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Why are caffeinated alcoholic beverages especially risky?

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HIGHLIGHTS

- More caffeine consumption was associated with more alcohol consumption and adverse consequences.
- These relationships were not explained by caffeine's influence on subjective intoxication.
- High doses of caffeine served as an independent risk factor for heavier and more harmful alcohol use.

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ABSTRACT

Purpose: Evidence suggests that people drink more alcohol and experience more adverse alcohol-related consequences (ARCs) on occasions when they also consume caffeine. The current study examined whether this increase in risk is a result of caffeine attenuating the subjective effects of alcohol intoxication (i.e., the masking hypothesis).

Methods: Undergraduate students ($n = 148$) reported their drinking patterns using a modified Timeline Followback approach. For each recalled drinking occasion, alcohol consumption, caffeine consumption, perceived blood alcohol concentration, and ARCs were assessed. Generalized linear mixed models were used to examine the influence that alcohol and caffeine consumption had on perceived intoxication and the experience of ARCs.

Results: At the occasion level, greater caffeine consumption was associated with increased consumption of alcohol and increased ARCs. There was also a significant curvilinear relationship between the amount of alcohol consumed and perceived intoxication, such that the more alcohol was consumed on each occasion the less each additional drink increased perceived intoxication. Increased caffeine consumption weakened the association between alcohol consumption and perceived intoxication and it also weakened the association between alcohol consumption and ARCs. Specifically, the weakest relationship between ARCs and alcohol consumption existed at the highest level of caffeine consumption (240+ mg). Caffeine increased subjective intoxication.

Conclusions: These findings do not support the masking hypothesis. Caffeine was strongly associated with ARCs when consumed at high doses and this effect does not appear to be the result of drinking more alcohol or underestimating one's blood alcohol content. Efforts to reduce caffeinated alcohol beverage use are greatly needed.

1. Introduction

Slightly over a third of young people consume caffeinated alcoholic beverages (CABs; Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2016; Lubman et al., 2013). CABs include drinks such as espresso martinis, rum and coke, vodka Red Bull®, and Jägerbombs. These latter

Abbreviations: AmED, Alcohol Mixed with Energy Drink; ARCs, alcohol related consequences; BAC, blood alcohol content; CABs, Caffeinated Alcoholic Beverages; cBAC, calculated BAC; ED, Energy Drink; pBAC, perceived BAC; SDUs, Standard Drink Units

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two drinks involve alcohol mixed with energy drinks (AmEDs). Energy drinks (EDs) typically contain taurine, guarana, and Vitamin B, in addition to caffeine (McLellan & Lieberman, 2012). The masking hypothesis proposes that caffeine attenuates or 'masks' some of the depressant effects of alcohol, which then causes individuals to underestimate their blood alcohol content (BAC), drink more alcohol, and engage in riskier behavior than they would if they were able to gauge their actual intoxication level (Brache & Stockwell, 2011; O'Brien, McCoy, Rhodes, Wagoner, & Wolfson, 2008). In support of this hypothesis, event-level studies using multilevel modeling have demonstrated that young people consume more alcoholic beverages and experience more alcohol related consequences (ARCs) when drinking AmEDs and other CABs than when drinking alcohol on its own (Linden-Carmichael & Lau-Barraco, 2017b; Norberg, Newins, & Henry, 2018; Patrick & Maggs, 2014). However, we do not know if caffeine increases risk as a function of increasing BAC underestimation and alcohol consumption as specified by the masking hypothesis. Outside of the CAB literature, greater BAC underestimation is associated with riskier decision making and more ARCs, even when controlling for alcohol consumption (Aston & Liguori, 2013; Grant, LaBrie, Hummer, & Lac, 2011; Proestakis et al., 2013; Turner, Bauerle, & Shu, 2004). Understanding the connection between caffeine, alcohol intake, and ARCs will help researchers and clinicians reduce the negative health impact of CABs by developing interventions that target the mechanisms that contribute to risk.

