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Interactive Influence of Alcohol and Stress on Learning and Intrusive Memories: A Preliminary Report

Jaycee L. Kirkland[1], Mercedes L. Stanek[1], Koen N. Suzelis[1], Caeden M. Gurganus[1], Kelsey M. Siereveld[1], Jasmine I. C. Ifeakanwa[1], Matthew S. Risner[1], John G. Blasco[1], Katherine R. Fleetham[1], Jeremy R. Yuen[1], Aiden J. Farrell[1], Boyd R. Rorabaugh[2], Phillip R. Zoladz[1*]

[1]Department of Psychology and Education, The School of Health, Life Sciences, and Education, Ohio Northern University, Ada, OH, USA
[2]Department of Pharmaceutical Sciences, Marshall University School of Pharmacy, Huntington, WV, USA
Email: *p-zoladz@onu.edu

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Abstract

Traumatic events, such as sexual/physical assaults and motor vehicle accidents, frequently involve individuals who are under the influence of alcohol. Research suggests that peri-traumatic alcohol ingestion can increase the risk for PTSD symptomatology, particularly intrusive memories. However, no studies have examined the impact of alcohol on what participants remember about a laboratory-controlled stressful event or the number of intrusive memories that result from such an event. Thus, in the present study, participants ingested ethanol or a placebo and were then exposed to a modified version of the Trier Social Stress Test (TSST) or the friendly-TSST (f-TSST). The TSST required participants to deliver a ten-minute speech in front of two lab panel members as part of a mock job interview; the f-TSST required participants to casually converse with panel members about their interests and hobbies. In both conditions, the panel members interacted with (central) or did not interact with (peripheral) several objects sitting on a desk in front of them. The next day, participants' memory for the objects that were present on Day 1 was assessed with recall and recognition tests. We also quantified participants' intrusive memories on Days 2, 4, 6, and 8. Participants exposed to the TSST exhibited greater recall of central and peripheral objects and fewer falsely recalled objects than participants exposed to the f-TSST. Importantly, on Days 2 and 4, participants exposed to the TSST reported a greater number of intrusive memories related to the speech task, and this effect was augmented by alcohol. Our findings suggest that memory for a stressful event is enhanced, relative to memory for a non-stressful event, and that alcohol may increase the development of intrusive memories related to a stressful experience.

Keywords

Stress, Alcohol, Intrusive Memories, Cortisol, PTSD

1. Introduction

Research examining what individuals remember about a stressful event can provide important information about a victim's memory of a crime, the accuracy of eyewitness testimony, and the mechanisms underlying the formation of intrusive memories, a cardinal symptom of post-traumatic stress disorder (PTSD). People remember stressful, emotionally arousing events better than emotionally neutral events; in particular, they exhibit better memory for aspects that they consider "central" to the event. However, enhanced memory for these central details can come at a cost of impaired memory for peripheral details [1] [2], which aligns with research showing that stress enhances or preserves memory for arousing information, while impairing memory for non-arousing (*i.e.*, neutral) information [3]-[5]. This trade-off effect adaptively prioritizes the storage of information that may be related to the stressor (*i.e.*, "central," arousing information), which could subsequently enhance survival, over unrelated information (*i.e.*, "peripheral," nonarousing information). The enhanced memory for central details is suspected to occur due to stress turning the prefrontal cortex (PFC) "offline," leading to narrowed attention, and causing an individual to focus on what is most important at the time of the stress [6] [7]. Numerous studies over the past half-century have revealed that the stress- or arousal-induced modulation of memory is produced by an interaction between corticosteroid and noradrenergic mechanisms in the amygdala, which alters memory storage in other cognitive brain regions, such as the hippocampus and PFC [8]-[10].

Most research studying the effects of stress on memory has utilized stressors that are extrinsic to the learning task (see [11] for a discussion). This has helped us understand how stress impacts memory for other information, but it has generally failed to tell us what individuals remember about the stress experience itself. Recently, some investigators modified the Trier Social Stress Test (TSST) to study what participants remembered about the stressful experience [12]. In the study, participants in the stress condition prepared (anticipatory phase) and delivered (speech phase) a speech while being video recorded in front of a panel of laboratory assistants who were dressed in white lab coats and expressing a neutral demeanor. Participants in the control group spoke in front of the panel members, but they were explicitly told that they were in a control group, introduced to the panel members prior to speaking to them, asked to discuss their hobbies and career aspirations, and were not video recorded; this control manipulation has been referred to as the friendly TSST (f-TSST) [13]. To test participants' memory for the speech experience in each condition, the investigators placed numerous office objects on a desk at which the panel members sat. Half of the objects were used by the panel members during the speech task (*i.e.*, "central" objects), and half of the objects were not used by the panel members during the speech task (*i.e.*, "peripheral" objects). The investigators found that participants exposed to the TSST remembered significantly more central, but not peripheral, objects than did participants exposed to the f-TSST. These results were replicated in several subsequent publications [14]-[17], including recent work from our own laboratory [11]. In this study, we extended upon the initial findings by showing that participants exposed to the TSST reported intrusive memories up to 4 days following the speech task. Thus, the modified TSST paradigm is a useful tool to study factors that could impact an individual's memory for a stressful event.

One factor that has been overlooked by nearly all researchers who study the impact of stress on learning and memory is that stressful, often traumatic, events, such as motor vehicle accidents and physical or sexual assaults (and most crimes), frequently involve individuals who are under the influence of alcohol [18]-[26]. Due to these findings, it is essential to gain knowledge on how alcohol influences memory during stressful events to allow for a better understanding of an individual's ability to recall these events accurately. Similar to stress, alcohol causes individuals to focus more on central details of an experience, at the cost of peripheral details [27]. In addition, Bisby and colleagues found, on two separate occasions [28] [29], that a low (0.4 g/kg), but not high (0.8 g/kg), dose of alcohol resulted in greater emotionally-arousing memory intrusions in healthy participants than placebo. These findings relate to work showing that alcohol-related trauma was associated with more severe memory intrusions [30] and a slower recovery of PTSD-related intrusion symptoms [31] in traumatized individuals. The impact of alcohol on stress-related memories could be attributed to its well-known impairing effect on hippocampal function. Many controlled laboratory studies have shown that alcohol impairs explicit, hippocampus-dependent learning, while enhancing or leaving implicit, hippocampus-independent learning intact [32]-[41]. Furthermore, hippocampal function has been inversely associated with the frequency and severity of intrusive memories in PTSD patients [42]-[47]. Thus, we predict that, by compromising hippocampal function, a low dose of alcohol will alter the storage of the memory for a stressful event and promote the development of intrusive memories in participants.

