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Caffeine increases false memory in nonhabitual consumers

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Insight into caffeine's equivocal effects on memory can be derived from work suggesting both emotional arousal and psychosocial stress increase false memory rates without increasing veridical memory. This study investigated how a range of caffeine doses affect veridical and false memory formation in nonhabitual consumers. A double-blind, repeated-measures design with caffeine (0 mg, 100 mg, 200 mg, 400 mg caffeine) was used to examine memory using the Deese-Roediger-McDermott (DRM) paradigm. Results showed that caffeine modulated arousal levels, peaking at 200 mg and returning to near baseline levels at 400 mg. Main effects of caffeine demonstrated higher critical lure recall and recognition ratings (i.e., false memory) as a function of dose, again peaking at 200 mg. Those who showed the highest arousal increases as a function of caffeine also tended to produce the highest false recall and recognition rates. Veridical memory was not affected. Results demonstrate that consumption of as little as 100 mg of caffeine elicits reliable inverted-U shape changes in arousal and, in turn, false memories in individuals who do not habitually consume caffeine.

Keywords: Arousal; Caffeine; False memory; Veridical memory.

Caffeine (1,3,7-trimethylxanthine) is found naturally in food and beverages, such as coffee, tea, and chocolate, and has recently become popular as a supplement in commercially available energy drinks and food bars. Population surveys of consumption in the United States indicate that over 80% of adults habitually consume caffeine (average 280 mg/day; Barone & Roberts, 1996). Caffeine has well known effects on the central nervous system that include increased alertness, wakefulness, motivation, and motor activity, as well as increased neuronal activity (for a review,

see Lieberman, 2003). It is often cited for its positive effects on attention and basic psychomotor tasks, such as visual vigilance, simple reaction time, and choice reaction time (for example, Lieberman, Tharion, Shukitt-Hale, Speckman, & Tulley, 2002). The beneficial effects of caffeine on performance are commonly attributed to caffeine's antagonistic role at adenosine A₁ and A_{2A} receptors in dopamine-rich brain areas (Garrett & Griffiths, 1997).

However, arousal does not always have positive effects on cognitive processes. Data regarding

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The opinions expressed herein are those of the authors and do not reflect those of the United States Army.

caffeine's effects on memory are equivocal, with some studies reporting positive effects and others reporting no effects or negative effects (for example, Childs & de Wit, 2006). Some insights into caffeine's effects on memory, however, can be derived from recent work suggesting both emotional arousal, such as that accompanying anger or fear, and arousal following psychosocial stress, increased false memory recall, and recognition rates without necessarily increasing veridical (true) memory (Corson & Verrier, 2007; Payne, Nadel, Allen, Thomas, & Jacobs, 2002). The effects of emotional arousal on false recall and recognition may be due to increased relational processing (Brunyé, Mahoney, Augustyn, & Taylor, 2009; Corson & Verrier, 2007), possibly resulting from arousal induced changes in cortisol in the PFC and hippocampal systems (Mayer & Gaschke, 1988). In general, false memories for word lists are thought to reflect the associative nature of verbal memory (Hutchinson & Balota, 2005; Roediger & McDermott, 1995). The extent to which participants recall or recognise previously presented words provides an indication of veridical verbal memory, whereas spontaneously recalling or giving high recognition ratings to highly associated, but never presented, words provides a measure of false memory. Given the ubiquity of caffeine consumption and the applied and legal implications of both veridical and false memory formation, it is important to understand if the physiological arousal resulting from caffeine consumption also affects false recall and recognition.

To understand how arousal affects both veridical and false memory formation, a double-blind, within-participants repeated-measures design tested the effects of a range of caffeine doses (0–400 mg) on recall and recognition in nonhabitual caffeine consumers using the Deese-Roediger-McDermott (DRM) paradigm. This design allowed for a targeted (i.e., isolated effects of arousal) and comprehensive (i.e., a parametric range of doses) evaluation of arousal effects on veridical and false memory. The DRM paradigm involves learning lists of words that are each highly associated with one nonpresented word, the *critical lure* (Roediger & McDermott, 1995). After encoding, participants complete recall and recognition tests. Veridical memory rates are determined by measuring correctly recalled and recognised words, and false memory rates are determined by measuring falsely recalled or recognised critical lures. Given earlier work suggest-

ing emotional arousal may increase rates of false memory without affecting veridical memory in the DRM paradigm (Corson & Verrier, 2007; Payne et al., 2002), it can be predicted that if caffeine consumption leads to increased arousal and, in turn, increased relational processing (i.e., associative lexical activation; Hutchinson & Balota, 2005), then rates of false recall and recognition should follow a positive dose–response relationship and veridical memory should remain relatively unaffected.

