 19$). Men and women were similar across most variables of interest, with the exception of lifetime frequency of discrimination and typical monthly alcohol use, with men reporting statistically significant higher values for both (discrimination: $M = 2.0$, $SD = 0.7$ vs. $M = 1.8$, $SD = 0.6$, $p < .05$; alcohol use: $M = 42$, $SD = 48$ vs. $M = 36$, $SD = 25$, $p < .05$).

Structural equation model

Measurement model. A CFA using WLSMV estimation was computed to examine whether the individual indicators loaded on the constructs as expected. An SRE item about having to “take drastic steps” in response to racism was dropped as an indicator because its factor loading was relatively low (.47) compared with the other items in the factor (.75–.82). The CFA with all eight constructs correlated provided good fit to the data, $\chi^2(800) = 1499.4$, $p < .0001$; comparative fit index (CFI) = .95; root mean square error of approximation (RMSEA) = .04, 90% CI [.03, .04]. All standardized factor loadings were .50 or larger, except for a BDI item asking about appetite (.36). In keeping with standard use of the BDI, we retained all depression items.

FIGURE 1. Structural equation model of alcohol consumption and alcohol-related problems. $\chi^2(1717) = 2,234.7$, $p < .0001$; comparative fit index = .96; root mean square error of approximation = .03, 90% CI [.03, .04]. $\Rightarrow$ moderated by gender [men/women]; $\rightarrow$ path between variables; $\leftrightarrow$ correlations between predictors. Italicized numbers indicate variance explained (R^2); all included paths and correlations are significant at $p < .05$. ns = not significant.

TABLE 1. Total and indirect effects on alcohol use and alcohol-related problems

Variable	Men (<i>n</i> = 292)	Women (<i>n</i> = 327)
Alcohol use		
Lifetime discrimination		
Total effect	.13 [†]	.02
Family riskiness		
Total effect	.72***	.72***
Direct effect	.55*	.55*
Indirect effect	.17**	.17**
Alcohol problems		
Lifetime discrimination		
Total effect	.13***	.11***
Direct effect	.09***	.09***
Indirect effect	.04**	.02
via depression	.01*	.01*
via generalized anger	.02*	.00
via generalized anger and use	.01 [†]	.00
Family riskiness		
Total effect	.09***	.09***
via depression	.02*	.02*
via generalized anger	.03***	.03***
via use	.03*	.03*
via generalized anger and use	.01**	.01**

Note: Standardized values.

[†]*p* ≤ .06; **p* ≤ .05; ***p* ≤ .01; ****p* ≤ .001.

Structural model. The final model (Figure 1) was computed using WLSMV estimation and provided excellent fit to the data, $\chi^2(1717) = 2,234.7$, $p < .0001$; CFI = .96; RMSEA = .03, 90% CI [.03, .04]. Releasing the equality constraint across genders resulted in significantly improved model fit only for the path from discrimination to generalized anger, $\chi^2(1) = 52.0$, $p < .0001$. The final model explained 6% of the variance in alcohol consumption and 37% of the variance in alcohol problems.

Table 1 lists all total and indirect effects separately by gender. The total effect of lifetime discrimination on alcohol consumption was marginally significant for men but nonsignificant for women. The path from discrimination to generalized anger was moderated by gender such that it was significant for men only. Generalized anger then predicted consumption for both genders. Discrimination also predicted discrimination-specific anger and depression for both men and women, but neither construct predicted consumption. The total effect of discrimination on alcohol problems, however, was significant for both men and women. Both genders showed a significant direct relation from discrimination to alcohol problems and a significant indirect path via depression. Men also showed mediated paths through generalized anger because of the initial moderated path originating from discrimination.

Discussion

The purpose of these analyses was threefold. First, we wanted to establish whether discrimination was associated

with alcohol-related outcomes in a large sample of African American HBCU students. Second, we were interested in the mediating role of both internalizing and externalizing affective responses to discrimination and, in particular, generalized versus discrimination-specific anger. Last, we wanted to provide some explanation for the paradox that African Americans, on average, consume less alcohol than European Americans and yet suffer from disproportionately high levels of alcohol-related consequences (Chartier and Caetano, 2010; Mulia et al., 2009).