The present study was designed to examine the impact of alcohol on an individual's memory for a stressful experience. Based on previous research [28] [29], participants ingested a low dose (0.4 g/kg) of alcohol or a placebo prior to undergoing the TSST of f-TSST. We then measured participants' memory for office objects that were (central) or were not (peripheral) manipulated by the panel members, as described above. We also tracked participants' intrusive memories for a period of one week following the TSST or f-TSST manipulation. We predicted that alcohol may produce an additive effect and augment the stress-induced enhancement of memory for central details. Most importantly, because of its impact on hippocampal function, we predicted that alcohol would lead to a greater number of intrusive memories in TSST-exposed participants.

2. Material and Methods

2.1. Participants

The present study was pre-registered at the Open Science Framework (https://doi.org/10.17605/OSF.IO/96P8E). A priori power analyses were performed with G*Power. These analyses suggested that we would need 140 participants to detect medium-sized ($f = 0.25$) interactive effects of 3 independent variables (stress, alcohol, and sex; 6 groups), with 80% power and an alpha equal to 0.05. However, because of the exclusion criteria for participating in our study (see below) and the size of our institution, we were unable to achieve this overall sample size. Therefore, we eliminated the factor of sex in our analyses and examined the interactive effects of 2 independent variables (stress and alcohol; 4 groups) on our measures. We acknowledge that our sample is still underpowered to detect medium-sized interactive effects between these variables, and as such, encourage the reader to consider our findings preliminary in nature.

We were able to recruit 35 healthy undergraduate students [17 males, 18 females; age: $M = 21.94$, $SD = 1.85$] from Ohio Northern University to participate. To participate, individuals had to be at least 21 years of age, self-reported social drinkers (report consuming alcohol at least once per month), and if female, taking some form of hormonal contraception or be sexually inactive for at least one month prior to the first experimental session. Individuals were excluded from participating if they met any of the following conditions: pregnant; allergic to cranberry juice; problematic drinking (defined as scores $\geq$ 10 on the Alcohol Use Disorders Identification Test); past or present substance use disorder; direct family history (*i.e.*, parent and/or sibling) of alcohol use disorder; self-reported aggressive behavior associated with past alcohol ingestion; current daily use of anticonvulsants, antipsychotics, muscle relaxants, sleep medication, benzodiazepines, antiarrhythmic agents, or beta blockers; use of acetaminophen or NSAIDs within 6 h of the first experimental session; past or present cardiovascular disease, hypertension, liver disease, pancreatic disorders, diabetes, Addison's disease, adrenal gland disorders, peripheral vascular disease, social anxiety disorder, or panic disorder; resting heart rate < 55 bpm; alcohol consumption within 24 h of the first experimental session; body weight less than 110 lbs (females) or 132 (males); history of syncope or vasovagal response to stress. Participants were also required to avoid engaging in strenuous exercise for 24 h prior to the first experimental session and not to eat or drink anything but water for 2 h prior to the first experimental session. All experimental procedures were approved by the Institutional Review Board at Ohio Northern University, carried out in accordance with the Declaration of Helsinki, and undertaken with the understanding and written consent of each participant.

2.2. Experimental Procedures

Participants were randomly assigned to stress (TSST)/no stress (f-TSST) and alcohol/placebo conditions. This resulted in 4 cells: stress + alcohol ($n = 9$; 4 males, 5 females), stress + placebo ($n = 9$; 5 males, 4 females), no stress + alcohol ($n = 9$; 5 males, 4 females), no stress + placebo ($n = 8$; 3 males, 5 females). A participant flow diagram can be found in Figure 1.

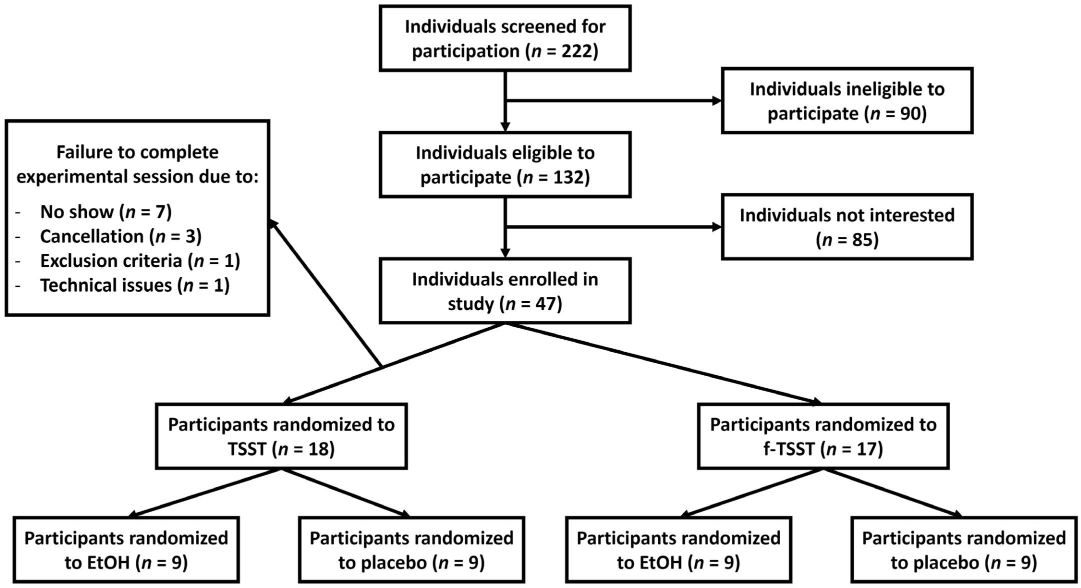

Figure 1. Participant flow diagram.

