Separate and Joint Effects of Alcohol and Caffeine on Conflict Monitoring and Adaptation

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Abstract

Rationale—Caffeine is commonly believed to offset the acute effects of alcohol, but some evidence suggests that cognitive processes remain impaired when caffeine and alcohol are co-administered.

Objectives—No previous study has investigated the separate and joint effects of alcohol and caffeine on conflict monitoring and adaptation, processes thought to be critical for self-regulation. This was the purpose of the current study.

Methods—Healthy, young adult social drinkers recruited from the community completed a flanker task after consuming one of four beverages in a 2×2 experimental design: Alcohol + Caffeine; Alcohol + Placebo caffeine; Placebo alcohol + Caffeine; or Placebo alcohol + Placebo caffeine. Accuracy, response time, and the amplitude of the N2 component of the event-related potential (ERP), a neural index of conflict monitoring, were examined as a function of whether or not conflict was present (i.e., whether or not flankers were compatible with the target) on both the previous trial and the current trial.

Results—Alcohol did not abolish conflict monitoring or adaptation. Caffeine eliminated conflict adaptation in sequential trials but also enhanced neural conflict monitoring. The combined effect of alcohol and caffeine was apparent only in how previous conflict affected the neural conflict monitoring response.

Conclusions—Together, the findings suggest that caffeine leads to exaggeration of attentional resource utilization, which could provide short-term benefits but lead to problems conserving resources for when they are most needed.

Keywords

Alcohol and caffeine; conflict monitoring; conflict adaptation; cognitive control; event-related potentials; ERPs

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The practice of mixing caffeine with alcohol has a long history and recently has become particularly popular among younger drinkers (e.g., Howland et al., 2011; Kponee et al., 2014). This practice is motivated, in part, by the belief that caffeine will attenuate the effects of alcohol, allowing the individual to "sober up" more quickly (Marczinski et al., 2013; Verster et al., 2013). Consistent with this lay belief, recent research indicates that caffeine decreases subjective feelings of intoxication (Attwood et al., 2012; Marczinski et al., 2011, 2012). However, these same studies indicate that caffeine does little to attenuate alcohol-related impairment of inhibitory control or motor coordination, implying that drinkers might feel more in control than they really are (see Brache & Stockwell, 2011; O'Brien et al., 2008).

Such findings suggest that mixing caffeine with alcohol might interfere with *cognitive control*—a set of cognitive operations that support self-regulation by allowing individuals to vary behavior adaptively according to current goals or situational demands (see Alexander & Brown, 2010; Braver, 2012). Alcohol is known to impair some aspects of cognitive control (Guillot et al., 2010), as evidenced by increased response conflict in laboratory tasks that require inhibition of prepotent but goal-incompatible responses (e.g., Stroop or go/no-go tasks; Bartholow et al., 2006; Curtin & Fairchild, 2003; Fillmore & Vogel-Sprott, 2000; Weafer & Fillmore, 2004), and by reduced behavioral adaptation (Bartholow et al., 2012; Ridderinkhof et al., 2002) and delayed recovery of control (Bailey et al., 2014) following errors. This impairment of cognitive control is often cited as the cause of alcohol-related increases in risk-taking (Fromme et al., 1997; George et al., 2005). Some reports suggest that caffeine might counter alcohol's impairment of cognitive performance, particularly by speeding up RT (Mackay et al., 2002; Marczinski & Fillmore, 2003, 2006), but findings have been inconsistent (Fudin & Nicastro, 1988; Verster et al., 2012). The current study went beyond characterizing the drugs' effects on overall response accuracy and RT (which do not directly address the ability to dynamically adapt behavior as situations dictate) by testing their separate and combined effects on a hallmark of cognitive control, namely, the ability to adapt to conflict.

The essence of cognitive control is the ability to flexibly adjust performance in response to changing situational demands. According to the *strategic adjustment hypothesis* (Gratton et al., 1988, 1992), an optimal balance between resource utilization and conservation is struck by determining, on a moment-by-moment basis, the extent to which resource-intensive cognitive control is required in order to maintain desired levels of performance. Situations in which conflict (e.g., between goal-relevant stimuli and goal-irrelevant distractors) is occurring or can be anticipated call for a focused mode of processing (i.e., heightened cognitive control). By contrast, in situations where conflict is low or unlikely, adequate performance can be maintained using a less resource-demanding parallel mode of processing, in which attention control can be relaxed.

Applied to laboratory task performance, the strategic adjustment hypothesis (and the related conflict-monitoring hypothesis; see Botvinick et al., 2001, 2004) predicts that encountering conflict on any given trial results in control adjustments, which reduce the influence of conflict on subsequent trials. First described by Gratton and colleagues (Gratton et al., 1992), this *conflict adaptation effect* (CAE) refers to the finding that responses on high-

conflict trials (e.g., incompatible trials in a flanker task, in which the central target stimulus and the flanker stimuli call for opposing responses; HH**S**HH) are faster and more accurate following other incompatible trials (iI trials) than following low-conflict, compatible trials (cI; trials in which the target and flankers are mapped to the same response; SS**S**SS). Likewise, compatible trial responses are faster following other compatible trials (cC) than following incompatible trials (iC). In other words, the size of the *compatibility effect* (CE; the difference between incompatible and compatible trial RTs) is reduced following the experience of conflict, a phenomenon linked to strategic adjustments in cognitive control (Blais et al., 2014; Braver, 2012; Gratton et al., 1992).