For both men and women, discrimination was a significant predictor of alcohol-related problems but not of level of use. These effects held after traumatic life events and a risky-family background were accounted for. However, the affective processes mediating the effects of discrimination differed between men and women. Specifically, men who reported higher levels of discrimination were more likely than women to report higher levels of generalized anger (cf. Chao et al., 2012). Generalized anger, in turn, mediated the links between discrimination and both alcohol consumption (full mediation) and alcohol problems (partial mediation) for men only. This pattern of results may be explained by African American men more frequently being the targets of discrimination than are African American women (Seaton et al., 2008; cf. Swim et al., 2003), which could result in greater erosion of their self-regulatory capacities (Gibbons et al., 2012a). Prior evidence also suggests that African American women enact more effective coping responses when encountering discrimination, which may partially buffer negative affect resulting from discriminatory experiences (Swim et al., 2003).

In addition, we tested whether the influence of discrimination on drinking behavior was mediated by generalized or discrimination-specific anger. For both genders, discrimination was associated with discrimination-specific anger, but this construct did not subsequently predict alcohol use or problems. These results indicate that anger in response to specific discriminatory experiences is common; however, for some individuals (particularly men), repeated exposure to racial discrimination may lead to poorer overall emotional self-regulation and higher emotional reactivity across situations (Gibbons et al., 2012a; Richman et al., 2010). These changes may then increase risk for subsequent alcohol misuse and associated negative health outcomes. Discrimination also predicted higher levels of depression, although, contrary to expectation, this association was not moderated by gender (cf. Brody et al., 2006). Depression, therefore, partially mediated the association between discrimination and alcohol problems for both men and women but did not predict alcohol use. Although prior evidence has indicated no relation between discrimination, depression/anxiety, and levels of substance use when anger is accounted for (Gibbons et al., 2010), our model suggests that depression does predict problems stemming from alcohol consumption.

Finally, our model provides further evidence that discrimination may explain why certain racial/ethnic minorities, in this instance African Americans, are disproportionately affected by alcohol issues. Variance explained for alcohol consumption was fairly modest at 6%, in stark contrast to the 37% of variance explained for alcohol problems. Furthermore, risky-family background was not strongly related to alcohol problems, suggesting that discrimination may be a more potent antecedent of alcohol problems for this group. Discrimination, therefore, may modify trait affect in young African Americans, possibly by dysregulating their capacity for emotional and behavioral self-control (Gibbons et al., 2012a); these changes, in turn, appear to be related much more to alcohol problems than to level of use. In sum, although African Americans are at lower risk for increased alcohol consumption, a certain subset of African Americans (i.e., those with greater experiences of discrimination and poor emotional self-regulation) may be especially vulnerable to the negative effects of alcohol.

Limitations and future directions

Although informative, these data carry with them all of the limitations associated with retrospective recall (e.g., attribution errors, cognitive biases). Furthermore, data were cross-sectional, and, although the time frames associated with each measure approximated a causal chain of events, we cannot definitively argue for causality. In addition, the BDI (Beck and Beck, 1972) assesses depression/psychological distress only for the past week, whereas the other affective mediators (i.e., generalized and discrimination-specific anger) used a more expansive time frame. Because recent depressive symptoms have been shown to be moderately stable over weeks to months (Beck et al., 1988), we believe that our results reasonably represent how general affective processes mediate the association between lifetime discrimination and alcohol-related outcomes. Future studies, however, would benefit from prospective methods exploring the long-term and potentially cumulative deleterious effects of discrimination on alcohol use and problems.

The direct path from discrimination to alcohol problems in the final model suggests that emotional dysregulation is not the only mechanism underlying the link between discrimination and alcohol. Discrimination likely works through a variety of processes, including biological/genetic (Gibbons et al., 2012b), physiological (Richman et al., 2010), structural (LaVeist and Wallace, 2000), and, notably, psychological (Gerrard et al., 2012; Richman et al., 2013). Finally, gender appears to play a role in how African Americans respond to discrimination and how those responses may lead to problematic drinking. Examination of gender differences in alcohol-related coping responses to discrimination would be a valuable avenue of further inquiry. Overall, our findings add to the accumulating body of evidence that a more

detailed understanding of discrimination, and its subsequent affective consequences, would be invaluable in the development of more effective alcohol-related interventions for certain racial and ethnic minority groups.

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