Although caffeine increases alertness and offsets alcohol-related fatigue (McKetin, Coen, & Kaye, 2015), most experimental studies (Benson & Scholey, 2014; Marczinski, Fillmore, Henges, Ramsey, & Young, 2012; Peacock, Bruno, Martin, & Carr, 2013; Ulbrich et al., 2013; van de Loo et al., 2016), but not all (Heinz, De Wit, Lilje, & Kassel, 2013), have not shown that adding caffeine to alcohol reduces subjective intoxication. These experimental findings might be limited to low levels of caffeine (~80 mg). A recent field study identified that individuals who consumed three or more EDs (i.e., 240 mg + of caffeine) reported feeling more intoxicated than those who had not consumed EDs when BAC levels were low, but these individuals reported feeling less intoxicated than those who had not consumed EDs when BAC levels were high (Kaestle, Droste, Peacock, Bruno, & Miller, 2018). This study also found that, regardless of ED consumption, individuals were somewhat able to judge their intoxication levels at lower BAC levels, but became insensitive to the intoxicating effects of alcohol at approximately .08% BAC (Kaestle et al., 2018). These combined findings suggest that the masking hypothesis may need a revision. The influence that caffeine has on subjective intoxication and ARCs may be a complicated interaction between how much caffeine and alcohol is consumed during a drinking episode.

After considering the accumulating research findings, we arrived at four specific hypotheses. First, we hypothesized that, consistent with the masking hypothesis, increased caffeine consumption would predict increased alcohol consumption. Second, we hypothesized that there would be a curvilinear relationship between our calculation of BAC, based on the Hustad and Carey (2005) equation for calculated BAC (cBAC), and individuals' perceived BAC (pBAC). Specifically, we expected that the effect of cBAC on individuals' pBAC would diminish as cBAC increased. Third, we hypothesized that level of caffeine consumption would moderate the relationship between cBAC and pBAC. Specifically, we expected that at high levels of caffeine use, cBAC would have a weaker relationship with pBAC compared to lower levels of caffeine use. Finally, we hypothesized that caffeine consumption, cBAC, and pBAC would all influence the probability of experiencing ARCs and that caffeine consumption would moderate the relationship between calculated BAC and ARCs, such that at higher levels of caffeine, cBAC would have less of an effect on ARCs.

2. Methods

2.1. Participants and procedures

To be eligible for participation, participants were required to have consumed caffeine within 30 min of alcohol consumption at least once in the previous 90 days and to be between 18 and 25 years old. These criteria were posted on SONA, an online research management system that enabled psychology students to sign-up and participate in research in exchange for course credit. One hundred sixty-four Australian undergraduates met these criteria. Participants had an average age of 18.95 (SD = 1.58). Most were first-year university students (84.1%), women (74.4%), of Anglo Australian heritage (55.5%), and single (54.3%). All study procedures were approved by the Human Research Ethics Committee at Macquarie University. All participants read and signed an informed consent document prior to beginning the study. Data were collected during an in-person interview.

2.2. Measures

2.2.1. Demographics

Participants completed a questionnaire assessing their age, gender, ethnicity, relationship status, year in school, and weight.

2.2.2. Modified timeline followback

A mTLFB interview (mTLFB; Norberg et al., 2018; Norberg, Norton, & Olivier, 2009; Schry & Norberg, 2013) was used to assess frequency and quantity of alcohol and caffeine use, drinking duration, perceived peak BACs (pBAC), and ARCs over the past 90 days. This data-driven calendar approach permitted examination of the real-world consequences of drinking alcohol and caffeine, while reducing participant and research effort as compared to long-term daily diary approaches (Norberg et al., 2018). Moreover, it also allowed for greater participant inclusion and more efficient use of resources compared to a one-day diary approach given that most individuals drink CABs much less frequently than they drink alcohol alone (Rossheim, Suzuki, & Thombs, 2013). Importantly, prior research has consistently found the mTLFB to have good reliability and validity for assessing self-reported alcohol and caffeine use, drinking durations, perceived peak BACs, and ARCs (Agrawal, Sobell, & Sobell, 2008; Norberg et al., 2018; Schry & Norberg, 2013).