Psychophysiological research has identified the medial prefrontal cortex (mPFC) as the neural source of conflict monitoring (see Carter & van Veen, 2007). In particular, event-related potential (ERP) studies consistently show that the amplitude of the fronto-central N2, a transient negativity emerging 200–350 ms after stimulus onset and known to be generated in mPFC (see van Veen & Carter, 2002), is larger to incompatible than to compatible trials in conflict tasks (see Larson et al., 2014). Moreover, neuroimaging and ERP data consistently show that N2 amplitude and other indices of mPFC activation are greatest for cI compared to iI trials (Botvinick et al., 1999; Clayson & Larson, 2011; Forster et al., 2010), and the behavioral CAE has been linked to dynamic, trial-level shifts in mPFC activation (Forster et al., 2010; Kerns, 2006; Kerns et al., 2004). In the current study, variability in N2 amplitude following compatible and incompatible flanker arrays was used to characterize effects of alcohol and caffeine on conflict monitoring and adaptation.

To date, no research has investigated effects of alcohol or caffeine on the CAE. Differences in their pharmacodynamics suggest the two drugs might have opposing effects. In particular, the fact that caffeine is considered an anxiogenic agent (Fredholm et al., 1999) whereas alcohol has anxiolytic properties (see Sayette, 1999) has implications for their effects on conflict adaptation. According to recent models (see Cavanagh & Shackman, 2015; Inzlicht et al., 2015), the averseness of conflict (Fritz & Dreisbach, 2013; Hajcak & Foti, 2008) plays an integral role in motivating adaptive control adjustments. Previous research (Tieges et al., 2004) has shown that caffeine increases mPFC activation to errors, an extreme form of conflict, consistent with other work showing that increased anxiety-like symptoms exaggerate conflict monitoring (e.g., Weinberg et al., 2012). In contrast, alcohol reduces mPFC activation when errors occur (Bailey et al., 2014; Ridderinkhof, et al., 2002), an effect directly tied to its anxiolytic effects (Bartholow et al., 2012). The drugs' effects on conflict monitoring appear to have implications for conflict management. Whereas caffeine reduces the CE during conflict task performance (Brunyé et al., 2010), alcohol appears to exacerbate effects of conflict, leading to impaired performance adjustment following errors (Bailey et al., 2014; Bartholow et al., 2012; Ridderinkhof, et al., 2002). Considered together, such findings suggest caffeine may counteract the effects of alcohol on conflict adaptation and its neural underpinnings.

However, it is important to underscore that these previous studies have focused mainly on post-error adjustments—that is, the recovery of control once it has failed—not on control adjustments that occur in response to successfully resolved conflict. Though related, post-error and post-conflict control adjustments are not identical phenomena (see Forster & Cho,

2014). Recent research shows that alcohol strongly affects post-error adjustments but has little impact on sequences of correct-response trials in conflict tasks (Bailey et al., 2014). Thus, it remains unclear whether alcohol, caffeine or their combination will adversely affect conflict adaptation.

Participants completed a flanker task after consuming one of four beverages (Alcohol + Caffeine [AC]; Alcohol + Placebo caffeine [AP]; Placebo alcohol + Caffeine [PC]; or Placebo alcohol + placebo caffeine [PP]). The flanker is a prototypical cognitive control task with a trial structure that permits examination of conflict adaptation and adjustment (Gratton et al., 1992). We hypothesized that caffeine would enhance conflict monitoring (Tieges et al., 2004), and that this effect might counteract any impairment of conflict adaptation by alcohol (Bailey et al., 2014; Ridderinkhof et al., 2002). Moreover, based on the strategic adjustment and conflict-monitoring hypotheses (Botvinick et al., 2001; Gratton et al., 1992) and previous findings linking dynamic shifts in N2 amplitude to behavioral conflict adaptation (Forster et al., 2010; Clayson & Larson, 2011), we predicted that beverage-related differences in behavioral conflict adaptation would correspond to similar differences in N2 amplitude.

Materials and Methods

Participants

Ninety-four adults (42 women), ages 21–32 years ($M = 23$, $SD = 2$), were recruited from a Midwestern community using email circulars, advertisements, and posted flyers.[1] Eligibility was determined using a structured telephone interview. Individuals who self-reported conditions that contraindicate alcohol or caffeine administration (pregnancy; abstention from alcohol; history of alcohol or drug treatment or serious mental or physical illness; prescription medication other than oral contraception) or who reported history of head trauma or neurological disorder were excluded. Participants were not subjected to a clinical interview to determine previous or current alcohol dependence or other psychiatric conditions, nor were they assessed for the presence of global internalizing or externalizing psychopathology. Thus, it is possible that some individuals with psychiatric histories participated in the study. However, given random assignment of participants to beverage groups we assume that any such conditions were represented equally across those groups. Eligible individuals attended individual lab appointments, for which they were paid $12/hour.

Beverage Administration

Participants were randomly assigned to one of four beverage conditions: AC ($n = 21$ [12 women]); AP ($n = 26$ [11 women]); PC ($n = 20$ [8 women]); PP ($n = 27$ [11 women]). Beverage condition was manipulated between subjects due to concerns with the viability of placebo effects and practical constraints on participants' willingness to return to the lab

[1]An additional 48 individuals participated in this study, but their data are not included in this report because they were assigned to beverage conditions (e.g., alcohol with no caffeine placebo) that are not relevant for testing the current hypotheses. Note, too, that despite use of the same behavioral task and alcohol dosing procedure, the current study is based on an entirely different sample than the one used for the study reported in Bailey et al. (2014).

repeatedly for additional sessions. All beverages were composed of one part alcohol/placebo alcohol to four parts caffeine/placebo mixer. Participants in the alcohol conditions expected to receive alcohol and consumed 100-proof Smirnoff® vodka (mixed with tonic water), dosed so as to produce a peak breath alcohol concentration (BrAC) of .08%. The placebo alcohol conditions expected alcohol and consumed a solution of 10% vodka mixed with tonic water. This solution was poured from a vodka bottle in view of participants to bolster the placebo manipulation. The caffeine conditions expected caffeine and consumed a dose of 4 mg/kg of tasteless, anhydrous caffeine powder dissolved in carbonated tonic water. The caffeine dose was selected to approximate the amount of caffeine in 2–3 commercial energy drinks. The placebo caffeine conditions expected to receive caffeine and consumed tonic water. Alcohol dosing was calculated based on gender, age, and the duration of the drinking period (15 min; Watson, 1989). Beverages were divided into three isovolumetric drinks, each consumed within 5 minutes. A 15-min absorption period followed completion of the final drink. BrAC values and subjective intoxication ratings are presented in Figure S1. All doses were administered in a double-blind manner.