2.2.1. Waiting Period

Upon entering the laboratory on Day 1, participants provided informed consent in Room 1. After providing consent, participants were weighed to calculate the appropriate dose of ethanol (EtOH) and escorted to the restroom, where they were asked to void. Female participants also completed a self-administered pregnancy test to confirm they were not pregnant. Participants were then returned to Room 1 for a 15-min waiting period, after which the drinking phase began.

2.2.2. Drinking Phase

1) *Alcohol condition*. Participants in the alcohol condition consumed a mixture of 75.5% EtOH and cranberry juice, which was combined to produce a dose of 0.4 g/kg EtOH. This dose was chosen based on previous work showing that it led to increased intrusive memories in healthy participants [28] [29]. The mixture was equally divided into ten 50-ml portions, each of which was sprayed with a mist of EtOH. Drinks were administered every 3-min throughout the 30-min drinking phase.

2) *Placebo condition*. Participants in the placebo condition consumed a mixture of 498 ml cranberry juice and 2 ml EtOH. The mixture was equally divided into

ten 50-ml portions, each of which was sprayed with a mist of EtOH. Drinks were administered every 3-min throughout the 30-min drinking phase.

2.2.3. Stress Procedure

1) *Trier Social Stress Test* (*TSST*). Participants assigned to the TSST condition were exposed to a modified TSST paradigm, based on previous work [12] [14]-[17] [48] [49], that enabled one's memory for the stress experience to be quantified. Participants were taken to Room 2 of the laboratory and instructed that their objective was to deliver a 10-min speech to a company's staff managers (two laboratory panel members) to show they were the best applicant for a vacant position. Participants were told that the panel members were trained to monitor nonverbal behaviors and that their speech would be recorded to analyze their voice frequency and performance. Participant questions were only answered if they pertained to the directions; participants were told "that is unspecified" to unrelated questions. Following the instructions, participants were guided back to Room 1 by the lead experimenter and given 5 min to take notes for the interview (*i.e.*, anticipation phase); participants in the TSST condition were not permitted to use these notes during the speech. After 5 min had elapsed, the lead experimenter guided participants back to Room 2 and turned on the camera. Participants then began their speech. Throughout the speech, the two panel members (one male and one female) systematically interacted with various objects placed on a desk in front of them. Similar to the original TSST procedure, panel members wore standard white laboratory coats and kept a neutral demeanor, and an "active" panel member responded to the participant with standardized instructions when necessary. For consistency, "active" panel members were always the opposite sex of the participant.

2) *Friendly TSST* (*f-TSST*). As in previous work [13], the control condition was a modified version of the TSST known as the f-TSST; this manipulation seemingly removed the stress component from the task while maintaining the manipulation of desktop objects by the panel members. Participants were taken to Room 2 of the laboratory and explicitly told that they were in the control condition and that they would be having a 10-minute conversation about their career goals and aspirations, hobbies, and favorite movies/books with two students from the laboratory (*i.e.*, the two laboratory panel members). Participants were also told that they would not be videotaped. Following the instructions, participants were guided back to Room 1 by the lead experimenter and given 5 min to take notes for the conversation; participants in the f-TSST condition were permitted to use these notes during the conversation. After 5 min had elapsed, the lead experimenter guided participants back to Room 2, and participants began conversing with the panel members. Throughout the conversation, both panel members (active and passive) engaged in conversation with the participants and acted interested and friendly. The panel members did not wear laboratory coats, but their outfits were matched in appearance to those worn during the TSST. As in the TSST condition,

the panel members systematically interacted with various objects placed on a desk in front of them. Additionally, as in the TSST condition, the panel member of the opposite sex sat in the "active" panel member position.

3) *Object interactions*. The objects utilized during the experiment were the same for both conditions. A total of 22 different objects were located within the laboratory room in the view of the participants as they gave their speech or had their conversation with the panel members. Objects that were manipulated during the speech/conversation were deemed "central" to the event, and objects that were not interacted with were deemed "peripheral" to the event. The object interactions were standardized for each panel member. Additionally, each panel member sat on the same side of the table for each condition. From the participant's view, the active panel member was always on the left, and the passive panel member was always on the right.

2.2.4. Memory Assessments

The next day, participants returned to the laboratory and were instructed to recall as many of the 22 objects that were on or around the desk of the panel members on the previous day as they could. Participants were not timed; instead, they were instructed to inform the experimenter when they could no longer remember any more objects. Participants then completed a recognition assessment. A PowerPoint presentation was projected on a screen in front of participants and included 66 objects. Before each object was presented, a black fixation cross was presented in the middle of a white background for 1 s, after which an object was presented in the middle of a white background for 2 s. Participants then had 5 s to place an "x" in a box of the recognition assessment questionnaire, on a scale of 1 to 6, with 1 being very confident that they did not see the object on the previous day and 6 being very confident that they did see the object on the previous day. Objects included 22 old objects, 22 new objects, and 22 similar objects. Old objects were those present on or around the desk on the previous day; old "central" objects were those that were interacted with by the panel members, and old "peripheral" objects were those that were not interacted with by the panel members. New objects were those never present on the previous day but could have been (e.g., clock, tape dispenser, notebook). Similar objects were foils for the old objects, which included both central and peripheral foils.

2.2.5. Intrusive Memory Assessment

The intrusive memory questionnaire was administered on Days 2, 4, 6, and 8 of the experiment. The first questionnaire was completed in person by participants after the recognition assessment on Day 2; the remaining questionnaires were completed online via the Qualtrics survey platform. The questionnaire was an adaptation of similar questionnaires that have been employed to measure intrusive memories [50]-[53]. The first 3 questions were created by modifying the Impact of Event Scale [54], similar to prior work [51], to assess memory intrusions related to the speech (TSST) or conversation (f-TSST) from Day 1. Participants were instructed to think about the speech or conversation from Day 1 and rate, on a scale from 0 (not at all) to 4 (extremely) how often 1) other things kept making them think about it, 2) pictures about it popped into their mind, and 3) they thought about it when they didn't mean to. Participants were then given a definition of an intrusive memory. They were asked to report how many intrusive memories they had experienced in the past 24 h and whether they were mental images, verbal thoughts, or a combination of the two. The final three questions asked participants to rate how intense, upsetting, and bothersome the intrusive memories were on a scale of 0 (not at all) to 100 (extremely). Responses to the "type of intrusive memories experienced" and "how intense, upsetting, and bothersome the intrusive memories were" depended on whether participants experienced any intrusive memories. The responses to these questions thus declined across days, because participants were experiencing fewer intrusive memories.

