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Reports

Alcohol-related cues promote automatic racial bias

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ABSTRACT

Previous research has shown that alcohol consumption can increase the expression of race bias by impairing control-related processes. The current study tested whether simple exposure to alcohol-related images can also increase bias, but via a different mechanism. Participants viewed magazine ads for either alcoholic or nonalcoholic beverages prior to completing Payne's (2001) Weapons Identification Task (WIT). As predicted, participants primed with alcohol ads exhibited greater race bias in the WIT than participants primed with neutral beverages. Process dissociation analyses indicated that these effects were due to automatic (relative to controlled) processes having a larger influence on behavior among alcohol-primed relative to neutral-primed participants. Structural equation modeling further showed that the alcohol-priming effect was mediated by increases in the influence of automatic associations on behavior. These data suggest an additional pathway by which alcohol can potentially harm inter-racial interactions, even when no beverage is consumed.

Introduction

Recent research has demonstrated the intriguing fact that behaviors often associated with alcohol use (e.g., aggression, perceptions of sexual attractiveness, tension-reduction) can be observed without consumption, following exposure to alcohol-related cues (see Bartholow & Heinz, 2006; Friedman, McCarthy, Förster, & Denzler, 2005; Subra, Muller, Bègue, Bushman, & Delmas, 2010). Such work suggests that cognitions or behavioral scripts pertaining to alcohol's effects can be triggered by perception of alcohol-related cues, thereby instigating relevant behaviors. However, specific, underlying mechanisms for such effects have not been identified. Moreover, no previous study has tested whether the mere presence of alcohol-related cues can increase racial bias. Such a finding could have important practical implications; for example, simply entering a bar could be enough to increase the likelihood of bias expression. The current research aimed to address these issues by testing whether simple exposure to alcohol-related cues increases the expression of racial bias in a standard laboratory paradigm, and by investigating the underlying processing mechanisms responsible for this effect.

Considerable research has focused on effects of alcohol on social behaviors such as aggression (see Bushman & Cooper, 1990; Giancola, 2000) and sexual risk-taking (see Cooper, 2002). In recent years some researchers have begun to expand this literature by investigating effects of alcohol on expression of various group biases. Although small, this literature consistently shows that alcohol increases expression of prejudice, stereotyping and discrimination. For example, Reeves and Nagoshi (1993) reported that participants who had consumed alcohol (or believed they had) judged a Black confederate as more aggressive compared to participants who were in a simple no-alcohol control condition. Parrott, Gallagher, Vincent, and Bakeman (2010) found that aggression towards sexual minorities was twice as common on days when participants consumed alcohol compared to days when they did not drink. Other recent work has more directly shown that consuming alcohol leads to increased expression of race bias in standard laboratory paradigms. For example, Bartholow, Dickter and Sestir (2006) found that, relative to a placebo beverage, alcohol led to a decrease in participants' ability to inhibit responses indicative of bias in a racial priming task (see also Bartholow, Henry, Lust, Saults, & Wood, 2012; Schlauch, Lang, Plant, Christensen, & Donohue, 2009).

Of greater interest for the present work, recent evidence indicates that alcohol can influence social behavior even when no beverage is consumed. For example, when presented with alcohol-related cues, even outside of conscious awareness, participants exhibit increased aggressive behaviors (Friedman, McCarthy, Bartholow, & Hicks, 2007; Subra et al., 2010) and a tendency to perceive others' behaviors as hostile (Bartholow & Heinz, 2006), increased sexual attraction (Friedman et al., 2005), increased willingness to engage in more anxiety-provoking social interactions (Friedman et al., 2007), and increased expression of socially-sensitive topics (Freeman, Friedman, Bartholow, & Wulfert, 2010).

The work of Freeman et al. (2010) is particularly relevant to the current study, given their investigation of social disinhibition.