Materials

Flanker task—As in previous research (Bailey et al., 2014; Ridderinkhof et al., 2002), on each of 800 experimental trials (8 blocks of 100 trials) participants viewed arrow arrays and were to identify the direction of the central arrow (right or left) via button press. On compatible trials, the flanker (peripheral) and target arrows pointed in the same direction (i.e., →→→→→ or ←←←←←); on incompatible trials, the flankers and target pointed in opposite directions (i.e., →→←→→ or ←←→←←). Compatible and incompatible arrays were presented pseudorandomly, with the constraints that they occurred with equal probability and left- and right-hand responses occurred equally often. Arrays were presented for 100 ms with an unlimited response window followed by a randomly-varying inter-trial interval (1100 or 1500 ms). Participants completed 100 practice trials prior to the experimental trials.

Electrophysiological recording—The electroencephalogram (EEG) was sampled at 1,000 Hz (filtered online at 0.05–40 Hz) with 32 tin electrodes placed in standard locations (American Encephalographic Society, 1994). Scalp electrodes were referenced online to the right mastoid; an average mastoid reference was derived offline. Impedance was kept below 5 kΩ. Blinks were removed from the EEG off-line using a regression-based procedure. EEG data were segmented into epochs of −100 to 1200 ms of post-stimulus activity, scanned for artifacts (trials containing voltage deflections of +/− 100 μV [< 2% of all trials] were discarded), and averaged according to stimulus and participant conditions. Visual inspection of the grand average waveforms indicated that the N2 occurred ~220–320 ms post-stimulus and was most prominent at midline frontal and fronto-central scalp sites (electrodes Fz and FCz). Thus, the N2 was defined as the average amplitude 220–320 ms post-stimulus at those two electrodes.[2]

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[2]Note that including N2 data from a broader array of electrodes (see Bailey et al., 2014) does not change any of the findings, and thus for simplicity we opted to focus analyses on the sites where N2 effects were most pronounced.

Procedure

Participants abstained from alcohol for 24 hr and other drugs for 48 hr, and fasted from food and caffeine for at least 4 hr, prior to the session. Upon arrival, participants provided informed consent, completed affidavits assessing compliance with pre-session protocols, and provided a breath sample to ensure initial sobriety. Women provided a urine-stream pregnancy test (all were negative), and men voided the bladder prior to continuing. Participants then were escorted to an EEG acquisition suite where experimenters placed and tested the electrodes. Next, participants completed the practice trials, after which an experimenter (unaware of beverage contents) mixed and served their beverage. After beverage consumption and absorption, participants completed the flanker task, after which electrodes were removed and participants were debriefed. Participants who had not consumed alcohol were then dismissed; those who had were retained in the lab until their BrAC descended to 0.02%.

Data Analysis

Behavioral Data

Four participants' data ($n = 1$ AC; $n = 3$ PP) were excluded due to noncompliance with pre-experiment protocols ($n = 1$) or computer error ($n = 3$). To eliminate very slow and fast guessing responses (< 1% of trials), RT analyses were limited to responses made 100–2000 ms after target onset. Still, RT distributions remained skewed, and therefore remaining outliers (values > 2 *SD* from an individual's mean RT in any condition; 1.9% of trials) were replaced with the 2 *SD* value (see Ratcliff, 1993).[3] To normalize variance across conditions, accuracy data (proportion correct) were transformed using arcsine of the square root. For ease of interpretation, untransformed means are presented in the text and Table 1. In order to avoid confounding conflict adaptation effects with control recovery processes following errors, analyses of accuracy and RT data were limited to trials preceded by at least two correct responses (see Bailey et al., 2014). Accuracy and RT data meeting this criterion (see Figure 1 and Table 1) were submitted to separate 2 (Alcohol: yes, no) × 2 (Caffeine: yes, no) × 2 (Previous trial: compatible, incompatible) × 2 (Current trial: compatible, incompatible) mixed factorial analyses of variance (ANOVAs) with repeated measures on the latter factors.

ERP Data

Due to equipment failure, EEG data were not collected for 14 participants ($n = 3$ AC; $n = 3$ AP; $n = 4$ PC; $n = 4$ PP). Data from an additional nine participants ($n = 3$ AC; $n = 4$ AP; $n = 2$ PP) were excluded from analyses due to excessive EEG artifact (i.e., > 25% of trials exceeded the artifact rejection criteria). N2 amplitudes were analyzed with hierarchical linear modeling (HLM), which provides several advantages over repeated-measures ANOVA for analyzing psychophysiological data (see Page-Gould, in press), including robustness to missing values, simultaneous estimation of both within- and between-

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[3]Given that this method might not adequately eliminate skew from the RT distributions, RT analyses were replicated using natural log-transformed data. These analyses produced findings virtually identical to those we report, and therefore the trimmed, untransformed analyses were retained for ease of interpretation.

participants effects (see Bryk & Raudenbush, 1992), and the ability to specify separate error terms at each level of nesting, all of which increases statistical power, particularly when sample sizes are modest. Initial analyses were carried out using a 2 (Alcohol: yes, no) × 2 (Caffeine: yes, no) × 2 (Previous trial: compatible, incompatible) × 2 (Current trial: compatible, incompatible) × 2 (Coronal: frontal-central, frontal) HLM, analogous to the behavioral data models. Additional comparisons tested associations between N2 amplitude and behavioral performance.