2.2.6. Objective and Subjective Measures of Stress

1) *Salivary alpha-amylase and cortisol*. Saliva samples were collected from participants on Days 1 and 2 to assay for levels of alpha-amylase and cortisol. On Day 1, saliva samples were collected at the end of the 15-min waiting period, after the 5-min anticipatory phase, immediately following the speech (TSST) or conversation (f-TSST), and 15 min after the speech or conversation. On Day 2, saliva samples were collected upon participants entering the laboratory and after completing the intrusive memory questionnaire. Saliva samples were collected via SalivaBio Oral Swabs (Salimetrics, LLC; State College, PA). Participants were given a swab to place under their tongue, which was left for at least 1.5 min, after which it was collected and stored at −20˚C until assayed.

The first three samples from Day 1 were assayed for salivary alpha-amylase. All samples from Days 1 and 2 were assayed for salivary cortisol. The alpha-amylase and cortisol concentrations were determined by an investigator (BRR) blind to the condition of participants via enzyme immunoassay according to the manufacturer's protocols. The alpha-amylase assay (product # 1 - 1902; Salimetrics, LLC; State College, PA) has a range of 2 - 400 U/ml and a sensitivity of 0.4 U/ml. The cortisol assay (product # 500360; Cayman Chemical Co.; Ann Arbor, MI) has a range of 6.6 - 4000 pg/ml and a sensitivity of 35 pg/ml. The intra-assay coefficients of variation were less than 3%, and the inter-assay coefficients of variation were less than 15%.

2) *State anxiety*. Participants' self-reported anxiety levels were assessed at multiple time points on Day 1. Participants completed the "state" portion of the State-Trait Anxiety Inventory (STAI) [55] during the last 5 min of the 15-min waiting period, following 5-min anticipatory phase, and immediately after the speech (TSST) or conversation (f-TSST).

2.2.7. Objective and Subjective Measures of Intoxication

1) *Breath alcohol concentration* (*BrAC*). BrAC was measured in participants with a BACtrack S80 throughout Day 1. BrAC was collected before the consent procedure to confirm sobriety, at the end of the 15-min waiting period, at minute

14 and 29 of the drinking phase, after the 5-min anticipation phase, after the speech task, and 15-min after the speech task.

2) *Subjective intoxication ratings*. Participants rated their perceived level of intoxication on a scale from 1 ("I feel completely sober") to 10 ("I feel completely drunk") at the end of the 15-min waiting period, at minute 14 and 29 of the drinking phase, after the 5-min anticipation phase, after the speech task, and 15-min after the speech task.

2.2.8. Sobering Room

Upon completion of Day 1 experimental procedures, participants were escorted to a sobering room by the lead experimenter and a sobering room chaperone. The sobering room chaperone checked participants' BrAC every 10 min. Participants who ingested alcohol had to stay in the sobering room at least 30 min, and their BrAC had to be below 0.02 before they were allowed to leave. Participants in the placebo condition were released after 1 h.

2.3. Statistical Analyses

Recall was divided into correct recall (central, peripheral) and false recall (*i.e.*, recalling objects not present on Day 1). Recognition memory was measured by first converting participant confidence ratings (scale of 1 to 6) to "seen" (scores 4, 5, 6) and "not seen" (scores 1, 2, 3), which enabled the computation of hits and/or false alarms for old central, old peripheral, similar central, and similar peripheral objects. Discrimination indices were then calculated for central and peripheral objects as p (hits) – p (false alarms), according to the Two-High Threshold Model [56] [57]. As in prior work [58] [59], confidence ratings from the recognition memory assessment were used to create receiver operating characteristic (ROC) curves for central and peripheral objects by plotting the p (hits) for old objects and the p (false alarms) for similar objects (*i.e.*, foils). The area under each ROC curve was then calculated for statistical analysis.

Mixed-model ANOVAs were used to analyze breath alcohol content (BrAC), subjective intoxication ratings, salivary cortisol levels, salivary alpha amylase levels, and state anxiety, with stress (TSST, f-TSST) and alcohol (alcohol, placebo) serving as the between-subjects factors and time point of measurement serving as the within-subjects factor. Mixed-model ANOVAs were also used to analyze correct recall, discrimination indices, and area under the ROC curves, with stress and alcohol serving as the between-subjects factors and object type (central, peripheral) serving as the within-subjects factor. A two-way ANOVA was used to analyze false recall, with stress and alcohol serving as the between-subjects factors. Repeated measures multivariate ANOVAs (MANOVAs) were used to analyze the first three questions of the intrusive memory questionnaire and the number of intrusive memories reported by participants (collectively referred to as "intrusive memory phenomena"); stress and alcohol served as the between-subjects factors, and question (Q1, Q2, Q3, number of intrusive memories) and time point of measurement (Day 2, Day 4, Day 6, Day 8) served as the within-subjects factors.

Responses to the "type of intrusive memories experienced" and "how intense, upsetting, and bothersome the intrusive memories were" depended on whether participants experienced any intrusive memories. The responses to these questions thus declined across days, because participants were experiencing fewer intrusive memories. Thus, we used separate MANOVAs for each day to analyze these measures; stress and alcohol served as the between-subjects factors, and question served as the within-subjects factor.

All statistical analyses were performed with IBM SPSS Statistics Version 29.0.2.0. Data points that were more than 3 standard deviations beyond the exclusive group mean were removed from the analyses. Less than 1% of all data were excluded. Alpha was set at 0.05 for all analyses, and Bonferroni-corrected post hoc tests were employed when the omnibus F indicated a significant effect. If the assumption of sphericity was violated, Greenhouse-Geisser corrections were employed, with reduced degrees of freedom reported.