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Specifically, in three studies participants were primed with alcohol-related or neutral images and then completed a computerized free-associations task in which they saw provocative/taboo words (related to sex or excretion) and neutral words and were instructed to respond with the first word that came to mind. Participants who were primed with alcohol-related images, but not those primed with neutral images, responded faster to provocative than to neutral words when under high evaluative pressure. To the extent that the "social disinhibition" observed by Freeman et al. generalizes to expression of other socially sensitive topics, it could be that the mere presence of alcohol-related cues will also increase the expression of racial bias.

Considerable research in both the race bias literature (e.g., Conrey, Sherman, Gawronski, Hugenberg, & Groom, 2005; Devine, 1989; Payne, 2001, 2005) and the literature on acute effects of alcohol (e.g., Easdon & Vogel-Sprott, 2000; Fillmore, Vogel-Sprott, & Gavrilescu, 1999) has focused on understanding the influence of controlled versus automatic aspects of cognition on behavioral responses. Most pertinent to the current research are studies indicating that the expression of racial biases is affected by variations in control, or what Sherman and colleagues refer to as the ability to overcome bias (see Conrey et al., 2005; Sherman et al., 2008). According to this perspective, stereotypic associations are activated spontaneously upon perceiving a member of a racial outgroup (Devine, 1989), but the extent to which such associations influence behavior depends upon a perceiver's ability to exert cognitive control over their response (see Bartholow & Henry, 2010; Stewart, von Hippel, & Radvansky, 2009).

Similarly, numerous alcohol experiments point to the conclusion that alcohol dysregulates behavior largely by impairing cognitive control, not by influencing the strength of automatic associations (Casbon, Curtin, Lang, & Patrick, 2003; Curtin & Fairchild, 2003; Fillmore & Vogel-Sprott, 2000; Fillmore et al., 1999). For example, Fillmore et al. (1999) had participants study a word list prior to consuming a moderate dose of alcohol or a placebo. Process dissociation procedure analyses (PDP; see Jacoby, 1991) indicated that alcohol (relative to placebo) reduced the influence of controlled processes on stem completion performance, but that the influence of automatic processes was unaffected by alcohol. In accordance with these findings, both Bartholow and colleagues (2006, 2012) and Schlauch and colleagues (2009) similarly concluded that increased expression of race bias following alcohol consumption results from reductions in cognitive control, and not increases in the strength of automatic associations (Bartholow et al., 2006) or the influence of automatic processes on behavior (Schlauch et al., 2009). This conclusion is consistent with theories positing that alcohol's effects on social behavior stem from impairment of frontal and prefrontal neural circuits responsible for implementation of cognitive control (e.g., Curtin & Fairchild, 2003; Giancola, 2000).

However, such an explanation cannot account for alcohol cue exposure effects such as those reviewed previously (e.g., Bartholow & Heinz, 2006; Freeman et al., 2010; Friedman et al., 2005, 2007). Rather, the literature on explicit alcohol outcome expectancies and alcohol-related implicit memory associations suggests that exposure to alcohol-related cues can activate associations in long-term memory that influence behavior in ways consistent with consumption, but for reasons differing from alcohol's pharmacological effects. Alcohol outcome expectancies are (generally explicit) beliefs concerning the effects that drinking alcohol will produce (e.g., see Goldman, Darkes, & Del Boca, 1999). Expectancies generally are thought to be related to a number of specific behavioral and experiential domains, including that alcohol increases aggression, reduces tension and enhances social interactions (e.g., Fromme, Stroot, & Kaplan, 1993; Goldman, Brown, & Christiansen, 1987). In addition, both direct and indirect experiences with alcohol lead to implicit memory associations pertaining to alcohol's effects, which may or may not be associated with specific behavioral domains (Stacy, 1995, 1997; Stacy, Leigh, & Weingardt, 1994; Stacy, Widaman, & Marlatt, 1990; Wiers & Stacy, 2006).