Results

Accuracy

Figure 1a presents accuracy rates in each beverage group as a function of current trial and previous trial flanker compatibility. Accuracy suffered when the current trial was incompatible (versus compatible) and also when the previous trial was compatible (versus incompatible), as indicated by main effects of current trial, $F(1, 86) = 262.7$, $p < .001$, $\eta_p^2 = .75$, and previous trial, $F(1, 86) = 52.9$, $p < .001$, $\eta_p^2 = .38$. Consistent with the conflict adaptation hypothesis (Gratton et al., 1992), the compatibility effect on the current trial was larger when the previous trial was compatible (i.e., low conflict) than when it was incompatible (i.e., high conflict), as indicated by a significant Previous trial × Current trial interaction, $F(1, 86) = 8.73$, $p = .004$, $\eta_p^2 = .09$. Specifically, participants were more accurate on iI trials ($M = 0.893$, $SD = 0.11$) compared to cI trials, ($M = 0.864$, $SD = 0.12$), $F(1, 86) = 49.2$, $p < .001$, $\eta_p^2 = .36$, and on iC ($M = 0.984$, $SD = 0.03$) compared to cC trials ($M = 0.978$, $SD = 0.03$), $F(1, 86) = 9.59$, $p = .003$, $\eta_p^2 = .10$.

Of greater interest was whether this CAE differed by beverage, and indeed the analyses showed a significant Previous trial × Current trial × Caffeine interaction, $F(1, 86) = 4.34$, $p = .040$, $\eta_p^2 = .05$. To simplify this complex interaction, we computed accuracy compatibility effects (i.e., incompatible trial accuracy – compatible trial accuracy, representing the 'Current trial' effect in the interaction) as a function of previous trial compatibility, separately for each beverage group (see Figure 1b). This method provides a means to understand conflict adaptation, in that the compatibility effect typically is reduced on post-incompatible trials (black bars in Figure 1b) relative to post-compatible trials (open bars in Figure 1b). Inspection of the means in Figure 1b suggests this typical pattern was more evident in the groups where caffeine was not consumed (AP and PP) than in the groups where caffeine was consumed (AC and PC). In other words, caffeine appears to have eliminated the conflict adaptation effect in response accuracy. Focused contrasts confirmed that the compatibility effects for post-compatible and post-incompatible trials did not differ for the caffeine groups (AC and PC), $M = .0175$; $F(1, 86) = 0.34$, $p = .56$; $\eta_p^2 = .00$, but did differ for the no-caffeine groups (AP and PP), $M = .0325$; $F(1, 86) = 14.3$, $p < .001$; $\eta_p^2 = .14$).

The only other significant effect in this analysis was a main effect of alcohol, $F(1, 86) = 8.57$, $p = .004$, $\eta_p^2 = .09$. Participants who consumed alcohol were less accurate overall ($M = .923$) than those who did not ($M = .955$). Importantly, consuming caffeine did not ameliorate alcohol's impairment of accuracy (Alcohol × Caffeine interaction $F < 1$).

RTs

Figure 1c presents RTs in each beverage group as a function of current trial and previous trial flanker compatibility. As with the accuracy data the RTs were strongly affected by both current trial and previous trial flanker compatibility ($Fs[1, 86] = 663.0$ and 8.46, $\eta_p^2 = .88$ and $.09$, ps .004, respectively), and the compatibility effect in RT was larger overall on trials following conflict compared to trials following no conflict, $F(1, 86) = 8.55$, $p < .001$, $\eta_p^2 = .09$. There were also main effects of both alcohol, $F(1, 86) = 3.84$, $p = .053$, $\eta_p^2 = .04$, and caffeine, $F(1, 86) = 4.63$, $p = .034$, $\eta_p^2 = .05$. The effects of the two drugs essentially opposed one another—consuming alcohol slowed RT ($M = 420.7$ ms) relative to no alcohol ($M = 400.8$ ms), whereas consuming caffeine speeded RT ($M = 399.8$ ms) relative to no caffeine ($M = 421.7$ ms). There was no Alcohol × Caffeine interaction ($F < 1$).

A significant Previous trial × Current trial × Caffeine interaction indicated that the CAE differed according to beverage contents, $F(1, 86) = 4.23$, $p = .043$, $\eta_p^2 = .05$. As with the accuracy data, we simplified this complex interaction by computing RT compatibility effects (i.e., incompatible RT – compatible RT) as a function of previous trial compatibility, separately for each beverage group (see Figure 1d). Focused contrasts within each beverage group showed significant conflict adaptation in the AP ($F[1, 25] = 6.15$, $p = .020$, $\eta_p^2 = .20$) and PP groups ($F[1, 23] = 4.19$, $p = .052$, $\eta_p^2 = .15$), but not in the AC or PC groups ($Fs < .82$, $ps > .376$, $\eta_p^2s < .02$). None of the interactions with alcohol approached significance ($Fs < 1.23$, $ps > .27$).

Close inspection of the data in Figure 1d suggests the caffeine effect on conflict adaptation was due to compatibility effects on post-compatible trials being smaller in the caffeine groups (AC and PC; $Ms = 50.4$ and 50.2 ms, respectively) compared to the no-caffeine groups (AP and PP; $Ms = 61.3$ and 63.8 ms, respectively). A one-way ANOVA confirmed this difference, $F(1, 86) = 7.85$, $p = .006$, $\eta_p^2 = .06$. In contrast, the magnitude of compatibility effects on post-incompatible trials did not differ for the caffeine ($M = 48.7$ ms) and no-caffeine groups ($M = 53.6$), $F(1, 86) = 1.09$, $p = .30$, $\eta_p^2 = .003$.