As per the basic TLFB, participants were asked to recall their drinking history for the last 90 days (Sobell & Sobell, 1996). They were shown a calendar, asked to use memory aids to help them recall their drinking history, and started the assessment by reporting on their most recent drinking occasion. For each alcohol use episode, participants reported on its duration, the number of standard drink units (SDUs) they consumed, their pBAC, the number of ARCs they experienced, and the quantity of caffeine they consumed during or within 30 min of drinking alcohol. This approach to CAB assessment was used because caffeine takes up to 45 min to be absorbed from the digestive tract (Ferré & O'Brien, 2011) and is consistent with prior research methodology (Price, Hilchey, Darredeau, Fulton, & Barrett, 2010).

Participants used four handouts to assist with their reporting (see Norberg et al., 2018). The first was a visual display of various alcoholic beverages and the number of standard drinks contained within each. The second was a visual display of various caffeinated beverages that are commonly added to alcohol (e.g., different types of energy drinks, soda, coffee) with information about caffeine content noted for each beverage. The third handout assisted participants in reporting on their pBAC. The handout specified various levels of blood alcohol concentration [BAC; from 0 (no impairment) – 0.40% (death)] and their corresponding level of impairment. Effects displayed on the figure were derived from the "Blood Alcohol Chart" (Washington State University, 2016) and from a National Institute on Alcohol Abuse and Alcoholism (2001) online bulletin. The final handout was a list of 26 ARCs that

assessed social (e.g., had a verbal argument), role (e.g., not able to do school-work or study for a test), personal (e.g., did something I later regretted or was embarrassed by), and physical consequences (e.g., felt sick or vomited). For each drinking episode, participants reported which, if any of these ARCs, they experienced. Prior research has shown that this method demonstrates more sensitivity as a measure of ARCs compared with the Rutgers Alcohol Problem Index, a retrospective standardized measure that does not assess ARCs at the event-level (Norberg et al., 2009). Additionally, test-retest reliabilities for ARCs collected from the mTLFB for alcohol only, non-energy drink CAB, AmED, and mixed AmED and non-energy drink CAB occasions have been good to excellent (ICC = 0.70–0.82; Norberg et al., 2018).

2.3. Analytic plan

Data from the mTLFB were used to determine the following variables for each drinking occasion: number of standard drink units (SDUs) consumed, milligrams of caffeine consumed, number of ARCs experienced, duration of the drinking occasion, and pBAC. We calculated participants' peak BAC (cBAC) using Matthews and Miller (1979) equation as adapted by Hustad and Carey (2005). In this equation, $BAC = [(c / 2) \times (GC / w)] - [\beta 60 \times t]$, where c refers to the number of standard drinks, GC reflects the gender constant (9.0 for women and 7.5 for men), w refers to weight, β60 is the metabolism rate of alcohol per hour (0.017 g/dl), and t is the number of hours spent drinking. As this formula was developed based on U.S. measurements (standard drinks: 14 g of alcohol; weight: pounds), our Australian data first had to be converted to U.S. measurements to apply the formula. We collected weight in kilograms and then converted it to pounds (2.20 kgs = 1 lb) and we collected Australian SDUs and then converted them into American SDUs (1 Australian SDU = 0.71 American SDU). To test for a curvilinear relationship, quadratic terms were added to regressions of pBAC on cBAC. For analyses involving interactions and quadratic terms, predictors were mean centered, and interaction and quadratic terms were created using the mean centered predictors. When testing for an interaction that included a variable expected to have a curvilinear effect (i.e., caffeine and cBAC), the interaction for both the linear and quadratic terms was examined to determine whether the interaction was occurring at the linear or quadratic level.