MATERIALS AND METHODS

Design

The present study used a double-blind, repeated-measures design with four levels of treatment (0 mg, 100 mg, 200 mg, 400 mg caffeine). The highest dose of caffeine matches that found in a 20 oz coffee portion served at a major franchise coffee house (i.e., 415 mg; www.starbucks.com). Treatment order was counterbalanced across participants.

Participants

Thirty six male (16) and female (20) volunteers between the ages of 18 and 35 were recruited from the Tufts University student population. They were 19.0 ($SD = 1.26$; range 18–22) years old, 171.5 ($SD = 8.6$; range 62–75) cm in height, and weighed 68.3 ($SD = 11.5$; range 110–208) kg.

All participants were low caffeine consumers (self-report of $M = 41.3$, $SD = 28.8$, range 0–102 mg/day), nonsmokers, in good health, did not use prescription medication other than oral contraceptives, and did not use nicotine in any form. Written informed consent was obtained, and all procedures were jointly approved by the Tufts University Institutional Review Board and the Human Use Review Committee of the US Army Research Institute for Environmental Medicine.

Manipulation check

Participants used the Brief Mood Introspection Scale (BMIS) to rate their current mood and arousal state in accordance with 16 adjectives (eight positive and eight negative) on a series of

Likert scales anchored at 1 (“definitely do not feel”) to 4 (“definitely feel”).

Cognitive tests

DRM paradigm. Fifty word lists were used. Each list contained 15 words and was randomly assigned to one of five sets. Thus, each participant saw 10 new word lists on each of the five test sessions. The sets of word lists were counter-balanced across test sessions. Lists were presented via headphones, at a rate of one word every 2 s. Lists were presented in random order. Immediately after each list was presented, participants engaged in a 1-minute recall test. After the 10th list was presented and recall completed, participants completed a recognition test consisting of 60 randomly ordered words including 30 “old” or presented words (the first, eighth, and 10th item of each list), the 10 critical lures and 20 “new” unrelated words. Participants rated each word on a scale ranging from 1 (“sure it was new”) to 4 (“sure that the item was studied”).

Caffeine or placebo administration

In order to control for taste, caffeine or placebo was administered in capsule form. All treatment doses were administered in an identical colour, size, weight, and shape capsule. Capsules contained either 0 mg, 100 mg, 200 mg, or 400 mg of caffeine. Placebo capsules were filled with physiologically inert microcrystalline cellulose powder, which was also used as filler material in the two lower-dose caffeine capsules. The caffeine was 99.8% pure anhydrous USP-grade powder.

Procedure

Participants completed all four treatment conditions and a normal consumption day on separate days, resulting in five test sessions. There was a minimum 3-day wash out period between test sessions. Participants were instructed not to eat or drink anything (with the exception of water) after 9:00 p.m. the night before a test session and not to use any over-the-counter medications or herbal supplements 24 hours prior to testing. During the normal consumption day, participants were allowed to consume their normal amount of

caffeine prior to arrival for testing. Test sessions began between 8:00 and 9:30 a.m.

When participants arrived in the morning, they completed a baseline BMIS and then consumed a capsule containing varying doses of caffeine or placebo along with a cup of water. Sixty minutes after consuming the capsule, participants completed another BMIS and then immediately began the DRM task. Timing of testing was based on literature showing peak plasma concentrations of caffeine occur approximately 45–60 minutes after ingestion. Participants completed the same test sequence for each of the treatment conditions.

RESULTS

Statistical analyses

DRM data were analysed with repeated measures analysis of variance (ANOVA) with treatment (0 mg, 100 mg, 200 mg, and 400 mg) as a within-subject variable. Baseline BMIS data were analysed by ANOVA with treatment (0 mg, 100 mg, 200 mg, and 400 mg) and adjective (16 separate adjectives) as within-subjects variables. Post-treatment BMIS difference score data [(60 minutes post-treatment) – (baseline)] were analysed by ANOVA with treatment (0 mg, 100 mg, 200 mg, and 400 mg) and adjective (16 separate adjectives) as within-subject variables. An effect was deemed statistically significant if the likelihood of its occurrence by chance was $p \leq 0.05$. If an ANOVA yielded a significant main effect, planned comparisons in the form of *t*-tests were performed (results of planned comparisons are provided in Figure 1). All statistical analyses were performed using SPSS 12.0. Two participants were excluded for failing to perform one or more recall tests.