3. Results

3.1. Subjective and Objective Intoxication Measures

3.1.1. Breath Alcohol Concentration (BrAC)

At the end of the waiting period, there were no significant differences in BrAC between participants who ingested alcohol and participants who ingested placebo (see **Figure 2(a)**). However, halfway through the drinking phase, participants who ingested alcohol exhibited significantly greater BrAC than participants who ingested placebo; this difference remained significant throughout the remainder of the first experimental session (effect of alcohol: $F(1, 30) = 427.28$, $p < 0.001$, $\eta_p^2 = 0.93$; effect of time: $F(1.47, 43.95) = 63.78$, $p < 0.001$, $\eta_p^2 = 0.68$; Alcohol x Time interaction: $F(1.47, 43.95) = 62.57$, $p < 0.001$, $\eta_p^2 = 0.68$). In participants who ingested alcohol, BrAC spiked at the end of the drinking session ($M = 0.102$), but by the end of the anticipation phase, it significantly decreased to ~0.04 and remained at that level until the end of the session. No other effects were significant (all $F < 2.03$; all $p > 0.16$).

3.1.2. Subjective Intoxication Ratings

At the end of the waiting period, there were no significant differences in subjective intoxication ratings between participants who ingested alcohol and participants who ingested placebo (see **Figure 2(b)**). However, halfway through the drinking phase, participants who ingested alcohol exhibited significantly greater subjective intoxication ratings than participants who ingested placebo; this difference remained significant throughout the remainder of the first experimental session (effect of alcohol: $F(1, 30) = 12.64$, $p = 0.001$, $\eta_p^2 = 0.30$; effect of time: $F(2.88, 86.41) = 16.83$, $p < 0.001$, $\eta_p^2 = 0.36$; Alcohol x Time interaction: $F(2.88, 86.41) = 5.92$, $p = 0.001$, $\eta_p^2 = 0.17$). In participants who ingested alcohol, subjective intoxication ratings spiked at the end of the drinking session ($M = 2.82$) and remained at that approximate level until the end of the session. No other effects were significant (all $F < 2.44$; all $p > 0.12$).

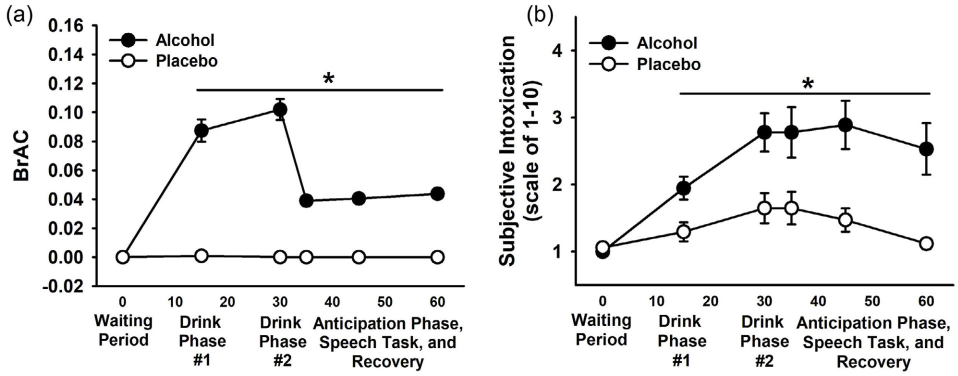

Figure 2. Participants who ingested alcohol exhibited a significant increase in BrAC during the drinking phase (a). Following the drinking phase, BrAC decreased significantly in these participants, but remained elevated, relative to participants who ingested placebo, throughout the remainder of the experimental session. The effects for subjective intoxication levels mirrored those for BrAC (b). Participants who ingested alcohol exhibited a significant increase in subjective intoxication levels during the drinking phase, and these levels remained elevated, relative to participants who ingested placebo, throughout the remainder of the experimental session. Data are presented as means ± SEM. * $p < 0.05$ relative to placebo.

3.2. Subjective and Objective Stress Response Measures

3.2.1. Salivary Cortisol

On Day 1, all participants, independent of alcohol or stress condition, exhibited a significant increase in salivary cortisol levels between the end of the anticipation phase and the end of the speech task (see Figure 3(a)) (effect of time: $F(3, 93) = 3.16$, $p = 0.028$, $\eta_p^2 = 0.09$). No other effects were significant (all $F < 2.43$; all $p > 0.07$). Because of *a priori* hypotheses, we probed the Day 1 salivary cortisol data further to determine whether stressed participants may have exhibited a greater increase in salivary cortisol levels than non-stressed participants. To do so, we calculated a percent change score by subtracting the post-anticipation phase salivary cortisol levels from the post-speech task salivary cortisol levels and dividing the difference by the post-anticipation phase salivary cortisol levels. A two-way ANOVA was performed on the resulting percent change scores, with stress and alcohol serving as the between-subjects factors. This analysis revealed a marginally significant effect of stress, $F(1, 31) = 4.10$, $p = 0.052$, $\eta_p^2 = 0.12$, suggesting that stressed participants exhibited a greater increase in salivary cortisol levels than non-stressed participants.

The analysis of Day 2 salivary cortisol levels revealed that, prior to memory testing, stressed participants who ingested alcohol exhibited significantly lower salivary cortisol levels than stressed participants who ingested placebo (Stress x Alcohol x Time interaction: $F(1, 31) = 4.75$, $p = 0.037$, $\eta_p^2 = 0.13$). No other effects were significant (all $F < 2.56$; all $p > 0.12$).

3.2.2. Salivary Alpha-Amylase

All participants, independent of alcohol or stress condition, exhibited a significant increase in salivary alpha-amylase between the waiting period and the anticipation phase (see Figure 3(b)) (effect of time: $F(2, 60) = 10.12$, $p < 0.001$, $\eta_p^2 = 0.25$).