A series of generalized linear mixed model (GLMM) analyses were conducted using PROC GLIMMIX in SAS, Version 9.4 to examine the relationships among variables from the TLFB. A mixed modeling framework was required for the analyses because events were clustered within persons (Searle, Casella, & McColloch, 1992). A negative binomial distribution was specified when the outcome was SDUs or ARCs because both were positively skewed count variables. Gender (0 = male, 1 = female) was entered as a covariate in all analyses. Significant interactions were probed using the procedures outlined in Holmbeck (2002). Simple slopes were computed for 0 mg, 80 mg, 160 mg, and 240 mg of caffeine, as these correspond to the approximate caffeine content of 0, 1, 2, and 3 EDs, respectively. Least square means were computed to examine predicted levels of pBAC at differing levels of cBAC and caffeine consumption in order to further evaluate the effects of caffeine on pBAC. Because least square means must be computed based on categorical variables, caffeine use was broken into four groups (i.e., 0 to 39 mg, 40 to 119 mg, 120 to 199 mg, and 200+ mg) such that mean values approximated the levels for which simple slopes were computed.

3. Results

Of the 2084 drinking occasions reported, 67.03% (n = 1397) involved only alcohol. Caffeine consumption ranged from 7 mg to 560 mg (M = 107.29; SD = 88.32) for the 687 CAB occasions.

Results of the GLMMs are presented in Table 1. Consistent with our first hypothesis, caffeine consumption was positively associated with the number of standard drinks consumed. As predicted by our second hypothesis, the statistically significant quadratic term indicated a curvilinear relationship between cBAC and pBAC. As the interaction between the quadratic term and caffeine was not statistically significant (see Table 1), this interaction was removed and the model was re-run with only the linear interaction to test our third hypothesis. The new model revealed a statistically significant cBAC × caffeine use interaction. Examination of the simple slopes revealed that cBAC was positively associated with pBAC at all levels of caffeine use examined, but as hypothesized, the relationship was weaker at higher levels of caffeine use (see Table 2 and Fig. 1). Examination of least square means (see Table 3) revealed three patterns. First, when cBAC was low (i.e., cBAC = .05), participants overestimated their BAC the most at the highest levels of caffeine consumption. Second, when cBAC was moderate (i.e., cBAC = .10), participants were more accurate in their BAC estimates when they consumed higher levels of caffeine. Third, at high levels of cBAC (i.e., cBAC = .15 and .20), almost all participants underestimated their BAC, but those who consumed more caffeine perceived their BAC to be closer to their cBAC (i.e. they underestimated their intoxication less so than those who consumed less caffeine). As predicted by our last hypothesis, caffeine, cBAC, and pBAC were all positively associated with ARCs and a significant interaction between cBAC and caffeine occurred. Analyses examining the simple slopes revealed that the relationship between cBAC and ARCs was weaker at higher levels of caffeine consumption, and the effect of cBAC on ARCs became non-significant at 240 mg of caffeine (see Table 2 and Fig. 2).[1]

4. Discussion

Previous studies have shown that on occasions when people drink caffeine with alcohol, they tend to experience more ARCs than they do on occasions when they drink alcohol alone (Linden-Carmichael & Lau-Barraco, 2017b; Norberg et al., 2018; Patrick & Maggs, 2014). We aimed to elucidate the mechanisms connecting CABs to ARCs by exploring the complex relationships among caffeine, alcohol, perceived and objective intoxication, and ARCs using event-level analyses. As hypothesized, we found that on occasions when participants consumed higher levels of caffeine, they also consumed more alcohol. However, contrary to the assumptions of the masking hypothesis – that caffeine causes individuals to underestimate their blood alcohol content – participants' perceptions of their BAC tended to be *higher* as caffeine intake increased. We also found that greater caffeine consumption, cBAC, pBAC, and the interaction between cBAC and caffeine were associated with experiencing more ARCs. At lower levels of caffeine consumption, risk of experiencing ARCs was low when cBAC was low, but increased as cBAC increased; however, cBAC was not predictive of ARCs at the highest level of caffeine consumption (240 mg +). At the highest level of caffeine consumption, the risk of experiencing ARCs was high for every level of cBAC. Together, these results suggest that the connection between CAB consumption and risk may not be due to underestimation of BAC.