Along these lines, Freeman et al. (2010) recently postulated that alcohol cues might come to serve as discriminant stimuli signaling that certain types of behavior (e.g., disinhibition) will be reinforced (e.g., encouragement from peers), and that such associations do not depend on any specific, explicit expectancy in order for related behavioral effects to occur (see also Bolles, 1972). Therefore, simple exposure to alcohol-related cues could activate implicit memory associations that prompt certain cognitive and behavioral scripts, and implementation of such scripts can occur without awareness of their priming or previous encoding (Freeman et al., 2010). Thus, and consistent with the general notion of behavioral priming (see Dijksterhuis & Bargh, 2001), exposure to alcohol-related cues could initiate relevant behaviors even if no beverage is consumed, and in the absence of specific, explicit expectancies directly linking alcohol with the behavior in question. In other words, whereas alcohol consumption is thought to impair behavior via pharmacological effects on neural control mechanisms, exposure to alcohol-related cues could influence behavior via contextual activation of implicit memory associations and the behavioral scripts with which they are related.

The purpose of the current study was to test the effects of simple exposure to alcohol-related cues on the expression of racial bias, and to investigate potential mechanisms for this effect in automatic and control-related processes. On the basis of previous alcohol cue-exposure research (Bartholow & Heinz, 2006; Freeman et al., 2010; Friedman et al., 2005, 2007), we predicted that participants exposed to alcohol-related cues would subsequently show increased race bias on the Weapons Identification Task (WIT; Payne, 2001), a commonly-used laboratory measure of so-called "implicit" race bias that has been shown to be sensitive to the intoxicating effects of alcohol consumption (Bartholow et al., 2012; Schlauch et al., 2009).

Additionally, based on theory and research indicating that exposure to alcohol-related cues leads to spontaneous activation of implicit associations pertaining to alcohol's effects (see Wiers & Stacy, 2006), previous studies indicating that effects of alcohol-related cues on behavior occur "automatically" because they can be observed even when cues are presented subliminally (Subra et al., 2010) or sub-optimally (Friedman et al., 2005, 2007), and research showing that such activated associations can automatically compel relevant behaviors (see Dijksterhuis & Bargh, 2001), we predicted that the increase in bias following exposure to alcohol-related cues would be driven by a stronger reliance on automatic associations, and not by impairment of cognitive control as typically occurs following alcohol consumption (Bartholow et al., 2006, 2012; Schlauch et al., 2009).

Method

Participants

One hundred and seventy-two undergraduate students (M_{age} = 19.28, SD_{age} = 1.72, 53% male) were recruited from Introductory Psychology courses at the University of Missouri. Most participants (66.3%) identified their racial/ethnic group as White/NonHispanic, 18% self-identified as African American, 7.6% as Asian American, 2.9% as Hispanic American or Latino/Latina, and 5.3% as multicultural or "Other."

Cue priming task

Participants were randomly assigned to one of two conditions: alcohol-cue priming or nonalcohol-cue priming, as in previous research (see Bartholow & Heinz, 2006, Study 2). In the alcohol-cue priming condition, participants were presented with six (randomly-ordered) print advertisements for alcohol. In the neutral-cue priming condition, participants were presented with six print advertisements

for nonalcoholic beverages (e.g., milk, juice).[1] Participants were asked to rate the ads on five dimensions (how pleasing, interesting, and persuasive they found each ad, how clear the message of the ad was, and how likely they would be to purchase the product) using a 7-point scale anchored at 1 (*not at all*) and 7 (*extremely*).

Race bias task

Participants completed 192 trials of the WIT (Payne, 2001). On each trial, a pattern mask (1 s) was followed by a prime (a White or Black man's face), shown for 200 ms, followed by a target (a gun or tool), also shown for 200 ms before being replaced by a pattern mask. Participants were to categorize the target as a gun or a tool by pressing one of two keys (counter-balanced across participants) as quickly as possible (before a 500 ms response deadline). Failure to respond within 500 ms elicited a warning ("Too Slow!"), presented for 500 ms, to encourage quicker responding. Trials were separated by an inter-trial interval of 1100 ms. Target stimuli were obtained from Payne (2005) and facial primes were selected from a set developed for the web-based Implicit Associations Test (Nosek, Banaji, & Greenwald, 2002). Participants completed 16 practice trials prior to the experimental trials.

