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EFFECTS OF PERITRAUMATIC ALCOHOL INTOXICATION ON INTRUSIVE MEMORIES FOLLOWING EXPOSURE TO AN ANALOG TRAUMA

by

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A DISSERTATION

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EFFECTS OF PERITRAUMATIC ALCOHOL INTOXICATION ON INTRUSIVE MEMORIES FOLLOWING EXPOSURE TO AN ANALOG TRAUMA

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University of Nebraska, 2018

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Intrusive memories and associated symptoms of posttraumatic stress disorder (PTSD) represent a significant public health problem, often leading to persistent physical and psychological difficulties experienced by victims long after the traumatic event, contributing to healthcare costs and loss of productivity. Research examining etiological factors that contribute to PTSD is needed in order to expand basic knowledge and to inform the development of prevention and intervention strategies. Although acute alcohol intoxication has the potential to impact established risk factors for the development of intrusive memories (e.g., via stress response, cognitive processing), and traumaparticularly sexual assault-often occurs under the influence of alcohol, the influence of peritraumatic (i.e., at the time of assault) alcohol intoxication on post-assault trauma symptoms is not well understood. To address this issue, the current study utilized an experimental design, including lab-based alcohol administration (high dose of .72 g/kg, low dose of .36 g/kg, and a placebo beverage), a well-accepted analog trauma exposure paradigm (a film with distressing or "traumatic" content), and ecological momentary assessment of intrusive memories. Results from 98 community women (ages 21 to 30, without a personal history of victimization) revealed peritraumatic intoxication did impact the occurrence of intrusive memories. Specifically, a marginally significant indirect effect showed that alcohol myopia disrupted cognitive processing and formation

of trauma memories, resulting in increased intrusive memories at high levels of intoxication. At the same time, those who consumed high or low doses of alcohol displayed a dampened stress response, which reduced intrusive memories. Findings highlight the influence of peritraumatic cognitive impairment and stress response on the development of intrusive memories. Though alcohol influenced these risk factors simultaneously and in opposite directions, overall, participants in the high dose condition reported more intrusive memories than those in the placebo and low dose conditions. These findings reflect the importance of prevention and intervention programs aimed at reducing alcohol-involved victimization.

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CHAPTER 1: INTRODUCTION

Sexual assaults against women occur with startling frequency. About 18.3% of women in the U.S. report a history of rape and 44.6% of women report experiencing other sexual violence in their lifetime (Black et al., 2011). Sexual assaults often lead to a host of negative outcomes for victims, including the development of posttraumatic stress disorder (PTSD). In fact, about 2 in 5 rape victims develop symptoms of PTSD (Kilpatrick, Resnick, Ruggiero, Conoscenti, & McCauley, 2007), which include intrusions, avoidance, cognitive and emotional changes, and hyperarousal (American Psychiatric Association [APA], 2013). PTSD symptoms, in turn, are associated with an increased risk of suicide attempts, comorbid mental health diagnoses (Pietrzak, Goldstein, Southwick, & Grant, 2011), and physical health problems (Pacella, Hruska, & Delahanty, 2013). The high prevalence and suffering associated with assault-related intrusions make research examining risk factors essential to informing interventions. As reviewed below, current theoretical models have illuminated key processes involved in the development of intrusive memories, and point to certain peritraumatic (i.e., occurring at the time of assault) cognitive processes and stress-related responses that appear to be key in the development of intrusions. However, current models do not account for other peritraumatic factors that may affect cognitive and stress-related reactions associated with intrusions. In the case of sexual assault, alcohol intoxication is important to consider, given that about half of victims are under the influence of alcohol at the time of assault (Abbey, Zawacki, Buck, Clinton, & McAuslan, 2004). Because alcohol has the potential to impact key mechanisms contributing to intrusive memories, it is imperative to

understand the role of peritraumatic intoxication in the development of post-assault intrusions. The present study fills this need.

Intrusive Memories

Intrusion symptoms (referred to as "re-experiencing symptoms" in the DSM-IV; APA, 2000) have long been considered a hallmark of PTSD (e.g., Foa, Steketee, & Rothbaum, 1989). Although individual symptoms and symptom clusters that make up PTSD have recently been at the focus of much debate (see Brewin, 2013; Friedman, 2013; Maercker & Perkonigg, 2013), diagnostic criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; APA, 2013) and recent theoretical reviews (e.g., Brewin, 2013; Ehlers, Hackman, & Michael, 2004) reaffirm the centrality of intrusive memories to the understanding of PTSD. In fact, intrusion symptoms were retained as the first listed symptom cluster in the DSM-5 despite other changes in diagnostic criteria.

Although the term, "intrusive memories," has previously been used interchangeably with "involuntary memories" and "flashbacks," these terms actually represent distinct phenomena (Kvavilashvili, 2014). Intrusive memories refer to spontaneous recollections that are typically experienced as disturbing. Although positive intrusive memories are possible, intrusive memories are most often accompanied by negative affect. In addition, intrusive memories are also repetitive in nature. That is, intrusive memories often involve recurrent recollections of the same image or memory. Not only are intrusive memories symptoms of PTSD (APA, 2013), but due to the distressing nature of repetitive intrusions, an individual may also make attempts to avoid intrusive memories, which is also a symptom of PTSD. In contrast to intrusive memories, involuntary autobiographical memories can include a broad range of personal memories. Specifically, involuntary autobiographical memories refer to spontaneous recollections of past events that can be positive, negative, or neutral. Involuntary autobiographical memories were first noticed by Ebbinghaus (1885/1964), but were relatively neglected in cognitive psychology until Berntsen (1996, 1998) began to research this phenomenon. Although occasionally referenced in the context of trauma memories and PTSD (e.g., Berntsen, 2009), involuntary autobiographical memories do not necessarily evoke a desire to avoid the recollections in the way intrusive memories do (Kvavilashvili, 2014).

Intrusive memories (as defined by PTSD symptom B1) can also be distinguished from flashbacks (B3), which involve "dissociative reactions" (APA, 2013). This dissociative quality of flashbacks can refer to a sense that the memory is occurring "here and now" (Ehlers & Clark, 2000) or that there is a sense of "nowness" associated with the trauma memory (Brewin, 2014; Ehlers et al., 2004). In other words, a flashback may be experienced as "reliving" the event (Kvavilashvili, 2014) and may involve a temporary loss of connection with the present (Brewin, Gregory, Lipton, & Burgess, 2010). Flashbacks are also typically accompanied by a high degree of physiological arousal and involve negative affect (Kvavilashvili, 2014). Moreover, although intrusive memories can occur in the context of a variety of other mental disorders (see Hackmann & Holmes, 2004) including depression (e.g., Reynolds & Brewin, 1999) and complicated grief (Boelen & Huntjens, 2008), flashbacks are specific to PTSD (Bryant, O'Donnell, Creamer, McFarlane, & Silove, 2011). Further, compared to intrusive memories in depressed patients, flashbacks (defined as intrusions experienced by those with PTSD) appear to have a more dissociative or "here-and-now" quality (Birrer, Michael, & Munsch, 2007; Reynolds & Brewin, 1998, 1999) and involve more sensory information (Parry & O'Kearney, 2014).

One way to resolve the use of inconsistent terminology in past research is to conceptualize spontaneous memories along a continuum from involuntary autobiographical memories to flashbacks, with intrusive memories falling in the middle (Kvavilashvili, 2014). Intrusive memories, which are the focus of the present study, include distressing thoughts or images of a prior event. Intrusive memories involve more negative emotion than memories that are simply involuntary, but may not involve a sense that the event is happening in the moment, as is the case with flashbacks. Intrusive memories that can be evoked through experimental laboratory procedures, without risk for long-term symptoms of PTSD (see Holmes & Bourne, 2008; James et al., 2016).

Intrusive Memories and Other PTSD Symptoms

Intrusive memories (and the more severe experience of flashbacks) are considered a hallmark of PTSD due to their potential to increase and perpetuate associated PTSD symptoms. For example, the disturbing nature of intrusive memories may motivate an individual to avoid reminders of the trauma (e.g., going out of one's way to avoid seeing the location where the trauma occurred). An individual may also worry that the experience of intrusive memories is abnormal and make negative appraisals of intrusion symptoms, such as "I'm going mad" or "I'll never get over this" (Ehlers & Clark, 2000, p. 322). These negative appraisals may produce maladaptive attempts to reduce intrusive memories (e.g., avoidance of trauma reminders, thought suppression). Avoidance is problematic because it may prevent the individual from experiencing the trauma memory in the absence of fear. A lack of disconfirming evidence for this memory-fear response pairing can contribute to the persistence of a pathological fear structure associated with trauma reminders (Foa, Huppert, & Cahill, 2006; Foa & Kozak, 1986). Regularly experiencing a fear response may also contribute to a sense of current threat and hyperarousal. In addition, avoidance is known to interfere with making meaning of the trauma memory and integrating it into an autobiographical context, a process that is conceptually linked to the maintenance of PTSD (Ehlers & Clark, 2000). On the other hand, successful elaboration of a trauma memory into a meaningful narrative account is a key component of empirically supported PTSD interventions (e.g., Cognitive Processing Therapy [CPT]; Resick, Monson, & Chard, 2017). Given that intrusive memories are integral to the development and maintenance of PTSD, understanding their etiology is of crucial importance to informing intervention and prevention efforts.

Risk Factors for Intrusions

Cognitive processes. PTSD has been conceptualized as a disorder of memory (e.g., McNally, 2006). In addition to experiencing intrusive memories and flashbacks, individuals with PTSD often exhibit difficulty intentionally recalling the traumatic event. Instead, trauma memories are frequently remembered in a disorganized and fragmented manner (see Brewin 2014; Foa & Riggs, 1993). In addition, trauma narratives in those with PTSD often involve a focus on sensory and perceptual information (see O'Kearney & Perrott, 2006). Ehlers and Clark (2000) propose that the apparently discrepant experiences of increased intrusive memories and deficits in intentional recall are related in that one serves to increase the other. These authors suggest that disruption in the trauma memory (e.g., increased sensory information, lack of contextualization) can lead to a persistent sense of current threat, and is therefore a core ingredient for the development and maintenance of intrusive symptoms. In addition, a disorganized trauma memory can simultaneously reduce one's ability to voluntarily retrieve the memory and increase the likelihood of cue-driven recollections (Ehlers & Clark, 2000), which may manifest as intrusive memories. Supporting Ehlers and Clark's theoretical supposition, disorganized trauma memories have been linked empirically to both intrusive memories (Halligan, Clark, & Ehlers, 2002) and PTSD symptoms broadly (Halligan, Michael, Clark, & Ehlers, 2003; Harvey & Bryant, 1999; Jones, Harvey, & Brewin, 2007).

Yet, what causes this disruption of the trauma memory in the first place? The answer may lie in the way the trauma is first encoded into memory. Theorists have identified two forms of cognitive processing that are of relevance here: data-driven processing and conceptual processing (Ehlers & Clark, 2000; Roediger, 1990). Data-driven processing refers to low-level cognitive processes in which an individual focuses on the sensory information and perceptual input. On the other hand, conceptual processing refers to higher-level cognitive processes in which one makes meaning of the experience as well as organizes and integrates the trauma memory into the context of other autobiographical memories. In a parallel manner, the well-known dual representation model of PTSD (Brewin, 2001, 2014; Brewin, Dalgleish, & Joseph, 1996; Brewin et al., 2010) distinguishes between contextualized representations of a traumatic event that can be integrated into personal memories over time (C-reps) and sensation-based representations (S-reps). In each of these conceptualizations, data-driven processing (or S-reps) in the absence of conceptual processing (or C-reps) is associated

with increased intrusive memories. That is, data-driven processing has been associated with strong perceptual priming (Ehlers & Clark, 2000; Roediger, 1990), which can in turn increase the likelihood that later perceptual cues in the environment will evoke poorly elaborated and disorganized trauma memories (e.g., Halligan et al., 2002), resulting in intrusive and distressing memories (e.g., Ehring, Ehlers, & Glucksman, 2008; Halligan et al., 2002, 2003). These cognitive risk factors have been examined and supported in both correlational studies with trauma survivors and lab-based trauma analog studies in participants without a trauma history (see Holmes & Bourne, 2008).

Stress response. In addition to cognitive processing of traumatic events, increased peritraumatic stress, as indicated by strong physiological reactivity (e.g., heart rate) and emotional responding during the trauma event, has been identified as a risk factor for the development of intrusive memories. Conceptually, the importance of the stress response can be understood in terms of classical conditioning, in which specific sensory cues in the environment become conditioned stimuli by virtue of pairing with a traumatic event (see Rescorla, 1988). Later encounters with these sensory cues may elicit a conditioned stress response similar to that experienced at the time of the trauma. Pitman (1989) suggests that strong stress responses lead to "super conditioning" and subsequent increases in intrusive memories. Similarly, Ehlers and Clark (2000) discuss the role of associative priming in the development of intrusions.

Prior research supports the role of a strong stress response in the development of intrusive memories and related PTSD symptoms. Specifically, increased heart rate assessed immediately after a traumatic event has been associated with increased risk for PTSD and associated intrusions up to six months later (e.g., Kuhn, Blanchard, Fuse,

Hickling, & Broderick, 2006; Shalev et al., 1998). Although some studies report nonsignificant or even negative associations between peritraumatic heart rate and subsequent PTSD (e.g., Blanchard, Hickling, Galvoski, & Veazey, 2002; Buckley et al., 2004; Ehring, Ehlers, Cleare, & Glucksman, 2008), a meta-analysis suggests that, overall, resting heart rate immediately after a trauma is positively associated with subsequent PTSD (weighted effect size of r = .20; Pole, 2007). Likewise, increased heart rate while viewing a sexual trauma film has been linked to greater reporting of intrusive memories (Weidmann, Conradi, Gröger, Fehm, & Fydrich, 2009). Together, these studies support the role of fear conditioning in the development of intrusions and associated PTSD symptoms.

In addition to heart rate, negative emotional responding to a traumatic event has been linked to intrusive memories and other PTSD symptoms. Although the predictive validity of specific negative emotional responses associated with the DSM-IV-TR's Criterion A2 (e.g., fear, helplessness, or horror; APA, 2000) has not been supported for PTSD (see for example Friedman, Resick, Bryant, & Brewin, 2011), this criterion does not capture all peritraumatic reactions. Bovin and Marx (2011) recommend that the peritraumatic experience be considered more broadly, including a focus on the emotions of fear, anger, sadness, and disgust, each of which have been associated with PTSD. These negative emotions may have implications for peritraumatic cognitive processing, and thus the development of intrusive memories.

Although strong emotions generally enhance memory, memory can be impaired when strong emotions are accompanied by heightened physiological arousal (Bennion, Ford, Murray, & Kensinger, 2013). As first explicated by Yerkes and Dodson (1908), moderate arousal can improve memory, whereas extreme arousal can impair performance. Easterbrook's (1959) cue utilization hypothesis suggests that when one is highly aroused or experiencing a strong emotion such as fear, one can only attend to the most central and important cues (at the cost of peripheral, irrelevant cues). Consistent with these hypotheses, strong negative emotions during stressful events have been shown to produce a narrowing of attention to the most central information (e.g., a perpetrator's knife; Christianson, 1992). Although this narrowing of attention to a weapon ("Weapon Focus"; e.g., Loftus, Loftus, & Messo, 1987; Loftus & Macworth, 1978) can be partially attributed to the unusual nature of the object, physiological arousal also contributes to this effect (see Fawcett, Russell, Peace, & Christie, 2013).

Such narrowing of attention during a trauma can impair processing of peripheral information, and in turn contribute to disorganization of the trauma memory (Foa & Riggs, 1993), thereby increasing intrusive memories (e.g., Halligan et al., 2002). Further, empirical research also supports the role of strong peritraumatic emotions in the development of intrusive memories. For example, the content of intrusive memories often reflects stimuli temporally associated with the most distressing aspect of the event (i.e., "hotspots"; Holmes, Grey, & Young, 2005), possibly because these stimuli are interpreted as warning signals of upcoming distress (the Warning Signal Hypothesis; Ehlers et al., 2002). Empirical support for the role of peritraumatic affect as a contributor to intrusions also comes from meta-analytic findings linking emotional arousal during trauma exposure to increased PTSD symptoms (Ozer, Best, Lipsey, & Weiss, 2003).

Overall, although physiological arousal and valence of emotion have been conceptualized as orthogonal components of affect (LaBar & Cabeza, 2006), both have implications for the development of intrusive memories. Extreme peritraumatic arousal (which can be observed through increased heart rate) can lead to strong associative priming (Pitman, 1989), increasing the likelihood that subsequent reminders will lead to a conditioned fear response and associated intrusions (Ehlers & Clark, 2000). When accompanied by heightened arousal, strong emotions can also lead to a narrowing of attention and impaired cognitive processing (Bennion et al., 2013). Accordingly, both increased heart rate and strong negative emotions at the time of the trauma have been associated with intrusive memories and related PTSD symptoms (e.g., Ozer et al., 2003; Pole, 2007).

Summary of risk factors for intrusions. In sum, the occurrence of intrusive memories can be impacted by cognitive, physiological, and emotional factors. Specifically, the frequency of intrusive memories has been linked to more disorganized trauma memories (Halligan et al., 2002), more data-driven processing (Ehring et al., 2008; Halligan et al., 2002, 2003), and heightened physiological (e.g., heart rate; Weidmann et al., 2009) and emotional arousal (Holmes et al., 2005; Ozer et al., 2003). When one experiences physiological arousal in the presence of strong, negative emotions, one's focus becomes limited to only the most central, important information (Easterbrook, 1959). This focus often involves processing basic sensory information (i.e., data-driven processing), which may limit capacity for higher-level processing (i.e., conceptual processing). Data-driven processing (in the absence of conceptual processing) during the trauma can contribute to poorly elaborated trauma memories characterized by a high degree of sensory information without appropriate context (i.e., disorganization in trauma memory). In turn, when sensory cues are experienced in the environment, intrusive memories are easily triggered and, without appropriate contextualization, can evoke a sense of current threat.

Peritraumatic Alcohol Intoxication

The theory and findings reviewed above illuminate key processes involved in the development of intrusions. Alcohol intoxication at the time of the event may also impact this process. As early as World War I, some suggested that the sedative properties of alcohol could protect against traumatic stress by producing "a decrease of critical selfconsciousness and anxiety" (Mott, 1919, p. 224). More recently, Ehlers and Clark (2000) remarked that alcohol use could be one of many factors that influence cognitive processing of a traumatic event (but did not elaborate on the possible nature of this influence). Similarly, Kaysen, Bedard-Gilligan, and Stappenbeck (2017) suggested acute alcohol intoxication might impact one's subjective experience of a potentially traumatic event and, in turn, affect post-trauma symptoms and recovery (though they too acknowledged that mechanisms remain unclear). Despite these conjectures, researchers have only recently considered the potential for peritraumatic alcohol use to alter later intrusive memories in empirical studies. As detailed below, this work reveals that alcohol intoxication during exposure to a traumatic event may indeed impact key cognitive, physiological, and emotional risk factors for intrusive memories.

Alcohol myopia. The Alcohol Myopia Model (AMM; Giancola, Josephs, Parrott, & Duke, 2010; Steele & Josephs, 1990) provides a useful theoretical lens through which to consider the impact of peritraumatic intoxication on symptom development. This well-supported model holds that the pharmacological effects of alcohol intoxication produce a narrowing of attention, which restricts the range of internal and external cues perceived

and processed by the intoxicated individual. Myopia is said to produce a shortsightedness characterized by an increased focus on immediate, superficial information to the exclusion of a longer-term perspective (Steele & Josephs, 1990). One lab study involving alcohol administration found that myopia was positively associated with impaired memory for peripheral aspects of events while leaving memories for central information unaffected (as reported while still intoxicated; Schreiber Compo et al., 2011). Alcohol myopia has also been demonstrated using the Simons and Chabris (1999) "gorillas in our midst" clip. Specifically, compared to those in a placebo condition, participants who drank alcohol were more likely to miss the gorilla due to a narrowed focus on counting the number of passes (Clifasefi, Takarangi, & Bergman, 2006). In further support of alcohol myopia, Harvey, Kneller, and Campbell (2013) showed that alcohol narrowed the focus of foveal attention to central information using an eyetracker.

In the present study, alcohol myopia is expected to impact previously identified risk factors on intrusive memories. Specifically, the shortsightedness brought on by alcohol myopia is expected to interfere with conceptual processing of traumatic events (Ehlers & Clark, 2000), limiting individuals to data-driven processing of salient perceptual cues. Alcohol myopia is also expected to exacerbate the narrowing of attention associated with affect-laden stress responses (Christenson, 1992). In this manner, alcohol-related myopia is predicted to increase intrusive memories by reducing conceptual processing, thereby increasing reliance on data-driven processing and creating associated disruptions in trauma memories.

Alcohol and stress-response dampening. At high doses, alcohol is also known to have anxiolytic effects, decreasing one's physiological response to stress—a

phenomenon known as stress-response dampening (Levenson, Sher, Grossman, Newman, & Newlin, 1980; Sayette, 1999). Stress-response dampening appears to be dosedependent (in the absence of distracting information), operating only at levels of intoxication high enough to achieve physiological anxiolytic effects (Donohue, Curtin, Patrick, & Lang, 2007). In support of this process are findings that high doses of alcohol are associated with reduced heart rate (Sayette & Wilson, 1991; Sayette, Smith, Breiner, & Wilson, 1992), as well as reduced facial affect (Sayette et al., 1992). As such, stressresponse dampening at high levels of peritraumatic alcohol intoxication is expected to override previously discussed myopic effects on cognitive processing displayed at low doses. Instead, at high doses of alcohol intoxication, stress-response dampening is expected to decrease data-driven processing, reduce disruption in the trauma memory, and ultimately lead to a reduction in intrusive memories when compared to a placebo.

Summary of peritraumatic alcohol intoxication. In sum, acute alcohol intoxication has implications for cognitive, physiological, and emotional risk factors for intrusive memories. One important manifestation of intoxication is alcohol myopia, which narrows one's of attention to the central, most salient cues at the cost of more peripheral cues (Steele & Josephs, 1990). Reminiscent of attentional narrowing displayed during times of high physiological and emotional arousal, alcohol myopia is similarly expected to exacerbate cognitive processes that lead to intrusive memories. However, at high doses, alcohol is also known to dampen responses to stress, potentially interfering with the physiological arousal required to activate the cognitive risk factors for intrusive memories.

Peritraumatic Alcohol Intoxication and Intrusions

Correlational studies. Despite this evidence that alcohol intoxication impacts mechanisms responsible for intrusive memories, little work has focused on the role of peritraumatic alcohol use in the development of intrusive symptoms. This lack of knowledge is concerning in light of findings that about 50% of women who are sexually assaulted were drinking at the time (Abbey et al., 2004). A few studies have examined current PTSD symptoms in relation to peritraumatic alcohol use, as reported retrospectively by victims. For instance, some studies examining self-reported drinking prior to a sexual or physical assault (Kaysen et al., 2010; Peter-Hagene & Ullman, 2016) or other traumatic event (Maes, Delmeire, Mylle, & Altamura, 2001) indicate that peritraumatic alcohol use initially buffers against intrusions and other PTSD symptoms. However, findings from Kaysen and colleagues' (2010) study showed that alcohol use prior to an assault was associated with more persistent intrusive symptoms up to six months post-assault.

Some recent studies have asked female victims of sexual assault to report retrospectively how intoxicated they were prior to the assault. Those studies involving college students with a history of sexual assault revealed that greater peritraumatic intoxication (as assessed subjectively and retrospectively) was associated with more current distress (Blayney & Read, 2015) and PTSD symptoms (Blayney, Read, & Colder, 2016). Similar findings were revealed in a community sample of women who experienced sexual assault (Jaffe et al., 2017). Specifically, when controlling for severity of the assault, greater levels of peritraumatic intoxication (but not alcohol use alone) were associated with more severe symptoms of PTSD, particularly intrusive symptoms (Jaffe et al., 2017).

In contrast to the above studies that have focused on the presence or absence of victim substance use, as well as level of intoxication during the assault, other research has categorized sexual victimization experiences based on perpetrator tactics (e.g., giving a potential victim alcohol, using force). For example, in a national study (Kilpatrick et al., 2007), sexual assault experiences were categorized as drug- or alcohol-facilitated rape (in which the perpetrator gave drugs or alcohol to a woman to facilitate a sexual assault), incapacitated rape (in which the woman became voluntarily intoxicated but was so impaired she could not consent), and forcible rape. Findings from this sample suggest that compared to those who did not experience a given type of assault, victims of drug- or alcohol-facilitated rape (OR = 1.87) and forcible rape (OR = 3.46) were at increased risk for PTSD, but victims of incapacitated rape were not (Zinzow et al., 2010). Further, rapes involving both victim intoxication/incapacitation and force were more likely to result in PTSD than either intoxication/incapacitation or force alone (Zinzow et al., 2012). Other work has identified three classes of sexual assault using cluster analyses: alcohol-related, high-violence, and moderate-severity sexual assaults (Peter-Hagene & Ullman, 2014). These authors reported that alcohol-related assaults led to more severe symptoms of PTSD than modest-severity assaults, but were associated with similar or lower levels of PTSD than high-violence assaults. Consistent with these findings, Brown, Testa, and Messman-Moore (2009) found that incapacitated rape led to an intermediate degree of PTSD compared to sexual assaults that involved verbal coercion alone, and forcible rapes. On the other hand, no differences in PTSD symptoms were found between victims who were non-impaired, impaired, or incapacitated at the time of a sexual assault (Littleton, Grills-Taquechel, & Axsom, 2009).