There was also a statistical trend suggesting that, by the end of the speech task, stressed participants who ingested alcohol displayed greater salivary alpha-amylase levels than stressed participants who ingested placebo (Stress x Alcohol interaction: $F(1, 30) = 4.21$, $p = 0.049$, $\eta_p^2 = 0.12$; Stress x Alcohol x Time interaction: $F(2, 60) = 3.01$, $p = 0.057$, $\eta_p^2 = 0.09$). No other effects were significant (all $F < 1.66$; all $p > 0.20$).

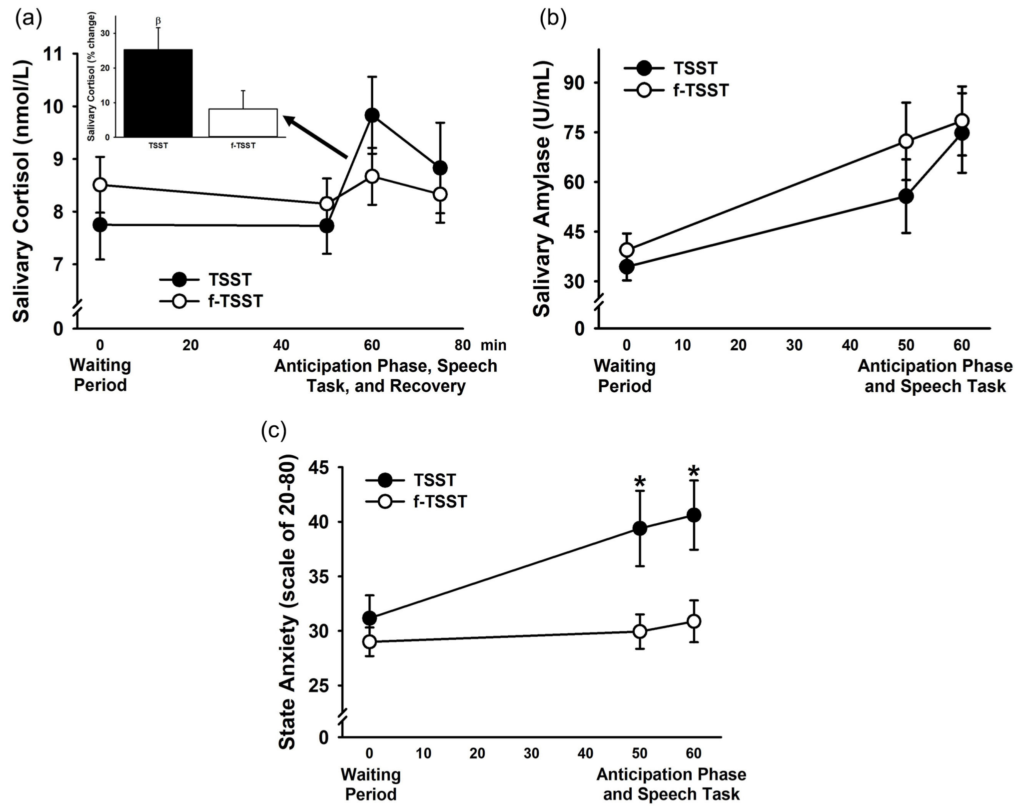

Figure 3. There were no significant group differences for salivary cortisol levels during the Day 1 experimental session (a). However, we calculated a percent change score for the difference in salivary cortisol levels between the end of the anticipation phase and the end of the speech task. Analysis of this percent change score revealed a greater increase in salivary cortisol levels in participants exposed to the TSST. All participants, independent of stress or alcohol condition, exhibited a significant increase in salivary alpha-amylase levels during the anticipation phase and speech task (b). Participants exposed to the TSST reported significantly greater state anxiety levels than participants exposed to the f-TSST at the end of the anticipation phase and following the speech task (c). Data are presented as means ± SEM. $\beta = p = 0.052$ relative to f-TSST; * $p < 0.05$ relative to f-TSST.

3.2.3. State Anxiety

At the end of the waiting period, there were no significant differences in state anxiety between stressed and non-stressed participants (see Figure 3(c)). However, stressed participants exhibited greater state anxiety than non-stressed participants at the end of the anticipation phase and at the end of the speech task (effect of stress: $F(1, 31) = 6.57$, $p = 0.015$, $\eta_p^2 = 0.18$; effect of time: $F(2, 62) = 6.83$, $p = 0.002$, $\eta_p^2 = 0.18$; Stress × Time interaction: $F(2, 62) = 3.80$, $p = 0.028$, $\eta_p^2 = 0.11$). No other effects were significant (all $F < 1.39$; all $p > 0.25$).

3.3. Memory Assessments

3.3.1. Free Recall (True)

Alcohol had no significant impact on object memory (see **Figure 4(a)**). Both stressed and non-stressed participants recalled significantly more central objects than peripheral objects (effect of object type: $F(1, 27) = 53.55$, $p < 0.001$, $\eta_p^2 = 0.67$). However, stressed participants recalled significantly more central and peripheral objects than non-stressed participants (effect of stress: $F(1, 27) = 11.82$, $p = 0.002$, $\eta_p^2 = 0.30$). The effect of stress on object memory appeared to be stronger for central objects than for peripheral objects; however, the Stress × Object Type interaction was only marginally significant, $F(1, 27) = 3.16$, $p = 0.087$, $\eta_p^2 = 0.11$. No other effects were significant (all $F < 0.08$; all $p > 0.78$).