The WIT is well suited for investigating the influence of automatic and controlled processes on the expression of bias using PDP analyses (see Payne, 2001, 2005). The PDP approach assumes that any given behavior is determined by both automatic and controlled processes. The structure of the WIT, in which some trials allow these processes to act in concert while others place these processes in opposition, provides a context in which the relative influence of these processes can be estimated. On black-gun trials in the WIT, both automatic stereotyping and the goal-driven "gun" response call for the same response (i.e., congruent trials). In contrast, on black-tool trials automatic stereotyping calls for the "gun" response, which opposes the goal-directed "tool" response (i.e., incongruent trials). The critical set of equations for deriving PDP estimates of controlled (C) and automatic (A) processing components is:

$$C = P(\text{correct}|\text{congruent trials}) - P(\text{stereotypic error}|\text{incongruent trials})$$

$$A = P(\text{stereotypic error}|\text{incongruent trials})/(1 - C)$$

That is, for each participant, C is computed as the proportion of congruent trials on which they responded correctly minus the proportion of incongruent trials on which they committed a stereotype-related error (e.g., responding with the "gun" key on black-tool trials), and A is that same proportion of incongruent error trials divided by the inverse of C. Automatic and controlled components can be estimated for both White and Black faces. The full set of PDP equations can be found in Payne (2001, 2005).

Procedure

Participants completed the experiment in groups of one to five (seated in individual cubicles). Participants were told that the experiment was comprised of two separate studies; the purpose of the first study was to examine their evaluations of various magazine advertisements and the purpose of the second study was to test their ability to quickly and accurately distinguish guns from other objects. After providing informed consent, participants were randomly assigned to the alcohol cue-priming ($n = 87$) or nonalcohol cue-priming condition ($n = 85$). After completing the cue priming procedure, participants were given instructions for the WIT and then completed the practice and experimental trials. Following the WIT, participants completed questionnaire measures of demographic information. Finally, participants were debriefed, thanked for participating, and dismissed.

Results

Data from one participant was discarded due to high error rates (0.98) for both Black-tool and White-tool trials, leaving the sample for data analyses at $n = 171$. Due to insufficient statistical power, it was not possible to compare the responses of white versus nonwhite participants. However, when analyses were restricted to data from white participants only, the pattern of results was very similar to what is reported here. Participants' gender did not moderate any of the effects that we describe, so all analyses collapsed across this factor as well.

Preliminary analyses

To ensure that differences in the experience of the ads (other than their content) could not account for our predicted effects, we examined participants' ratings of the ads as a function of condition. One of the rating dimensions, Purchasing Intention, differed significantly between the groups. In the alcohol condition, participants were less likely to indicate Purchasing Intention ($M = 3.26$, $SD = 1.38$) than in the nonalcohol condition ($M = 4.13$, $SD = 1.35$), $t(169) = 4.47$, $p < .001$. In the nonalcohol condition, only one participant (1.2% of this subsample) indicated no Purchasing Intention for any of the six advertised products, whereas 8 participants in the alcohol condition (9.2% of the subsample) did so. Therefore, we controlled for Purchasing Intention by entering it as a covariate in the models described below.

Race bias in the WIT

Previous research consistently has shown that race bias in the WIT is expressed in terms of increased errors in identifying tools that follow Black compared to White face primes; in contrast, errors identifying guns typically are not affected by the race of the prime (e.g., Amodio, Devine, & Harmon-Jones, 2008; Amodio et al., 2004; Bartholow et al., 2012; Payne, 2001, 2005). Thus, analysis of WIT performance data was focused on error rates for tool trials, using a 2 (Cue Priming Condition: Alcohol vs. Nonalcohol) × 2 (Race of Prime: Black or White) mixed analysis of covariance with the first factor varying between participants and Purchasing Intention included as a continuous covariate. In accordance with predictions, a significant Race of Prime × Cue Priming Condition interaction emerged, $F(1, 168) = 5.06$, $p < .05$, $\eta_p^2 = .03$, indicating that the race bias effect differed across Cue Priming Conditions. Follow-up comparisons within the two priming conditions indicated that misidentification of tools when preceded by Black versus White primes was particularly prominent in the alcohol priming condition, $t(86) = 4.98$, $p < .001$, $d = 1.07$, relative to the neutral priming condition, $t(83) = 2.70$, $p = .008$, $d = 0.59$ (see Fig. 1). No other effects of interest were significant.