Manipulation check: BMIS

As expected, baseline affect measures did not vary as a function of treatment condition, across any of the 16 adjectives in a 4 (treatment condition: 0, 100, 200, 400 mg) \times 16 (adjectives) ANOVA ($F_s < 1$).

An ANOVA on BMIS difference scores revealed an effect of adjective, $F(15, 495) = 10.12$, $p < .01$, $\eta^2 = .09$; more importantly,

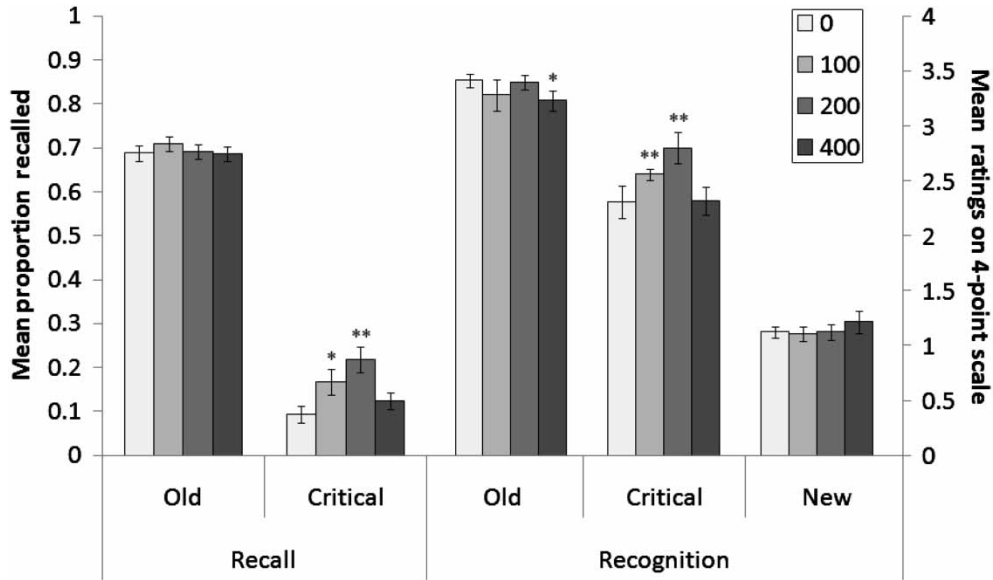


Figure 1. Results from the recall and recognition tests, with standard error bars, for each of the four caffeine doses (0, 100, 200, 400). Asterisks indicate results from paired *t*-tests comparing to 0 mg condition: ***p* < .01, **p* < .05, ****p* < .10. Within critical recall, 200 mg > 100 mg^m, and within critical recognition, 200 mg > 100 mg*.

adjective interacted with treatment condition, $F(45, 1485) = 2.79, p < .01, \eta^2 = .05$. To specifically examine treatment condition effects on individual adjective ratings, we conducted a series of ANOVAs. Eight adjectives (caring, content, gloomy, jittery, grouchy, loving, fed up, active) did not vary as a function of treatment condition. The other eight adjectives showed effects of treatment condition (all ANOVA $ps < .05$); these are detailed in Table 1, along with results from paired *t*-tests comparing each treatment condition to the 0 mg condition. Note that in no case did any individual adjective rating differ between the 200 mg and 400 mg conditions. Exploratory analysis included gender in the

earlier ANOVA; no main or interactive effects were identified ($p_{min} = .14$).

Recall

A main effect of caffeine demonstrated higher critical lure recall as a function of treatment condition, peaking at 200 mg, $F(3, 99) = 8.83, p < .01, \eta^2 = .20$ (see Figure 1). Exploratory analysis included gender in the previous ANOVA; no main or interactive effects were identified ($p_{min} = .07$). Veridical recall of old items did not vary as a function of treatment condition, $F(3, 99) = 1.39, p = .25$.