A related literature involving victims of motor vehicle accidents and other injuries has examined associations between drug and alcohol screenings at hospital admission and subsequent PTSD. Like findings with sexual assault victims, these results have been mixed, with some studies linking intoxication to increased risk for PTSD (McFarlane et al., 2009; Richmond & Kauder, 2000), while others report lower risk for PTSD (Mellman, Ramos, David, Williams, & Augenstein, 1998), or no association between alcohol intoxication and later PTSD (Zatzick et al., 2002).

Summary of correlational studies. In sum, most prior research on the impact of peritraumatic intoxication on PTSD symptoms has been correlational in nature. These studies involve participants who have personally experienced traumatic events, and are therefore strong in external validity. However, these studies also contain a number of threats to internal validity that could contribute to the mixed findings. For instance, the use of alcohol prior to an actual trauma may be confounded with other variables that affect the development of PTSD. As an example, alcohol-related sexual assaults differ from other sexual assaults in a variety of ways, including degree of perceived threat and type of perpetrator (Peter-Hagene & Ullman, 2014). Correlational studies with trauma victims therefore cannot isolate the influence of alcohol. Mixed correlational findings may also be a consequence of inconsistent methodologies. Measures of intoxication vary across studies (e.g., positive vs. negative alcohol screen, blood alcohol content above or below .08, retrospective self-report of alcohol use). Within studies using objective measures of intoxication at hospital admission, there is inevitable variability between subjects in the amount of time since the trauma. Studies also vary in the length of time between trauma exposure and PTSD symptom assessment, as well as in the type and

degree of trauma exposure being studied (e.g., assault vs. motor vehicle accident). Moreover, because this work is correlational, there is no way to test causal relations between intoxication and intrusive symptoms.

Experimental studies. Compared to such correlational studies, experimental studies offer a number of methodological advantages. Specifically, manipulating acute alcohol intoxication in the laboratory setting prior to an analog trauma exposure allows for the examination of causal effects of intoxication. Further, the increased internal validity afforded by experimental control permits a more precise examination of the pharmacological effects of alcohol on intrusive memories. Though ethical and safety concerns necessitate limits to the amount of alcohol administered and the degree of stress induced in a lab setting, potentially limiting external validity, highly controlled experimental studies provide an important complement to correlational work in this area.

To date, two such experimental studies have examined the dose-dependent effects of peritraumatic alcohol intoxication on intrusive memories (Bisby, Brewin, Leitz, & Curran, 2009; Bisby, King, Brewin, Burgess, & Curran, 2010). In these studies, participants were administered either a placebo, a low dose of alcohol, or a high dose of alcohol, then were exposed to a 12.5-minute film of road traffic accidents, which served as an analog trauma. Participants were asked to record subsequent intrusive memories of the film in an online daily diary for seven days following the film. These studies also involved other self-report questionnaires, physiological assessments, and memory tasks. Results from the first of these studies (Bisby et al., 2009) supported the proposed direct effects of alcohol on intrusive memories, in that participants in a low-dose alcohol condition had greater intrusions than those in the placebo condition, while those in a high-dose alcohol condition had fewer intrusions than those consuming a placebo beverage. Although these authors attributed the increased intrusions at low doses to reduced explicit memory, they were unable to account for the reduced intrusions at high doses. In the second study by this team, Bisby and colleagues (2010) again found an inverted U-shaped association between alcohol dose and intrusions, such that low doses of alcohol produced a significant increase in intrusions over the placebo dose. Findings supported viewpoint-dependent memory as one possible mechanism underlying the alcohol-intrusions relationship. These studies provide important initial evidence that alcohol intoxication may have direct effects on the development of intrusive memories following exposure to an analog trauma. Notably, however, this work does not examine the effects of alcohol on other established contributors to intrusive memories as potential underlying mechanisms accounting for alcohol's dose-dependent effects on intrusive memories. It also remains unclear whether these findings would extend to other types of (analog) traumas, including sexual assault.

Summary and Current Study

To summarize, intrusive memories are an important component of PTSD that are experienced as distressing and often follow a sexual assault. Prior research implicates various risk factors in the development of intrusive memories following sexual assault. Data-driven processing in the absence of conceptual processing has been associated with disorganization of the trauma memory, which in turn has been linked to increased intrusive memories. Heightened physiological arousal and strong emotional responses, which can lead to fear conditioning and attentional narrowing, have also been implicated in the development of intrusive memories and related PTSD symptoms. A separate literature suggests that acute alcohol intoxication can impact these cognitive, physiological, and emotional processes relevant to intrusive memories. Specifically, alcohol can exacerbate the attentional narrowing associated with strong affective responses, and may limit one to data-driven processing. Although alcohol myopia is expected to increase with level of intoxication, at high doses of intoxication, alcohol can dampen one's physiological stress response. This effect may override myopic processes at high levels of intoxication, reducing fear conditioning and subsequent intrusions.

The present study integrates theoretical and empirical literatures on traumarelated intrusive memories and acute alcohol intoxication. Building on this work, the current study involved examining a model accounting for the direct and indirect effects of known risk factors and alcohol use on trauma-related intrusions (see Figure 1.1). Specifically, this model was examined using an experimental lab design involving alcohol administration, a well-accepted analog trauma exposure paradigm (a trauma film), and behavioral measures of key constructs (e.g., alcohol myopia, stress responses). Findings from this study should clarify the role of alcohol intoxication in the development of intrusive memories and are expected to highlight the importance of prevention and intervention programs aimed at reducing alcohol-involved victimization.

Drawing on Bisby et al.'s (2009, 2010) recent studies—the only prior work examining intrusive symptoms following lab-based alcohol administration—a basic premise of the model is that low levels of alcohol intoxication will produce greater intrusive memories than the placebo drink, but that high levels of intoxication will produce fewer intrusive memories than the placebo drink (path 1A). Although intrusive memories are expected to decrease over time for all levels of alcohol intoxication, the

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rate at which intrusions decrease is expected to vary by dose. Specifically, low levels of intoxication should be associated with more persistent memories, but high levels of intoxication will be associated with intrusions that decrease more rapidly (path 1B). Alcohol's influence on cognitive factors will inform these overall effects. Consistent with the Alcohol Myopia Model (Steele & Josephs, 1990), intoxication is expected to produce a narrowing of attention to the most salient, or central, features of the trauma film, resulting in impaired memory for peripheral information (Schreiber Compo et al., 2011; path 2A). This attentional narrowing is predicted to interfere with conceptual processing of the film in a meaningful, organized manner, thereby limiting intoxicated participants' processing of the film to a sensory level (i.e., data-driven processing, path 2B) and disrupting the formation of trauma memories (path 2C). Similarly, alcohol dose is expected to relate to stress response as indicated by heart rate and facial affect, with high doses dampening stress responding (Donohue et al., 2007; path 3A). An increased stress response, which is associated with attentional narrowing in the absence of alcohol (Christianson, 1992), is expected to be associated with increased data-driven processing (path 3B) and disruption of the trauma memory (path 3C). Finally, increased data-driven processing of sensory information, which is expected to lead to increased temporal disorganization in trauma-related memories (Halligan et al., 2002; path 4A), should be associated with increased intrusive memories (e.g., Ehring, Ehlers, & Glucksman, 2008; Halligan et al., 2003; paths 4B, 4C). These aims and hypotheses can be summarized as follows. (Path identifiers in Figure 1.1 correspond to the listed hypotheses below.)

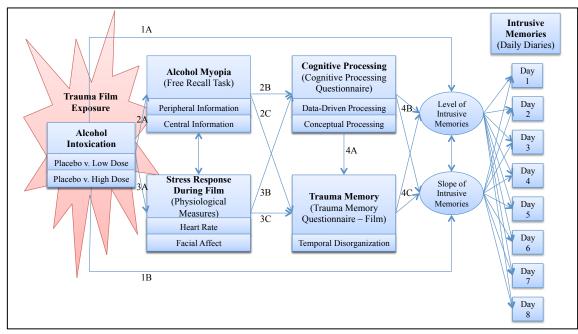


Figure 1.1. Conceptual model.

Aim 1: Examine the direct effects of peritraumatic alcohol intoxication on intrusive memories.

Hypothesis A: Compared to a placebo condition, low levels of alcohol intoxication will lead to higher levels of intrusive memories following the trauma film, while high levels of intoxication will lead to lower levels of intrusive memories following the trauma film.

Hypothesis B: Although intrusions will decrease over time for all participants, reductions will be slower for those with low levels of alcohol intoxication and faster for those with high levels of intoxication.

Aim 2: Investigate the impact of alcohol myopia on cognitive factors.

Hypothesis A: Greater alcohol intoxication will be associated with reduced memory for peripheral details of the trauma film (reflecting alcohol myopia); memory for central details of the film will not vary by intoxication level.

Hypothesis B: Greater alcohol myopia will be associated with increased processing of sensory information (i.e., data-driven processing) in the trauma film and reduced ability to make meaning of the trauma film (i.e., conceptual processing).

Hypothesis C: Greater alcohol myopia will be associated with more temporal disorganization in memory for the trauma film.

Aim 3: Investigate the impact of stress responding on cognitive factors.

Hypothesis A: High levels of alcohol intoxication will result in stress-response dampening as indicated by reduced heart rate and negative facial affect.

Hypothesis B: Increased stress responding (i.e., heart rate and facial affect) will be associated with increased processing of sensory information (i.e., data-driven processing) in the trauma film and reduced ability to make meaning of the trauma film (i.e., conceptual processing).

Hypothesis C: Increased stress responding will be associated with more temporal disorganization in the memory for the trauma film.

Aim 4: Examine the effects of cognitive factors on intrusive memories.

Hypothesis A: Data-driven processing of the trauma film will be positively associated with disorganization in memory for the trauma film.

Hypothesis B: Increased data-driven processing and reduced conceptual processing will be associated with more frequent and persistent intrusive memories of the film.

Hypothesis C: Increased temporal disorganization in memories of the trauma film will be associated with more frequent and persistent intrusive memories.

CHAPTER 2: METHOD

Overall Design

This investigation involved a between-subjects experimental design to examine the influence of cognitive, physiological, and emotional factors and acute alcohol intoxication on intrusive symptoms during a weeklong period following an analog trauma exposure in a community sample of women. Participants were randomized into an alcohol condition (placebo, low dose, high dose) and then viewed a film clip depicting a sexual assault. Facial affect and heart rate during the film were recorded. After film viewing, participants completed measures to assess their cognitive processing and memory of the event. During the next seven days, participants completed daily diaries assessing the frequency, content, and distress associated with intrusive memories of the film. At the week's end, participants returned to the lab to complete one final intrusion diary, an additional self-report measure of memory for the trauma film, a follow-up assessment of distress, and a thorough debriefing.

Participants

Participants were 98 women recruited from Lincoln, Nebraska. To participate, individuals had to be between 21 (legal drinking age) and 30 years of age, an age range at high risk for sexual assault victimization (Ogle, Rubin, Berntsen, & Siegler, 2013). Because of risks associated with alcohol consumption, a number of additional exclusion criteria were employed based on previously developed procedures (Eckhardt, 2007; Giancola, 2002, 2004; Giancola et al., 2009; Watkins, DiLillo, & Maldonado, 2015; see Appendix A for the full telephone screening interview.) Specifically, individuals were required to endorse social drinking (defined as drinking three or more standard drinks per occasion, at least twice a month; e.g., Godlaski & Giancola, 2009) to participate. Individuals were excluded if they reported: (a) current or past alcohol treatment, or hospitalization due to alcohol use; (b) serious psychological symptoms; (c) a condition or medication in which alcohol consumption is medically contraindicated; (d) any past serious head injuries (as indicated by the HELPS Brain Injury Screening Tool; Picard, Scarisbrick & Paluck, 1991); (e) current pregnancy (also assessed via a urine pregnancy test administered upon arrival); (f) nursing an infant; or (f) the presence of a positive breath alcohol concentration (BrAC) upon arrival. Given that the amount of alcohol administered was based upon participant weight, individuals under 6 feet and weighing over 250 pounds, and individuals over 6 feet and over 300 pounds, were excluded for safety reasons.

To reduce the likelihood of an adverse reaction to the potentially distressing film, additional inclusion/exclusion criteria were employed. Drawing on procedures used by Weidmann and colleagues (2009), participants were excluded if they reported a personal history of sexual victimization, which is the traumatic event depicted in the film used in the present study. In particular, participants were excluded if they endorsed any sexual abuse prior to the age of 14, assessed via three screening questions of the Computer Assisted Maltreatment Inventory sexual abuse subscale (CAMI; DiLillo et al., 2010). Participants were also excluded if they endorsed a history of forcible sexual assault since the age of 14, assessed via four questions from the Sexual Experiences Survey (SES-F; Koss & Gidycz, 1985). Further, because daily diary data were collected through an app, ownership of an Android or Apple smartphone or tablet was required for participation. Of 364 individuals who responded to recruitment materials (see procedures below), 316 completed a telephone screening interview to determine eligibility. Figure 2.1 depicts the flow of participant recruitment and reasons for exclusion. Of 124 participants who were eligible to participate, 98 participants completed the first laboratory session. The 26 individuals lost to follow-up did not differ from the 98 participants by age, t(122) = -0.87, p = .386, or ethnic minority status, $\chi^2(1) = 0.89$, p = .346, as indicated on the telephone screen.

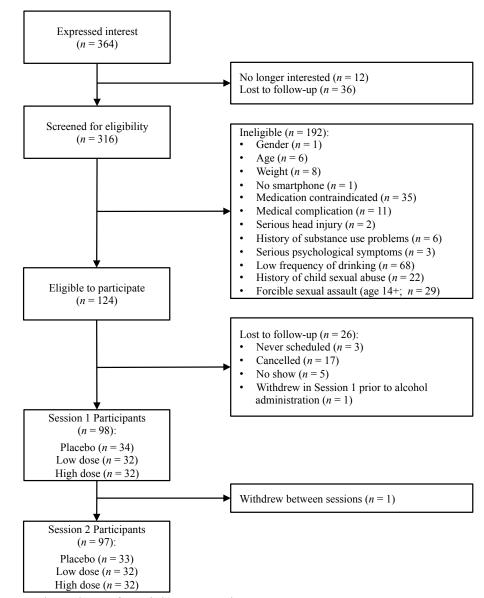


Figure 2.1. Flow chart of participant recruitment.

The final sample of 98 participants had an average age of 23.03 years (SD = 2.19, range = 21 - 30). All participants identified as female (recruitment materials advertised this as a study for "women"), though other options for gender identity were given (including male, transgender – female to male, transgender – male to female, and other). With regard to sexual orientation, 87.8% identified as heterosexual, 10.2% as bisexual, and 2.0% as lesbian/gay. Regarding ethnicity, 14.3% reported they were Latino, Hispanic, or of Spanish origin. In addition, 84.7% identified as White, 13.3% as Asian, 4.1% as African American, 0.0% as American Indian, and 5.1% as "Other" (participants could endorse more than one category). Although women were recruited from the community at large, 76.5% reported they were currently a student. Given this large percentage, we began asking more detailed questions about student status mid-way through data collection. Of 50 participants who answered these detailed questions, 20.0% were not students, 46.0% were undergraduates at University of Nebraska-Lincoln (UNL), 4.0% were undergraduate students at other local institutions, and 30.0% were graduate students at UNL. Participants reported between 12 and 23 years of formal education including kindergarten (M = 17.10, SD = 2.04). With regard to marital status, 90.8% were single, 8.2% were married, and 1.0% was divorced. Regarding relationship status, 66.7% were either in a romantic relationship (n = 52) or married (n = 8). Participants reported a wide range of annual income (or family income if a financially dependent student), with 9.2% reporting income of 0 - 5,000, 9.2% reporting 5,000 - 10,000, 24.5%reporting 10,000 - 20,000, 15.3% reporting 20,000 - 30,000, 7.1% reporting \$30,000 - \$40,000, 7.1% reporting \$40,000 - \$50,000, 3.1% reporting \$50,000 -

\$60,000, 2.0% reporting \$60,000 – \$70,000, and 22.4% reporting an annual income over \$60,000.

Measures of Primary Study Variables

Lab Tasks.

Alcohol administration. Alcohol administration procedures mirrored those established by Bisby and colleagues (2009, 2010) and currently in use in the lab of Dr. David DiLillo, primary sponsor of this project. Participants were randomly assigned to a placebo or, similar to prior studies (Bisby et al., 2009, 2010), a low or high dose alcohol condition. A single-blind, no-information design was utilized to understand the pharmacological effects of alcohol (Martin & Sayette, 1993). Specifically, participants were told they might be assigned to a high dose alcohol condition, a low dose alcohol condition, or a no-alcohol condition, but were not informed about which beverage they received until after all study procedures were complete.

Alcohol conditions. Women in the alcohol conditions were administered a dose of .36 g/kg (for the low dose condition) or .72 g/kg (for the high dose condition) of 95% alcohol (Everclear) mixed at a 1:5 ratio with a mixer (orange juice). For example, a 150-lb woman in the low dose condition would have been given 32.58 ml Everclear and 162.90 ml orange juice. A 150-lb woman in the high dose condition would have been given 65.15 ml Everclear and 325.75 ml orange juice. Participants were allotted 20 minutes for beverage consumption, or up to 30 minutes if more time was needed. BrAC was assessed using the Intoximeter Alco-Sensor FST Breathalyzer (calibrated monthly; tolerance of \pm .003% immediately following calibration). Participant BrAC was tested following an absorption period of 15 minutes (and if needed, again at 22 and 30 minutes

post-drink consumption). After reaching a BrAC of at least .030% in the low dose condition or .070% in the high dose condition (or after 30 minutes absorption if the target BrAC was not reached), participants completed the Alcohol Consumption Questions, which included questions about beverage taste, perceived intoxication and impairment, and presumed beverage condition (see Appendix A). Then, participants proceeded with a baseline heart rate assessment and trauma film viewing (detailed below).

Placebo condition. Four milliliters of alcohol were added to each placebo beverage and alcohol was sprayed on the rim of the placebo beverage glass. Because alcohol placebo manipulations are effective for only approximately 30 minutes after beverage consumption (Bradlyn & Young, 1983), immediately after drink consumption, participants in the placebo group had their BrAC tested, then proceeded with the Alcohol Consumption Questions, baseline heart rate assessment, and trauma film viewing.

Trauma film paradigm. Participants viewed a 10.5-minute clip showing a sexual assault from the French-language feature film *Irreversible* by Gaspar Noé, originally released in France in 2003. The film was shown to participants on a 17-inch display laptop via *OpenSesame*, an open-source software program used to design psychology experiments. English subtitles were not displayed to avoid detracting attention from the visual images. The clip consists of a single unbroken shot, mimicking memory formation of personal events. A recent study (Weidmann et al., 2009) comparing a number of film clips with distressing content recommends the *Irreversible* segment for use in trauma film studies, due to its consistent ability to induce increased heart rate, negative affect, and intrusive memories, all of which are variables of interest in the present study. Although Weidmann et al. (2009) used a 13.5-minute clip, which showed both a sexual and

physical assault (physical assault not depicted in the current study), Arnaudova and Hagenaars (2017) validated the use of a trauma film with sexual content alone. Specifically, they found that a two-minute clip of the sexual assault in *Irreversible* was associated with a faster heart rate than a positive film with sexual content.

Participants were informed of the distressing nature of this film before viewing it, as well as their right to stop the film (and participation) at any time. Immediately prior to the film, participants received written and verbal reminders that they could stop the film by pressing the spacebar. (One participant briefly paused the film, but decided to finish the film and continue participation.) After film viewing, participants were asked how distressing they found the film on a scale from 0 (*not at all*) to 10 (*very much*). Participants responded with a mean distress rating of 8.42 (*SD* = 1.50, range = 5 to 10). Based on this item, there were no differences between alcohol conditions in how distressing participants found the film, F(2, 95) = .20, p = .817. Participants were also asked whether they had seen the film before (2.0% said yes) and to estimate the percentage of the French dialogue they understood (M = 9.59%, SD = 17.20%, range = 0% to 100%). (See Appendix A for Post-Film Questions.)

Facial affect. Participants' facial expressions while watching the trauma film were video recorded and coded in real time using *FaceReader* v.5.1.1, a software program by Noldus. To improve image quality and facial recognition, a neutral (light blue) curtain was hung behind the participant and LED lights were placed in front of the participant. Videos of participants were recorded with Logitech c920 HD Pro Webcam (placed above the laptop) at 15 frames per second. Video recordings of participants corresponded to the exact start and stop times of the trauma film, which was administered

via *OpenSesame* software and connected to *FaceReader* using the Application Programming Interface (Zosky, 2015). Continuous calibration procedures in *FaceReader* were utilized to adapt to any individual biases in facial responses.

The *FaceReader* program uses advanced modeling techniques (see Noldus, 2012) to code action units in accordance with the Facial Action Coding System (FACS; Ekman & Friesen, 1978; Ekman, Friesen, & Hager, 2002) and compute continuous scores for each of the six basic emotions (happy, sad, angry, surprised, scared, disgusted) described by Ekman (1970) and "neutral" affect. Prior research has demonstrated high convergence with trained human raters (den Uyl & van Kuilenberg, 2005; Lewinski, den Uyl, & Butler, 2014), high test-retest reliability (r's > .80), and construct validity (correlations to clinical symptoms of schizotypy; Cohen, Morrison, & Callaway, 2013). *FaceReader* measurements of happiness also correlate with facial electromyography data (D'Arcey, Johnson, Ennis, Sanders, & Shapiro, 2013). The *FaceReader* software has also been successfully used in past psychological studies (e.g., Chentsova-Dutton & Tsai, 2010; Chóliz & Fernández-Abascal, 2012).

For each frame recorded of the participant, *FaceReader* generates a value to represent the strength of each emotion from 0 (*not present*) to 1 (*fully present*). In addition, *FaceReader* computes an overall value for valence ranging from -1 (*negative*) to +1 (*positive*). Specifically, *FaceReader* considers "happy" to be a positive emotion, "sad," "angry," "scared," and "disgusted" to be negative emotions, and "surprise" to be either positive or negative. A valence score, reflecting the overall degree of negativity or positivity expressed, is computed by subtracting the value of the most intense negative emotion from the intensity of the "happy" score. Given the wide range of negative

emotions that could be experienced (and potentially impacted by acute alcohol intoxication) during the film, the valence score was utilized in the current study. More specifically, a mean change score was computed to represent the change in valence of facial affect from the first 105 seconds of the trauma film in which there is no violence (a woman is simply walking) to the remainder of the trauma film, which contains violent and potentially distressing content.

Some data recorded from *FaceReader* were missing at random. Technical issues at study onset led to brief crashes in *FaceReader* for three participants. Occasionally, the participant's face could not be found or modeled by *FaceReader* in certain frames. Although participants were asked to wear contacts instead of glasses if they were equally comfortable, 24.1% participants wore glasses, including 5.1% with particularly thick frames. Though participants were asked not to cover their face while watching the film, other factors could have interfered with modeling participants' faces (e.g., bangs, facial piercings, turning away from the screen). Only one participant had insufficient data (i.e., no data coded during the violent portion of the film) to compute a change score. For participants with change scores, *FaceReader* generated data for an average of 99.7% (*SD* = 0.7%, range = 96.2% to 100%) of frames in the baseline period of 105 seconds, and 98.0% (*SD* = 5.3%, range = 68.1% to 100%) of frames in the remainder of the film.

Heart rate. Prior to viewing the trauma film, participants were asked to place the Polar H2 Heart Rate Sensor on their chest using an adjustable strap. Sensors on the strap were dampened to improve signal conduction. The monitor communicated heart rate data to the *Polar ProTrainer* v.5 software, installed on a nearby computer (not accessible to participants), via Bluetooth technology. Three heart rate assessments were conducted:

prior to the film to establish a 5-minute baseline, during the 10.5-minute film, and for a 5minute period after the film. Consistent with prior trauma film studies (e.g., Chou, La Marca, Steptoe, & Brewin, 2014; Holmes, Brewin, & Hennessy, 2004), the increase in the heart rate (i.e., beats per minute) during the film (relative to the pre-film baseline) was used as a measure of stress response.

During each heart rate assessment, study personnel made notes of spikes, dips, and signal losses visible on the heart rate monitor, as well as any corresponding participant movements. Although some efforts were made to improve the signal during the session (e.g., asking the participant to reposition the chest strap), priority was given to viewing the film as soon after the alcohol absorption period as possible. Data cleaning procedures involved deleting the following artifacts from each heart rate assessment file: (a) spikes or dips coinciding with visible participant movements, (b) flat-lines over 10 seconds, (c) dips below 40 beats per minute, (d) artifacts identified by the Polar Error Corrections function, and (e) spikes or dips at the beginning of a data file suggestive of calibration. Assessments in which the signal never stabilized or with less than 45 seconds of clean data were not utilized and therefore considered missing data. Sufficient data were collected during both the pre-film baseline and film assessment to create change scores for 91.8% of participants.

Free recall task. A novel approach to assess alcohol myopia developed by Schreiber Compo et al. (2011) was adapted for use in the present study. The film viewing (and heart rate assessment) took place in a separate, staged room. After viewing the film, participants returned to the original room and were asked to recall, in writing, all aspects of their experience viewing the film, no matter how trivial. A written prompt (see Appendix A) was provided to participants, as well as read aloud by study personnel via intercom to confirm all participants comprehended the instructions while intoxicated. Participants were given approximately 10 minutes to complete the recall and were not informed ahead of time about the task. Trained research assistants subsequently coded each response for the number of accurate details reported for (a) each item in the room, and (b) each person and object in the film (for coding instructions and record form, see Appendix A). A second rater coded 50% of responses and achieved excellent inter-rater agreement (ICC = .98). Measurement models described below were utilized to determine whether each piece of information recalled best represented central or peripheral information. Alcohol myopia was operationalized as impaired recall of peripheral details, but no impairment in recall of central details.