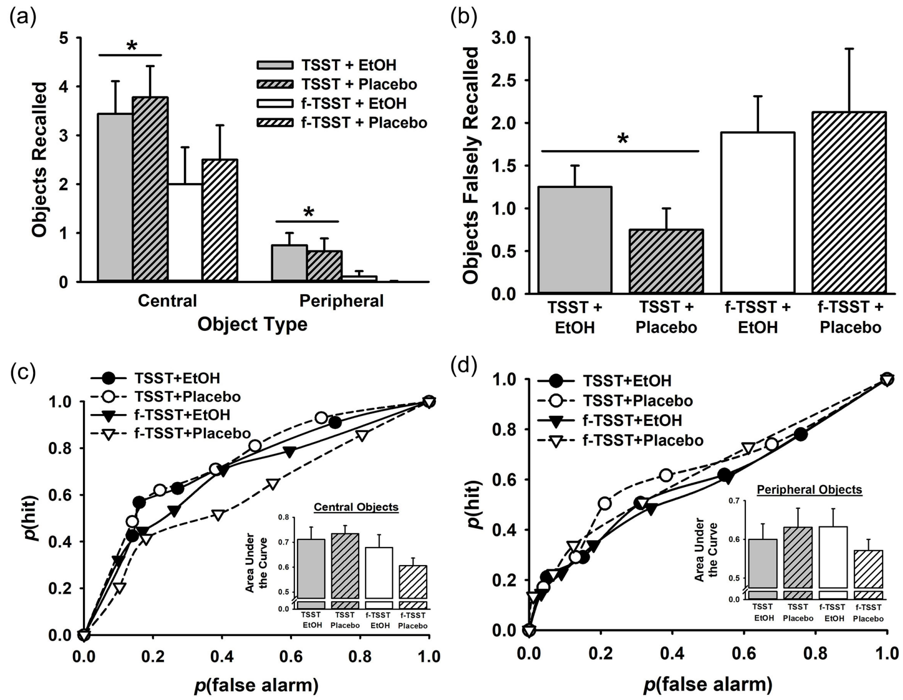

Figure 4. Participants exposed to the TSST recalled significantly more central and peripheral objects than participants exposed to the f-TSST (a). Participants exposed to the TSST also falsely recalled fewer objects than participants exposed to the f-TSST (b). No significant effects were observed for recognition of central (c) or peripheral (d) objects. Data are presented as means ± SEM. * $p < 0.05$ relative to f-TSST.

3.3.2. Free Recall (False)

Alcohol had no significant impact on false memory for objects (see **Figure 4(b)**). Stressed participants falsely recalled fewer objects than non-stressed participants, $F(1, 29) = 4.77$, $p = 0.037$, $\eta_p^2 = 0.14$. No other effects were significant (all $F <$ 0.64; all $p > 0.77$).

3.3.3. Recognition

The analysis of discrimination indices for recognition memory revealed no significant effects (all $F < 1.91$; all $p > 0.17$). Confidence ratings from the recognition memory assessment were used to plot ROC curves for central and peripheral objects, and the areas under each curve (AUC) were analyzed (see **Figure 4(c)** and **Figure 4(d)**). The AUC for central objects was significantly greater than the AUC for peripheral objects (effect of object type: $F(1, 29) = 6.29$, $p = 0.018$; $\eta_p^2 = 0.18$). No other effects were significant (all $F < 2.89$; all $p > 0.10$).

3.3.4. Intrusive Memories (See Figure 5)

The completion rates of the intrusive memory questionnaires were as follows: Day 2 (100%), Day 4 (100%), Day 6 (100%), Day 8 (97%).

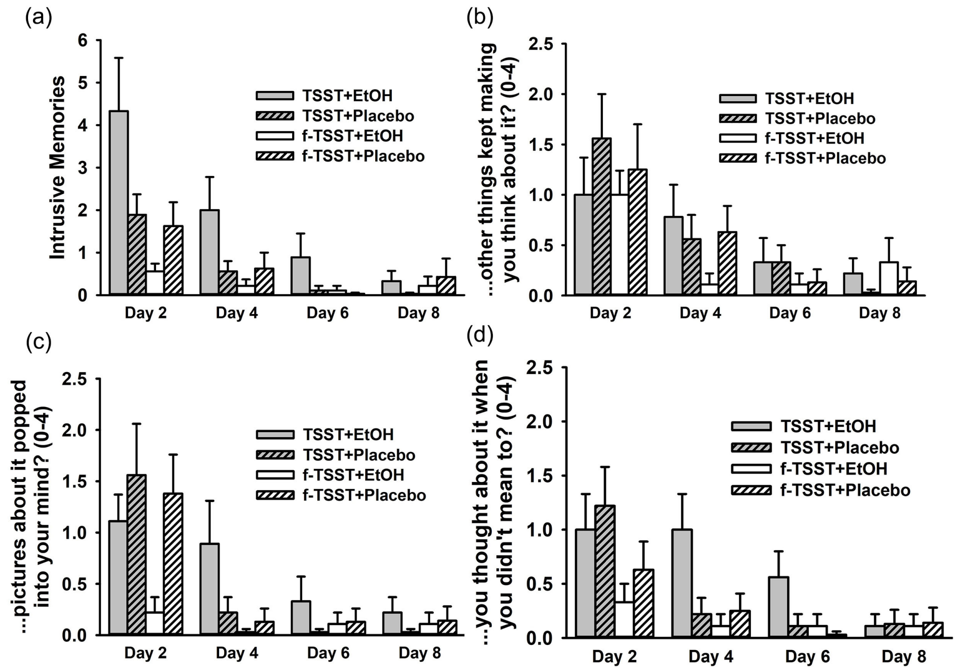

Figure 5. Participants exposed to the TSST reported more intrusive memory phenomena than participants exposed to the f-TSST. This effect was driven by the ingestion of alcohol. Participants who ingested alcohol and were exposed to the TSST reported a greater number of intrusive memories than all other groups (a). The intrusive memory phenomena observed in this group also tended to decrease more slowly over time ((a) (c) (d)). Data are presented as means ± SEM.