We found a curvilinear relationship between cBAC and pBAC, such that each extra drink consumed had less of an impact on perceived intoxication than the previous drink. This finding is consistent with prior studies that have shown pBAC increases as cBAC increases (Quinn & Fromme, 2011), but that subjective intoxication hits a ceiling, beyond which more alcohol consumption has little to no effect (Kaestle et al., 2018). Also consistent with prior research (Grant, Labrie, Hummer, &

[1] We re-ran these analyses with number of standard drinks as a predictor in place of cBAC. The pattern of results was the same. Specifically, the direction of the effects and the statistical significance of effects were identical in both models. The only difference was in the analyses of simple slopes, as the simple slope of standard drinks remained statistically significant at 240 mg of caffeine; however, the simple slopes were still attenuated as caffeine increased.

Table 1
Multilevel generalized linear model results.

Dependent variable	Predictors	*b*	*SE*	*β*	*t*	*df*	*p*
SDUs	Gender	−.26	.08	−5.47	−3.16	1919	.002
	Caffeine	.003	.000	9.90	17.31	1919	< .001
pBAC	Gender	−.004	.003	−.08	−1.15	1915	.249
	Caffeine	.0001	.0000	.38	12.13	1915	< .001
	cBAC	.47	.01	1.46	33.51	1915	< .001
	$cBAC^2$	−1.03	.10	−.11	-2.39	1915	< .001
	cBAC × Caffeine	−.0004	.0002	−.40	−10.21	1915	.017
	$cBAC^2$ × Caffeine	.0004	.0011	.02	.40	1915	.687
pBAC	Gender	−.004	.003	−.08	−1.15	1916	.248
	Caffeine	.0001	.0000	.38	12.88	1916	< .001
	cBAC	.47	.01	1.45	33.53	1916	< .001
	$cBAC^2$	−1.02	.10	−.39	−10.31	1916	< .001
	cBAC × Caffeine	−.0004	.0001	−.10	−3.37	1916	< .001
ARCs	Gender	.03	.15	.68	.22	1916	.830
	Caffeine	.002	.000	7.90	7.31	1916	< .001
	cBAC	4.17	.51	12.85	8.18	1916	< .001
	pBAC	17.54	.80	31.29	21.83	1916	< .001
	cBAC × Caffeine	−.02	.00	−5.10	−5.14	1916	< .001

Notes. ARCs = alcohol-related consequences; cBAC = calculated blood alcohol concentration; pBAC = perceived blood alcohol concentration.

Table 2
Simple slopes of cBAC on pBAC or ARCs by level of caffeine.

Level of caffeine	*b*	*SE*	*β*	*t*(1916)	*p*
DV: pBAC					
0 mg	.48	.01	1.49	33.48	< .001
80 mg	.46	.01	1.41	30.48	< .001
160 mg	.43	.02	1.32	21.88	< .001
240 mg	.40	.03	1.23	15.26	< .001
DV: ARCs					
0 mg	4.83	.56	14.90	8.67	< .001
80 mg	3.33	.49	10.27	6.77	< .001
160 mg	1.83	.59	5.64	3.12	.002
240 mg	.33	.79	1.01	.42	.676

Notes. DV = dependent variable; cBAC = calculated blood alcohol concentration; pBAC = perceived blood alcohol concentration; ARCs = alcohol-related consequences.

earlier findings, we found that caffeine consumption intensified these effects. When greater amounts of caffeine were consumed, people tended to overestimate their intoxication levels more at low levels of cBAC and underestimate it less at high levels of cBAC than did individuals who consumed little or no caffeine. This implies that consuming large amounts of caffeine makes people feel more intoxicated than they would if consuming smaller amounts caffeine at all levels of alcohol consumption.