N2 Amplitude

Figure 2 presents ERP waveforms elicited by compatible and incompatible trials as a function of beverage condition and previous trial compatibility. As with the behavioral measures the N2 was sensitive to both current trial and previous trial flanker compatibility ($Fs[1, 448] = 78.6$ and 10.5, ps .001, respectively). Previous research has shown that conflict monitoring as reflected in N2 amplitude adapts to the experience of conflict (see Clayson & Larson, 2011; Forster et al., 2010). Consistent with this idea, the analysis showed a Previous trial × Current trial interaction, $F(1, 448) = 5.62$, $p = .018$. To understand this interaction, we computed separate models comparing N2 amplitude for current-compatible and current-incompatible trials as a function of previous trial compatibility. These models showed that N2 amplitude elicited by compatible arrays did not differ according to previous trial compatibility ($Ms = 2.80$ and 2.91 μV for previous-compatible and previous-incompatible, respectively), $t(448) = -0.61$, $p = .543$. However, N2 amplitude elicited by incompatible arrays was significantly greater (less positive) when the previous trial was compatible ($M = 1.41$ μV) than when it was incompatible ($M = 2.11$ μV), $t(448) = 7.95$, $p < .$

001. Stated differently, the CE in N2 amplitude was larger for post-compatible trials (M = −1.39 μV) than for post-incompatible trials (M = −0.80 μV), $t(448)$ = 2.37, p = .018.

Of greater interest here was whether these effects varied according to beverage contents. A significant Alcohol × Caffeine interaction, $F(1, 448)$ = 4.87, p = .028, was qualified by a significant Alcohol × Caffeine × Previous trial interaction, $F(1, 448)$ = 5.13, p = .024, indicating that the effect of previous trial compatibility on current trial N2 amplitude varied according to both alcohol and caffeine intake. Follow-up models tested the effect of caffeine on the N2 difference (previous compatible vs. previous incompatible) separately for the alcohol and no-alcohol groups (see Figure 3a). These models showed that, relative to the other beverage groups, the combination of alcohol and caffeine significantly reduced N2 amplitude following conflict. When alcohol was *not* present (groups PC and PP) the effect of caffeine was minimal, as indicated by a nonsignificant Caffeine × Previous trial interaction, $F(1, 243)$ = 1.07, p = .302. However, when alcohol *was* present (groups AC and AP) the Caffeine × Previous trial interaction was significant, $F(1, 229)$ = 12.55, p < .001, owing to a much smaller (less negative) N2 on post-incompatible (M = 4.10 μV) than post-compatible trials (M = 2.64 μV) in the AC group, $t(229)$ = −4.53, p < .001; no such difference emerged in the AP group (Ms = 1.78 and 1.83 μV, respectively).

Finally, the analysis showed a marginally nonsignificant Alcohol × Caffeine × Current trial interaction, $F(1, 448)$ = 3.10, p = .079. This interaction was deconstructed by computing separate Caffeine × Current trial models for the alcohol and no-alcohol groups (see Figure 3b). The compatibility effects in the alcohol groups did not differ (Ms = −0.70 and −1.06 μV in the AP and AC groups, respectively), as indicated by the lack of a Caffeine × Current trial interaction (F < 1). However, in the no-alcohol groups the compatibility effect was larger in the PC group (M = −1.88 μV) compared to the PP group (M = −0.74 μV), as indicated by a significant Caffeine × Current trial interaction, $F(1, 243)$ = 13.55, p < .001.

Relating Brain and Behavioral Responses

To the extent that variability in neural conflict monitoring contributed to conflict adaptation effects in RT, we should expect correspondence between the RT and N2 data in terms of the effects of the beverage manipulations. In particular, three patterns should emerge in comparing these data. First, in theory conflict is better managed—in this case, indicated by smaller RT compatibility effects—when the neural conflict-monitoring system is more strongly engaged (see Carter & van Veen, 2007). Recall that, compared to the other groups the caffeine groups (AC and PC) experienced smaller RT compatibility effects when conflict was low on the previous trial (previous-compatible trials), but when conflict was high on the previous trial (previous-incompatible trials) the magnitude of RT compatibility effects did not differ across groups (Figure 1d). To the extent that this beverage-related variability in conflict adaptation derives from dynamic shifts in N2 conflict-monitoring, we should find that the post-compatible CE in N2 amplitude was larger for groups who consumed caffeine (M = −2.01 μV) than for groups who did not (M = −0.80 μV)—this difference was significant, $t(204)$ = 2.87, p = .004—and that the post-incompatible CE in N2 amplitude did not differ for the caffeine (M = −1.27 μV) and no-caffeine groups (M = −0.65 μV); this difference was not significant, $t(204)$ = 1.39, p = .166. Finally, if sensitivity in the conflict

monitoring system is associated with adaptive behavior (see Forster et al., 2010; Kerns, 2006), we might expect the magnitude of the compatibility effects in RT and N2 amplitude to correlate across our entire sample, which they did, $r = .29$, $p = .020$.

Discussion

A number of previous studies have documented effects of alcohol, caffeine and their combination on behavioral and neural measures related to cognitive performance. In large part the general performance findings in the current study are consistent with those previous studies. Alcohol decreased response accuracy (e.g., Jennings et al., 1976) and increased RT (e.g., Tzambazis & Stough, 2000), whereas caffeine decreased RT (e.g., Smith et al., 1977). However, there was no evidence that caffeine ameliorated the deleterious effects of alcohol on these basic performance measures (also see Oborne & Rogers, 1983). Also consistent with previous findings, at a descriptive level alcohol generally blunted (e.g., Rohrbaugh et al., 1987) and caffeine generally enhanced ERP amplitude (e.g., Kawamura et al., 1996), the latter finding appearing broadly consistent with the general arousal hypothesis of caffeine's effects on neural responding (Barry et al., 2014; Lorist et al., 1994a, 1994b).