1) *Q1…other things made you think about it*, *Q2…pictures popped into your mind*, *Q3…thought about it when you didn't mean to*, *Q4 number of intrusive memories* (*collectively referred to as* "*intrusive memory phenomena*"). The repeated measures MANOVA revealed significant effects of question, $F(1.57, 47.19) = 8.67$, $p < 0.001$, $\eta_p^2 = 0.22$, and day, $F(1.86, 55.93) = 40.42$, $p < 0.001$, $\eta_p^2 = 0.57$, as well as significant Stress × Question, $F(1.57, 47.19) = 3.73$, $p = 0.041$, $\eta_p^2 = 0.11$, Question × Day, $F(2.78, 83.36) = 5.89$, $p = 0.001$, $\eta_p^2 = 0.16$, Question × Stress × Alcohol, $F(1.57, 47.19) = 4.47$, $p = 0.024$, $\eta_p^2 = 0.13$, and Question × Day × Stress, $F(2.78, 83.36) = 3.50$, $p = 0.022$, $\eta_p^2 = 0.11$, interactions. The Question × Day × Stress × Alcohol interaction was initially significant, but after the Greenhouse-Geisser correction due to a violation of sphericity, it remained only a statistical trend, $F(2.78, 83.36) = 2.74$, $p = 0.053$, $\eta_p^2 = 0.08$. The significant

effects revealed that the magnitude of intrusive memory phenomena reported by participants decreased each day. On Days 2 ($p = 0.021$) and 4 ($p = 0.05$), stressed participants reported greater intrusive memory phenomena than non-stressed participants. These effects seemed to be largely driven by the ingestion of alcohol. For instance, stressed participants who consumed alcohol reported a greater number of intrusive memories, overall, than all other groups. Although the Question × Day × Stress × Alcohol interaction was only a statistical trend, we examined the post hoc comparisons for any meaningful differences. These comparisons revealed that the greater number of intrusive memories reported by alcohol-treated stressed participants was primarily evident on Days 2 and 4, but that these participants also reported greater scores on Q2 (pictures) and Q3 (thoughts) than other groups on Days 2, 4, and 6.

4. Discussion

The purpose of the present study was to examine the impact of alcohol on an individual's memory for a laboratory-controlled stressful event, as well as any intrusive memories that developed subsequent to the event. As in past work [11] [12] [14]-[17], exposing participants to the TSST paradigm resulted in greater recall of central objects, relative to controls. Interestingly, TSST exposure also enhanced the recall of peripheral objects, an effect that has not been consistently observed in previous work. We also replicated our previous finding [11] that TSST exposure led to fewer falsely recalled objects than controls, supporting the idea that exposure to the stressful TSST experience results in greater memory accuracy for objects associated with the event. Alcohol, alone or in combination with stress, had no significant impact on recall or recognition memory. However, the ingestion of alcohol did exacerbate the effects of TSST exposure on intrusive memory phenomena. TSST exposure, alone, led to greater intrusive memory phenomena than what was observed in controls, particularly on Days 2 and 4, but alcohol intensified these effects and resulted in a greater number of intrusive memories in stressed participants. Collectively, these findings provide important replication of past memory-related observations with this paradigm; they also suggest that alcohol may result in greater deleterious psychological effects (e.g., intrusive memories) when ingested in combination with stressful experiences.

Previous work with the TSST paradigm that was used in the present study has shown that stress exposure results in greater recall [11] [14]-[16] and/or recognition [12] [14]-[17] of central objects, relative to non-stressed participants. Some of this work has also revealed greater memory for peripheral objects in stressed participants [14] [15], as observed here. Some research has shown that increased levels of circulating corticosteroids, in conjunction with increased physiological arousal, can broaden memory and selectively enhance the recognition of peripheral details [48]. However, even when TSST exposure has enhanced memory for peripheral objects in past work, the effect of TSST exposure on central object memory has been stronger [14] [15]. In theory, TSST exposure results in narrowed attention for stress-related information, which enhances the encoding of objects tied directly to the stress experience. This perspective is consistent with the finding of Herten *et al.* [15], in which stressed participants exhibited greater eye fixation on central objects than did controls. It also aligns with recent work from Bierbrauer and colleagues [14] who found that, in stressed participants, the neural representations of central objects in the amygdala became more similar to one another as a result of "emotional binding" of the stress-related information, thus hypothetically making retrieval of this information easier. Both the TSST and f-TSST appear to increase participant arousal (e.g., salivary alpha-amylase); however, only the TSST has led to a consistent increase in salivary cortisol [12] [14]-[17]. Thus, the differential impact of the TSST and f-TSST on memory for central objects is likely associated with corticosteroid-related effects and/or interactions with other neuromodulators in cognitive brain regions, such as the amygdala.

Most previous work studying the formation of intrusive memories has involved exposing participants to arousing films and quantifying intrusive memories thereafter. However, measuring intrusive memories that result from viewing a film is vastly different from measuring intrusive memories that result from an actual stress experience. We previously reported that TSST exposure increased intrusive memory phenomena up to 4 days after the experience [11]. In the present study, we replicated and extended these findings by revealing that alcohol exacerbates such effects. Specifically, alcohol led to a greater number of intrusive memories in stressed participants, particularly on Days 2 and 4. It also appeared to delay the decrease in subjectively reported symptoms associated with intrusive memories (e.g., see **Figure 5(c)** and **Figure 5(d)**). These findings are comparable to those reported by Bisby and colleagues [28] [29], in which alcohol increased intrusive memories in participants who observed arousing films. What is unique about our findings is that alcohol increased intrusive memories in participants who were actually exposed to a stressful experience.

Extensive work has shown that high doses of alcohol impair hippocampus-dependent, explicit memory [32]-[41]. A 0.4 g/kg dose of alcohol is a moderate dose that we did not expect to impair memory, in and of itself; it is also the dose that Bisby and colleagues [28] [29] found to increase intrusive memories in healthy participants. In theory, alcohol ingestion altered the manner in which the TSST experience was stored. Though it did not completely impair hippocampal function (as it did not worsen recall or recognition performance), the alcohol likely did change how the hippocampus was involved in storing the memory of the event. Presumably, compromising hippocampal function would result in less memory contextualization and cohesion and increase the likelihood of memory intrusions [60] [61].

It is important to consider the findings presented in this manuscript as preliminary in nature. Due to the size of the institution at which this study was conducted, we were unable to achieve a sample size that adequately powered our statistical analyses. The low sample size also prevented us from considering participant sex as a factor in the analyses. This is an important limitation, as previous work has shown that both stress [62] and alcohol [63] can differentially impact cognitive performance in males and females. Still, our findings support the idea that the modified TSST paradigm in this study can be a useful tool for studying emotional memories and the development of intrusive memories. In addition, they suggest that alcohol could result in greater deleterious psychological effects when combined with stressful experiences. Future research is necessary to provide a more thorough assessment of how alcohol interacts with stress to influence the development of intrusive memories.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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