Self-Report Measures.

Peritraumatic cognitive processing. The Cognitive Processing Questionnaire (Halligan et al., 2002; see Appendix A) was administered immediately after viewing the trauma film to assess the degree to which participants engaged in data-driven and conceptual processing of the film. The questionnaire consisted of 17 items, including eleven items designed to assess data-driven processing (i.e., a focus on surface, sensory-level details) and six items designed to assess conceptual processing (i.e., making meaning of the film). Participants responded to each item on a scale from 1 (*not at all*) to 5 (*very strongly*). Prior studies show satisfactory internal reliability (coefficient alpha of .69 for an 8-item data-driven processing scale and .76 for a 7-item conceptual processing scale; Halligan et al., 2002). In the current study, coefficients alpha were .78 for the 11-item data-driven processing scale and .56 for the 6-item conceptual processing scale.

Although mean scores were examined in preliminary analyses, measurement models were evaluated and modified to obtain more internally consistent representations of these constructs.

Intrusive memory diary. Ecological momentary assessment was used to assess intrusive memories. Intrusive memory diaries have proven to be more sensitive than other retrospective measures administered at follow-up (e.g., Regambal & Alden, 2012) and are commonly used in trauma film studies (Holmes & Bourne, 2008; James et al., 2016). Recently, intrusive memories diaries have been successfully administered via daily online surveys in trauma film studies (Bisby et al., 2009, 2010) and via handheld devices in participants with PTSD (Kleim, Graham, Bryant, & Ehlers, 2013; Pedersen, Kaysen, Lindgren, Blayney, & Simpson, 2014; for a review, see Chun, 2016).

In accordance with procedures used by Holmes et al. (2004) and Bisby et al. (2009, 2010), participants were asked to complete diaries at least once daily to report on spontaneously occurring thoughts, images, or memories related to the trauma film. In each assessment, intrusive memories were differentiated from dreams (through the use of a separate dream-related question). Intrusive memories were then described to participants as "a memory of the film that pops into your mind spontaneously, out of the blue, without deliberately thinking about it. This could be a fleeting memory or more vivid." For each intrusion, participants were asked to provide a description of the content, specify the nature of the intrusion (an image, thought, or both), and rate their associated distress (Holmes et al., 2004; see Appendix A). Present analyses focus on the number of intrusive memories reported each day.

Mobile data collection software called *MetricWire* was used to administer intrusive memory diaries via an app on each participant's smartphone or tablet. Using this software, participants were asked to complete a "Daily Diary" survey at the end of each day, whether or not they had experienced any intrusive memories. In addition to the intrusive memory survey questions, beginning the day after film viewing, additional questions (not central to current study aims) assessed daily alcohol use and sexual activity. Participants were prompted to complete this survey at 8pm each day, with a reminder at 10pm. Surveys were available for up to five hours. The first Daily Diary survey was administered on the same day the film was viewed; participants who were still present in the lab completing study procedures (and therefore had their phones turned off) during the initial prompt were reminded before leaving to complete this survey. In addition to the automized daily prompts, participants were also encouraged to initiate a "Single Intrusive Memory" diary at any time throughout the day (e.g., immediately after an intrusive memory) to reduce rehearsal efforts and minimize the time required for the Daily Diary. To reduce participant burden for those who reported a large number of new intrusive memories on the Daily Diary (even after excluding previous reports on the Single Intrusive Memory survey), event-level details were assessed for a maximum of three intrusive memories on each Daily Diary report. Compensation for diary completion (as well as the variable assessing total number of diaries completed) was based solely on the Daily Diary survey. (For written instructions provided to participants about the difference between the Daily Diary and Single Intrusive Memory survey, see Appendix A.)

On the eighth day after the film viewing, participants returned to the laboratory and completed a final intrusion diary online in the lab. At that time, participants were also asked to indicate "how accurate" they thought their diaries were on a scale from 1 (*not at all accurate*) to 10 (*extremely accurate*). At the end of the second laboratory session, participants were told they could delete the *MetricWire* app off their phone at any time; no additional data were collected after this lab session. Therefore, those who returned to the laboratory more than seven days after the initial laboratory session were only asked to complete daily diaries through the seventh day; although a daily diary was completed inlab during the second session, this data was not utilized for the four participants were not provided the opportunity to report on intrusive memories after the eighth day.

Trauma memory. During the second lab session, participants also completed a version of the Trauma Memory Questionnaire (Halligan et al., 2003) adapted for a film (consistent with Halligan et al., 2002; see Appendix A). Though this scale also includes an 8-item subscale involving retrospective assessment of intrusions, only the 5-item subscale assessing temporal disorganization of the memory for the trauma film was of interest here. Sample items include, "I have trouble remembering the order in which things happened during the film" and "My memory of the film is muddled." Participants were asked to rate the extent to which each statement applied to their memory of the film on a scale from 0 (*not at all*) to 4 (*very strongly*). In past research, this temporal disorganization scale has shown acceptable internal reliability, with coefficient alpha of .88 (Halligan et al., 2003). In the current study, the coefficient alpha was .74.

Demographics. Participants completed a measure assessing demographics including age, education, ethnicity, marital and relationship status, sexual orientation, and annual income (see Appendix A).

Procedure

All study procedures were approved by the University of Nebraska-Lincoln Institutional Review Board (see approval letter in Appendix B).

Recruitment. Participants were recruited through flyers, newspaper advertisements (placed in the classified section of the local Journal Star newspaper), webbased advertising in the "et cetera jobs" section of Craigslist, Facebook desktop ads, and emails sent to a random subset of students enrolled at the University of Nebraska-Lincoln identified by the office of Registration and Records as 21 to 30 years old. All recruitment materials advertised the study as recruiting women ages 21 to 30 who drank alcohol. To ensure sample diversity, ethnic minority participants were oversampled by posting flyers in grocery stores and libraries in ethnically diverse neighborhoods, as well as through emails to students identified by the office of Registration and Records as ethnic minorities. Participants received compensation at a rate of \$10 per hour for the laboratory portions of the study, \$5 for each of 7 days of complete intrusive memory diary entries, and a \$10 bonus for completing all daily diaries. Thus, participants were told they could earn between approximately \$70 and \$120 for completing all portions of the study, depending on condition and associated length of the first laboratory session. Actual compensation for participants who completed all study procedures (including all daily diaries) ranged from \$75 to \$150, and was largely dependent on randomly assigned condition and individual variability in the metabolism of alcohol. (For example, the

participant who received \$150 took 9.5 hours to complete the first lab session, including drinking a high dose of alcohol and returning to a BrAC of .030%).

Data collection. Participants who expressed interest in the study after being reached through the various recruitment methods were screened over the telephone to assess initial eligibility. Detailed telephone screening procedures are described in Appendix A. Those who initially met the inclusion criteria were scheduled for two lab visits exactly one week apart. Participants were asked to refrain from drinking alcohol or using recreational drugs 24 hours prior to the first laboratory session, and to refrain from eating at least four hours prior to the appointment. Participants were asked to arrange their own transportation to the laboratory, but were informed that they would either have to find a ride or take a taxi home after the first laboratory session (regardless of alcohol condition). Participants were sent a reminder email or text message at least 24 hours prior to each scheduled lab session.

Upon arrival, participants provided full verbal and written informed consent (see Appendix C). They then completed additional in-lab screening procedures to confirm eligibility. These screening procedures involved participants (a) completing a breathalyzer test to ensure sobriety, (b) showing study personnel photo identification to verify age, (c) completing a urine pregnancy test (i.e., the Alere hCG Combo test, which can detect as little as 20 mIU/mL human chorionic gonadotropin [hCG] in urine within 3 minutes), (d) verbally reviewing their responses to the telephone screening with trained study personnel, (e) responding to questions on the HELPS Brain Injury Screening Tool (Picard et al., 1991), administered verbally by study personnel, and (f) completing height and weight measurements. No participants had a positive breathalyzer or pregnancy test. Four individuals were deemed ineligible in person (indicated as ineligible in the flow diagram, Figure 2.1), specifically due to weight as measured in the laboratory (n = 1) and responses not reported on the telephone, including a history of sexual assault (n = 1) and low frequency of drinking (i.e., below the study-defined criterion of a "social drinker;" n = 2). These individuals were compensated \$10 for their time before leaving the laboratory. Eligible participants handed any car keys to study personnel for safe keeping during alcohol administration.

After eligibility was confirmed, participants completed a 45-minute battery of questionnaires online (not central to current study aims). Study personnel then provided participants with verbal and written instructions for the intrusive memory diaries and helped participants set up the *MetricWire* app on their smartphone or tablet. Participants were then asked to place the heart rate monitor and strap around their chest and turn off their mobile device to prevent signal interference with the heart rate monitor. Next, participants completed alcohol administration and absorption periods while engaging in an emotionally neutral task (e.g., word search puzzles). They then moved to the separate, staged room for viewing of the trauma film. Study personnel monitored participants through a two-way mirror and provided verbal instructions via an intercom. While in the staged room, participants (a) completed a 5-minute baseline heart rate assessment while relaxing with their eyes open, (b) completed pre-film ratings of current emotions (not central to current study aims), (c) watched the 10.5-minute trauma film while having their heart rate and facial affect recorded, (d) completed post-film ratings of emotions (also not central to current study aims), and (e) completed a 5-minute post-film heart rate assessment while simultaneously monitoring their thoughts for any film-related intrusive

memories (not central to current study aims). Therefore, all participants sat in the staged room for approximately 20 to 30 minutes, including at least 5 minutes with no distractions during the pre-film heart rate assessment. Participants then left the staged room and completed additional self-report questionnaires online, including the Cognitive Processing Questionnaire (to assess for disruption in peritraumatic cognitive processing) and the written free recall task.

All participants were asked to remain in the lab until their BrAC dropped to .030% (National Advisory Council on Alcohol Abuse and Alcoholism, 2005) and they could pass a field sobriety test. Though this meant participants in the placebo condition could leave soon after study procedures were complete, to reinforce the possibility they had consumed alcohol, they were still required to complete procedures confirming sobriety and to obtain a ride home. Before leaving, participants were compensated for the first lab session. Study personnel confirmed each participant left the laboratory with a friend, a family member, or in a taxicab paid for by the study.

During the seven days following the lab visit, participants completed the Daily Diaries of film-related intrusive memories through their smartphones or tablets. One week after the first lab session, they returned to the lab, where they completed one final intrusive memory diary, the film-related Trauma Memory Questionnaire (to assess disorganization in trauma memories), and other self-report measures. Participants then completed an intrusion provocation task (not central to current study aims) in which they returned to the staged room, viewed screenshots of the film, and monitored film-related intrusions for 2 minutes. Participants then viewed a comedy film of their choice for approximately 5 minutes before leaving the staged room. Following the completion of experimental procedures, study personnel asked each participant to guess the study hypotheses ("What do you think this study is about?"; "What do you think the hypotheses of the study are?"; and "Do you think alcohol might have affected your responses to the film you watched, and if so, how?"). Then, participants were told which beverage condition they had received and completed a full verbal and written debriefing detailing the purposes of the study (see Appendix D). Participants were assessed for any persistent distress and provided referrals to mental health services as needed. Before leaving, participants were compensated for completing the daily diaries and the second lab session.

Data Analyses

Preliminary analyses and manipulation check. All data were examined for errors; any data entered by study personnel were double-entered and checked for data entry errors. Descriptive analyses, including sample characteristics and bivariate correlations, were examined for all study variables. Group differences between conditions were examined for all study variables by estimating between-subjects analyses of variance (ANOVAs) in SAS PROC MIXED using restricted maximum likelihood estimation (i.e., equivalent to least squares); variances for each condition were estimated separately using the GROUP command on the REPEATED line. To ensure that alcohol intoxication manipulation was successful, between-subjects ANOVAs were specifically used to examine group differences in peak BrAC levels following the absorption period and at any time in the lab. Individual BrAC levels were considered as a predictor in analyses, centered within each alcohol intoxication group.

Specific Aims 1-4. The specific aims and corresponding hypotheses (as shown in Figure 1.1) focus on the direct and indirect effects of alcohol intoxication on intrusive memories, as mediated through alcohol myopia, stress responses, cognitive processing, and disturbances in trauma memory. All aims were examined simultaneously within a path model in Mplus v.8 (Muthén & Muthén, 2017). The significance of indirect effects was evaluated using maximum likelihood estimation and bias-corrected bootstrap standard errors. Models without bootstrapping were estimated using robust maximum likelihood (MLR). The MLR estimator provides model fit statistics and parameter standard errors that are robust to deviations of normality; however, alternate distributions were considered for outcomes whose residuals showed large deviations from normality. The influence of alcohol intoxication was examined via two dummy codes representing if a participant was assigned to a low dose or high dose alcohol condition, and placebo as the reference group; model-implied comparisons between non-reference groups were obtained using the MODEL CONSTRAINT option. Finally, as shown in Figure 1.1, the outcomes of interest derived from the eight daily diary entries were latent variables that indicate individual differences in level of intrusive memories and their rate of change over time (i.e., slope). Both linear and nonlinear (i.e., quadratic) functions of change were evaluated in order to best describe the pattern of change across days prior to examining the specific aims.

The structural equation model was built sequentially. First, measurement models for the cognitive processing and trauma memory questionnaires were examined using confirmatory factor analyses. Empirically indicated changes in measurement models (based on criteria detailed below) were considered to improve fit. Once the best-fitting

measurement model was determined, tau-equivalence was examined by constraining all loadings within a factor to be equal. If the fit of the tau-equivalent model was not significantly worse than the model with unconstrained factor loadings, the factor could have been represented by a mean subscale score (an observed variable) within the larger structural equation model. However, in cases where tau-equivalence was not supported, factor scores and standard errors were estimated using maximum likelihood with robust standard errors for use in subsequent analyses. (MLR estimation was used to create the factor scores, regardless of the original estimation method of the measurement model, in order to use the expected a priori [EAP] method to estimate factor scores.) Specifically, reliability-corrected factor scores (Brown, 2006) were considered observed variables in the larger structural model. Corrections involved fixing the residual variance of the factor scores to $(1 - \rho)(variance of factor scores + error variance of factor scores)$ where $\rho = \left(\frac{factor variance}{factor variance + error variance of factor scores}\right)$. For models with categorical indicators, an average reliability estimate was computed and utilized in the larger structural model.

Measurement models were identified by fixing latent factor means to 0 and factor variances to 1. In order for factor variance to be predicted in multidimensional structural models, factor variance was freely estimated and a factor loading was fixed to 1 for identification. Nested model comparisons were conducted using a rescaled likelihood ratio test. Non-nested models were compared using a χ^2 test of absolute model fit (which compares the current model to the best-fitting saturated model, such that non-significant *p* values indicate good fit), the Comparative Fit Index (CFI, which compares fit to a null model; values greater than .95 indicate good fit), the Tucker-Lewis Index (TLI, a non-

normed fit index that also compares fit to a null model; values greater than .95 indicate good fit), and the Root Mean Square Error of Approximation (RMSEA, a test of close fit; values and 90% confidence intervals falling under .05 indicate good fit). Local misfit was examined using standardized loadings and the residuals for the estimated correlations.

CHAPTER 3: RESULTS

Preliminary Analyses

The 98 participants detailed above completed the first laboratory session. One participant withdrew from the study after completing the first Daily Diary (no reason was provided), but was retained in analyses. Of the 97 participants who returned for the second laboratory session, 93 (95.9%) completed the second session on time (i.e., exactly 7 days after the first session), 2 (2.1%) participants completed the second session one day late (i.e., 8 days between sessions), 1 (1.0%) participant completed the second session two days late (i.e., 9 days between sessions), and 1 (1.0%) participant completed the second session for days late (i.e., 9 days between sessions). As detailed above, participants who completed the second lab session late were considered to have missing a missing daily diary on Day 8 (which was typically completed in lab). Although the assessment of trauma memory disorganization also took place during this second lab session, this measure was not considered to be as time-sensitive, so data were used from all participants regardless of day of completion.

Awareness of study aims. At the conclusion of study procedures, just prior to debriefing, participants were asked "suspicion questions" to determine whether they could guess the study hypotheses. Given that the study was advertised to participants as a study on "alcohol, memories, and emotions," many of participants' open-ended verbal responses involved acknowledgement of one or more of these three factors. However, only 11.3% recognized the purpose of the study was to examine the influence of alcohol specifically on frequency of intrusive memories. Other common responses suggested participants thought the study might be about alcohol's influence on the vividness or

accuracy of memories. Others thought the study was designed to assess the influence of post-film self-initiated drinking episodes on intrusions. Still others guessed that the study was about eyewitness memory or bystander responses to sexual assault when intoxicated. Of those whose responses did suggest awareness of the current study hypotheses, 60.0% thought alcohol intoxication and intrusive memories would be positively related, and the other 40.0% thought they would be negatively related. Because these perceptions were distributed across all conditions (one to two participants in each condition believed there would be a negative association; two participants in each condition believed there would be a positive association), these assumptions were not expected to impact the overall pattern of results.

Alcohol-related variables. Participants were randomized into a placebo condition (n = 34), low dose condition (n = 32), and high dose condition (n = 32). As shown in Table 3.1, all participants in the low dose condition reached a BrAC of at least .030% after the absorption period and before film viewing. Of those in the high dose condition, three (9.4%) participants did not reach a BrAC of .070% prior to film viewing. Instead, following a 30-minute absorption period, these participants had a BrAC of .054%, .061%, and .062%. Though the first participant only reached a peak BrAC of .069% during the session, the other two participants eventually reached a BrAC of at least .070% in the laboratory.

Between-subjects ANOVAs allowing variances to differ between conditions revealed significant differences between conditions in post-absorption BrAC, F(2, 95) =170.30, p < .001 (see Table 3.1). Specifically, pairwise comparisons revealed that participants' post-absorption BrAC in the high dose condition (M = .091, SD = .019) was significantly higher than those in the low dose condition (M = .051, SD = .012), which was in turn significantly higher than those in the placebo condition (M = .020, SD = .012). Although some participants in the placebo condition did register a BrAC up to .047% (beyond measurement error of the device), this test was administered immediately after drink consumption and therefore may have been impacted by the small amount of alcohol on the glass (though participants were asked to rinse their mouth with water). Similarly, between-subjects ANOVAs revealed significant differences between conditions in peak observed BrAC, F(2, 95) = 317.67, p < .001. Specifically, pairwise comparisons revealed that participants' peak observed BrAC in the high dose condition (M = .110, SD = .017) was significantly higher than those in the low dose condition (M = .053, SD = .010), which was in turn significantly higher than those in the placebo condition in Figure 3.1. Visual inspection of curves reveals that film viewing typically took place near the peak BrAC in alcohol conditions.

	Р	lacebo	Lo	w Dose	High Dose		F(2, 95)
	()	n = 34)	()	n = 32)		n = 32)	
	М	Range	М	Range	М	Range	-
	(SD)		(SD)		(SD)		
Post-absorption	.020	.000047	.051	.030 – .075	.091	.054 – .126	170.30*
BrAC ^{a, b. c}	(.012)		(.012)		(.019)		
Peak observed	.020	.000047	.053	.036 – .075	.110	.069 – .150	317.67*
BrAC ^{a, b. c}	(.012)		(.010)		(.017)		
Beverage tasted	3.32	3 - 4	2.34	1 - 4	2.22	1 - 3	40.91*
pleasant (1 to 4) ^{a, c}	(0.48)		(0.65)		(0.66)		
Beverage tasted	3.29	3 - 4	2.44	1 - 4	2.34	1 - 4	27.94*
good $(1 \text{ to } 4)^{a, c}$	(0.46)		(0.72)		(0.75)		
Perceived intoxication	2.06	0 - 8	3.47	0 - 7	5.34	2 - 8	27.78*
$(0 \text{ to } 11)^{a, b. c}$	(1.98)		(1.90)		(1.64)		
Perceived impairment	1.71	0 - 6	3.16	0 - 7	4.81	0 - 9	20.61*
$(0 \text{ to } 10)^{a, b. c}$	(1.75)		(2.19)		(2.16)		

Table 3.1

Descriptive Statistics and ANOVAs for Alcohol-Related Variables				
	Descriptive Statistics	and ANOVAs	for Alcohol-Related	Variables

^aHigh Dose and Placebo are significantly different, p < .05

^bHigh Dose and Low Dose are significantly different, p < .05

^cLow Dose and Placebo are significantly different, p < .05

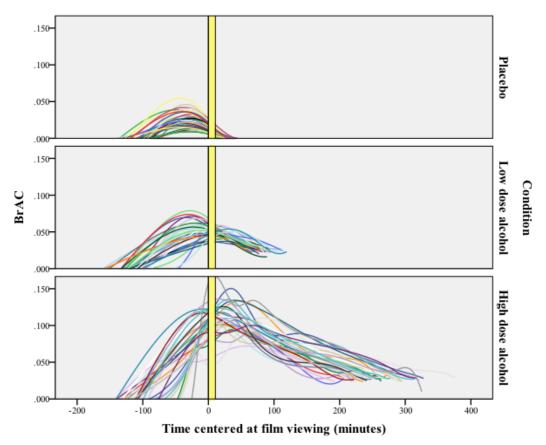


Figure 3.1. Individual BrAC curves centered at film viewing (indicated by the yellow reference line).

Participants in the low and high dose alcohol conditions both reported the beverage tasted worse than participants in the placebo condition; there were no differences between either question assessing taste between low and high dose participants. As shown in Table 3.1, perceived intoxication and impairment also differed between conditions, with those in the placebo condition reporting the least perceived intoxication and impairment, and those in the high dose condition reporting the greatest perceived intoxication and impairment. As indicated in Table 3.2, most (85.3%) participants in the placebo condition, and all those in the low and high dose conditions believed they consumed alcohol. Thus, only 14.7% of those in the placebo condition

guessed their beverage condition correctly. Those who believed they consumed alcohol were also asked to guess which condition they were in; the percentage of low dose participants who guessed correctly (65.6%) was not significantly different from the percentage of high dose participants who guessed correctly (75.0%), p > .05.

Table 3.2Perceptions of Alcohol Conditions

^ ×	Placebo $(n = 34)$	Low Dose $(n = 32)$	High Dose $(n = 32)$
Believe consumed alcohol	29 (85.3%)	32 (100%)	32 (100%)
Guessed low dose (1-2 drinks) Guessed high dose (3-4 drinks)	25 (73.5%) 4 (11.8%)	21 (65.6%) 11 (34.4%)	8 (25.0%) 24 (75.0%)
Correctly guessed condition	5 (14.7%)	21 (65.6%)	24 (75.0%)

Intrusive memories. Daily diary compliance was good, as the majority of participants completed all seven daily diaries (71.4%), 17.3% completed six diaries, 6.1% completed five diaries, 3.1% completed four diaries, 1.0% completed three diaries, and 1.0% completed only one diary (before withdrawing from the study). Participants' own evaluation of the accuracy of their daily diaries ranged from 6 to 10 (M = 8.82, SD = 0.92), with 10 being *extremely accurate*. Across all intrusive memory reports provided on the app, intrusive memories had a mean distress rating of 1.53 (SD = 1.14, range = 0 to 4). Participants categorized 25.7% of intrusive memories as thoughts, 33.8% were images, and the remaining 40.5% were a combination of a thought and image. Of intrusions that involved any imagery, the average vividness of the memory was 4.41 (SD = 1.50, range = 1 to 7).

Descriptive statistics and bivariate correlations for daily reports of intrusive memories are shown in Table 3.3. Across all eight days (including the report made during the second laboratory session), the total number of intrusive memories per participant ranged from 0 to 22. The vast majority of participants (94.9%) reported at least one intrusive memory. The number of intrusive memories reported on any given day was correlated with the number of intrusive memories reported on most, but not all other days. On average, participants reported the most intrusive memories on the day of the film viewing (Day 1); the number of intrusive memories declined each day thereafter. There was variability in the time of day participants viewed the film and therefore, variability in the amount of time remaining in Day 1 to report intrusive memories. However, the hours remaining in the first day (after film viewing) was not significantly correlated to the number of intrusive memories in Day 1, r = -.07, p = .531.

	п	Mean (SD)	Range		Correlations					
Intrusive Memories				1	2	3	4	5	6	7
Day 1	95	2.21 (2.09)	0-12							
Day 2	93	1.61 (1.50)	0 - 8	.47						
Day 3	89	0.87 (1.14)	0 - 8	.23	.11					
Day 4	92	0.54 (0.89)	0 - 4	.00	.09	.28				
Day 5	92	0.43 (0.79)	0 - 4	.22	.25	.46	.29			
Day 6	92	0.35 (0.56)	0 - 2	.28	.27	.24	.24	.29		
Day 7	88	0.20 (0.48)	0 - 2	.14	.28	.18	.17	.41	.44	
Day 8	94	0.20 (0.56)	0 - 4	.05	.40	.18	.04	.01	02	03

Table 3.3Descriptive Statistics and Bivariate Correlations for Daily Intrusive Memory Reports

Note: Bold, italicized correlations are significant at p < .01. Bold correlations are significant at p < .05

Between-subjects ANOVA comparisons for intrusive memories (allowing

variances to differ between conditions) are displayed in Table 3.4 (results of pairwise comparisons are indicated by superscripts). Participants in the high dose condition (M = 2.25, SD = 1.86) reported significantly more intrusive memories on Day 2 than those in the low dose (M = 1.39, SD = 1.33) or placebo conditions (M = 1.29, SD = 1.17). However, no other differences between conditions were observed between the raw

number of intrusive memories reported on a daily basis. The trends for number of

intrusive memories reported per day in each condition can be viewed in Figure 3.2.