TABLE 1
Mean and standard error BMIS difference scores (pre- to post-capsule administration) for each treatment condition

Adjective	NCD		0 mg		100 mg		200 mg		400 mg	
	M	SE	M	SE	M	SE	M	SE	M	SE
Lively	0.13	0.13	-0.09	0.17	0.24	0.14	0.29*	0.14	0.29*	0.16
Happy	-0.22	0.14	-0.56	0.12	0.02**	0.12	0.00**	0.09	-0.21*	0.13
Sad	-0.10	0.13	-0.09	0.11	-0.21*	0.10	0.18	0.11	-0.15	0.06
Tired	-0.57	0.13	-0.03	0.18	-0.50*	0.13	-0.59**	0.12	-0.45*	0.06
Drowsy	-0.42	0.15	0.12	0.18	-0.41**	0.13	-0.53**	0.15	-0.59**	0.16
Peppy	-0.01	0.12	-0.12	0.15	0.15	0.19	0.24*	0.13	0.30*	0.14
Nervous	-0.22	0.12	0.06	0.12	-0.12	0.08	0.18	0.19	0.41	0.15
Calm	-0.13	0.11	-0.12	0.16	-0.09	0.11	-0.12	0.16	-0.33	0.15

NCD: Normal Consumption Day. Asterisks indicate significance in *t*-tests comparing each treatment level to the 0 mg condition (**p* < .05, ***p* < .01). Nervous and calm showed only nonsignificant differences. All other BMIS adjectives $p > .05$.

Recognition

As with recall data, critical lure recognition ratings increased as a function of caffeine dose, again peaking at 200 mg, $F(3, 99) = 7.53, p < .01, \eta^2 = .19$ (see Figure 1). Exploratory analysis included gender in the earlier ANOVA; no main or interactive effects were identified ($p_{min} = .55$). Veridical ratings of old items decreased marginally across caffeine doses, $F(3, 99) = 2.67, p = .052, \eta^2 = .07$, and correct rejection of unrelated items did not change ($F < 1$).

We also assessed false recognition using a gist memory d' measure, which compares critical lure ratings to ratings of unstudied unrelated lures (with a 1 or 2 rating recoded as “no”, and a 3 or 4 rating recoded as “yes”). This method treats “yes” responses to critical lures as hits and “yes” responses to unrelated lures as false alarms. As such, higher d' reveals higher global associative processing (i.e., gist memory; Koutstaal & Schacter, 1997). An ANOVA comparing d' across treatment conditions revealed a main effect, $F(3, 99) = 11.87, p < .01, \eta^2 = .26$. Overall, relative to placebo ($M = 1.55, SE = 0.18$) there were higher d' levels in the 100 mg ($M = 1.91, SE = 0.19$), $t(33) = 2.63, p = .01$, and the 200 mg ($M = 2.16, SE = 0.23$), $t(33) = 3.48, p < .01$, conditions (400 mg, $M = 1.27, SE = 0.19$), $p > .05$.

A more traditional d' measure, calculated by comparing hits to old words versus false alarms to critical lures (using recoded ratings, as before), also showed a main effect, $F(3, 99) = 6.52, p < .01, \eta^2 = .16$. Overall, relative to placebo ($M = 1.17, SE = 0.21$) there were lower d' levels in the 100 mg ($M = 0.64, SE = 0.21$), $t(33) = 3.44, p < .01$, and the 200 mg ($M = 0.51, SE = 0.22$), $t(33) = 3.41, p < .01$, conditions (400 mg, $M = 1.08, SE = 0.20$), $p > .05$. We also calculated response criterion, which demonstrated a main effect of treatment, $F(3, 99) = 8.28, p < .01, \eta^2 = .20$, with a more lenient criterion at 200 mg ($c = -0.70$), relative to placebo ($c = -0.40$), $t(33) = 3.29, p < .01$ (all other $ps > .05$). This type of criterion shift often accompanies higher critical lure false alarms (e.g., Koutstaal & Schacter, 1997).

Withdrawal effects

To confirm that our results are not attributable to caffeine withdrawal effects, we conducted t -tests to compare the normal consumption day recall

and recognition data to performance on the 0 mg day. No significant differences were found (all $ps > .05$). Normal consumption day data are detailed in Table 2, for both the BMIS and memory tasks.