According to the masking hypothesis (Brache & Stockwell, 2011; O'Brien et al., 2008), CABs increase the risk of ARCs because caffeine attenuates the depressant effects of alcohol, which makes people feel less intoxicated, and consequently causes them to drink more alcohol and misjudge the risk involved in undertaking certain behaviors. However, the current findings suggest that CABs may be associated with a greater risk of ARCs because caffeine makes people feel *more* intoxicated. Inherent to this assumption is prior research demonstrating

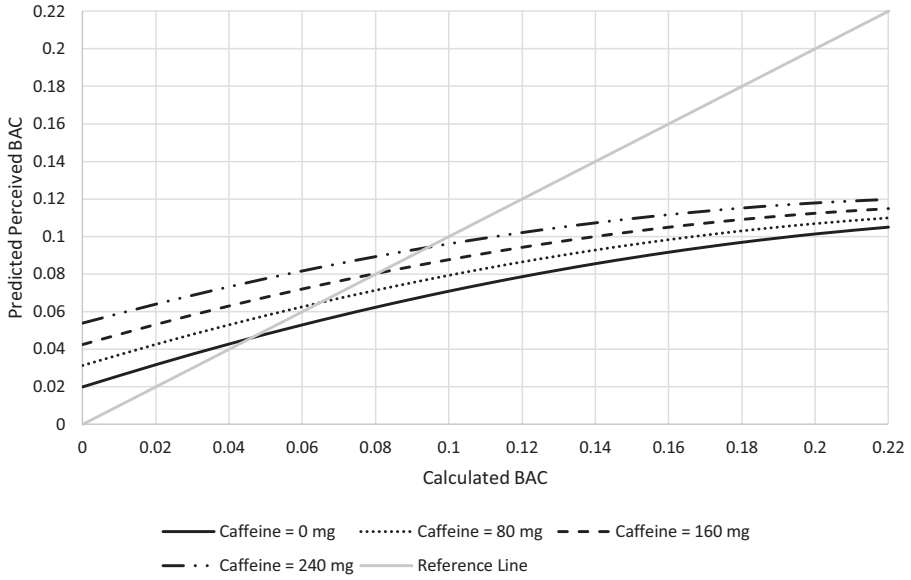

Fig. 1. Relationship between cBAC and pBAC by caffeine level.

Lac (2012); Rossheim et al., 2017; Thombs, Olds, & Synder, 2003), we found that students tended to overestimate their intoxication at low BAC levels but underestimate it at high BAC levels. Adding to these that the stimulant effects of alcohol exert more influence on subjective intoxication than do its sedative effects (Celio et al., 2014) and that caffeine increases alertness and energy when consuming alcohol

Table 3
Estimated mean pBAC by cBAC and caffeine.

cBAC	Caffeine range (mg)	pBAC	95% CI of pBAC
.05	0–39	.041	.038 to .044
	40–119	.052	.047 to .056
	120–199	.063	.057 to .068
	200+	.073	.066 to .079
.10	0–39	.065	.062 to .069
	40–119	.076	.072 to .080
	120–199	.087	.081 to .092
	200+	.097	.090 to .103
.15	0–39	.089	.085 to .093
	40–119	.100	.100 to .104
	120–199	.111	.105 to .116
	200+	.121	.114 to .128
.20	0–39	.113	.108 to .118
	40–119	.124	.118 to .129
	120–199	.135	.129 to .141
	200+	.145	.138 to .152

Note. cBAC = calculated blood alcohol concentration; pBAC = perceived blood alcohol concentration; CI = confidence interval.