Between-Group Comparison of Intrusive Memories									
Placebo	Low Dose	High Dose	F	df	р				
M (SD)	M (SD)	M (SD)							
2.18 (1.38)	1.77 (1.69)	2.68 (2.89)	1.25	2, 92	.290				
1.29 (1.17)	1.39 (1.33)	2.25 (1.86)	2.91	2,90	.060				
0.65 (0.75)	0.89 (1.52)	1.07 (1.05)	1.67	2,86	.193				
0.39 (0.76)	0.68 (0.94)	0.57 (0.97)	0.94	2, 89	.395				
0.38 (0.83)	0.45 (0.72)	0.48 (0.83)	0.14	2, 89	.870				
0.33 (0.60)	0.32 (0.48)	0.39 (0.62)	0.12	2, 89	.894				
0.13 (0.34)	0.21 (0.49)	0.29 (0.60)	0.82	2,85	.444				
0.16 (0.45)	0.17 (0.38)	0.28 (0.77)	0.34	2, 91	.715				
	Placebo M (SD) 2.18 (1.38) 1.29 (1.17) 0.65 (0.75) 0.39 (0.76) 0.38 (0.83) 0.33 (0.60) 0.13 (0.34)	Placebo Low Dose M (SD) M (SD) 2.18 (1.38) 1.77 (1.69) 1.29 (1.17) 1.39 (1.33) 0.65 (0.75) 0.89 (1.52) 0.39 (0.76) 0.68 (0.94) 0.38 (0.83) 0.45 (0.72) 0.33 (0.60) 0.32 (0.48) 0.13 (0.34) 0.21 (0.49)	Placebo Low Dose High Dose M (SD) M (SD) M (SD) 2.18 (1.38) 1.77 (1.69) 2.68 (2.89) 1.29 (1.17) 1.39 (1.33) 2.25 (1.86) 0.65 (0.75) 0.89 (1.52) 1.07 (1.05) 0.39 (0.76) 0.68 (0.94) 0.57 (0.97) 0.38 (0.83) 0.45 (0.72) 0.48 (0.83) 0.33 (0.60) 0.32 (0.48) 0.39 (0.62) 0.13 (0.34) 0.21 (0.49) 0.29 (0.60)	$\begin{tabular}{ c c c c c c c c c c c c c c c c c c c$	$\begin{tabular}{ c c c c c c c c c c c c c c c c c c c$				

Table 3.4

^aHigh Dose and Placebo are significantly different, p < .05^bHigh Dose and Low Dose are significantly different, p < .05

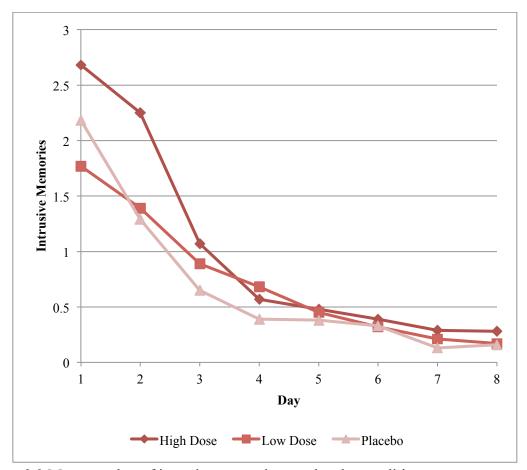


Figure 3.2 Mean number of intrusive memories per day, by condition.

To examine potential group differences in *total* number of intrusive memories during the eight days, a linear regression was estimated with restricted maximum likelihood in SAS PROC MIXED to determine the adjusted mean differences between conditions, controlling for the number of diaries completed. Controlling for number of diaries completed, those in the high dose condition ($M_{adjusted} = 7.42$, SE = 0.89) reported significantly more intrusive memories than those in the placebo condition ($M_{adjusted} =$ 5.28, SE = 0.56), t(94) = -2.04, p = .044. Number of intrusive memories reported by those in the low dose condition ($M_{adjusted} = 5.60$, SE = 0.89) did not differ from the placebo or high dose condition, ps > .05. Adjusted means (controlling for number of diaries completed) are displayed in Figure 3.3.

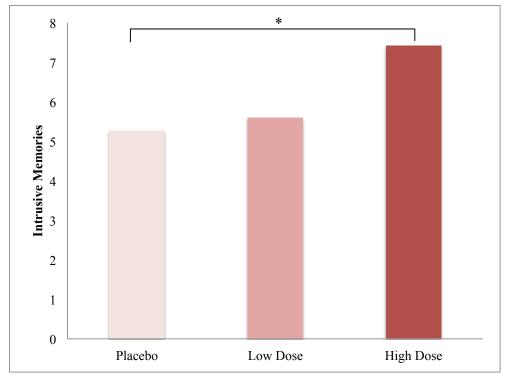


Figure 3.3. Total number of intrusive memories reported per condition, controlling for number of daily diaries completed. *p < .05

Potential mechanisms. Descriptive statistics and correlations for all variables considered to be potential mechanisms (related to alcohol myopia, stress response, and cognitive processing) are included in Table 3.5. For the purposes of these descriptive statistics, conceptual processing, data-driven processing, and disorganization mean scores were computed in accordance with published scoring recommendations. Given that no scoring recommendations were available for the myopia variables, total sum scores for central and peripheral details were computed for items indicated in the measurement model described below.

Descriptive Statistics and Bivariate Correlations for Potential Mechanisms									
	п	Mean (SD)	Range		Correlations				
				1	2	3	4	5	6
1. Total peripheral details	98	22.48 (14.61)	0-71						
2. Total central details	98	20.09 (16.30)	0 - 61	26					
3. Change in heart rate	90	5.63 (8.43)	-30.88 - 34.37	17	07				
4. Change in facial affect	97	-0.12 (0.20)	-0.57 - 0.57	13	.05	.05			
5. Data-driven processing	98	2.42 (0.57)	1.36 - 4.55	08	09	.09	.02		
6. Conceptual processing	98	3.58 (0.64)	1.83 - 5.00	.20	.02	09	06	14	
7. Disorganization of	97	0.36 (0.41)	0.00 - 2.40	08	10	01	06	.29	17
trauma memory									

Table 3.5Descriptive Statistics and Bivariate Correlations for Potential Mechanisms

Note: Bold, italicized correlations are significant at p < .01. Bold correlations are significant at p < .05

With regard to the free recall task, the total number of details reported for peripheral and central information (as determined in the measurement model detailed below) ranged from 0 to 71, and 0 to 61, respectively. Though participants were prompted to write about both the film and room, 11.2% only reported details about the film, and 19.4% only reported details about the room. Relatedly, the total number of central and peripheral details recalled were negatively correlated, r = -.26, p = .010.

As described in the methods section, heart rate data were available for 90 participants. The average film-related change in heart rate was 5.63 (SD = 8.43) beats per

minute. Without considering the degree of change, 78.9% experienced at least some filmrelated increase in heart rate, and the remaining 21.1% experienced at least some slowing in heart rate. In a similar manner, while the overall change in facial affect reflected more negative emotions during the film (M = -0.12, SD = 0.20), 79.4% participants had a filmrelated decrease in valence and 20.6% had at least some increase in valence.

Few bivariate correlations between potential mechanisms were significant. One significant correlation revealed a positive association between the number of peripheral details recalled and conceptual processing, r = .20, p = .046. In addition, data-driven processing was positively correlated with the degree of disorganization in the memory for the trauma film, r = .29, p = .003.

Between-subjects ANOVAs allowing for heterogeneous variances were used to assess differences in potential mechanisms by alcohol condition (see Table 3.6). With regard to the free recall task, the number of peripheral details freely recalled was significantly lower in the high dose condition (M = 16.38, SD = 10.98) compared to both the low dose (M = 23.66, SD = 12.08) and the placebo condition (M = 27.12, SD = 17.82). Consistent with expectations, this suggests a narrowing of attention associated with high doses of alcohol. In addition, film-related increases in heart rate were greatest in the placebo condition (M = 9.52, SD = 6.99) compared to the dampened film-related increases in heart rate for both the low dose (M = 4.53, SD = 10.78) and high dose conditions (M = 2.79, SD = 5.91). No other significant group differences between potential mechanisms were observed.

Between-Group Comparison of Pote	ential Mechanish	15				
	Placebo	Low Dose	High Dose	F	df	p
	M (SD)	M (SD)	M (SD)			
Total peripheral details ^{a, b}	27.12 (17.82)	23.66 (12.08)	16.38 (10.98)	5.62	2,95	.005
Total central details	21.12 (17.82)	20.19 (17.52)	18.91 (13.57)	0.17	2,95	.845
Change in heart rate ^{a, c}	9.52 (6.99)	4.53 (10.78)	2.79 (5.91)	8.60	2,87	< .001
Change in facial affect	-0.15 (0.16)	-0.09 (0.22)	-0.10 (0.22)	0.99	2,94	.374
Data-driven processing	2.39 (0.59)	2.36 (0.42)	2.53 (0.68)	0.81	2,95	.448
Conceptual processing	3.65 (0.64)	3.63 (0.64)	3.46 (0.64)	0.82	2,95	.442
Disorganization of trauma memory	0.35 (0.36)	0.34 (0.36)	0.40 (0.50)	0.16	2,94	.857

 Table 3.6
 Between-Group Comparison of Potential Mechanisms

^aHigh Dose and Placebo are significantly different, p < .05

^bHigh Dose and Low Dose are significantly different, p < .05

^cLow Dose and Placebo are significantly different, p < .05

Latent Growth Curve Model for Intrusive Memories

The initial level and change in number of intrusive memories reported each day was predicted within a latent growth curve model. Indicators were eight daily variables representing the number of intrusive memories reported each day. Because the first report occurred during the same day as the film viewing, time remaining in Day 1 was included as a predictor for Day 1 intrusive memories. All indicators were count variables (i.e., non-negative integers) and therefore modeled using Poisson distributions (e.g., Atkins & Gallop, 2007). A log link is used for Poisson distributions to keep estimated values positive. Poisson distributions also assume the mean and variance of the distribution are equal. An extension of the Poisson distribution, a negative binomial distribution, allows the mean and variance to differ through a dispersion parameter. Although negative binomial distributions were considered for the intrusive memory variable indicators, the dispersion parameters were not significant, and therefore not empirically indicated. The more parsimonious model with Poisson distributions was therefore retained.

The latent growth curve model was specified such that an intercept was represented by a factor with all loadings fixed to 1, a slope was represented by a factor with loadings ranging from 0 to 7 (corresponding to Day 1 to 8, increasing by 1 for each day), and a quadratic trend was represented by a factor with loadings ranging from 0 to 49 (such that the factor loadings of the slope were squared). Fixed and random effects were considered for both linear and quadratic trends. Rescaled likelihood ratio tests revealed that a model with a random intercept and linear slope fit better than a model with a random intercept only, p = .001. Rescaled likelihood ratio tests also indicated that including a random quadratic effect would have further improved the fit of the model, p = .001. However, in the larger structural model, it was necessary to remove the random quadratic trend to 0) to facilitate estimation (i.e., the larger model would not converge otherwise). Therefore, although a fixed deviation from linearity was estimated within the latent growth curve model, only the intercept and linear trend were predicted by study variables. This approach lends itself to a clear interpretation; the influence of each study variable on the rate of change in intrusions is represented by a single coefficient (instead of separate coefficients associated with linear and quadratic components of change).

Alcohol variables, including two dummy codes representing alcohol conditions and group-mean centered BrAC for both the low and high conditions, were considered as predictors of the intercept and slope. Group-mean centered BrAC variables were not significant predictors and therefore removed from all subsequent analyses. The more parsimonious model representing differences between alcohol conditions in the level and rate of change in intrusive memories is represented in Figure 3.4 and Table 3.7. Given the inclusion of count variables, unstandardized estimates are presented.

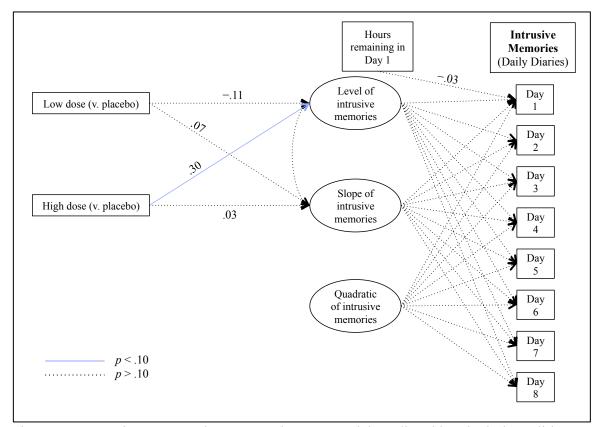


Figure 3.4. Intrusive memory latent growth curve model predicted by alcohol conditions.

Table 3.7Unstandardized Estimates of Intrusive Memory Latent Growth Curve M	odel
	Estimate
Low dogo (v. placebo) - Lowel of intrusive memories	0.11

	Estimate	SE	р
Low dose (v. placebo) \rightarrow Level of intrusive memories	- 0.11	0.17	.510
High dose (v. placebo) \rightarrow Level of intrusive memories	0.30	0.18	$.084^{\dagger}$
High dose (v. low dose) \rightarrow Level of intrusive memories (model-implied)	0.42	0.20	.033*
Low dose (v. placebo) \rightarrow Slope of intrusive memories	0.07	0.07	.305
High dose (v. placebo) \rightarrow Slope of intrusive memories	0.03	0.07	.619
High dose (v. low dose) \rightarrow Slope of intrusive memories (model-implied)	- 0.04	0.06	.567
Hours remaining on Day 1 \rightarrow Day 1 intrusive memories	- 0.03	0.02	.123
Covariance between level and slope	0.02	0.02	.303
Level Intercept	0.86	0.19	< .001*
Slope Intercept	- 0.69	0.15	< .001*
Quadratic Mean	0.03	0.02	.153
Level Residual Variance	0.24	0.07	.001*
Slope Residual Variance	0.02	0.01	.078
$\frac{1}{10}$ = 10 * = < 05			

 $^{\dagger}p < .10, * p < .05$

Although it was hypothesized that the low dose condition (v. placebo) would produce more frequent and persistent intrusive memories, and the high dose condition (v. placebo) would produce fewer and less persistent intrusive memories (Hypotheses 1a and 1b), a different pattern of results was found. With regard to initial level of intrusive memories (i.e., number of intrusive memories at Day 1 controlling for number of hours remaining), the high dose condition was marginally greater than the placebo (p = .084), and significantly greater than those in the low dose (p = .033). The level for the placebo was greater than the level for the low dose, but this difference was not significant (p =.510). There were no significant differences between conditions in the slope of intrusive memories. In sum, instead of observing the expected inverted U-shaped effect of alcohol in which the low dose condition produced the most intrusions immediately after the film, the high dose condition produced the most intrusions. Contrary to expectations, the persistence of intrusive memories was not found to be associated with alcohol condition.

Measurement Models

Measurement model for alcohol myopia. Confirmatory factor analysis was used to evaluate the distinction between central and peripheral information. To first evaluate fit, robust maximum likelihood estimation was used in order to create fit statistics and standard errors that were robust to non-normality (all variables were considered to be continuous). Given that representing each individual object as a separate indicator would have resulted in more parameters than participants, objects were combined into larger groupings (i.e., sum scores were created) based on location in the room or film and salience. The following four groupings were trimmed from analyses due to low standardized loadings on all possible factors: items in a peripheral corner of the room, items under the participant desk, mention of drug use in the film, and context (i.e., language and setting) of the film. Although distinctions were originally examined between central and peripheral information within the film, all details for the film were highly correlated and therefore collapsed into one factor. The final, best fitting model involved two subscales: (1) details of the film, reflecting Central information, and (2) details of the room, reflecting Peripheral information. Details specific to the perpetrator and victim in the film were highly correlated and allowed to covary, r = .55, p < .001. Though the χ^2 test of absolute model fit was significant, χ^2 (133) = 165.82, p = .021, other indices suggest the fit of this final model was good, CFI = .948, TLI = .941, RMSEA = .050 (90% CI = .018, .073). Standardized factor loadings ranged from .56 to .86 for the Central factor and .34 to .69 for the Peripheral factor. Central and Peripheral factors were marginally correlated, r = -.24, p = .096.

After determining a factor model with sufficient fit, appropriate count distributions were allowed for each indicator, including Poisson and—where indicated and estimable—zero-inflated Poisson distributions. (Although dispersion factors via negative binomial distributions were also considered, they could not be estimated.) Given that covariances between count variables are not allowed in *Mplus*, the covariance between perpetrator and victim details was represented equivalently by a latent variable (loadings constrained to 1). Absolute fit statistics are not available for factor models with count distributions and standardized loadings are not interpretable for count variables.

Measurement model for cognitive processing style. Based on recommendations by Halligan and colleagues (2002), a two-factor model representing data-driven processing (11 items) and conceptual processing (6 items) was hypothesized to fit data

from the Cognitive Processing Questionnaire. Likert-type responses were modeled as ordinal with multinomial distributions. Given that absolute fit indices are not available for maximum likelihood estimation with categorical indicators, fit was first determined using mean- and variance-adjusted weighted least-squares (WLSMV) estimation. This original model fit the data poorly as indicated by a significant χ^2 test of absolute model fit and other fit indices, CFI = .824, TLI = .798, RMSEA = .116 (90% CI = .098, .134). Modifications to the factor structure were made after considering standardized factor loadings, residual correlations, modification indices, and conceptual groupings. As can be seen in Table 3.8, the resultant model included three factors representing data-driven processing (8 items), a separate factor for particularly sensory-based impressions (3 items), and conceptual processing (4 items). Two items were dropped due to low standardized factor loadings and vague wording. Though the χ^2 test of absolute model fit of this revised model was still significant, other fit indices suggest this model achieved adequate fit, CFI = .955, TLI = .946, RMSEA = .068 (90% CI = .040, .093). Standardized loadings ranged from .56 to .85 for the data-driven processing factor, .38 to .70 for the sensory-based impression factor, and .44 to .62 for the conceptual processing factor. As expected, the data-driven and conceptual processing factors were negatively correlated, r= -.66, p < .001. However, the sensory-based impression factor was positively correlated to the conceptual processing factor, r = .71, p < .001, and not correlated to the data-driven processing factor, r = -.05, p = .658. Therefore, the sensory-based impression factor was deemed theoretically ambiguous and subsequently dropped from the model.

The resulting two-factor model also had adequate fit, CFI = .966, TLI = .957, RMSEA = .076 (90% CI = .042, .106). Standardized loadings ranged from .56 to .85 for

the data-driven processing factor, and .37 to .64 for the conceptual processing factor. The data-driven and conceptual processing factors were again negatively correlated, r = -.69, p < .001. A tau-equivalent model in which the loadings in each factor were constrained to be equal fit significantly worse than a model with freely-estimated loadings, $\chi^2(10) = 28.00$, p = .002. Therefore, reliability-corrected factor scores for data-driven and conceptual processing were used for subsequent analyses.

Table 3.8

Cognitive Processing Questionnaire Factor Models

Item	Original	Revised
3. I could not really take everything in.	DDP	DDP
4. I did not fully understand what is going on.	DDP	DDP
7. It was like a stream of unconnected impressions following each other.	DDP	DDP
8. I didn't think clearly.	DDP	DDP
9. There were so many sensations I could not put everything together.	DDP	DDP
11. I got confused.	DDP	DDP
15. It all became a bit of a blur.	DDP	DDP
16. My mind was racing but my thoughts were disjointed.	DDP	DDP
2. My mind was filled with immediate impressions and reactions.	DDP	SBI
13. My mind was fully occupied with what I saw, heard, smelled, and felt.	DDP	SBI
14. I was aware of very immediate things, just going from moment to moment.	DDP	SBI
1. My thinking was very clear, not muddled.	CP	CP
10. I had a clear impression of how one thing followed from another.	CP	CP
12. I thought about what was happening.	CP	CP
17. I stayed focused.	CP	CP
5. I thought ahead.	СР	
6. In my mind I talked myself through what was happening in the film.	CP	

Note: DDP = Data-Driven Processing, CP = Conceptual Processing, SBI = Sensory-Based Impressions. Items not included in the final analyses are shaded gray.

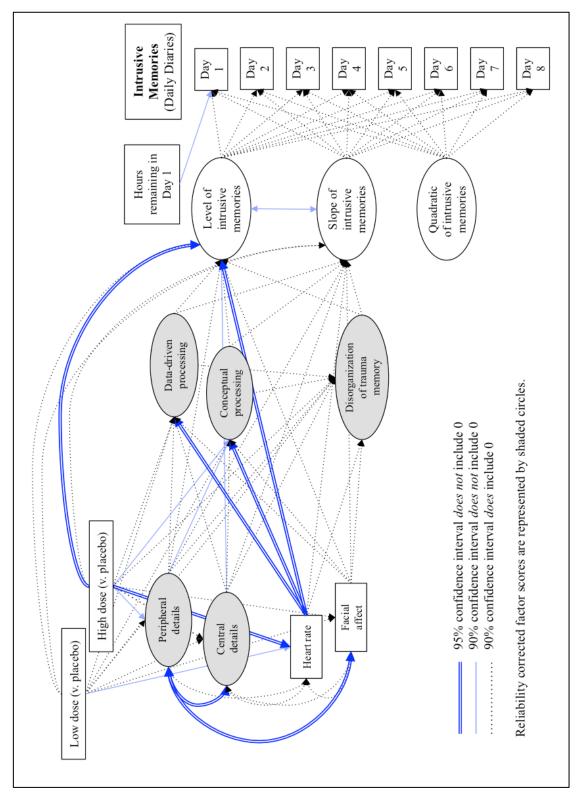
Measurement model for disorganization of trauma memory. Based on

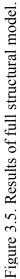
Halligan et al. (2003), a two-factor model representing temporal disorganization (5 items) and sensory information (8 items) was hypothesized to fit data from the Trauma Memory Questionnaire–Film. However, only the 5-item temporal disorganization scale was conceptually of interest as a possible mediator. Therefore, a one-factor model with five categorical indicators and WLSMV estimation was examined and determined to have good fit, $\chi^2(5) = 8.05$, p = .153; CFI = .988, TLI = .977, RMSEA = .079 (90% CI = .000,

.176). Standardized loadings ranged from .27 to .85. A tau-equivalent model in which the loadings were constrained to be equal fit significantly worse than a model with freely-estimated loadings, $\chi^2(4) = 45.52$, p < .001. Therefore, a reliability-corrected factor score for temporal disorganization of the trauma memory was used in subsequent analyses.