While reassuring, these replications do not directly address the primary aim of this study, which was to understand how alcohol and caffeine, separately and in combination, affect the adaptive control of behavior in response to conflict. In this regard, the current data make a number of contributions. In particular, the combination of alcohol and caffeine eliminated the typical CAE in both accuracy and RT, whereas consuming alcohol without caffeine did not. This comparison is relevant to concerns that the combination of alcohol and caffeine might ultimately prove more hazardous than consumption of alcohol alone (O'Brien et al., 2008). However, this effect was not specific to the combination of alcohol and caffeine— participants who consumed caffeine without alcohol also showed no conflict adaptation.

Close inspection of the data showed that caffeine eliminated the typical CAE not because the drug reduced control following conflict, but rather because caffeine significantly improved performance when conflict on the previous trial was low (i.e., following compatible trials). In the context of the strategic adjustment hypothesis (Gratton et al., 1992), caffeine appears to eliminate the need to adjust attention following conflict, possibly because attention control is maximally deployed even when conflict has not occurred. That is, caffeine appears to enhance sustained attentional focus by preventing the relaxation of attention control. This interpretation is consistent with other findings indicating that caffeine enhances focused attention and vigilance performance (Hewlett & Smith, 2007; Lorist & Tops, 2003). On the one hand, the effect of caffeine in the current study could be considered adaptive, in that sustained maintenance of task goals and attentional focus should provide the basis for better performance. On the other hand, prolonged deployment of focused attention requires considerable cognitive resources, and arguably the most optimal strategy for performance is one in which deployment of resource-intensive attentional focus is offset by opportunities for resource conservation (see Bartholow et al., 2005; Braver, 2012).

The fact that alcohol alone did not eliminate conflict adaptation could be considered surprising given previous demonstrations that alcohol impairs control-related processes

(Guillot et al., 2010), including post-error adjustment (Bartholow et al., 2012; Ridderinkhof et al., 2002). However, the current findings are consistent with those of another recent report (Bailey et al., 2014), which showed that alcohol's deleterious effects on control adjustment emerge only once control has failed (i.e., following errors). This situation is analogous in the current study to previous trials involving conflict versus those that did not. Bailey et al. found that, among participants who consumed alcohol, conflict monitoring (as indicated by N2 amplitude) was intact on trials that followed correct responses but was compromised on trials following errors. Here, although alcohol alone did not appear to disrupt conflict monitoring, the combination of alcohol and caffeine significantly reduced N2 amplitude following high-conflict, incompatible trials (see Figure 3a). Interestingly, however, the AC group still showed significant neural conflict effects. Indeed, there was some suggestion in the N2 data that alcohol alone (AP) was associated with the smallest neural conflict response and caffeine alone (PC) showed the largest response, with the combination of the two (AC) falling in-between (see Figure 3b). This pattern is consistent with the possibility, suggested previously, that the pharmacodynamics of alcohol (anxiolytic) and caffeine (anxiogenic) might produce offsetting effects on conflict-related neural responding (see Inzlicht et al., 2015).

The patterns in the N2 data further suggest that, despite caffeine having apparently enhanced attentional focus throughout the task, it did not produce more efficient conflict monitoring. Typically, increasing sustained attention reduces the need for reactive adjustments in control because conflict is better anticipated and therefore requires a smaller-magnitude mPFC response to overcome it (see Braver, 2012). Here, participants who consumed caffeine experienced the largest compatibility effects in the N2, suggesting the most pronounced mPFC response to conflict. Thus, consuming caffeine led to both exaggerated sustained attention and increased sensitivity to conflict, suggesting a nonoptimal deployment of cognitive resources. This is generally consistent with the conclusions of Lorist and Tops (2003), who characterized caffeine's effects on cognition in terms of its impact on mechanisms involved in regulation of energy expenditure.

The current study had some limitations. First, although the overall number of participants was large by the standards of both alcohol challenge and psychophysiological studies, the ambitious goals of the project—to compare pharmacological effects of both drugs and their combination—required that participants be divided into several groups, thereby resulting in relatively small numbers of individuals per group. The between-groups differences reported here should be replicated using larger samples. Additionally, the constraints of ethical laboratory protocol precluded administration of alcohol and caffeine doses as large as those frequently self-administered by young people, limiting the generalizability of our conclusions.

The current study also suggests additional avenues for future research. As this was the first study to investigate effects of alcohol and caffeine on conflict adaptation and its neural underpinnings, we chose to use the behavioral and electrophysiological data in ways that are typical for this literature. There are numerous other ways to use the kind of data we obtained, however, which could prove useful in future studies. For example, rather than trimming or transforming the RT data to remove or modify extremely slow responses prior

to mean-based modeling, researchers could examine RT distributional properties using ex-gaussian parameters (e.g., Lacouture & Cousineau, 2008), or could count the number of extremely long RTs as a way of modeling beverage effects on the frequency of attentional lapses (see Seli et al., 2012). In addition, the EEG data could be used to provide measures of global field power across the entire scalp or could be decomposed into frequency band oscillations, which in theory provide a different level of information on cognitive processes than do mean component amplitudes (Fingelkurts & Fingelkurts, 2014).