PDP analyses

To simplify reporting and highlight the most relevant effects of interest (i.e., the extent to which controlled and automatic processes were differentially involved on Black versus White prime trials depending upon the priming condition), we created Black–White difference scores for both automatic and controlled PDP estimates. These difference scores were submitted to a 2 (Cue Priming Condition: Nonalcohol vs. Alcohol) × 2 (PDP Component: Controlled vs. Automatic) mixed analysis of covariance with priming condition as a between-subjects factor and with Purchasing Intention included as a continuous covariate. This analysis revealed a Cue Priming

[1] A separate pretest sample ($n = 15$) rated all of the ads to ensure that they did not differ in terms of global favorability or the affect (positivity and negativity) and arousal they elicited (no differences were found).

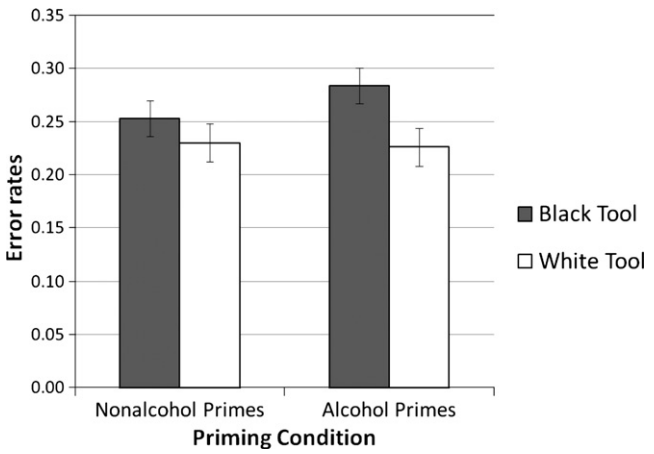

Fig. 1. Proportion of errors on tool trials as a function of priming condition and race of primes. Vertical bars indicate standard errors.

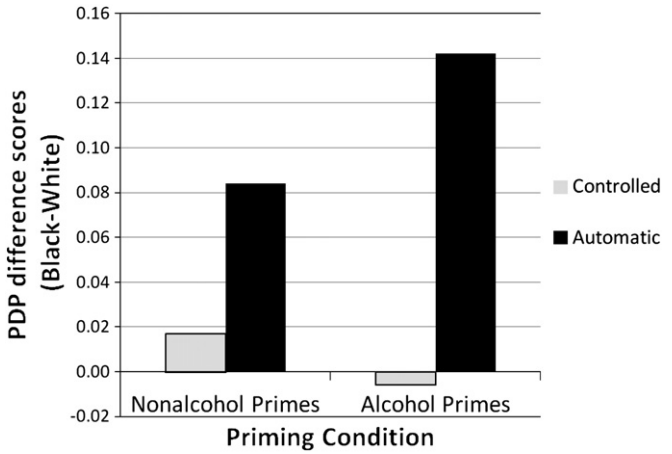

Fig. 2. Difference scores (Black prime trials − White prime trials) as a function of PDP estimates and priming condition.

Condition × PDP Component interaction, $F(1, 168) = 4.08$, $p < .05$, $\eta_p^2 = .024$. Follow-up comparisons indicated that automatic estimates were higher than controlled estimates in both conditions, but that this effect was almost twice as large in the alcohol cue priming condition, $t(86) = -5.19$, $p < .001$, $d = 1.12$, compared to the nonalcohol cue priming condition, $t(83) = -2.87$, $p = .005$, $d = .63$ (see Fig. 2).[2] Original data for estimates of the influence of automatic and controlled processes on responding for both Black and White primes in each of the Cue Priming Conditions are presented in Table 1. No other significant effects emerged.