Structural Models

Full structural model. After establishing each component measurement model, the larger structural model was built, as shown in Figure 3.5. The full latent growth curve model for intrusive memories with Poisson distributions was included in the larger structural model (and therefore, fit indices were not produced). All previously discussed measurement models were represented as reliability-corrected factor scores. Change scores for heart rate and facial affect were observed variables. This model was estimated with maximum likelihood and 1000 bootstrap resamples. Unstandardized estimates and bias-corrected bootstrap confidence intervals are displayed in Table 3.9.





nstanaaraizea Estimates for Fuii Structurai M	10401		95%	6 CI	90%	6 CI
	Estimate	SE	Lower	Upper	Lower	Upper
nth Coefficients						
Y X Level of Intrusive Memories						
Low dose (v. placebo)	0.02	0.18	-0.278	0.387	-0.244	0.308
High dose (v. placebo)	0.02	0.18	0.112	0.387	0.140	0.308
High dose (v. low dose; model-implied		0.21	0.010	0.350	0.010	0.733
Change in heart rate	0.03	0.22	0.004	0.048	0.008	0.045
Change in facial affect	0.05	0.34	-0.498	0.933	-0.390	0.744
Data-driven processing	0.03	0.23	-0.282	0.933	-0.222	0.504
Conceptual processing	0.12	0.23	-0.538	0.845	-0.458	0.683
Disorganization of trauma memory	0.15	0.16	-0.161	0.472	-0.105	0.384
Peripheral details	-0.03	0.10	-0.309	0.472	-0.280	0.186
Central details	0.14	0.14	-0.022	0.237	0.018	0.309
	0.14	0.07	-0.022	0.545	0.010	0.507
Slope of Intrusive Memories Low dose (v. placebo)	0.08	0.07	-0.075	0.214	-0.046	0.184
High dose (v. placebo)	0.08		-0.130	0.214		
e (1)		0.09		0.217	-0.091	0.188
High dose (v. low dose; model-implied		0.07	-0.178		-0.154	0.057
Change in heart rate	0.00	0.01	-0.007	0.012	-0.005	0.010
Change in facial affect	-0.09	0.17	-0.420	0.213	-0.357	0.146
Data-driven processing	-0.02	0.09	-0.187	0.183	-0.142	0.174
Conceptual processing	-0.07	0.14	-0.322	0.261	-0.280	0.140
Disorganization of trauma memory	-0.01	0.05	-0.123	0.072	-0.106	0.048
Peripheral details	0.00	0.05	-0.112	0.084	-0.080	0.078
Central details	-0.03	0.04	-0.105	0.038	-0.090	0.032
Disorganization of Trauma Memory						
Low dose (v. placebo)	-0.02	0.23	-0.529	0.407	-0.512	0.324
High dose (v. placebo)	-0.03	0.24	-0.634	0.400	-0.451	0.270
Change in heart rate	-0.01	0.01	-0.041	0.014	-0.029	0.011
Change in facial affect	-0.14	0.53	-1.281	0.752	-1.176	0.567
Peripheral details	0.04	0.15	-0.225	0.386	-0.182	0.353
Central details	-0.12	0.11	-0.297	0.112	-0.271	0.101
Data-driven processing	0.21	0.23	-0.342	0.555	-0.219	0.531
Conceptual processing	-0.28	0.31	-0.964	0.196	-0.749	0.163
Data-Driven Processing						
Low dose (v. placebo)	0.14	0.20	-0.239	0.534	-0.190	0.492
High dose (v. placebo)	0.36	0.24	-0.079	0.780	-0.010	0.710
Change in heart rate	0.03	0.01	0.008	0.053	0.012	0.047
Change in facial affect	0.13	0.55	-0.982	1.017	-0.819	0.964
Peripheral details	-0.19	0.18	-0.550	0.132	-0.511	0.089
Central details	0.05	0.12	-0.208	0.279	-0.169	0.244
Conceptual Processing						
Low dose (v. placebo)	-0.17	0.20	-0.591	0.194	-0.517	0.133
High dose (v. placebo)	-0.34	0.21	-0.887	0.018	-0.712	-0.048
Characteristic haracteristic	-0.02	0.01	-0.049	-0.008	-0.043	-0.010
Change in heart rate	-0.02					
Change in facial affect	0.12	0.52	-0.707	1.196	-0.643	1.032
e				1.196 0.571 0.139	-0.643 0.041 -0.235	1.032 0.546 0.125

Table 3.9Unstandardized Estimates for Full Structural Model

				6 CI		6 CI
	Estimate	SE	Lower	Upper	Lower	Upper
Path Coefficients (cont.)						
Y X						
Peripheral Details						
Low dose (v. placebo)	-0.03	0.19	-0.347	0.376	-0.281	0.34
High dose (v. placebo)	-0.43	0.24	-0.862	0.006	-0.770	-0.01
Central Details						
Low dose (v. placebo)	-0.06	0.23	-0.447	0.406	-0.379	0.38
High dose (v. placebo)	0.09	0.22	-0.407	0.475	-0.317	0.40
Change in Heart Rate						
Low dose (v. placebo)	-4.96	2.34	-9.245	0.019	-8.628	-1.26
High dose (v. placebo)	-6.85	1.54	-9.835	-3.628	-9.368	-4.47
Change in Valence of Facial Affect						
Low dose (v. placebo)	0.06	0.05	-0.042	0.166	-0.017	0.14
High dose (v. placebo)	0.05	0.04	-0.043	0.125	-0.019	0.11
Intrusive Memories on Day 1						
Hours remaining in Day 1	-0.03	0.02	-0.068	0.006	-0.058	-0.00
Covariances						
Peripheral details & Central details	-0.29	0.08	-0.469	-0.150	-0.450	-0.17
Peripheral details & Heart rate	0.61	0.49	-0.383	1.492	-0.211	1.47
Peripheral details & Facial affect	-0.03	0.02	-0.062	-0.001	-0.058	-0.00
Central details & Heart rate	-0.66	0.61	-2.000	0.379	-1.914	0.17
Central details & Facial affect	0.02	0.02	-0.019	0.051	-0.013	0.04
Level & Slope of intrusive memories	0.02	0.02	-0.005	0.063	0.001	0.06
Heart rate & Facial affect	0.18	0.15	-0.036	0.510	-0.006	0.46
Means and Intercepts						
Level of Intrusive Memories	0.58	0.17	0.252	0.897	0.297	0.84
Slope of Intrusive Memories	-0.73	0.11	-1.043	-0.542	-0.950	-0.58
Quadratic of Intrusive Memories	0.03	0.02	0.005	0.053	0.011	0.05
Disorganization (uncorrected factor score)	-0.12	0.19	-0.505	0.219	-0.452	0.13
Data-driven (uncorrected factor score)	-0.35	0.19	-0.716	0.000	-0.654	-0.03
Conceptual (uncorrected factor score)	0.36	0.20	-0.023	0.747	0.052	0.71
Peripheral details (uncorrected factor score)	0.15	0.16	-0.232	0.415	-0.194	0.34
Central details (uncorrected factor score)	-0.02	0.16	-0.313	0.265	-0.262	0.23
Heart rate	9.63	1.19	7.481	12.01	7.818	11.8
Facial affect	-0.15	0.03	-0.206	-0.112	-0.201	-0.11
Residual Variances						
Level of Intrusive Memories	0.18	0.07	0.105	0.271	0.124	0.27
Slope of Intrusive Memories	0.01	0.01	-0.009	0.030	-0.003	0.02
Disorganization (uncorrected factor score)	0.48	0.09	0.385	0.628	0.411	0.62
Data-driven (uncorrected factor score)	0.66	0.10	0.543	0.859	0.568	0.85
Conceptual (uncorrected factor score)	0.42	0.07	0.322	0.577	0.341	0.57
Peripheral details (uncorrected factor score)	0.74	0.12	0.551	1.012	0.587	0.96
Central details (uncorrected factor score)	0.85	0.10	0.633	1.009	0.664	0.97
Heart rate	61.97	15.70	39.13	96.79	43.17	95.4
Facial affect	0.04	0.01	0.027	0.049	0.030	0.04

			95%	6 CI	90%	6 CI	
	Estimate	SE	Lower	Upper	Lower	Upper	
Total Effects							
Low dose (v. placebo) \rightarrow Level	-0.13	0.17	-0.389	0.223	-0.318	0.158	
High dose (v. placebo) \rightarrow Level	0.33	0.15	0.006	0.563	0.033	0.542	
Low dose (v. placebo) \rightarrow Slope	0.07	0.07	-0.068	0.198	-0.045	0.191	
High dose (v. placebo) \rightarrow Slope	0.02	0.07	-0.100	0.202	-0.067	0.184	
Total Indirect Effects							
Low dose (v. placebo) \rightarrow Level	-0.14	0.13	-0.393	0.114	-0.353	0.068	
High dose (v. placebo) \rightarrow Level	-0.17	0.15	-0.540	0.056	-0.458	0.025	
Low dose (v. placebo) \rightarrow Slope	-0.02	0.05	-0.104	0.079	-0.083	0.054	
High dose (v. placebo) \rightarrow Slope	-0.01	0.05	-0.104	0.110	-0.076	0.106	
Specific Indirect Effects: Low Dose (v. Placeb	$(0) \rightarrow Leve$	el of Int	rusive M	emories			
Facial affect	0.00	0.02	-0.028	0.078	-0.016	0.063	
Heart rate	-0.14	0.09	-0.350	0.010	-0.314	-0.024	
Data-driven	0.02	0.07	-0.066	0.265	-0.046	0.195	
Conceptual	-0.03	0.13	-0.607	0.062	-0.455	0.035	
Disorganization	0.00	0.06	-0.285	0.075	-0.191	0.045	
Peripheral	0.00	0.03	-0.066	0.041	-0.062	0.029	
Central	-0.01	0.04	-0.103	0.072	-0.068	0.058	
Facial affect \rightarrow Data-driven	0.00	0.01	-0.008	0.045	-0.004	0.035	
Heart rate \rightarrow Data-driven	-0.02	0.05	-0.151	0.049	-0.125	0.039	
Peripheral \rightarrow Data-driven	0.00	0.01	-0.014	0.038	-0.011	0.033	
Central \rightarrow Data-driven	0.00	0.01	-0.034	0.006	-0.032	0.003	
Facial affect \rightarrow Conceptual	0.00	0.02	-0.014	0.050	-0.008	0.050	
Heart rate \rightarrow Conceptual	0.02	0.06	-0.070	0.174	-0.044	0.143	
Peripheral \rightarrow Conceptual	0.00	0.02	-0.058	0.050	-0.037	0.028	
$Central \rightarrow Conceptual$	0.00	0.01	-0.008	0.045	-0.004	0.039	
Facial affect \rightarrow Disorganization	0.00	0.01	-0.058	0.004	-0.058	0.003	
Heart rate \rightarrow Disorganization	0.00	0.02	-0.013	0.083	-0.008	0.051	
Data-driven \rightarrow Disorganization	0.00	0.02	-0.006	0.140	-0.002	0.104	
Conceptual \rightarrow Disorganization	0.00	0.02	-0.006	0.204	-0.002	0.171	
Peripheral \rightarrow Disorganization	0.00	0.01	-0.017	0.012	-0.014	0.008	
Central \rightarrow Disorganization	0.00	0.01	-0.011	0.012	-0.007	0.003	
Facial affect \rightarrow Data-driven \rightarrow	0.00	0.01	-0.011	0.029	-0.007	0.021	
Disorganization	0.00	0.00	-0.001	0.014	0.000	0.013	
Heart rate \rightarrow Data-driven \rightarrow Disorganization		0.00	-0.050	0.002	-0.050	0.000	
Peripheral \rightarrow Data-driven \rightarrow Disorganization		0.00	-0.002	0.002	-0.001	0.009	
Central \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.010	0.001	-0.010	0.001	
Facial affect \rightarrow Conceptual \rightarrow	0.00	0.00	-0.010	0.001	-0.010	0.001	
Disorganization \rightarrow	0.00	0.00	-0.020	0.001	-0.020	0.000	
Heart rate \rightarrow Conceptual \rightarrow Disorganization		0.00	-0.020	0.001	-0.020	0.000	
Peripheral \rightarrow Conceptual \rightarrow Disorganization		0.03	-0.100	0.003	-0.082	0.001	
$Central \rightarrow Conceptual \rightarrow Disorganization$	0.00	0.00	-0.037	0.001	-0.013	0.000	

			95%	6 CI	90%	6 CI
E	stimate	SE	Lower	Upper	Lower	Upper
pecific Indirect Effects: High Dose (v. Placebo)						
Facial affect	0.00	0.02	-0.023	0.065	-0.014	0.061
Heart rate	-0.19	0.10	-0.369	-0.029	-0.337	-0.057
Data-driven	0.04	0.09	-0.113	0.340	-0.063	0.263
Conceptual	-0.06	0.15	-0.466	0.170	-0.423	0.080
Disorganization	-0.01	0.07	-0.328	0.066	-0.225	0.048
Peripheral	0.01	0.07	-0.109	0.213	-0.070	0.151
Central	0.01	0.04	-0.039	0.114	-0.033	0.099
Facial affect \rightarrow Data-driven	0.00	0.01	-0.009	0.022	-0.005	0.018
Heart rate \rightarrow Data-driven	-0.03	0.06	-0.190	0.056	-0.153	0.040
Peripheral \rightarrow Data-driven	0.01	0.04	-0.016	0.137	-0.009	0.125
Central \rightarrow Data-driven	0.00	0.01	-0.008	0.027	-0.006	0.021
Facial affect \rightarrow Conceptual	0.00	0.02	-0.012	0.079	-0.006	0.066
Heart rate \rightarrow Conceptual	0.03	0.09	-0.089	0.227	-0.082	0.186
Peripheral \rightarrow Conceptual	-0.02	0.07	-0.270	0.024	-0.252	0.013
Central \rightarrow Conceptual	0.00	0.01	-0.062	0.010	-0.039	0.006
Facial affect \rightarrow Disorganization	0.00	0.01	-0.068	0.005	-0.062	0.003
Heart rate \rightarrow Disorganization	0.00	0.02	-0.021	0.100	-0.002	0.073
Data-driven \rightarrow Disorganization	0.01	0.02	-0.003	0.077	0.000	0.059
Conceptual \rightarrow Disorganization	0.01	0.02	-0.007	0.244	-0.004	0.149
Peripheral \rightarrow Disorganization	0.02	0.03	-0.137	0.244	-0.086	0.011
Central \rightarrow Disorganization	0.00	0.03	-0.045	0.009	-0.038	0.005
Facial affect \rightarrow Data-driven \rightarrow	0.00	0.01	-0.045	0.007	-0.050	0.005
Disorganization	0.00	0.00	-0.001	0.008	0.000	0.008
Heart rate \rightarrow Data-driven \rightarrow Disorganization	-0.01	0.00	-0.044	0.008	-0.033	0.003
Peripheral \rightarrow Data-driven \rightarrow Disorganization	0.00	0.02	-0.002	0.000	0.000	0.005
Central \rightarrow Data-driven \rightarrow Disorganization	0.00	0.01	-0.002	0.005	-0.001	0.003
Facial affect \rightarrow Conceptual \rightarrow	0.00	0.00	-0.005	0.000	-0.001	0.005
Disorganization	0.00	0.00	-0.020	0.001	-0.020	0.001
Heart rate \rightarrow Conceptual \rightarrow Disorganization	-0.01	0.03	-0.130	0.001	-0.020	0.001
Peripheral \rightarrow Conceptual \rightarrow Disorganization	0.01	0.03	-0.002	0.128	-0.001	0.002
Central \rightarrow Conceptual \rightarrow Disorganization	0.00	0.00	-0.002	0.011	-0.001	0.010
	0.00	0.00	-0.002	0.011	-0.001	0.010
pecific Indirect Effects: Low Dose (v. Placebo)	\rightarrow Slop	e of Int	rusive M	emories		
Facial affect	-0.01	0.01	-0.034	0.011	-0.031	0.007
Heart rate	-0.01	0.03	-0.109	0.022	-0.075	0.017
Data-driven	0.00	0.02	-0.052	0.039	-0.048	0.025
Conceptual	0.01	0.04	-0.023	0.149	-0.010	0.134
Disorganization	0.00	0.01	-0.018	0.027	-0.012	0.025
Peripheral	0.00	0.01	-0.019	0.020	-0.013	0.015
Central	0.00	0.01	-0.022	0.029	-0.013	0.028
Facial affect \rightarrow Data-driven	0.00	0.00	-0.014	0.006	-0.007	0.004
Heart rate \rightarrow Data-driven	0.00	0.02	-0.034	0.032	-0.024	0.025
Peripheral \rightarrow Data-driven	0.00	0.00	-0.012	0.006	-0.009	0.004
1	0.00	0.00	-0.003	0.017	-0.002	0.008
Central \rightarrow Data-driven				0.005	-0.015	0.002
Central \rightarrow Data-driven Facial affect \rightarrow Conceptual	0.00	0.01	-0.024	0.005		
Facial affect \rightarrow Conceptual	0.00 -0.01	0.01 0.02	-0.024 -0.058			0.012
Facial affect \rightarrow Conceptual Heart rate \rightarrow Conceptual	-0.01	0.02	-0.058	0.026	-0.050	0.012 0.018
Facial affect \rightarrow Conceptual						0.012 0.018 0.001

			95%			6 CI
	stimate	SE	Lower	Upper	Lower	Upper
pecific Indirect Effects: Low Dose (v. Placebo)						
Heart rate \rightarrow Disorganization	0.00	0.00	-0.015	0.004	-0.008	0.002
Data-driven \rightarrow Disorganization	0.00	0.00	-0.023	0.003	-0.016	0.001
$Conceptual \rightarrow Disorganization$	0.00	0.01	-0.049	0.004	-0.029	0.002
Peripheral \rightarrow Disorganization	0.00	0.00	-0.004	0.004	-0.002	0.003
$Central \rightarrow Disorganization$	0.00	0.00	-0.007	0.002	-0.005	0.001
Facial affect \rightarrow Data-driven \rightarrow						
Disorganization	0.00	0.00	-0.006	0.000	-0.004	0.000
Heart rate \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.002	0.013	-0.001	0.009
Peripheral \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.004	0.001	-0.002	0.000
Central \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	0.000	0.001	0.000	0.001
Facial affect \rightarrow Conceptual \rightarrow						
Disorganization	0.00	0.00	0.000	0.007	0.000	0.004
Heart rate \rightarrow Conceptual \rightarrow Disorganization	0.00	0.01	-0.002	0.030	-0.001	0.014
Peripheral \rightarrow Conceptual \rightarrow Disorganization	0.00	0.00	-0.007	0.001	-0.004	0.001
$Central \rightarrow Conceptual \rightarrow Disorganization$	0.00	0.00	-0.001	0.001	0.000	0.001
pecific Indirect Effects: High Dose \rightarrow Slope of	Intrusiv	e Mem	ories			
Facial affect	0.00	0.01	-0.043	0.009	-0.034	0.006
Heart rate	-0.02	0.04	-0.084	0.034	-0.078	0.026
Data-driven	-0.01	0.04	-0.113	0.064	-0.075	0.040
Conceptual	0.01	0.04	-0.028	0.294	-0.016	0.150
Disorganization	0.00	0.00	-0.025	0.032	-0.014	0.024
Peripheral	0.00	0.01	-0.023	0.052	-0.037	0.024
Central	0.00	0.03	-0.047	0.000	-0.025	0.009
Facial affect \rightarrow Data-driven	0.00	0.01	-0.0037	0.014	-0.025	0.009
Heart rate \rightarrow Data-driven	0.00	0.00	-0.039	0.000	-0.032	0.004
Peripheral \rightarrow Data-driven	0.00	0.02	-0.039	0.044	-0.032	0.004
Central \rightarrow Data-driven	0.00	0.01	-0.027	0.017	-0.020	0.008
Facial affect \rightarrow Conceptual	0.00	0.00	-0.020	0.004	-0.003	0.003
Heart rate \rightarrow Conceptual	-0.01	0.01	-0.020	0.004	-0.020	0.002
Peripheral \rightarrow Conceptual	0.01	0.03	-0.074	0.029	-0.007	0.021
Central \rightarrow Conceptual	0.01	0.02	-0.010	0.072	-0.003	0.070
						0.009
Facial affect \rightarrow Disorganization	0.00	0.00	-0.002	0.005	-0.001	
Heart rate \rightarrow Disorganization	0.00	0.01	-0.033	0.005	-0.023	0.003
Data-driven \rightarrow Disorganization	0.00	0.01	-0.059	0.003	-0.020	0.002
Conceptual \rightarrow Disorganization	0.00	0.01	-0.076	0.004	-0.035	0.002
Peripheral \rightarrow Disorganization	0.00	0.01	-0.005	0.017	-0.002	0.011
Central \rightarrow Disorganization	0.00	0.00	-0.003	0.007	-0.002	0.005
Facial affect \rightarrow Data-driven \rightarrow	0.00	0.00	0.005	0.000	0.002	0.000
Disorganization	0.00	0.00	-0.005	0.000	-0.002	0.000
Heart rate \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.002	0.014	-0.002	0.009
Peripheral \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.009	0.001	-0.005	0.000
Central \rightarrow Data-driven \rightarrow Disorganization	0.00	0.00	-0.001	0.000	-0.001	0.000
Facial affect \rightarrow Conceptual \rightarrow	0.00	0.00	0.000	0.000	0.000	0.002
Disorganization	0.00	0.00	0.000	0.003	0.000	0.003
Heart rate \rightarrow Conceptual \rightarrow Disorganization	0.00	0.01	-0.004	0.027	-0.002	0.019
Peripheral \rightarrow Conceptual \rightarrow Disorganization	0.00	0.00	-0.024	0.002	-0.022	0.001
Control Concentual Discussioni-	0.00	0.00	0 002	0.000	0 001	0 000

Note: SE = Standard Error, CI = Confidence Interval. † 90% CI does not include 0, * 95% CI does not include 0.

0.00

0.00

-0.003

0.000

-0.001

Central \rightarrow Conceptual \rightarrow Disorganization

0.000

Results of the full structural model, estimated as proposed, revealed a significant direct effect of the high dose alcohol condition on level of intrusive memories, compared to both the placebo (95% CI: 0.112, 0.850) and the low dose condition (95% CI: 0.010, 0.795), such that those in the high dose condition reported more intrusive memories on Day 1, controlling for the time remaining in the day. No direct effect was found for the low dose condition v. placebo on level of intrusive memories. Similar to the above findings in the latent growth curve model, these findings run counter to expectations (Hypothesis 1a) that, compared to placebo, the low dose condition would lead to more intrusive memories and the high dose condition would lead to lower levels of intrusive memories. Although we also expected alcohol-related differences in rates of recovery (Hypothesis 1b), no direct effects of alcohol condition on slope of intrusive memories were observed.

The second study aim involved investigating the role of alcohol myopia. Specifically, higher levels of alcohol intoxication were expected to interfere with recall of peripheral, but not central details (Hypothesis 2a). Consistent with these expectations, there was a marginal direct effect between the high dose (v. placebo) condition and peripheral details (90% CI: -0.770, -0.013), such that those in the high dose condition reported fewer peripheral details than those in the placebo condition. Also consistent with Hypothesis 2a, no alcohol-related differences were observed in the number of central details recalled.

In turn, alcohol myopia (i.e., reduced recall of peripheral details) was expected to be associated with more data-driven processing and less conceptual processing (Hypothesis 2b). Though there was not a unique effect from peripheral details to datadriven processing, there was a marginally significant unique effect from peripheral details to conceptual processing (90% CI: 0.041, 0.546), such that those who recalled less peripheral information also engaged in less conceptual processing. Although associations were also expected between alcohol myopia (i.e., reduced recall of peripheral details) and greater disorganization in memory for the trauma film (Hypothesis 2c), there were no unique predictors of trauma memory disorganization.

The third aim involved examining the role of stress response, operationalized as film-related changes in heart rate and facial affect. Specifically, the higher levels of intoxication were expected to lead to reduced film-related increases in heart rate and reduced film-related changes in facial affect (Hypothesis 3a). Consistent with expectations, those in the high dose (v. placebo) condition displayed lower levels of film-related heart rate changes (95% CI: -9.835, -3.628), and those in the low dose (v. placebo) condition displayed marginally lower levels of film-related heart rate changes (90% CI: -8.628, -1.267). However, contrary to expectations, no direct effects from alcohol condition to facial affect were observed.

In turn, greater stress responding was expected to be associated with more datadriven processing and less conceptual processing (Hypothesis 3b). Consistent with expectations, greater film-related increases in heart rate were associated with more datadriven processing (95% CI: 0.008, 0.053) and less conceptual processing (95% CI: -0.049, -0.008). However, no unique effects of film-related changes in facial affect were observed on data-driven or conceptual processing. Although stress response (displayed via heart rate and facial affect) was also expected to be associated with more temporal disorganization in trauma memory (Hypothesis 3c), and processing type was expected to be associated with disorganization in trauma memory (Hypothesis 4a), no such effects were found.

Finally, although more data-driven processing and less conceptual processing were expected to be associated with more frequent and persistent intrusive memories (Hypothesis 4b) no such effects were found. Similarly, no effect of trauma memory disorganization was found on intrusive memories (Hypothesis 4c).

With regard to indirect effects, there was only one mechanism that emerged as uniquely significant. Specifically, an indirect effect of high dose (v. placebo) condition led to lower film-related changes in heart rate, which in turn led to fewer intrusive memories (95% CI: -0.369, -0.029). A similar indirect effect was marginally significant for the low dose condition (90% CI: -0.314, -0.024). These indirect effects reveal inconsistent mediation (see MacKinnon, Fairchild, & Fritz, 2007) in that there is a negative indirect effect and positive direct effect. That is, alcohol transmitted a negative effect on intrusive memories via heart rate, but alcohol led to more intrusive memories, overall. Notably, there was also a significant total effect (i.e., sum of all indirect and direct effects) from high dose (v. placebo) to initial level of intrusive memories (95% CI: 0.006, 0.563). In other words, when considering all observed and unobserved mechanisms (including the negative indirect effect via heart rate), the high dose condition (v. placebo) had a significant positive impact on the initial level of intrusive memories.

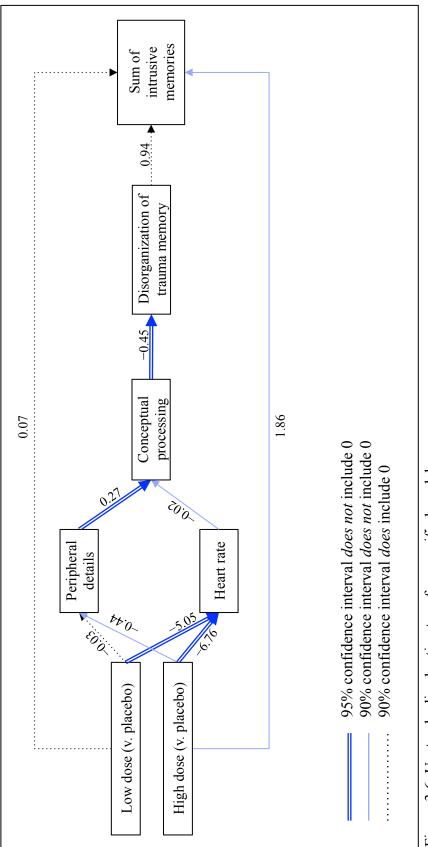
Model trimming and re-specification. To reduce the amount of shared variance between conceptually similar mechanisms represented in the model, the model was trimmed such that only one variable per concept (as defined in Figure 1.1) was retained. With regard to stress response, the change in heart rate was retained, given that it was consistently related to intoxication variables. With regard to alcohol myopia, the peripheral details variable was retained given that central details were not expected (nor shown) to vary by intoxication level. Similarly, with regard to cognitive processing variables, conceptual processing was expected to be most negatively impacted by alcohol (thereby increasing reliance on data-driven processing), and was therefore retained.

Direct paths within the overall model were trimmed in order to evaluate only the most parsimonious conceptual model. In this re-specified model, alcohol was only expected to predict stress response (i.e., heart rate) and alcohol myopia (i.e., peripheral details), in addition to intrusive memory variables. Stress and myopia were only expected to predict conceptual processing, which in turn was expected to predict disorganization, and in turn, disorganization of the trauma memory was expected to predict intrusive memories.