Using arousal to predict false memory

To test whether caffeine-induced BMIS arousal differences between the 200 mg and 0 mg conditions predict recall and recognition results, we conducted two linear regressions. First, for each participant we calculated a composite arousal measure for each treatment dose that averaged the four arousal-related (Mayer & Gaschke, 1988) adjectives that showed differences between 200 and 0 mg (i.e., Table 1): lively, tired (reverse-scored), drowsy (reverse-scored), and peppy. We then used this composite measure to calculate difference scores between the 200 mg and 0 mg conditions (200–0 mg). We used these BMIS arousal difference scores to predict, in separate regressions, recall and recognition critical lure false alarm difference scores (200–0 mg). For recall results, those with greater arousal differences as a function of caffeine dose also tended to show the greatest increases in critical lure recall, $\beta = 0.44, t(33) = 2.77, p < .01$; this was not true for veridical recall, $\beta = 0.15, t(35) = 0.84, p > .05$. For recognition results, the same effect was found for critical lure recognition, $\beta = 0.43, t(35) = 2.69, p < .05$, and inversely for old item recognition, $\beta = -0.38, t(35) = 2.32, p < .05$.

TABLE 2

Mean and standard error recall and recognition ratings for the normal consumption day

	<i>M</i>	<i>SE</i>
Recall rates		
Critical lure recall	0.12	0.02
Veridical recall	0.68	0.01
Recognition rates		
Critical lure recall	2.49	0.09
Veridical recall	3.51	0.04
CR of unrelated items	1.09	0.02
d' traditional sensitivity	1.09	0.14
d' gist sensitivity	1.75	0.12
c response criterion	-0.50	0.07

DISCUSSION

The present study examined effects of a range of caffeine doses (0–400 mg) on veridical verbal memory and false recall and recognition using the Deese-Roediger-McDermott (DRM) paradigm. Converging evidence from our manipulation check and memory tasks demonstrates that caffeine intake approximating a 12–16 oz coffee at major franchise coffee houses elicits reliable and parametric increases in arousal and, in turn, false memories, without substantially altering veridical memory. Specifically, we found higher phenomenological experiences of arousal and higher false recall and recognition critical lures after participants consumed as little as 100 mg of caffeine.

Our results are consistent with previous work demonstrating that increases in arousal lead to greater relational, sometimes at the cost of item-specific, processing (Brunyé et al., 2009) and are in agreement with research showing that arousal enhances elaborative encoding and increases suggestibility (for example, Porter, Spencer, & Birt, 2003). They also extend previous work examining caffeine influences on false memory. Although false memory did not continue to increase at the 400 mg dose, as was hypothesised, this finding is consistent with other work that suggests performance advantages diminish with high caffeine doses. In fact, cognitive processes such as vigilance, executive control, and mood (e.g., Lieberman et al., 2002) commonly show an inverted-U pattern of caffeine effects, particularly in low consumers.

To date, only two studies have examined the influence of caffeine-related arousal on false memory formation and both used only a single moderate dose (200 mg). The first study assessed whether caffeine administration would enhance recall memory for word lists as well as increase false recall for semantically related words (Capek & Guenther, 2009). The results are consistent with ours in that a 200 mg caffeine dose increased false recall, but differ from ours in that this dose improved veridical recall. The second study examined how sleep deprivation affects false recall and if caffeine administration prior to retrieval would attenuate this effect (Diekelmann, Landolt, Lahl, Born, & Wagner, 2008). Contrary to our findings, the results of this study suggest that sleep-deprived participants had more false memories and caffeine consumption led to fewer false memories. These findings

are contrary to ours, but it is difficult to compare data from well-rested individuals with those who are 9 hours sleep deprived. Further, it could be the case that the caffeine administered in Diekelmann et al. (2008) returned sleep-deprived participants to a baseline level of arousal at which, as in the present study, there were relatively moderate false alarm rates. In addition, both Capek and Guenther (2009) and Diekelmann et al. used between-participant designs and did not account for normal consumption habits. Therefore, it should be noted that not only does our study further existing work by providing a parametric assessment of arousal effects on false recall and recognition, but it also better controlled for individual differences in caffeine metabolism and possible effects of subjective experiences of arousal by using a within-subjects design and controlling for normal consumption habits. In addition, by testing only in the morning after an overnight fast, our study limited potential confounding effects associated with circadian rhythms and differences in macro- and micronutrient consumption prior to testing. Finally, testing only low consumers and including a normal consumption test session reduced the confound of changes in glucocorticoid release associated with habitual caffeine use (e.g., nonconsumer vs., regular consumer) and demonstrated that effects we observed were due to increased arousal associated with caffeine consumption, rather than symptoms of caffeine withdrawal.