Accounting for cue priming effects on race bias

The pattern of results seen in our primary analyses indicates that alcohol cue priming leads to increased bias (i.e., more errors on black-tool relative to white-tool trials), and enhanced influence of automatic processes on performance, relative to nonalcohol cue priming. To test the hypothesis that the difference in bias between the priming conditions was driven by stronger reliance on automatic associations (and not reduced reliance on control) following alcohol cue exposure, we constructed a structural equation model (using *M*plus version 6.1, Muthén & Muthén, 2010) of the associations among relevant variables to test the magnitude of indirect effects (i.e., mediation) of cue priming condition on bias via automatic and controlled PDP estimates. The endogenous variables (PDP estimates and bias score) were modeled as difference scores (Black prime trials minus White prime trials) to replicate the dependent variables of interest from our primary analyses. To emulate the analysis of covariance approach used in our primary analyses, the model included Purchasing Intention and the Purchasing Intention × Cue Priming Condition interaction as additional exogenous variables. Cue Priming Condition was effect coded as alcohol = 1, nonalcohol = −1. This model is given in Fig. 3.

Consistent with the previous analysis, inspection of Fig. 3 shows that the PDP-Automatic estimate was significantly higher among those in the alcohol cue priming condition, relative to the nonalcohol cue condition, while the PDP-Control estimate did not differ significantly between Cue Priming Conditions. Additionally, both enhanced PDP-Automatic and reduced PDP-Control estimates significantly predicted bias (i.e., more errors on Black-tool than White-tool trials), findings consistent with the idea that responses will be more biased if subject either to greater influence of automatic associations or reduced influence of control-related processes (e.g., Payne, 2005; Sherman et al., 2008). Of greatest interest here, the analysis showed that the indirect effect of condition via the PDP-Automatic estimate on bias-related errors was significant, standardized estimate = .11 ($SE = .051$), $t = 2.08$, $p = .03$. In contrast, the indirect effect of condition via the PDP-Control estimate was not significant, standardized estimate = .05 ($SE = .046$), $t = 1.17$, $p = .24$. Moreover, the fact that the direct path from condition to race-biased tool errors was not significant in the presence of these mediating variables, alongside the significant indirect effect just noted, indicates that the PDP-Automatic estimate fully mediates this association (see MacKinnon, Fairchild, & Fritz, 2007).

Discussion

On the basis of recent research showing that exposure to alcohol-related pictures or words can trigger behaviors often attributed to alcohol consumption (e.g., Bartholow & Heinz, 2006; Freeman et al., 2010; Friedman et al., 2007; Subra et al., 2010), we hypothesized that alcohol cue exposure would increase bias-related errors in the WIT, similar to the effects of alcohol consumption reported in recent studies (Bartholow et al., 2012; Schlauch et al., 2009). Unlike in

Table 1
Means and standard errors for PDP automatic and controlled components for White and Black prime trials of the WIT as a function of cue priming condition.

Condition	Prime	Automaticity	*M*	*SE*
Nonalcohol	Black	Controlled	.55	.03
		Automatic	.56	.02
	White	Controlled	.53	.03
		Automatic	.48	.02
Alcohol	Black	Controlled	.53	.03
		Automatic	.61	.02
	White	Controlled	.53	.03
		Automatic	.46	.02

Note. Standard errors reported here were obtained from a 2 (Cue Priming Condition: Nonalcohol vs. Alcohol) × 2 (Race of Prime: Black vs. White) × 2 (PDP Component: Controlled vs. Automatic) analysis of covariance with cue priming condition as a between-subjects factor and with Purchasing Intention included as a continuous covariate.

[2] Although this analysis utilizes difference scores (Black–White), we also ran a similar analysis of covariance with all three factors (i.e., Cue Priming Condition, PDP Component, and Race of Prime) with Purchasing Intention included as a continuous covariate. Follow-up comparisons indicated that the PDP-automatic component was larger for Black than for White primes, but especially so in the alcohol cue priming condition. The reason we decided to present difference scores is because neither of the simple effects (i.e., alcohol vs. nonalcohol for Black automatic estimates, and alcohol vs. nonalcohol for White automatic estimates) was significant, and thus the significant Cue Priming Condition × PDP Component interaction depends on the difference for both Black and White automatic components, as opposed to being driven by one or the other.