In addition, given that the rate individuals recovered from intrusive memories (i.e., slope of intrusive memories) was not significantly associated with any study variables, the dependent variables were changed from a latent growth curve model representing intrusive memories, to a single sum score representing number of intrusive memories reported during the 8 days (the one participant who withdrew from the study during this time was considered to have missing data for the intrusive memory sum). The number of diaries completed was not significantly associated with total number of intrusive memories reported, r = -.07, p = .498, and was therefore not included as a covariate.

The results of this revised model, as estimated with maximum likelihood and 5000 bootstrap resamples, are displayed in Figure 3.6 and Table 3.10. Unstandardized

estimates are presented for consistency. The fit of this model was excellent; the χ^2 test of absolute model fit of this revised model was not significant, $\gamma^2(10) = 11.932$, p = .290, suggesting this model fit no worse than the best-fitting model. Good fit is also reflected in other fit indices, CFI = .944, TLI = .889, RMSEA = .044 (90% CI = .000, .123). Though no indirect paths were significant as indicated by 95% confidence intervals, 90% confidence intervals did reveal some indirect effects that could reach significance with a larger sample. Specifically, there was a marginal indirect effect from the low dose (v. placebo) to intrusive memories via heart rate, conceptual processing, and disorganization of the trauma memory (90% CI: -0.176, -0.002). Specifically, compared to those in the placebo condition, participants who received a low dose of alcohol experienced a smaller film-related increase in heart rate, which in turn allowed for more conceptual processing, less disorganization of the trauma memory, and finally, fewer intrusive memories. A similar, marginally significant indirect effect was observed for the high dose (v. placebo) condition (90% CI: -0.190, -0.004). In addition, there was a marginally significant indirect effect for the high dose (but not low dose) condition due to alcohol myopia. That is, those in high dose condition (v. placebo) recalled fewer peripheral details, which was associated with less conceptual processing, which was associated with more temporal disorganization of the trauma memory, and in turn, more frequent associations (90% CI: 0.003, 0.222). This indirect effect was also present for the model-implied comparison between high and low dose conditions (90% CI: 0.001, 0.208).





Unstandardized Estimates for Re-Specified Model							
			959	95% CI	90% CI	CI	
	Estimate	SE	Lower	Upper	Lower	Upper	
Path Coefficients							
Y X							
Sum of Intrusive Memories							
Low dose (v. placebo)	0.07	1.02	-1.825	2.198	-1.519	1.843	
High dose (v. placebo)	1.86	1.04	-0.138	3.863	0.199	3.577	÷
High dose (v. low dose; model-implied)	1.79	1.23	-0.736	4.108	-0.298	3.743	
Disorganization of trauma memory	0.94	0.65	-0.261	2.297	-0.090	2.036	
Disorganization of Trauma Memory							
Conceptual Processing	-0.45	0.15	-0.742	-0.165	-0.696	-0.206	*
Conceptual Processing							
Peripheral details	0.27	0.10	0.082	0.473	0.112	0.441	*
Heart rate	-0.02	0.01	-0.033	0.000	-0.030	-0.003	*-
Peripheral Details							
Low dose (v. placebo)	-0.03	0.22	-0.450	0.392	-0.390	0.334	
High dose (v. placebo)	-0.44	0.24	-0.900	0.009	-0.827	-0.064	*
Change in Heart Rate							
Low dose (v. placebo)	-5.05	2.40	-9.871	-0.558	-9.128	-1.246	*
High dose (v. placebo)	-6.76	1.64	-9.969	-3.560	-9.450	-4.075	*
Intercepts							
Sum of intrusive memories	5.45	0.55	4.497	6.661	4.655	6.439	*
Disorganization of trauma memory (factor score)	-0.06	0.09	-0.229	0.135	-0.203	0.106	
Conceptual processing (factor score)	0.14	0.12	-0.090	0.367	-0.052	0.331	
Peripheral details (factor score)	0.16	0.17	-0.190	0.470	-0.134	0.421	÷
Heart rate	CC.4	CZ.1	1.197	12.021	175./	000.11	÷

	13.184 26.889 0.340 0.699 0.331 0.635 0.527 0.987 0.527 0.987 37.210 106.298 -1.868 2.177 -0.121 3.875 -0.725 4.153 -0.724 0.015 -0.724 0.132 -0.014 0.132	26.889 14.174 0.699 0.364 0.635 0.356 0.987 0.567 0.298 40.846 0.237 -1.551 3.875 0.203 4.153 -0.203 0.015 -0.196	25.787 0.665 0.613 0.955 99.511
rauma memory ing 0.46 0.74 0.74 0.74 0.74 0.04 0.04 0.04 0.04 0.04 1.87 0.03 0.03 0.03 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00	10		0.665 0.613 0.955 99.511
ing 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.74 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.78 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79 0.79	2		0.613 0.955 99.511
0.74 0.74 0.74 $0.0 \rightarrow \text{ Sum of intrusive memories}$ 0.04 0.04 0.04 1.87 $0.0 \rightarrow \text{ Sum of intrusive memories (model-implied)}$ 1.83 1.83 $0.0 \rightarrow \text{ Sum of intrusive memories (model-implied)}$ 1.83 0.03 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00			0.955 99.511
$61.89 1$ $61.89 1$ $61.89 1$ $61.89 $ $60 \rightarrow Sum of intrusive memories$ 0.04 1.87 $0.0 \rightarrow Sum of intrusive memories (model-implied)$ 1.83 1.83 1.83 1.83 $0.0 \rightarrow Sum of intrusive memories (model-implied)$ 0.03 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.00			99.511
(0) → Sum of intrusive memories 0.04 20) → Sum of intrusive memories 0.04 30) → Sum of intrusive memories (model-implied) 1.83 31.83 1.83 32.90 → Sum of intrusive memories (model-implied) 1.83 33.90 → Sum of intrusive memories (model-implied) 0.03 30.0 → Sum of intrusive memories (model-implied) 0.03 31.83 0.0 → Sum of intrusive memories (model-implied) 0.03 32.91 → Sum of intrusive memories (model-implied) 0.03 33.91 → Sum of intrusive memories (model-implied) 0.03 34.1 ← 0.00			
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$ose) \rightarrow Sum of intrusive memories (model-implied) 1.83 o) \rightarrow Sum of intrusive memories (model-implied) -0.03 oo) \rightarrow Sum of intrusive memories (model-implied) 0.03 ose) \rightarrow Sum of intrusive memories (model-implied) 0.03\overline{ts}$			3.592
00) → Sum of intrusive memories -0.03 20) → Sum of intrusive memories 0.00 0se) → Sum of intrusive memories (model-implied) 0.03 100 0.03 100 → Sum of intrusive memories			3.775
 → Sum of intrusive memories → Sum of intrusive memories 0.00 e) → Sum of intrusive memories (model-implied) 0.03 e) → Sum of intrusive memories 			
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e) → Sum of intrusive memories (model-implied) 0.03 → Sum of intrusive memories 0.00		-	0.097
→ Sum of intrusive memories 1 details → Concentual processing → Disorganization 0.00			0.180
→ Sum of intrusive memories I details → Concentual processing → Disorganization 0.00			
ino → Disoroanization 0.00			
nrocessing -> Disorganization 0.00			
t cupuctat uctans → Conceptuat processing → Disorganization Heart rate → Concentual processing → Disorganization -0.04 0.04	-0.047 0.0	0.091 -0.031	0.072
-			1
High dose (v. placebo) \rightarrow Sum of intrusive memories			
Peripheral details \rightarrow Conceptual processing \rightarrow Disorganization 0.05 0.06	-0.002 0.2	0.263 0.003	0.222
			-0.004
High dose (v. low dose) \rightarrow Sum of intrusive memories (model-implied)			
nization 0.05			0.208
Heart rate \rightarrow Conceptual processing \rightarrow Disorganization -0.01 0.02	-0.111 0.0	0.008 -0.091	0.002

Supplemental analyses. Given the strong associations between heart rate and alcohol conditions, a model with heart rate as a single mediator was estimated with maximum likelihood and 5000 bootstrap resamples. The model was just-identified and therefore had perfect fit. Unstandardized estimates are shown in Figure 3.7 and Table 3.11. Bias-corrected bootstrap confidence intervals revealed indirect effects from alcohol conditions to number of intrusive memories via heart rate. Specifically, compared to placebo, those in the high dose condition exhibited a dampened stress response to the film (i.e., a smaller increase in heart rate), which in turn was associated with fewer intrusive memories (95% CI: -1.921, -0.097). A similar indirect effect was observed for those in the low dose condition compared to placebo (95% CI: -2.121, -0.367). After accounting for heart rate, there remained a significant direct effect from high dose (v. placebo) to intrusive memories (95% CI: 0.918, 5.067), but not for low dose (v. placebo; 90% CI: -0.752, 2.568). As in the full model above, these results reveal inconsistent mediation (MacKinnon et al., 2007): the direct effect from high dose (v. placebo) to intrusive memories is positive, but the indirect effect via heart rate is negative.

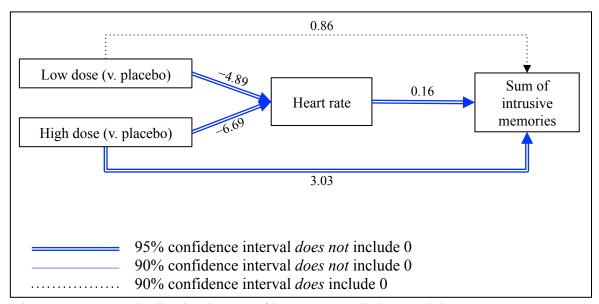


Figure 3.7. Unstandardized estimates of heart rate mediation model.

Estimate S.E Lower Upper Lower Upper 0.86 1.01 -1.082 2.905 -0.752 3.03 1.06 0.918 5.067 1.275 3.03 1.06 0.918 5.067 1.275 3.03 1.06 0.918 5.067 1.275 3.03 1.06 0.918 5.067 1.275 3.03 1.06 0.918 5.067 1.275 3.03 1.06 0.052 0.062 0.033 -4.89 2.38 -9.611 -0.422 -8.913 - -6.69 1.63 -9.857 -3.513 -9.366 - 3.96 0.63 2.764 5.309 2.366 - 9.48 1.24 7.163 11.937 7.469 1 1756 37.157 105.656 40.751 9 61.73 16.52 37.157 105.656 40.751 9 1.95 1.937	EstimateSELowerUppSee (v. placebo)See (v. placebo) 3.03 1.06 0.918 See (v. placebo) 3.03 1.06 0.918 0.062 See (v. low dose; model-implied) 2.17 1.24 -0.430 See (v. low dose; model-implied) 2.17 1.24 -0.430 See (v. low dose; model-implied) 2.17 1.24 -0.430 See (v. placebo) -4.89 2.38 -9.611 -9.857 See (v. placebo) -4.89 2.38 -9.611 -9.857 See (v. placebo) -6.69 1.63 2.764 1 See (v. placebo) -6.69 1.63 2.764 1 See (v. placebo) -5.69 1.63 2.764 1 See (v. placebo) -5.05 5.23 $3.71.57$ 10 See (v. placebo) -5.00 5.00 3.64 11.672 37.157 10 See (v. placebo) -5.00 5.00 1.03 -1.881 1.24 2.167 10 See (v. placebo) -5.00 -5.00 -5.00 -1.03 -1.03 -1.672 2.7157 10 See (v. placebo) -5.00 -5.00 -1.03 -1.03 -1.881 <th>Upp 32 18</th> <th>Lowe</th> <th>Upper</th>	Upp 32 18	Lowe	Upper
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-1.08 0.44 -2.121 -0.367 -1.909 -0.29 0.41 -1.331 0.386 -1.124	n of intrusive memories -0.79 0.46 -1.921			
-0.29 0.41 -1.331 0.386 -1.124	-1.08 0.44 -2.121			
	s (model-implied) -0.29 0.41 -1.331			

Table 3.11 Unstandardized Estimates for Heart Rate Mediation Model 78

Integrative Summary

Although the study aims were specifically evaluated within the context of the full structural model (detailed above), an integrated summary of results across all models is warranted. Table 3.12 shows all study aims and hypotheses, as well as expected and observed associations between variables across all analyses. With respect to Aim 1, Hypothesis 1a was not supported: whereas the low dose condition was expected to produce the most intrusive memories, the high dose condition was instead revealed to be associated with the most intrusive memories across almost all analyses. Although the rate of recovery was also expected to vary by condition (Hypothesis 1b), no analyses revealed significant predictors of linear trends of intrusive memories.

With respect to Aim 2, alcohol myopia was consistently demonstrated. Specifically, compared to those in the placebo condition, those who consumed a high dose of alcohol showed impairment in recall of peripheral information, but no impairment in recall for central information (Hypothesis 2a). Also as expected, no differences between the low dose and placebo conditions were observed for peripheral and central details. In turn, alcohol myopia was expected to be associated with cognitive processing. Hypothesis 2b was partially supported: impairments in peripheral information were consistently related to impairments in conceptual processing, but showed no association with data-driven processing. Recall of peripheral information was also not associated with disorganization of the trauma memory, contrary to Hypothesis 2c.

With respect to Aim 3, stress response was examined. Partial support was found for Hypothesis 3a; high levels of intoxication were associated with a dampened stress response observed via heart rate, but this was not observed in participants' facial affect. Stress response was in turn expected to impact cognitive processing (Hypothesis 3b). This hypothesis was partially supported in that increased stress response (observed as heart rate) was associated with less conceptual processing (in most analyses), but was not associated with data-driven processing. Facial affect showed no associations with datadriven or conceptual processing. Contrary to expectations, there was no association between stress response and disorganization in any analyses (Hypothesis 3c).

With respect to Aim 4, associations among cognitive factors and intrusive memories were examined. Though there was a significant bivariate association between trauma memory disorganization and the data-driven processing mean score, the datadriven processing scale was subsequently revised to provide a more internally consistent representation of the construct. Within the full structural model, there were no unique, direct predictors of trauma memory disorganization. However, the strong correlation between conceptual and data-driven processing may have interfered with detecting significant unique effects of either processing variable on disorganization. Thus, in the revised model, conceptual processing alone was examined as a potential direct predictor of disorganization. Indeed, a significant effect was revealed, such that less conceptual processing was associated with more disorganization in the trauma memory, in support of Hypothesis 4a. Although increased data-driven processing, reduced conceptual processing, and increased disorganization were in turn expected to be associated with more frequent and persistent intrusive memories of the film (Hypotheses 4b and 4c), no such associations were found.

Finally, although no specific indirect effects were hypothesized, indirect effects were examined to determine how these potential mechanisms might impact the effect of

alcohol on intrusive memories. Results of indirect effects across relevant models are summarized in Table 3.13. Although the overall impact of alcohol on intrusive memories was positive, there was a negative indirect effect via stress response. Specifically, relative to the placebo condition, both the low and high dose conditions displayed stress-response dampening (measured as film-related changes in heart rate); greater stress–response dampening was in turn associated with fewer intrusive memories. Cognitive mechanisms also appear to play a role here; a dampened stress response allowed conceptual processing and organization of the memory to be relatively uninterrupted, resulting in fewer intrusive memories.

There was also some support for alcohol myopia as a competing mechanism. Specifically, the high dose (v. placebo) displayed evidence of alcohol myopia, and this narrowing of attention interfered with conceptual processing of the trauma film, which led to greater disorganization of the trauma film memory, and in turn, more intrusive memories. This indirect effect was positive (albeit only marginally significant in the respecified model), and therefore may help to explain why high intoxication increased intrusive memories overall.

Integrative Summary of Results								
	(Hypothesized)	Between- subjects ANOVA	Bivariate correlations	Total IM, controlling for diaries	Latent growth curve model	Full structural model	Re-specified model	Heart rate mediation model
		Tables 3.4, 3.6	Table 3.5	Figure 3.3	Table 3.7, Figure 3.4	Table 3.9, Figure 3.5	Table 3.10, Figure 3.6	Table 3.11, Figure 3.7
Aim 1: Examine the direct effects of peritraumatic	peritraumatic	alcohol intox	alcohol intoxication on intrusive memories.	ve memories.				
Hypothesis A: Compared to a placebo condition,]	bo condition,	low levels of ;	alcohol intoxicat	ion will lead to h	higher levels of	intrusive mei	nories following	low levels of alcohol intoxication will lead to higher levels of intrusive memories following the trauma film,
while high levels of intoxication will lead to lower levels of intrusive memories following the trauma film.	l lead to lowe	er levels of intr	usive memories	following the trai	uma film.			
• Low v. placebo \rightarrow IM frequency	cy (+)	us		ns	su	ns	ns	ns
 High v. placebo → IM frequency 	Ĩ	ns		+	marg. +	+	marg. +	+
• High v. low \rightarrow IM frequency	(-)	su		ns	÷	÷	ns	ns
Hypothesis B: Although intrusions will decrease over time for all participants. reductions will be slower for those with low levels of alcohol intoxication and	will decrease	over time for ;	all participants. r	eductions will be	slower for the	ose with low l	evels of alcohol	intoxication and
faster for those with high levels of intoxication.	ntoxication.							
• Low v. placebo \rightarrow IM slope	+				ns	ns		
• High v. placebo \rightarrow IM slope	Ĺ				ns	ns		
• High v. low \rightarrow IM slope	Ĺ				ns	su		
Aim 2: Investigate the impact of alcohol myopia on cognitive factors.	ohol myopia c	m cognitive fac	ctors.					
Hypothesis A: Greater alcohol intoxication will be associated with reduced memory for peripheral details of the trauma film (reflecting alcohol myopia); memory for central details of the film will not vary by intoxication level	xication will h m will not var	be associated w v bv intoxicati	vith reduced men	nory for peripher	al details of th	e trauma film	(reflecting alco	hol myopia);
• I ow y nlaceho — Derinheral	(nc)	24				5 U	54	
• High v. placebo \rightarrow Peripheral	(cm) (-					marg. –	marg. –	
• Low v. placebo \rightarrow Central	(us)	ns				ns)	
• High v. placebo \rightarrow Central	(su)	SU				ns		
Hypothesis B: Greater alcohol myopia will be associated with increased processing of sensory information (i.e., data-driven processing) in the trauma film and	pia will be as	sociated with i	ncreased process	sing of sensory in	Iformation (i.e.	., data-driven	processing) in th	he trauma film and
• Deriveral - Note deriver (-)	the trauma fil	m (1.e., concer	otual processing)			54		
	23		SII -			eII	-	
 Peripheral → Conceptual 	(+)		÷			marg. +	÷	
Hypothesis C: Greater alcohol myopia will be associated with more temporal disorganization in memory for the trauma film.	pia will be as	sociated with r	more temporal di	sorganization in	memory for th	ie trauma film		
• Peripheral \rightarrow Disorganization	-		us			su		

Table 3.12 cont.								
(Hypot	(Hypothesized)	Between- subjects ANOVA	Bivariate correlations	Total IM, controlling for diaries	Latent growth curve model	Full structural model	Re-specified model	Heart rate mediation model
		Tables 3.4, 3.6	Table 3.5	Figure 3.3	Table 3.7, Figure 3.4	Table 3.9, Figure 3.5	Table 3.10, Figure 3.6	Table 3.11, Figure 3.7
Aim 3: Investigate the impact of stress responding on cognitive factors.	responding	on cognitive f	actors.					
Hypothesis A: High levels of alcohol intoxication	ntoxication	n will result in	stress-response (will result in stress-response dampening as indicated by reduced heart rate and negative facial affect.	licated by redu	iced heart rate	and negative fa	icial affect.
• Low v. placebo \rightarrow Heart rate	(us)	I				marg. –	I	I
 High v. placebo → Heart rate 	-	I				I	I	I
 Low v. placebo → Facial affect 	(us)	ns				ns		
• High v. placebo \rightarrow Facial affect	-	ns				su		
Hypothesis B: Increased stress responding (i.e., heart rate and facial affect) will be associated with increased processing of sensory information (i.e., data-	ling (i.e., h	teart rate and f	acial affect) will	be associated wi	th increased pi	rocessing of se	ensory informati	ion (i.e., data-
driven processing) in the trauma film and reduced a	nd reduced	l ability to mak	e meaning of the	ability to make meaning of the trauma film (i.e., conceptual processing).	., conceptual r	processing).		
• Heart rate \rightarrow Data-driven	+		ns			+		
 Heart rate → Conceptual 	-		ns			I	marg. –	
• Facial affect \rightarrow Data-driven	+		ns			ns		
• Facial affect \rightarrow Conceptual	-		su			ns		
Hypothesis C: Increased stress responding will be	d lliw guill b	e associated wi	ith more tempor:	associated with more temporal disorganization in the memory for the trauma film.	1 in the memor	ry for the trau	na film.	
• Heart rate \rightarrow Disorganization	(+) (+)		su			su		

	ns	ns	
	ns	ns	
0	÷	÷	
	Heart rate \rightarrow Disorganization	Facial affect \rightarrow Disorganization	
,	•	•	

(Hypc	(Hypothesized)	Between- subjects ANOVA	Bivariate correlations	Total IM, controlling for diaries	Latent growth curve model	Full structural model	Re-specified model	Heart rate mediation model
		Tables 3.4, 3.6	Table 3.5	Figure 3.3	Table 3.7, Figure 3.4	Table 3.9, Figure 3.5	Table 3.10, Figure 3.6	Table 3.11, Figure 3.7
Aim 4: Examine the effects of cognitive factors on	e factors or	n intrusive memories.	nories.					
Hypothesis A: Data-driven processing of the trauma film will be positively associated with disorganization in memory for the trauma film.	g of the trau	uma film will b	e positively asso	sciated with disor	ganization in r	nemory for th	e trauma film.	
 Data-driven → Disorganization 	+		+			ns		
 Conceptual → Disorganization 	-		su			ns	I	
Hypothesis B: Increased data-driven processing and reduced conceptual processing will be associated with more frequent and persistent intrusive memories of the film.	processing a	and reduced co	nceptual process	sing will be assoc	iated with mo	re frequent an	d persistent intr	usive memories of
 Data-driven → IM frequency 	+					ns		
 Conceptual → IM frequency 	-					ns		
 Data-driven → IM slope 	+					ns		
 Conceptual → IM slope 	1					ns		

Hypothesis C: Increased temporal disorganization in memories of the trauma film will be associated with more frequent and persistent intrusive memories.

	ns		nemories, depending on the specific	hypothesized association. Gray cells
	ns	ns	Note: Frequency of intrusive memories refers to either the initial level of intrusive memories or total number of intrusive memories, depending on the specific	ttent with expectations. Red text represents significant findings opposite the hypothesized association. Gray cells
•	Disorganization \rightarrow IM frequency (+)	isorganization \rightarrow IM slope (+)	ency of intrusive memories refers to	analyses. Green text represents findings consister
•	 Disoi 	Disor	Note: Frequ	analyses. G

represent associations not examined in a given analysis. IM = Intrusive memories; + = positive association; - = negative association; ns = non-significant association; marg. = marginally significant at <math>p < .10 or 90% confidence interval. ani

integrative summary of specific intartect effects			
	Full structural	Re-specified	Heart rate mediation
	model	model	model
	Table 3.9,	Table 3.10,	Table 3.11,
	Figure 3.5	Figure 3.6	Figure 3.7
Stress Response			
• Low v. placebo \rightarrow Heart rate \rightarrow IM frequency	marg. –		I
• High v. placebo \rightarrow Heart rate \rightarrow IM frequency	I		I
• Low v. placebo \rightarrow Heart rate \rightarrow Conceptual \rightarrow Disorganization \rightarrow IM frequency	ns	marg. –	
• High v. placebo \rightarrow Heart rate \rightarrow Conceptual \rightarrow Disorganization \rightarrow IM frequency	ns	marg. –	
<u>Alcollol Myopia</u> • Tawa njaraha - Darinhami - Concentral - Discretion - IM fractionana	2	20	
- LOW V. Placedo - Feripiteial - Conceptual - Disorganization - Livi itequency	CII	211	
• High v. placebo \rightarrow Peripheral \rightarrow Conceptual \rightarrow Disorganization \rightarrow IM frequency	ns	marg. +	
Note: Frequency of intrusive memories refers to either the initial level of intrusive memories or total number of intrusive memories, depending on the specific	s or total number of int	rusive memories, de	pending on the specific
analyses. Gray cells represent associations not examined in a given analysis. $IM = Intrusive$ memories; $+ =$ positive association; $- =$ negative association; $ns =$	memories; $+ = positiv$	e association; $- = ne$	gative association; ns =
non-significant association; marg. = marginally significant at $p < .10$ or 90% confidence interval.	erval.		

Table 3.13 Integrative Summary of Specific Indirect Effects

CHAPTER 4: DISCUSSION

The primary goal of this study was to examine whether acute alcohol intoxication during an analog trauma exposure would influence the occurrence of subsequent intrusive memories, and if so, why. Within this broader goal, there were four primary study aims. The first aim was to examine the direct effects of peritraumatic alcohol intoxication on intrusive memories. The second and third aims were to investigate the impact of alcohol myopia and stress response (respectively) on cognitive mechanisms. The fourth and last aim was to examine the effect of cognitive mechanisms on intrusive memories. Findings related to each of these aims are discussed in detail below, followed by a discussion of limitations, future directions, and clinical implications.