The mechanism by which caffeine may influence false memory formation is unknown. One possibility is that caffeine influences memory via an increase in glucocorticoid production. Glucocorticoids are produced by the hypothalamic-pituitary-adrenal (HPA) axis in response to physical and psychological stress. In humans, cortisol is the primary glucocorticoid. Cortisol is known to influence neuronal metabolism in the brain, particularly the hippocampus, a structure linked to memory function with dense concentrations of glucocorticoid receptors. For example, cortisol can reduce glucose uptake in the hippocampus as shown with positron emission tomography (PET) and reduce activation in the hippocampus during memory retrieval as shown with fMRI (Oei et al., 2007). Furthermore, evidence suggests that emotional arousal, psychosocial stress, and/or high levels of glucocorticoids that accompany stress can impair performance on memory tasks in normal participants and those with cognitive impairments

that can affect memory function, such as Cushing syndrome, Alzheimer's, schizophrenia, and depression (Corson & Verrier, 2007; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Payne et al., 2002; Wolf, 2009).

Consumption of caffeine typically results in increased cortisol release, especially in nonhabitual consumers given moderate or high doses of caffeine (Lovallo et al., 2005). This modulation of cortisol may disrupt the PFC and hippocampal systems contributions to contextual remembering. It is generally agreed the hippocampus and PFC are important sites for contextual remembering either by binding together elements of the contextual situation or through source monitoring (Mitchell, Johnson, Raye, & D'Esposito, 2000). According to the theory of spreading activation, the ability to distinguish between presented words and nonpresented words that were associatively activated depends in part on the ability to apply context, such as the order of a particular word on a list or what preceded it. Stress, or glucocorticoid release following stress, may disrupt formation of contextual representations via the modulation of the PFC and hippocampal systems, and therefore increase the difficulty in distinguishing words that were actually presented (i.e., "old" words) and semantically related words that were associatively activated (i.e., critical lures) (Payne et al., 2002). Of course, the present design does not allow us to disentangle the effect of caffeine on the different stages of memory formation; given that both encoding and retrieval processes were done under the influence of caffeine. This limitation presents a possibility for future research regarding the possible differential influence of caffeine administration on encoding and retrieval processes.

The present results might also be attributed to caffeine-modulated changes in the distinctiveness between studied and critical items. The distinctiveness heuristic proposes that word memory accuracy is determined by both verifying the correctness of list items and rejecting critical lures, and distinctive processing serves both functions (Dodson & Schacter, 2001). Distinctive processing allows people to recognise differences between items and lures within the context of high similarity. Predictions offered by the distinctiveness heuristic provide a potential explanation for the relatively lenient response criterion to critical lures at 200 mg: Caffeine-related arousal might promote nondistinctive processing of old items and critical lures. Given that caffeine-related memory effects were specific to critical

lures, this pattern suggests an effect on event- but not item-based distinctiveness (cf., Hunt, 2003). Future research might more specifically examine whether caffeine might interact with the application of a distinctiveness heuristic, for instance by manipulating the salience of encoded words.

CONCLUSION

In conclusion, the present study extends previous work by demonstrating that pharmaceutically induced physiological arousal can increase false memories in the DRM paradigm in a dose-dependent manner, without affecting veridical memory. This is most likely due to greater relational processing, perhaps as a result of glucocorticoid-mediated dysregulation of contextual binding and source monitoring within the PFC and hippocampus. It might also be partially attributed to shifted response criteria, perhaps indicating a differential application of the distinctiveness heuristic under high arousal conditions. Given the ubiquity of caffeine consumption in the form of coffee, tea, energy drinks, and foods such as chocolate and energy bars, the present results hold everyday implications for false memory formation, at least for individuals who do not regularly consume caffeine. One caveat to this is that the translation of DRM results to real-world behaviours is not known, suggesting a need for future work using more naturalistic situations. Future work could also attempt to determine the mechanism by which caffeine influences false memory by examining the effects of other stimulants that do not primarily affect adenosine receptors (e.g., amphetamine or modafinil). Studies to examine the relationship between caffeine consumption, cortisol release, and false memory in a dose-response fashion in habitual and non-habitual consumers should also be conducted, as the present effects, are likely to be less pronounced in habitual caffeine consumers.

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