In conclusion, the current study is the first to test the separate and joint effects of alcohol and caffeine on the CAE, which indexes an important function of cognitive control. Alcohol did not impair conflict adaptation; consuming caffeine with alcohol eliminated conflict adaptation by reducing the influence of conflict following compatible trials, as indexed by increased neural conflict monitoring. Whether this combined effect is helpful or harmful to self-regulation in the long run is open to debate.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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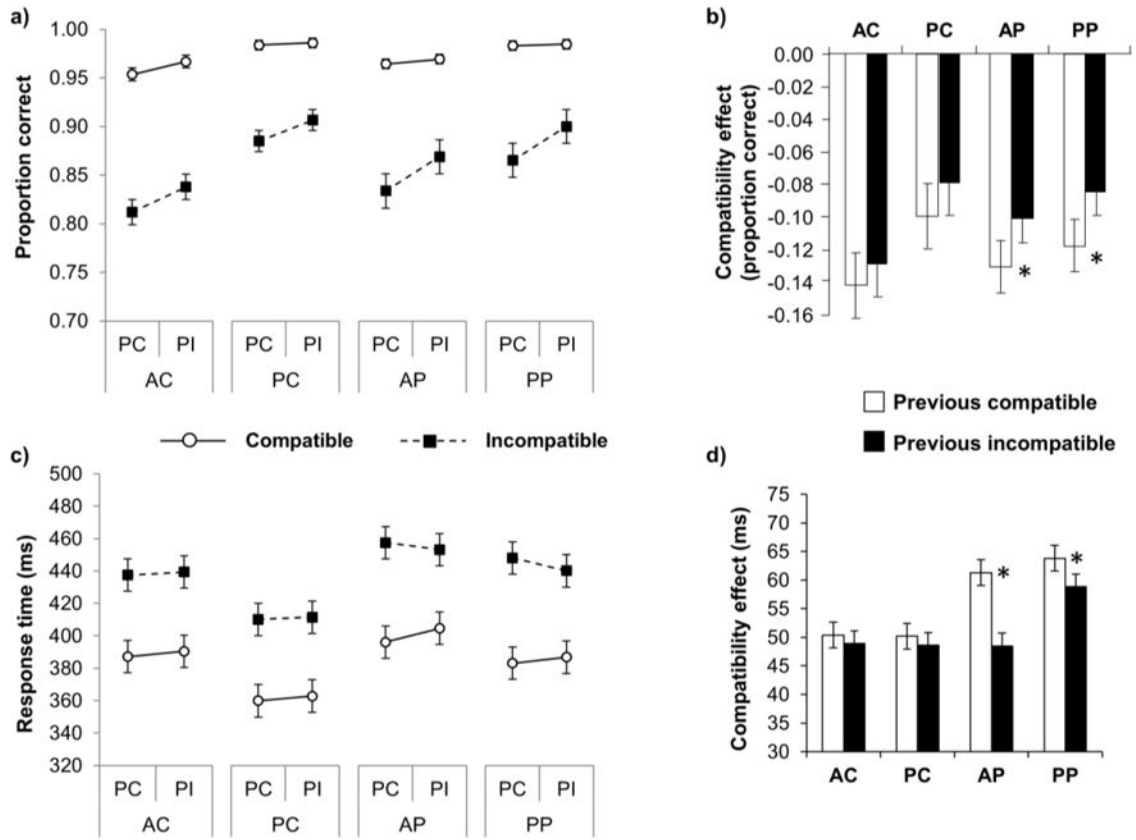
Fig. 1.