Descriptive Findings

Before discussing findings related to the primary hypotheses, a review of the descriptive statistics is warranted. Descriptives for alcohol-related variables were consistent with expectations. Specifically, achieved BrAC levels were significantly different between alcohol conditions in the expected directions such that the high dose was greater than the low dose, which was in turn greater than the placebo condition. Achieved BrAC levels were also comparable to other studies in this area. The two prior alcohol administration studies involving a trauma film paradigm (Bisby et al., 2009, 2010) both administered a low dose of .40g/kg, and a high dose of .80 g/kg. Though the current study involved administration of a low dose of .36 g/kg and a high dose of .72 g/kg, achieved alcohol levels were slightly higher in the current study than in Bisby and colleagues' (2009, 2010) studies. Specifically, for the low dose condition, Bisby et al. (2009) reported a mean post-absorption blood alcohol concentration (BAC) of .33 (*SD* =

.09), Bisby et al. (2010) reported a mean post-absorption BAC of .22 (SD = .11), and participants in the current study reached a BAC (as converted from BrAC) of approximately .51 (SD = .12). For the high dose condition, Bisby et al. (2009) reported a BAC of .74 (SD = .17), Bisby et al. (2010) reported a BAC of .48 (SD = .19), and participants in the current study reached a BAC of approximately .91 (SD = .19). Overall, these results suggest the alcohol manipulation was effective in achieving desired BrAC levels.

With regard to participants' subjective experience of drinking alcohol, those in the low and high dose alcohol conditions reported that the drink tasted worse than those in the placebo condition. This finding is expected given that the placebo beverage had a very low concentration of alcohol, whereas the low and high dose beverages had equivalent alcohol concentrations. Such differences in beverage taste ratings have been also been found in past alcohol administration studies (e.g., Watkins et al., 2015), though they have not been reported in any known study involving alcohol administration and an analog trauma exposure. Thus, there is no known evidence that beverage taste ratings impact the occurrence of intrusive memories. Though an aversive beverage taste might have led to more negative affect prior to film viewing, the use of change scores should account for any differences in pre-film affect, whether related to differences in beverage taste or otherwise. A strong aversive beverage taste might also be considered a strong sensory cue preceding the film viewing, but determining how subsequent drinking episodes (and therefore reminders of this sensory cue) might impact intrusive memories is beyond the scope of this study.

Despite differences in beverage taste ratings, nearly all participants (94.9%) believed they drank alcohol. However, perceived intoxication and impairment levels differed by condition, such that those in the high dose condition reported higher levels of subjective intoxication and impairment than those in the low dose condition, who in turn reported higher levels than those in the placebo condition. Such differences are consistent with past alcohol administration designs in which no information is given to participants about which dose they will receive (see Martin & Sayette, 1993). In the absence of experimenter-provided dosage information, participants rely on both external cues (e.g., taste, smell) and interoceptive cues to make judgments about intoxication (Martin & Sayette, 1993). It is therefore unreasonable to expect participants in this study (who drink regularly) to equate subjective experiences between alcohol and placebo conditions (Giancola, Godlaski, & Roth, 2012). Nevertheless, only 51.0% of participants correctly guessed their beverage condition. In all, the alcohol administration appears to have worked as designed; the blinding of beverage condition appears to have created sufficient ambiguity surrounding dosage, allowing the pharmacological effects of alcohol to be examined in the present study.

Further, although participants were blinded to beverage condition, alcohol expectancies (i.e., expectations of the effect of alcohol) were not anticipated to have a systematic influence on the occurrence of intrusive memories. Few participants reported awareness of the study aims, and those who did had inconsistent expectations regarding the manner in which alcohol might impact intrusive memories. This is not surprising given that researchers in this area disagree regarding the direction of alcohol's influence on intrusive memories (see Jaffe et al., 2017).

With regard to the analog trauma exposure, participants reported they found the film distressing (M = 8.42) at a level similar to participants in Weidmann and colleagues' (2009) study (M = 8.16 when transformed to the 0 to 10 scale used in the current study). The distressing nature of this film was also reflected in participants' facial affect, which became more negatively valenced (on average) in response to the violence depicted in the film. Consistent with prior research demonstrating the particularly distressing nature of trauma films with sexual assault (Weidmann et al., 2009), distress ratings in the current study were greater than in the Bisby et al. (2009) study, which employed a trauma film depicting traffic accidents.

To further evaluate the success of the trauma film in inducing stress, film-related increases in heart rate were examined. Other studies that have shown participants a film clip from *Irreversible* depicting a sexual assault (Lass-Hennemann, Peyk, Streb, Holz, & Michael, 2014; Ossewaarde et al., 2011; Schaich, Watkins, & Ehring, 2013; Weidmann et al., 2009) revealed an average increase in heart rate of six to eight beats per minute relative to baseline. Consistent with these findings, participants in the placebo condition of the current study experienced an average increase in heart rate of 9.52 beats per minute. Thus, the stressful nature of the analog trauma exposure in the current study is consistent with past work using this stimulus. Therefore, the trauma analog in the current study should have been similarly successful in inducing temporarily distressing intrusive memories. Though the vast majority of participants (94.9%) reported at least one intrusive memory, slight variations in methods (e.g., only counting distressing or image-based intrusive memories, excluding participants with no intrusive memories, excluding

intrusive memories under the influence of alcohol) make it difficult to compare the exact frequency of intrusive memories reported in this study to prior work.

With regard to cognitive mechanisms, increased data-driven processing was associated with more disorganization in the trauma memory. This finding is consistent with expectations and past research with trauma victims (Halligan et al., 2003) and studies using trauma film paradigms (Halligan et al., 2002). However, contrary to expectations, mean conceptual processing scores were not associated with either data-driven processing or disorganization. This null finding may have been due to insufficient internal reliability of the conceptual processing measure, as the original alpha of this subscale was particularly low (.56). Revised measurement models for data-driven and conceptual processing did reveal that the factors were highly negatively correlated. This finding is consistent with the notion that low-level, sensory-focused (i.e., data-driven) processing is inversely related to the degree of high-level conceptual processing of a stressful experience. Similarly, when measured appropriately through reliability-corrected factor scores, greater conceptual processing was also associated with less disorganization of the trauma memory.

Alcohol and Intrusive Memories

Based on prior studies involving alcohol administration and a trauma film paradigm (Bisby et al., 2009, 2010), an inverted U-shaped effect was hypothesized such that those in the low dose condition (v. placebo) would initially experience more intrusive memories, while those in the high dose condition (v. placebo) would experience fewer intrusive memories (Hypothesis 1a). This hypothesis was not supported. Rather, those in the high dose condition reported the most intrusive memories. Although differences in rate of recovery were expected between conditions (Hypothesis 1b), no such differences were observed.

The finding that high levels of intoxication led to more intrusive memories contrasts with Bisby and colleagues' (2009, 2010) results, and may reflect differences in study methodology. To begin, exact comparisons are difficult to make given idiosyncrasies in Bisby and colleagues' analyses. For example, Bisby et al. (2009) excluded participants from analyses who did not report any intrusive memories; the number of participants in each condition excluded for this reason is not known, and it is therefore not possible to determine if this might have impacted their findings. Additionally, Bisby et al. (2010) excluded intrusive memories that occurred under the influence of alcohol. Though it is plausible that intrusive memories may be more likely during drinking episodes for those who were intoxicated during encoding (i.e., intrusive memories could show state-dependent effects), future work is needed to examine this possibility. For the purposes of this study, participants in any condition may have been reminded of the trauma film and associated laboratory procedures during subsequent drinking episodes; this was therefore considered to be a naturally occurring trauma cue that should not be excluded.

In addition to differences in specific analyses, there are broad methodological differences between the current study and Bisby et al. (2009, 2010) that may have contributed to different findings. As detailed above, participants in the current study achieved slightly higher BrAC levels and reported more distress in response to the trauma film than those in Bisby's study. In this way, the current investigation might have more closely approximated conditions of real traumatic events. Further, Bisby and colleagues'

analog trauma exposure was a film clip showing five horrific and graphic scenes of road traffic accidents. In the current study, the trauma film involved a single unbroken scene of one woman being sexually assaulted. Therefore, while the focus in Bisby's studies appears to be on processing distressing images, the trauma film used here likely evoked a greater need for cognitive processing. Participants in the current study were women in an age range at high risk for sexual assault (Ogle et al., 2013); they were likely cognizant of this risk. In addition, although women who reported a personal history of sexual assault were excluded, most participants (54.1%) reported knowing someone who has been sexually assaulted. Thus, in addition to distressing and violent imagery, this film might have evoked a sense of current threat and increased awareness of distress experienced by real-life sexual assault victims. Though these factors may also be present for a film depicting car accidents, the current film clip also depicted willful acts of violence by one person perpetrated on another, as well as a passerby who chose not to intervene. The interpersonal violence portrayed in this film may challenge pre-existing beliefs (e.g., people are generally good and trustworthy) and therefore require more cognitive processing in order to integrate this new information. These increased cognitive processing demands may have been uniquely impacted by high levels of intoxication in the current study, whereas these cognitive mechanisms might not have been activated by the trauma film used by Bisby et al. (2009, 2010).

Although the experimental studies conducted by Bisby et al. (2009, 2010) were most analogous to the current study, the current findings can also be placed in the context of prior correlational research. As discussed in the introduction, much of the research examining peritraumatic alcohol and PTSD conflates the presence of alcohol use during a sexual assault with perpetrator tactics (e.g., use of force, giving the victims drugs/alcohol with or without her knowledge). While perpetrator tactics certainly have important implications for the development of PTSD, they are not applicable to the central question examined here regarding the pharmacological influence of peritraumatic intoxication. Correlational studies most closely addressing this question suggest that pre-assault alcohol use (without considering differences in level of intoxication) has a negative or non-significant influence on PTSD symptoms (Jaffe et al., 2017; Kaysen et al., 2010; Peter-Hagene & Ullman, 2016). Given that all participants in the current study technically had at least some exposure to a small amount of alcohol, this clear dichotomy would not be expected in the current study. Instead, dose-dependent effects are of interest. Indeed, recent studies of sexual assault victims found that retrospective, subjective reports of peritraumatic intoxication level were associated with more distress, more severe PTSD symptoms, and in particular, more intrusions (Blayney & Read, 2015; Blayney et al., 2016; Jaffe et al., 2017). These findings are consistent with the patterns shown in the current study; the highest levels of intoxication were associated with the most intrusive memories.

This convergence between prior correlational results and the current experimental finding is notable. Sexual assaults involve many unique dynamics that cannot be reproduced in the lab. For example, victims of alcohol-related sexual assault often report high levels of self-blame (e.g., Littleton et al., 2009), which is now considered a symptom of PTSD (APA, 2013). The fact that alcohol was associated with more film-related intrusions in a lab study in which participant self-blame was highly unlikely suggests that

pharmacological effects (rather than expectancies) of alcohol may play a key role in the development of intrusions, and potentially, PTSD.

Alcohol Myopia

In support of Hypothesis 2a, greater alcohol intoxication was associated with impaired recall of peripheral details for the trauma film, but had no effect on recall of central details. This finding suggests that intoxicated participants experienced alcohol myopia. More specifically, consistent with Schreiber Compo et al. (2011), participants who consumed a high dose of alcohol freely recalled less peripheral information than those who consumed a low dose or placebo drink. Also consistent with expectations and the Alcohol Myopia Model (Steele & Josephs, 1990), recall of central information did not vary by alcohol condition, suggesting that alcohol restricts the range of cues that can be attended to, but still allows for encoding important, central information.

Importantly, the free recall task was completed while the participant was still intoxicated, and therefore reflects alcohol-related deficits in cognitive capacity, as well as related attentional narrowing to the most central environmental cues. That is, alcohol myopia was observed when intoxication impacted both encoding and retrieval. Studies using *delayed* recall (and therefore sober retrieval) show no such impairment in recall of peripheral information (Crossland, Kneller, & Wilcock, 2016; Harvey et al., 2013). These findings suggest individuals may successfully encode peripheral information while intoxicated. Although a recognition task would be required to assess individuals' capacity for retrieval while intoxicated, the immediate free recall task used here reveals participants' choice to focus on recalling central (v. peripheral) information when intoxicated. In sum, although the free recall task was adapted from Schreiber Compo et al. (2011) and involved a novel coding system, results were consistent with alcohol myopia.

Alcohol Myopia and Cognitive Mechanisms

In turn, alcohol myopia was expected to be associated with increased data-driven processing and reduced conceptual processing (Hypothesis 2b). In support of this hypothesis, impaired recall of peripheral details was associated with less conceptual processing. Though no associations were shown between number of central details recalled and conceptual processing, this is consistent with expectations; alcohol myopia was only reflected in reduced recall for peripheral information and did not impact recall of central information. In turn, any differences in recall of central details was not considered to be a reflection of myopic processes, and therefore, not expected to influence cognitive processing. Thus, alcohol myopia impaired conceptual processing in the expected manner.

The hypothesis that alcohol myopia would increase data-driven processing was not supported. Specifically, although data-driven and conceptual processing were negatively correlated, and recall of peripheral details was associated with conceptual processing, recall of peripheral details was *not* associated with data-driven processing. Thus, it appears that alcohol myopia did not impact the degree of self-reported datadriven (i.e., sensory-based) processing during the trauma film. However, the observed alcohol myopia-related impairments in conceptual processing still suggest participants had to rely more on data-driven processing (in the absence of conceptual processing) to understand the analog trauma. Though no known work has directly examined how acute alcohol intoxication might impact self-reported processing of stressful stimuli in a lab, these findings overall are consistent with expectations and mechanisms hypothesized in prior work (e.g., Bisby et al., 2009; Jaffe et al., 2017).

In contrast to Hypothesis 2c, alcohol myopia (as observed in number of peripheral details recalled) was not associated with disorganization in the trauma film memory. No known studies have examined the influence of alcohol-related myopic processes on the organization (or lack thereof) of stressful memories. This lack of prior work makes it difficult to contextualize the present findings. Although trauma memories generally are characterized by temporal disorganization (e.g., Ehlers & Clark, 2000; Halligan et al., 2003), it is possible that alcohol-related myopia (at levels of intoxication that can safely be observed in a lab) does not lead to further disorganization by exacerbating underlying processes. However, other direct effects found here suggest that alcohol myopia does impact underlying processes, such as conceptual processing. A more likely explanation for the null finding in the present study is a floor effect with regard to disorganization; endorsement of disorganization in the trauma memory was minimal across conditions, leaving little variance to explain. One possible reason for such a floor effect is that the trauma film used here involved relatively few distinct events to organize. Thus, participants may not have endorsed items such as, "I have trouble remembering the order in which things happened during the film," because there were few things to order. It is also possible that participants had disorganized memories, but could not recognize them as such. Metacognitive judgments (e.g., confidence in memory) do not always reflect reality (e.g., accuracy of memory; Busey, Tunnicliff, Loftus, & Loftus, 2000). In support of this notion, a few participants spontaneously commented that the screenshots shown during the second lab session made them realize their memory for the film was worse

than previously thought. Further research on trauma memory disorganization is needed to shed light on these possibilities.

Alcohol and Stress-Response Dampening

High levels of alcohol intoxication were expected to result in stress-response dampening as indicated by reduced heart rate and negative facial affect (Hypothesis 3a). This hypothesis was partially supported in that stress-response dampening was observed via heart rate but not facial affect. Specifically, consistent with past work involving laboratory-based stress-induction and alcohol administration (Sayette et al., 1991, 1992), the average increase in heart rate from baseline to the trauma film was greater in the placebo condition than the high dose condition. This finding points to reduced reactivity to the trauma film at high levels of intoxication due to the physiological, anxiolytic effects of alcohol, consistent with past work (e.g., Donohue et al., 2007).

Stress-response dampening was expected only at high levels of intoxication; Donohue et al. (2007) suggested that reduced reactivity to aversive stimuli at low levels of intoxication would occur only in the context of complex cognitive demands, most often introduced by distracting tasks concurrent with a stressful stimuli. Though this current study was designed without any distracting tasks (participants were only asked to passively view the trauma film), participants in the low dose condition did demonstrate a dampened stress response. Specifically, the film-related increase in heart rate was lower for those in the low dose condition than those in the placebo condition. (Although the difference between low and high dose conditions was not significant, stress-response dampening generally appeared to increase linearly with greater intoxication, such that the low dose heart rate change fell in between the placebo and high dose conditions.) Though

contrary to expectations, this finding is revealing. Immediately prior to viewing the trauma film, participants were simply asked to sit still, try not to cover their face, and view the film. They were also reminded they could stop the film at any time by pressing the space bar. Although this film-viewing task was expected to involve low cognitive demand, the presence of stress-response dampening at low levels of intoxication suggests that the cognitive complexity involved in viewing and processing a film clip depicting a sexual assault may have been greater than anticipated. For example, during the film viewing, some participants may have considered whether to stop the film, started to cover their face but remembered they were not supposed to, been aware of the possible presence of study personnel behind a two-way mirror, all the while trying to make sense of the distressing film content. These cognitive demands may be more challenging and complex than some experimental tasks (e.g., passively viewing still pictures; Donohue et al., 2007), but are likely less complex than cognitive demands placed on individuals during real traumatic events. As an extension of the demands discussed for participants in the current trauma analog study, victims of a sexual assault would likely have much greater concerns about survival, may weigh their options regarding whether to try to leave the situation or stop the assault, consider the presence of bystanders, and simultaneously attempt to process the event. Thus, the cognitive demands of the current trauma film paradigm appeared to be sufficiently complex to lead to anxiolytic (i.e., stress-reducing) effects of alcohol at low levels of intoxication. Alcohol reduces one's cognitive capacity, and in the presence of these complex cognitive demands, fewer resources are available for an anxious, fear-based reaction. In this way, reactivity is

reduced and the stress-response is dampened. These effects are expected to be greater for real traumatic events, given the increased cognitive demands.

Contrary to expectations and prior research (Sayette et al., 1992), the film-related change in valence of participants' facial affect did not vary by alcohol condition. That is, although facial affect is generally considered to be an important form of nonverbal communication and a valid and universal reflection of internal states (e.g., Collier, 2014), and participants' facial affect did become more negative in response to the violence depicted in the film on average, the degree of change was not influenced by alcohol dose. It is possible that changes in emotional expression (or lack thereof) may not directly reflect underlying changes in emotional experience.

Further, discrepancies between expressed and experienced emotion could have been impacted by alcohol. Alcohol intoxication might have led to increased expression for some individuals. More specifically, despite any dampening in underlying stress response, alcohol myopia might have increased participants' focus on the salient, distressing film, while reducing any (peripheral) concerns about expressing emotion. On the other hand, alcohol intoxication could have reduced emotional expression in some individuals. Because only one participant was in the lab at a time, and participants watched the film near a two-way mirror while being video-recorded, it is possible that some participants were motivated to control their emotional expression. For these individuals, the two-way mirror and webcam may have served as a salient inhibitory cue. Further, some individuals have difficulty accepting their emotions (e.g., Gratz & Roemer, 2004) and tend to suppress their emotional reactions (Gross, 2002). These tendencies could have been exacerbated by a compensatory response to perceived alcohol intoxication (e.g., Newlin, 1986). That is, some individuals might have expected that alcohol would make them emotional, expressive, or loud (Fromme, Stroot, & Kaplan, 1993), and in turn, attempted to minimize and suppress their expression of emotions. However, suppression only reduces expression (not experience) of emotion (Gross, 2002). Thus, lack of facial affect would not reflect lack of internal distress in these participants.

In sum, although alcohol was expected to dampen participants' internal experience of distress, individual differences in factors such as emotion regulation may have interacted with alcohol intoxication to either hinder or enhance outward expressions of affect. In line with this possibility, one lab-based study found that intoxicated participants displayed more facial expressions of anger overall, but intoxicated participants with long-standing tendencies to control their anger displayed fewer facial expressions of anger (Parrott, Zeichner, & Stephens, 2003). Thus, a more nuanced examination of factors that influence expressivity (e.g., emotion regulation strategies), as well as other indicators of underlying emotional experience, may be required to determine alcohol's impact on emotional reactivity. Given that stress-response dampening was not demonstrated via facial affect in this study, facial affect will not be discussed with regard to subsequent hypotheses involving stress-response.

Stress-Response Dampening and Cognitive Mechanisms

Consistent with Hypothesis 3b, a greater stress response (operationalized as a larger film-related increase in heart rate) was associated with more data-driven processing and less conceptual processing. Given that all participants, regardless of alcohol condition, reported the film to be highly distressing, this finding is consistent

with past work demonstrating that heightened arousal in the presence of strong negative emotions can lead to impaired cognitive processing (Bennion et al., 2013), and thus a narrowing of attention to the most central, basic information (Easterbrook, 1959). As a result, participants who experienced a stronger physiological reaction to the film reported engaging in more data-driven processing (which involves a focus on the most basic, sensory information) and less conceptual (i.e., higher-level) processing. However, contrary to Hypothesis 3c, stress response (measured by heart rate) showed no direct association with disorganization in the trauma film memory. Potential limitations to the measure of memory disorganization are discussed above and may have also interfered with significant findings here.

Cognitive Mechanisms and Intrusive Memories

In support of Hypothesis 4a, and consistent with related research (Halligan et al., 2002), less conceptual processing was associated with more disorganization in trauma memory. However, this finding was not consistently demonstrated across analyses, and therefore not very robust. This inconsistency across analyses may be related to problems in the measurement of trauma memory disorganization, which was not associated with many other study variables (e.g., alcohol condition, alcohol myopia, stress response) and displayed floor effects, as discussed above.

Although increased data-driven processing, reduced conceptual processing, and increased disorganization were expected to be associated with more frequent and persistent intrusive memories of the film (Hypotheses 4b and 4c), no such associations were found. This null finding is inconsistent with past work in this area (Ehring et al., 2008; Halligan et al., 2002, 2003) and may reflect underlying problems in measuring

cognitive mechanisms. Though the self-report measures of cognitive processing and memory disorganization have been used in past studies, these measures have not undergone formal psychometric evaluation. In fact, substantial revisions to the measurement models were required in the current study to achieve adequate fit, but even then, did not support the use of aggregate mean scores (which were used in past studies). Thus, it is difficult to know whether these measures accurately represent the cognitive mechanisms they are designed to assess. These cognitive measures have also been used in European samples, but this is the first known administration of such measures with Americans. Future psychometric evaluation is needed to determine if these questionnaires reflect cognitive mechanisms in the same way between groups (i.e., measurement invariance should be examined). In addition, although memory disorganization was thought to be a relatively stable construct (at least during one week), both perceived and actual memory disorganization could plausibly change over time. Memory disorganization was assessed in the second lab session, and therefore after the occurrence of intrusive memories. Though not examined here, the frequency and nature of intrusive memories might have impacted participants' perceptions of the trauma film memory. More objective assessments of cognitive processing and disorganization are therefore needed to clarify the direct impact of cognitive mechanisms on intrusive memories.

Indirect Effects

Overall, high levels of peritraumatic alcohol intoxication led to more intrusive memories when compared to a placebo beverage. At the same time, alcohol-related reductions in stress response led to fewer intrusive memories. Specifically, alcohol dampened participant stress responses (consistent with prior work; Sayette et al., 1991, 1992), which led to fewer intrusive memories (also consistent with prior work; Pole, 2007; Weidmann et al., 2009), both directly and via cognitive mechanisms. These results comport with predictions from nearly a century ago that a substance-induced "decrease of critical self-consciousness and anxiety" (Mott, 1919, p. 224) during a traumatic event indeed protects against traumatic stress. However, alcohol's physiological impact is not limited to physiological stress reduction occurring at high levels of intoxication (Donohue et al., 2007). Instead, alcohol also causes cognitive impairments by narrowing one's attention.

In contrast to the impact of stress-response dampening, alcohol myopia appeared to have an opposite, exacerbating impact on intrusions. Specifically, alcohol-related narrowing of attention intensified cognitive processes unique to traumatic situations (e.g., reduction of cognitive resources and processing), consistent with expectations. Alcohol myopia was the only mechanism examined that helped to explain why, overall, high intoxication was associated with more intrusive memories. Though the effect of stressresponse dampening was more significant than the effect of myopia, stress-response dampening did not explain the overall trend, so therefore cannot be considered to outweigh the impact of myopia at high doses (as was expected). The fact that the indirect effect via myopia was only marginally significant suggests that the exacerbating impact of intoxication on intrusions remains largely unexplained by observed variables in this study. It is possible that the proposed mechanisms do underlie the alcohol-intrusions association, but measurement issues discussed above interfered with the adequate representation of the mechanisms (e.g., valence of affect, cognitive processing, disorganization of trauma memory). Thus, alcohol-related changes in cognition, including narrowing of attention and impairment of cognitive processing, are promising potential mechanisms that should continue to be examined in future studies with multimodal assessments.

On the other hand, there may be mechanisms at work that were not considered in this study. For example, possibly as a result of myopic processes, participants in the high dose condition may have felt more engaged in the film and lost some awareness of the film as a fictional depiction separate from themselves. Such engagement in the film may have made the film feel more like a stressful event personally experienced, even if as a bystander. High levels of intoxication, particularly in an unusual setting, may have also increased perceptions of vulnerability and helplessness. Perceived lack of control during a trauma has been implicated in PTSD (e.g., Foa, Zinbarg, & Rothbaum, 1992), may have influenced participants' experience of the film as a trauma analog. In addition, to reach a level of sobriety, participants in the high dose condition remained in the lab for substantially more time than participants in the other conditions. This may have allowed for an extended period to encode environmental cues related to the study, and thus cues related to the trauma film. With more cues encoded, these participants may have encountered more trauma reminders during the week that reminded participants of the lengthier experience. Finally, participants were allowed to complete intrusive memory reports after experimental procedures in the first session, whether they were waiting in the lab during this time or not. Participants in the high dose condition may have been more likely to report intrusive memories on the first day simply because they had fewer distractions that might prevent them from initiating a report on their phone. These

potential mechanisms should be examined in future work examining the association between acute alcohol intoxication and intrusive memories.