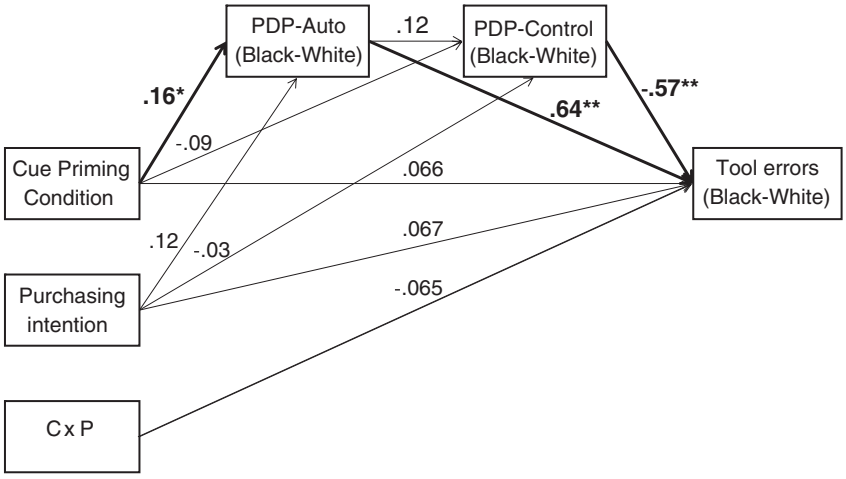

Fig. 3. Structural equation model depicting associations among study variables. PDP-Auto (Black–White) = Black–White difference score for Automatic PDP estimates. PDP-Control (Black–White) = Black–White difference score for Controlled PDP estimates. Tool errors (Black–White) = "Black tool–White tool" difference score on error rates. C × P = Condition × Purchasing Intention interaction effect. Effects from Condition to Tool errors: (a) total effect = .21, $t = 2.46$, $p = .014$; (b) total indirect effect = .14, $t = 2.19$, $p = .029$; (c) indirect effect via PDP-Auto = .10, $t = 2.08$, $p = .03$; and (d) indirect effect via PDP-Control = .05, $t = 1.17$, $p = .24$. $^{**}p < .01$, $^{*}p < .05$.

those recent reports, however, we hypothesized that exposure to alcohol cues would influence bias through effects on automatic rather than control-related processes.

Consistent with these hypotheses, participants who were primed with alcohol-related cues were more likely to misidentify tools when preceded by Black (compared to White) faces than those primed with neutral beverage cues. This finding has a number of implications. First and foremost, this finding indicates that alcohol-related increases in racial bias (see Bartholow et al., 2006, 2012; Schlauch et al., 2009) do not necessarily stem from alcohol's pharmacological effects. Second, the current findings extend other recent alcohol priming work (e.g., Bartholow & Heinz, 2006; Freeman et al., 2010; Friedman et al., 2005, 2007; Subra et al., 2010) into a different behavioral domain. Importantly, whereas most previous studies have addressed behaviors that expectancy research has shown to be explicitly associated with alcohol consumption (e.g., increased aggression, tension reduction or sexual attraction), we investigated a phenomenon that has not been explicitly addressed in the expectancy literature, but that recent findings nevertheless suggested could be affected by the presence of alcohol cues. Specifically, Freeman et al. (2010) found that alcohol priming increases expression of socially-sensitive topics, which they argued stems from the mere activation of implicit memory associations in which alcohol cues serve as discriminant stimuli signaling that socially disinhibited behavior will be reinforced.

Indeed, it seems likely that most people do not hold explicit alcohol outcome expectancies regarding race-based responding, and may have no awareness that they have a history of reinforcement/non-punishment for letting go of inhibitions in situations involving alcohol. Following this line of reasoning, the increase in race bias following alcohol cue exposure seen here seems likely to be rooted in a conditioning-based process like that proposed by Freeman et al. (2010), rather than in explicit expectancies or beliefs linking alcohol consumption with race prejudice.