Mean untransformed accuracy (a) and response time (c) by beverage group, previous trial compatibility, and current trial compatibility. PC = post-compatible; PI = post-incompatible. Accuracy compatibility effects as a function of previous trial compatibility, separately for each beverage group (b). More negative values represent larger compatibility effects, corresponding to more interference from incompatible flanker stimuli. Response time compatibility effects as a function of previous trial compatibility, separately for each beverage group (d). Larger (more positive) values represent larger compatibility effects, corresponding to more interference from incompatible (relative to compatible) flanker stimuli. Error bars represent standard error of the mean; asterisks indicate significant within-group mean differences, $p < .05$. AC = alcohol + caffeine; PC = placebo alcohol + caffeine; AP = alcohol + placebo caffeine; PP = placebo alcohol + placebo caffeine.
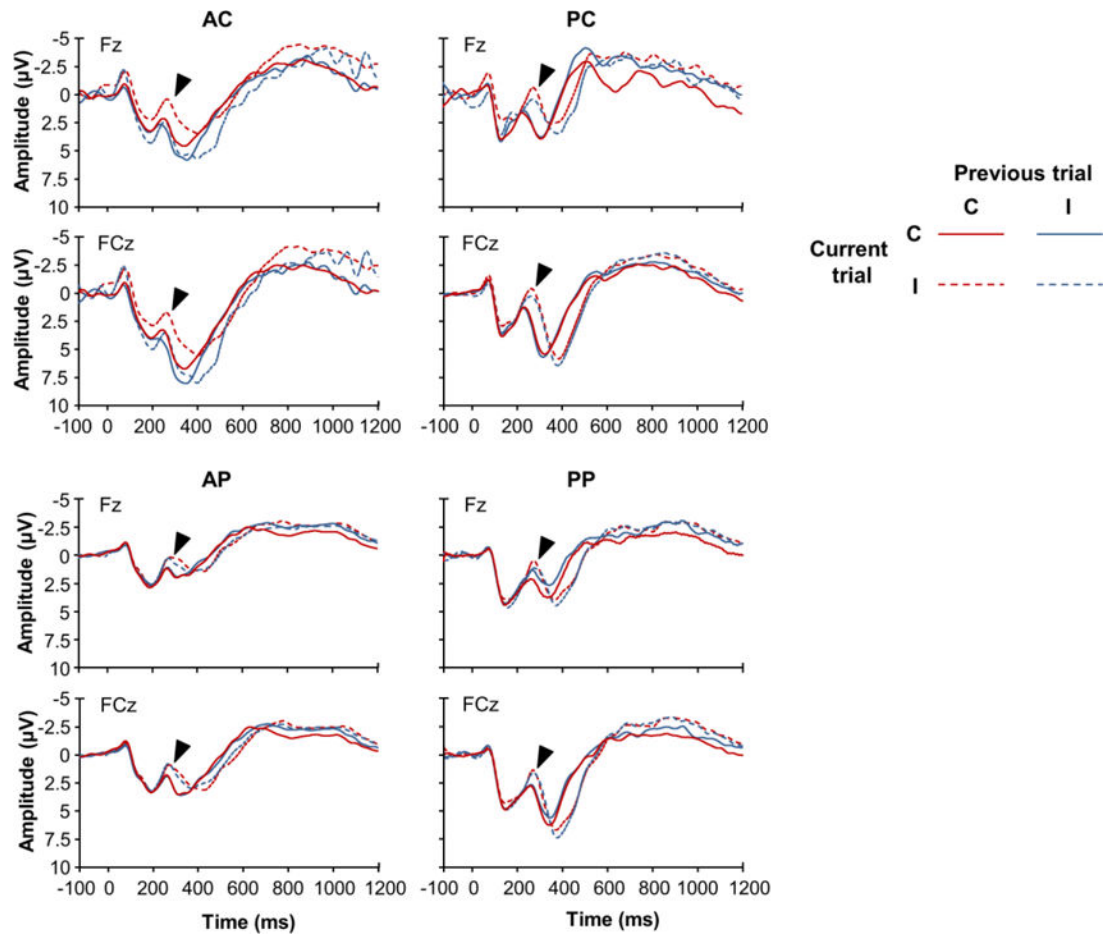
Fig. 2.
ERP waveforms measured at midline frontal (Fz) and fronto-central (FCz) electrodes elicited by compatible (C) and incompatible trials (I), as a function of previous trial compatibility, in each beverage group. AC = alcohol + caffeine; PC = placebo alcohol + caffeine; AP = alcohol + placebo caffeine; PP = placebo alcohol + placebo caffeine. The N2 is the prominent negative-going voltage deflection occurring around 250 ms post-stimulus onset, indicated by the black arrowheads in each panel.
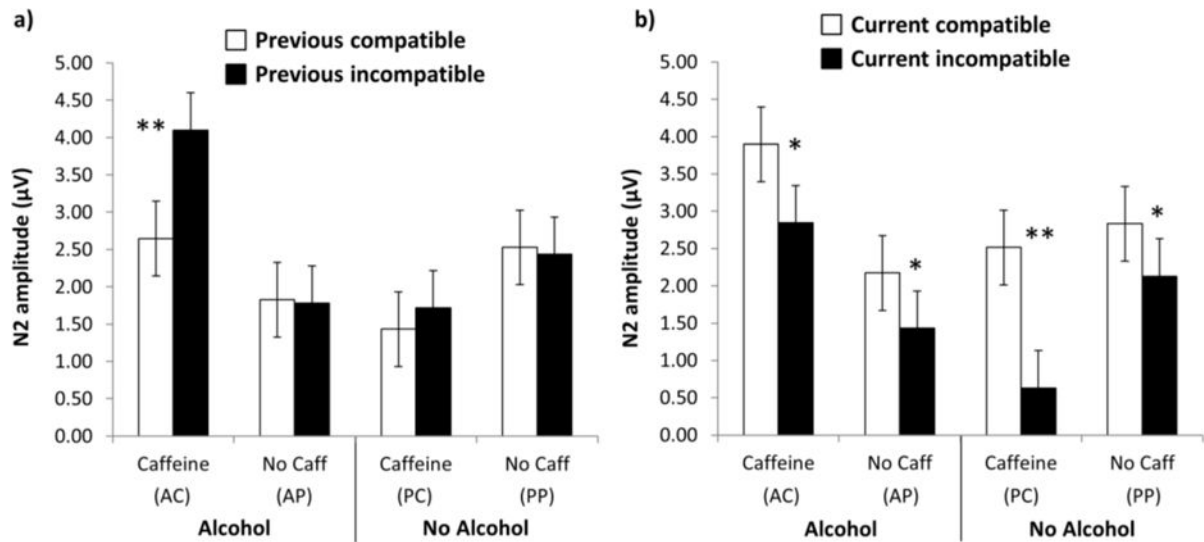
Fig. 3.
Mean N2 amplitude values for each beverage group as a function of previous trial (a) and current trial (b) flanker compatibility. Vertical bars indicate standard error of the mean; asterisks indicate significant within-group mean differences, *p* < .05; **p* < .01. More positive amplitude values indicate smaller (less negative) N2 responses. AC = alcohol + caffeine; PC = placebo alcohol + caffeine; AP = alcohol + placebo caffeine; PP = placebo alcohol + placebo caffeine.

Table 1

Mean Accuracy, RT (ms), and N2 amplitude (μV) as a Function of Beverage, Previous Trial, and Current Trial Compatibility.

Beverage Condition	Previous Trial	Current Trial					
		Accuracy		RT		N2	
		C	I	C	I	C	I
AC	C	0.95	0.81	387	437	3.30	1.56
		0.05	*0.20*	*36*	*31*	*3.37*	*3.36*
	I	0.97	0.84	390	439	3.44	2.92
		0.04	*0.19*	*38*	*30*	*3.78*	*3.78*
AP	C	0.96	0.83	396	457	1.77	1.05
		0.03	*0.13*	*48*	*59*	*2.34*	*2.51*
	I	0.97	0.87	405	453	1.77	0.93
		0.02	*0.11*	*55*	*56*	*2.79*	*2.17*
PC	C	0.98	0.88	360	410	1.99	−0.29
		0.02	*0.08*	*48*	*48*	*1.92*	*1.71*
	I	0.99	0.91	363	411	2.12	0.10
		0.02	*0.07*	*48*	*45*	*1.77*	*1.82*
PP	C	0.98	0.87	383	448	2.50	1.63
		0.01	*0.09*	*52*	*55*	*2.57*	*2.62*
	I	0.99	0.90	387	440	2.24	1.79
		0.01	*0.07*	*50*	*53*	*2.50*	*2.51*

Note. C = compatible; I = incompatible; AC = alcohol + caffeine; AP = alcohol + placebo caffeine; PC = placebo alcohol + caffeine; PP = placebo alcohol + placebo caffeine. Numbers in italics are standard deviations.