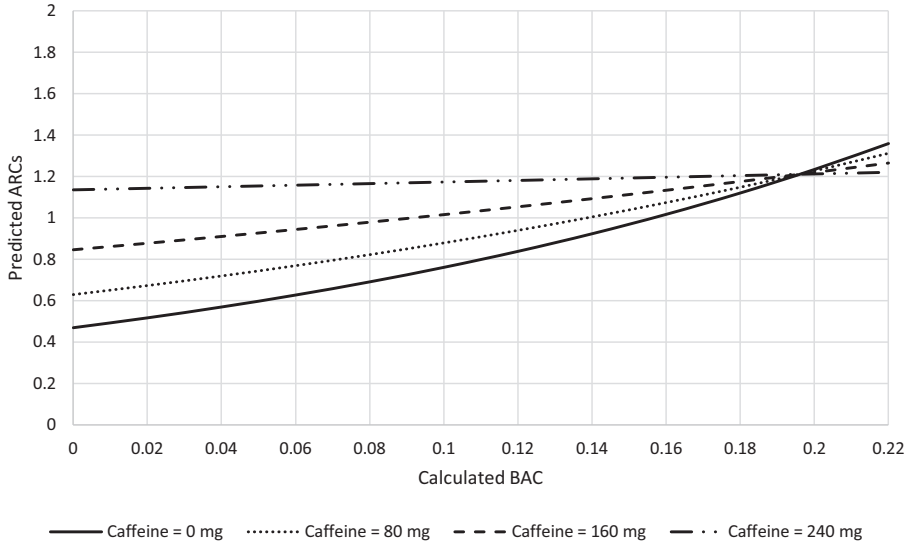

Fig. 2. Relationship between cBAC and ARCs by caffeine level.

(McKetin et al., 2015).

4.1. Limitations and future directions

This study is limited by several factors. First, although the mTLFB has been shown to reliably capture CAB drinking behavior and its associated outcomes (Norberg et al., 2018), we do not yet know if its data are valid. Perhaps participants report a certain pBAC level due to the consequences they experience during that drinking occasion rather than because of how they felt during the drinking occasion. These data highlight the pressing need to study these relationships using ecological momentary assessment, which would offer the additional benefit of reducing the recall bias inherent in retrospective measures. Second, the formula we used to calculate BAC assumed all participants metabolized alcohol at same rate (17 mg%/hr). This is an accurate average, but it should be noted that breath alcohol concentrations do not perfectly correlate with cBACs (Hustad & Carey, 2005). Third, the mTLFB did not assess the location of drinking episodes. Preliminary evidence suggests that individuals may drink more CABs and experience more ARCs in riskier contexts (e.g. bars, clubs, pre-gaming at home; Linden-Carmichael & Lau-Barraco, 2017a). Thus, future research may consider including an assessment of location for each CAB occasion. Fourth, this study did not measure the stimulant and sedative effects of alcohol, nor did it assess other factors (e.g., sleep quantity and sleep quality) that may contribute to ARCs. Future research should incorporate a broader assessment package to fully understand how caffeine influences ARC risk. Finally, the study was conducted on a predominantly female student sample at one university, reducing its generalizability. While gender was included as a covariate in the analyses, results may be more representative of the effects of caffeine and alcohol in female university students than male university students. Therefore, the moderating role of gender in these relationships should be examined in future studies with larger male student representation.

4.2. Implications and contributions

The current findings question the validity of the masking hypothesis because the connection between caffeine consumption and increased ARCs was explained by an increase in subjective intoxication, rather than a decrease. However, this is not to mean that caffeine caused people to more accurately judge their intoxication levels. Even though students more accurately judged their BAC levels when consuming large amounts of caffeine and alcohol as compared to occassions when only alcohol was consumed, they still grossly underestimated their intoxication levels when cBAC rose above 0.13. Caffeine additionally served as an independent risk factor for heavier and more harmful alcohol use. The current findings, along with other research showing that students radically misperceive their intoxication levels (Rossheim et al., 2013) and that subjective intoxication is the best predictor of ARCs (Quinn & Fromme, 2011), suggest that social marketing approaches should be used to inform individuals that BAC levels cannot be accurately judged. In regard to reducing ARCs, policy approaches that prevent pre-mixed CABs from being sold may be more advantageous than social marketing approaches that inform the public that caffeine increases subjective intoxication as many people report drinking CABs to get a bigger buzz (Peacock, Bruno, & Martin, 2013). Lastly, the findings and limitations of this research should be used to inform an ecological momentary assessment study to fully understand whether the masking hypothesis needs revision.

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Conflicts of interests

The authors have no conflicts of interests to report.

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