Although this account provides a plausible reason why the presence of alcohol cues might increase race bias, it does not address specific process mechanisms linking cue exposure with such an increase. However, the current analyses testing the role of PDP-derived estimates of automatic and controlled processes as indirect predictors linking cue exposure to bias do address this issue. Consistent with predictions rooted in the idea that exposure to alcohol cues automatically activates memory associations (Kramer & Goldman, 2003; Krank et al., 2005; Wiers & Stacy, 2006) and that such activated associations can compel relevant behaviors (see Dijksterhuis & Bargh, 2001), the current analyses indicated that the increase in bias-related errors on Black-face trials (relative to White-face trials) was influenced by heavier reliance on automatic processes in the alcohol cue condition compared to the nonalcohol cue condition (see Figs. 2 and 3). These data are the first in the alcohol cue-priming literature to show a potential underlying mechanism for the effects of alcohol cue exposure on behavior.

Although it remains to be determined whether alcohol cue exposure effects on other behaviors (e.g., aggression) also stem from enhancement of automatic processes, to the extent that this effect generalizes to other behavioral domains this finding could have important theoretical implications for models of alcohol's effects on social behavior. Current models (e.g., Giancola, 2000; Steele & Josephs, 1990) and considerable empirical data (e.g., Bartholow et al., 2006, 2012; Casbon et al., 2003; Curtin & Fairchild, 2003; Fillmore et al., 1999; Schlauch et al., 2009) point to an important role for impairment of cognitive control in understanding intoxicated behavior. The possibility that activation of alcohol-related implicit associations affects the extent to which automatic processes drive relevant behaviors would suggest modification of such models.

The specific reason(s) why alcohol cue exposure effects appear driven by increased automaticity (relative to control) remain to be investigated in future work. Recent work by Hicks, Friedman, Gable and Davis (in press) provides some initial evidence that exposure to alcohol-related images can reduce the breadth of perceptual attention, resulting in effects similar to those observed following alcohol consumption, such as a focus on more immediate, proximal cues at the expense of more distal cues (see Steele & Josephs, 1990). Considered in this light, it could be that alcohol cue-exposed participants in the current study were more likely to focus on and extract threat-related information stereotypically associated with Black male relative to White male faces, thereby increasing reliance on stereotypes when making gun/tool responses.

When comparing findings from alcohol administration and alcohol priming studies, it is important to consider how placebo conditions differ from alcohol cue exposure conditions. In both cases, participants are exposed to alcohol-related cues that arguably activate relevant expectancies, and in that sense one might expect similar effects in the two conditions. However, an important difference between them is that participants in a placebo condition believe that they are consuming alcohol, whereas no such belief exists for participants simply exposed to alcohol-related cues. This difference is important because considerable research indicates that participants who consume a placebo prior to completing a challenging cognitive

task increase control in an attempt to overcome anticipated cognitive impairment from alcohol (see Bartholow et al., 2012; Fillmore & Blackburn, 2002). Thus, any enhancement of automatic processes that might result from activation of alcohol-related memory associations would be counter-acted by enhanced control. In contrast, cue-exposed participants have no expectation of impairment and therefore are unlikely to attempt to enhance their control, potentially allowing for automatic associations to have a greater influence on behavior.

In conclusion, the findings reported here suggest that people could be more likely to act upon their prejudices simply for having entered a bar, watched an alcohol advertisement, or passed relevant billboard on the freeway. These findings have important implications for social engineering campaigns designed to limit the likelihood of alcohol-related harm, going beyond attempts to limit consumption. The automatic mechanisms by which these effects occur raise questions about the design of effective intervention techniques for reduction of bias. For example, some recent work suggests that stereotypic, automatic associations can be reduced by exposure to counter-stereotypic exemplars (e.g., Dasgupta & Greenwald, 2001; Gonsalkorale, Allen, Sherman, & Klauer, 2010), and exploring such possibilities with alcohol-priming research is an important next step. Perhaps educating the public about automatic effects of alcohol-related cues on racial biases will facilitate employment of control-related processes that can counteract these effects.

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