Limitations

There are a number of methodological limitations to the current study. With regard to possible threats to internal validity, the administration of beverages was singleblind. Though this was important for data collection purposes, as it allowed for study personnel to plan for study sessions that could last vastly different lengths of time, it is possible that study personnel could have subtly transmitted knowledge of condition assignment to participants. Moreover, although the placebo manipulation led most participants to believe they consumed alcohol, a few participants maintained that they did not drink alcohol. Though these factors could potentially influence study findings in unexpected ways, they were not expected to interfere with study of the pharmacological effects of alcohol on intrusive memories, especially because participant expectations of how alcohol might impact intrusions was ambiguous.

Further, although the multimodal nature of this study was a strength, some constructs were assessed via a single assessment type. For example, cognitive mechanisms were assessed only through self-report in the current study; future studies would benefit from the use of more comprehensive, psychometrically sound measures of cognitive processing constructs, post-film memory tasks to infer the type of processing (e.g., assessment of viewpoint-dependent memory; Bisby et al., 2010), as well as observational measures of disorganization in trauma narratives. Valence of affect was also assessed by computerized assessment of facial expressions. Automated modeling of facial expressions allows for a high degree of internal consistency, but is also computed without recognition of context. In the current study, for example, participant affect could have been coded as surprised (potentially positive), when human coders might have recognized a participant was looking above the screen to avoid viewing distressing content. Researchers assessing facial affect in future studies might consider converging evidence between both computerized and manualized coding systems, as well as assessing participants' internal experience of emotion.

With regard to external validity, there are necessarily limitations to a laboratorybased analog trauma exposure. For obvious ethical reasons, the trauma film paradigm is designed to induce only temporary distress and is therefore a much more mild stressor than real traumatic events. Although viewing a film under these conditions does not meet DSM-5 criteria for a traumatic event (APA, 2013), there is increased recognition that indirect exposure can lead to significant distress (see James et al., 2016). The trauma film paradigm also focuses only on the development of intrusions; although intrusive memories are a hallmark of PTSD that can exacerbate other PTSD symptoms, they do not capture the full range of possible PTSD symptomatology. Further, due to concerns about extreme film-related distress, individuals with a personal history of sexual abuse or forcible sexual assault were excluded in the current study. Though exclusion of trauma victims is standard practice in most trauma film studies (James et al., 2016), some studies have included victims. Salters-Pedneault and colleagues (Salters-Pedneault, Gentes, & Roemer, 2007; Salters-Pedneault, Vine, Mills, Park, & Litz, 2009) found no differences in reactions to a film clip of a sexual assault (from the 1988 feature film, *The Accused*) between participants who had experienced physical or sexual assault and those who had not. However, because such studies have rarely been conducted with trauma victims, it is

not known how the inclusion of victims might have changed the results of the present study. For example, a trauma film could have served as a trauma reminder in victims, evoking intrusive memories of a past assault and possibly impairing cognitive processing of new film content. In addition, the participants in the current study may differ from those who actually experience sexual assaults in terms of past experiences (e.g., history of child abuse has been associated with adult victimization; Finkelhor, Turner, Hamby, & Ormrod, 2011; Messman-Moore & Long, 2003) and psychopathology (see Ullman & Najdowski, 2011), both of which may have implications for the cognitive processing of the trauma. Overall, caution should be used when generalizing the results of trauma film studies to understand the development of PTSD in trauma victims.

The administration of alcohol in the current study further complicates this picture. Alcohol administration in a laboratory setting devoid of typical contextual cues encountered when drinking (e.g., a bar, other social drinkers) may lead to differences in alcohol expectancies regarding emotional experiences (Wall, Hinson, McKee, & Goldstein, 2001) and physiological tolerance (i.e., environment-dependent tolerance; Dafters & Anderson, 1982). Further, the amount of alcohol that can be safely administered in a lab is limited. Conditions of alcohol-induced blackouts or complete incapacitation that may be present during real traumatic events, including sexual assault (e.g., Kilpatrick et al., 2007), cannot be reproduced in a lab setting. Though the effects of alcohol on processing of traumatic events may depend on dose, with particularly unique effects on memory encoding during blackout (see White, 2003), the effects of alcohol at these highest doses were not examined here. The composition of the current sample should be factored into considerations of generalizability. Though reflective of the local community, the majority of participants identified as White and heterosexual. Given that differences in PTSD prevalence have been demonstrated in ethnoracial (Pole, Gone, & Kulkarni, 2008) and sexual minorities (Roberts, Austin, Corliss, Vandermorris, & Koenen, 2010), caution should be used when generalizing the present laboratory findings to communities at large. Future laboratory studies may consider the potential for minority stress to influence various factors, such as complexity of cognitive demands during the stressful film and experience of distress and intrusions. Finally, the trauma film used in the present study was a French film depicting a White female victim and a White male perpetrator; future studies should examine whether identification with the victim was impacted by differences in racial identity or sexual orientation, and if so, whether this translated to differences in intrusive memories. **Future Directions**

Future research is recommended, not only to address the above methodological limitations, but also to examine new questions evoked by this study. For example, future research is needed to assess whether and how alcohol myopia and stress-response dampening work in concert to impact intrusive memories and other responses to stressful events. Examination of dose-dependent effects on both myopia and stress response is encouraged. In addition, more research is needed to disentangle factors that may influence cognitive complexity of stimuli, as well as the resulting impact on alcoholrelated stress-response dampening. For example, future studies might consider whether cognitive demands during a trauma film paradigm are more complex for participants who personally know someone who experienced a similar traumatic event. As an extension of the current work on peritraumatic intoxication and frequency of intrusive memories, future work should also consider how peritraumatic intoxication might impact the *nature* of intrusive memories (e.g., image or thought, associated distress). The conditions that most often provoke intrusions for alcohol-involved traumas should also be explored. For example, state-dependent effects on memory have been observed for alcohol such that experiences encoded while under the influence of alcohol are retrieved best when also under the influence of alcohol (see Mintzer, 2007). However, it remains unclear whether involuntary or intrusive memories might also display a statedependent effect. Possible pharmacological influences on memory should be considered on such state-dependent effects, in addition to considering drinking as a behavioral cue and trauma reminder. If intrusive memories for traumatic events encoded under the influence of alcohol are more often reported during subsequent drinking episodes, this could have implications for understanding the high co-occurrence of PTSD and alcohol use disorders (e.g., Pietrzak et al., 2011).

Clinical Implications

The present findings have a number of implications for clinicians treating victims of alcohol-involved sexual assault. First, results support the existence of previously proposed cognitive mechanisms underlying PTSD. For example, findings highlight the role of poorly elaborated, sensory-based trauma memories in the development of PTSD. Thus, current findings suggest that standard treatment for PTSD involving elaboration and contextualization of trauma memories (as in Cognitive Processing Therapy; Resick et al., 2017) should similarly be effective in treating PTSD following alcohol-involved sexual assaults. Though levels of alcohol intoxication typically administered in a laboratory are not associated with impairments in accuracy of recall, even for stressful events (e.g., Schreiber Compo et al., 2012), alcohol-related blackouts experienced in natural settings may lead to more serious impairments in memory for an assault. For victims with alcohol-related limitations in trauma memory, engaging in exposure-based treatments focused on the trauma memory may be ill-advised, as this may promote rumination in an effort to recall the assault (Gauntlett-Gilbert, Keegan, & Petrak, 2004). Though limited attention has been paid to the unique challenges of treating PTSD in victims of alcohol-related sexual assaults (for exceptions, see Gauntlett-Gilbert et al., 2004; Padmanabhanunni & Edwards, 2013), current findings suggest the potential utility of targeting memories that are present (e.g., sensory-based memories of the assault, events identified as warning signs) to reduce intrusions and promote recovery from PTSD more broadly.

Second, it is important for clinicians to understand that there are individual differences in possible emotional and physiological reactions to a sexual assault. For example, alcohol was shown to dampen the stress response in the current study, yet high levels of alcohol were still associated with more intrusive memories. Thus, clients who experienced alcohol-related assault may present to treatment with PTSD symptoms while also reporting less fear or anxiety during the assault than might otherwise be expected. This reduced stress response may be a physiological consequence of alcohol intoxication, and should not be considered abnormal. By better understanding such underlying mechanisms, clinicians can help clients normalize and contextualize their experience, reducing self-blame and thereby promoting recovery from PTSD.

Third, current findings highlight the importance of prevention efforts. A high degree of alcohol intoxication not only increases risk for sexual assault (e.g., Abbey et al., 2004), but among those who experience an assault, the presence of alcohol during the assault appears to increase risk for subsequent PTSD. Thus, preventing alcohol-involved sexual assault is a particularly important public health issue. Recently, a web-based intervention targeting both alcohol use and sexual assault showed initial success in reducing alcohol-involved sexual assault (Gilmore, Lewis, & George, 2015). Further development and evaluation of such prevention programs would be crucial to prevent violent victimization and associated distress.

Fourth, findings suggest that a reduced physiological stress response during a traumatic event may protect against intrusive memories and associated PTSD symptoms. Unfortunately, the pharmacological agent studied here (ethanol) is associated with both stress-response dampening (which reduced intrusions) and cognitive impairment (which increased intrusions). However, should there be a way to induce a dampened physiological stress response without the accompanying cognitive impairments, prior to a traumatic event, this might allow for clearer cognitive processing and in turn, fewer intrusive memories. Recently, for example, the use of propranolol (a beta-blocker often used to treat high blood pressure) has been proposed as one such agent. Though a recent meta-analysis suggested that propranolol administration soon after a traumatic event did not reliably prevent PTSD symptoms (Argolo, Cavalcanti-Ribeiro, Netto, & Quarantini, 2015), administering propranolol immediately post-trauma may not be sufficient to impact the peritraumatic processes discussed here. Although the current study was designed to consider implications for alcohol-involved sexual assault, which cannot be

predicted, some jobs (e.g., emergency responders, active-duty military personnel) involve high risk for traumatic exposure. The use of an anxiolytic drug that does not impair cognition on a regular basis might allow for stress-response dampening during traumatic events, when they are experienced. Prior to field tests, this possibility could be examined in a trauma film paradigm. In addition to pharmacological prevention efforts, psychosocial trainings could be used to prepare individuals in high-risk professions for potentially traumatic situations. For example, there is some evidence that Stress Inoculation Training (Meichenbaum, 2007) can lower one's heart rate (Fontana, Hydra, Godfrey, & Cermak, 1999). In addition, biofeedback for heart rate variability has shown some success in treating PTSD (Tan, Wang, & Ginsberg, 2013), and could be explored as a means to prevent peritraumatic arousal.

Conclusions

Although sexual assault often occurs under the influence of alcohol, the specific influence of alcohol on cognitive risk factors for PTSD has rarely been examined. The current study involved comparing the influence of three alcoholic beverage conditions (placebo, low dose, high dose) on cognitive processing of an analog trauma exposure, and subsequent intrusive memories. Though not all hypotheses were supported, novel findings were revealed in this study. Specifically, high levels of intoxication were associated with more intrusive memories. Although only marginally significant, indirect effects point to the competing, yet simultaneous mechanisms underlying this process. On one hand, alcohol myopia exacerbated the attentional narrowing and cognitive impairment typical of highly stressful experiences, thereby increasing intrusive memories. On the other hand, alcohol-related stress-response dampening allowed for the experience to be perceived as less stressful, reducing intrusive memories. Despite these differing indirect effects, the overall effect remained such that higher levels of intoxication were associated with the occurrence of more intrusive memories. These findings suggest that women who are highly intoxicated at the time of a sexual assault may be at increased risk for PTSD symptoms. Thus, preventing alcohol-involved sexual assaults is an important public health issue to be addressed in future clinical and research efforts.

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APPENDIX A: Questionnaires Used

TELEPHONE SCREENING INTERVIEW

Say to participant: "This study is about alcohol, memory, and emotional responding. You will need to be able to participate in a phone screen, two laboratory sessions a week apart, and complete a few questions on your phone once a day between the laboratory sessions in order to complete the study. Your phone screen should take 20 minutes or less. The first laboratory session could take anywhere from 1.5 to 6.5 hours and the second laboratory session will take about 1 hour. The daily questions are expected to take 5-10 minutes per day.

During the phone screen I'm going to ask questions about your alcohol use, and your medical and psychiatric history. There will also be some questions about unwanted sexual experiences, to which I will only ask you to respond yes or no. This information is confidential and will only be seen by study personnel. This information is used to determine whether you are eligible, and whether it is safe for you to participate. Do you have any questions?

Participation in this study is voluntary. You can refuse to participate or withdraw at any time without harming your relationship with the researchers or the University of Nebraska-Lincoln, or in any other way receive a penalty or loss of benefits to which you are otherwise entitled."

"Do you consent to the phone interview?" Y N (if no, stop here and thank them for their time)

"Are you in a private situation and feel you can answer questions freely?" Y N (if no, stop here and ask them when would be a better time to complete the phone screen)

"Now, before we begin, I would like you to rate your level of distress right now on a scale from 1 to 7 where 1 is no distress and 7 is extremely distressed." _____ (If the participant is a 6 or 7, ask if this is their typical level of distress and use active listening. Reschedule the phone interview if needed.)

"What is your age?": _____ (exclude if under 21 or over 30)

"What is your gender?": F_____M____ (exclude if male)

"Where did you hear about our study?" _____

"What is your ethnic background?"_____

"How tall are you and how much do you weigh?" (exclude if participant is under 6 feet tall and weighs more than 250 lbs) (exclude if participant is over 6 feet tall and weighs more than 300 lbs)

"Do you own an Apple or Android smartphone?" Y N		
If Yes: "Is your device running iOS 7.0+ or Android 4.0+?" Y N		
(Android: Settings > About Phone / About Device > Android Version)		
(iPhone: Settings > General > About)		
If No: "Would you be willing/able to install an upgrade?" Y N		
If No:		
"Do you own an iPod Touch or iPad with iOS 7.0+, or a tablet with Android 4.0+?"	Y	Ν
If Yes: "Would you be willing to carry this device with you every night for one week?	"Y	Ν

If Yes: "Would you be willing to carry this device with you every night for one week?" Y

(exclude if participant does not own a smartphone, iPod Touch, or iPad with a supported operating system, or is not willing to carry this device with them regularly)

MEDICAL AND PSYCHIATRIC INFORMATION

(if YES, what are they, how of Medication Name	Dosage	How often take?		
	Dosage			
(Exclude if medication is	s listed on harmful inte	ractions list)		
2) "Can you drink alcohol with	this medication?"		Y	N
(exclude if answer is NO)			
If unknown, continue wi				
them to let them know if before being scheduled.	we can schedule. Medi	cations will need to be r	un by Di	. Grant
belore being seneauleu.				
3) "Is there any reason that yo	u should not drink alcohol,	medical or otherwise?"	Y	Ν
(exclude if YES)				
 <i>"Have you ever had, or do y</i> (seizures, gastric bypass, live 			Y	N
			Y	N
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(Exclude if any psychotic, paranoid, or bipolar disorders, or current major depression)

9) "Have you ever been treated for alcohol or drug problems?" (exclude if yes)	Y	Ν
10) "Have you ever been hospitalized due to alcohol use?" (exclude if yes)	Y	Ν
11) "Do you have a physical disability?" (exclude if necessary)	Y	Ν
12) "Do you have any hearing problems?" (exclude if significant hearing loss)	Y	Ν
13) "Do you have significant visual impairment that is not correctable with glasses or contacts?" (exclude if significant visual impairment)	Y	N
14) "Do you have a cardiac pacemaker?" (exclude if answer is YES)	Y	Ν
15) <i>"Do you have Asthma"</i> [if YES then ask the following questions]	Y	Ν
a) "Have you had an emergency room visit related to asthma in the past year?" (exclude if answer is YES)	Y	Ν
b) "Do you use your inhaler more frequently when drinking?" (exclude if answer is YES)	Y	Ν
c) "Have you used oral steroid treatments for asthma in the past year?" (exclude if answer is YES)	Y	Ν
IF subject reports having asthma but did not say yes to a, b, or c. AND they can regularly tolerate 3-4 alcoholic drinks per occasion. THEN they can participate.		
16) "Do you have any legal restrictions against your drinking (e.g. as a condition of probation or parole)?" (exclude if answer is YES)	Y	N
17) "Are you currently nursing (breastfeeding)?" (exclude if answer is YES)	Y	Ν
18) "Are you currently pregnant?" (exclude if answer is YES)	Y	Ν
19) "How often do you have 3 or more drinks containing alcohol?"		<u> </u>

(e.g. less than monthly, once a month, twice a month, three times a month or more) Person must consume 3 or more drinks at least twice monthly to be eligible.

20) "Immediately after drinking even a small amount of alcohol, have you ever had a reaction that included flushing, blushing, nausea, vomiting, or a feeling of impending doom? Y N

(exclude if answer is YES to a *consistent* response after *any* alcohol)

"Next I'm going to ask you about prior sexual experiences."

Select items from the Computer Assisted Maltreatment Inventory (CAMI)

"It is now commonly known that many people have sexual experiences during childhood or adolescence. These experiences may occur with other children, adolescents, or adults and can include a wide range of behaviors including witnessing sexual activity, touching or being touched in a sexual way, and sexual intercourse.

"We would like to ask you about some of the sexual experiences you may have had before you turned 14. First, I am going to read through a list of sexual experiences. Then, I will ask you a few questions that will require a simple yes or no answer."

- Someone kissed, touched, or fondled your body in a sexual way or you touched or fondled them.
- Someone attempted to have sexual intercourse with you (oral, anal, or vaginal).
- You and another person actually had sexual intercourse (oral, anal, or vaginal).

1. Before you were 14, did ANY of the above ever happen with <u>anyone</u> against your will or when you did not want it to happen?

[**For RAs to read when reviewing responses with participants:

→ Before you were 14, did you have any sexual experiences with anyone against your will or when you did not want to?]

Y N

2. Before you were 14, did ANY of the above ever happen with an immediate family member or other relative? (Please EXCLUDE any voluntary sexual play that may have occurred with a similar age peer – for example "playing doctor.")

[**For RAs to read when reviewing responses with participants:

→ Before you were 14, did you have any sexual experiences with an immediate family member or other relative?]

Y N

3. Before you were 14, did ANY of the above ever happen with anyone who was more than 5 years older than you? (Please EXCLUDE any VOLUNTARY activities that occurred with a dating partner.)

[**For RAs to read when reviewing responses with participants:

 \rightarrow Before you were 14, did you have any sexual experiences with anyone who was more than 5 years older than you?]

Y N

(Exclude if participant endorsed any of the above activities.)

"Now I will ask you some questions about experiences since the age of 14."

Select items from the Sexual Experiences Survey (SES-F)

1. Has anyone used some degree of physical force with you to try and make you engage in kissing and petting (fondling) when you didn't want to?	Y	N
2. Has anyone ever threatened you or used physical force (twisting your arm, holding you down), in an attempt to obtain sexual intercourse but for various reasons the person did not succeed?	Y	Ν
3. Have you ever had oral, anal, or vaginal sex with anyone when you did not want to, because that person threatened to use physical force if you did not cooperate?	Y	N
4. Have you ever had sex with anyone when you did not want to, because that person used physical force?	Y	N

(Exclude if participant endorsed any of the above activities.)

-----[RAs: STOP HERE] ------

"Do you wear contacts or glasses?"	Y	N
"Do you smoke cigarettes?"	Y	N
"Do you have a photo ID displaying your age?"	Y	N

"Could you please rate your level of distress right now on a scale from 1 to 7 where 1 is no distress and 7 is extremely distressed."

(If their self-reported level of distress increased <u>by 3 or more points</u> since their rating at the beginning of the study, talk the participant through strategies for tolerating distress. Assess distress again. Continue until back to pre-experiment levels. If the participant is at immediate risk of harm, contact Dr. David DiLillo immediately.)

If not eligible: "Thank you for taking the time to email me about the study and to answer these questions over the phone. Because this study involves alcohol consumption, this study has a number of very specific exclusion criteria. Many people are not eligible to participate for one reason or another. From the information you have provided, it appears that you are not eligible for this study at this time. Do you have any questions for me?" "Thank you again for your time."

If eligible: "Thank you for your time. Those are all the questions I have for you right now. It appears that you are eligible for the study at this time. Participating in this study would involve attending a laboratory session that may involve drinking alcohol. We would also ask you to watch a film that may be potentially distressing."

- "You will have to undergo a pregnancy test if you decide to participate because the study may include alcohol consumption. In order to complete the pregnancy test you will have to produce a urine sample within the first 1/2 hour of arriving at the laboratory."
- *"Please do not drink alcohol 24 hours before coming in. If you read a positive BAC, we will not run you."*
- "Please refrain from recreational drugs from the time of this interview."
- "Please do not eat 4 hours prior to arriving at the laboratory."
- "Food and water will be provided."
- "Please bring a form of ID displaying your age. If you do not bring a picture ID, you will not be able to participate."
- *"Please also remember to bring your smartphone with you to the study session and make sure you can use it; so if you need, bring a charger."*
- (IF WEAR BOTH CONTACTS & GLASSES:) "If it doesn't matter to you whether you wear contacts or glasses, we encourage you to wear contacts to the study session. If you would be more comfortable in glasses, that's fine too."
- *(IF SMOKES:) "You cannot smoke during the experimental part of the study (a couple hours). After is fine."*
- "If at all possible, we encourage you to walk or have someone drop you off to participate in the study, rather than driving yourself. You should also arrange if at all possible to have someone come and pick you up. If you are not able to find someone, we will provide a taxi for you to get home. If you walk to the building, you will have to arrange to have someone walk or drive you home."
- The lab is located on UNL City Campus in the 501 Building, which is across from the Stadium Garage. I will send you a link to a map, as well as a reminder of this information, before your first session.

"As I mentioned, you qualify for the study. Can I schedule you for an appointment to come to our lab?"

- Settle on a day and time for them to come in
- Schedule both the first and second session. These should be exactly one week apart, though the second session can be at any time in the day.

"Would you prefer to receive a reminder for the session by email or text?"

• Schedule reminder to be sent at least 24 hours prior to the session.

HELPS BRAIN INJURY SCREENING TOOL

Consumer Information: _

Agency/Screener's Information:

н	Have you ever H it your H ead or been H it on the H ead? Note: Prompt client to think about all incidents that may have occurred at any age, even those that did not seem serious: vehicle accidents, falls, assault, abuse, sports, etc. Screen for domestic violence and child abuse, and also for service related injuries. A TBI can also occur from violent shaking of the head, such as being shaken as a baby or child.
E	Were you ever seen in the Emergency room, hospital, or by a doctor because of an injury to your head? Note: Many people are seen for treatment. However, there are those who cannot afford treatment, or who do not think they require medical attention.
L	Did you ever Lose consciousness or experience a period of being dazed and confused because of an injury to your head? Note: People with TBI may not lose consciousness but experience an "alteration of consciousness." This may include feeling dazed, confused, or disoriented at the time of the injury, or being unable to remember the events surrounding the injury.
Ρ	Do you experience any of these P roblems in your daily life since you hit your head? \Box Yes \Box No Note: Ask your client if s/he experiences any of the following problems, and ask when the problem presented. You are

- looking for a combination of two or more problems that were not present prior to the injury.
 - headaches difficulty reading, writing, calculating
 - dizziness
- poor problem solving
- anxiety depression

S Any significant **S**icknesses?

- difficulty performing your job/school work
- □ change in relationships with others
- □ difficulty concentrating
- poor judgment (being fired from job, arrests,

fights)

□ difficulty remembering

Yes No

Note: Traumatic brain injury implies a physical blow to the head, but acquired brain injury may also be caused by medical conditions, such as: brain tumor, meningitis, West Nile virus, stroke, seizures. Also screen for instances of oxygen deprivation such as following a heart attack, carbon monoxide poisoning, near drowning, or near suffocation.

Scoring the HELPS Screening Tool

A HELPS screening is considered positive for a *possible* TBI when the following 3 items are identified: 1.) An event that could have caused a brain injury (yes to H, E or S), and

- 2.) A period of loss of consciousness or altered consciousness after the injury or another indication that the injury was severe (yes to L or E), and
- 3.) The presence of two or more chronic problems listed under P that were not present before the injury.

Note:

- A positive screening is **not sufficient to diagnose TBI** as the reason for current symptoms and difficulties - other possible causes may need to be ruled out
- Some individuals could present exceptions to the screening results, such as people who do have TBI-related problems but answered "no" to some questions
- Consider positive responses within the context of the person's self-report and documentation of altered behavioral and/or cognitive functioning

The original HELPS TBI screening tool was developed by M. Picard, D. Scarisbrick, R. Paluck, 9/91, International Center for the Disabled, TBI-NET, U.S. Department of Education, Rehabilitation Services Administration, Grant #H128A00022. The Helps Tool was updated by project personnel to reflect recent recommendations by the CDC on the diagnosis of TBI. See http://www.cdc.gov/ncipc/pub-res/tbi_toolkit/physicians/mtbi/diagnosis.htm.

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Demographics

- 1. Gender Identity Do you consider yourself to be:
 - 1 = Female
 - 2 = Male
 - 3 = Transgender Female to Male
 - 4 = Transgender Male to Female
 - 5 = Other Please specify _____
- 2. Sexual Orientation Do you consider yourself to be:
 - 1 = Heterosexual / Straight
 - 2 = Lesbian / Gay
 - 3 = Bisexual
 - 4 = Other please specify _____
- 3. What is your age (in years)?
- 4. Are you currently a student?
 - 0 = No
 - 1 = Yes

4a. (If yes:) What best describes your status as a student?

- 1 = Undergraduate student
- 2 =Graduate student
- 4b. Are you a student at the University of Nebraska-Lincoln?
- 1 = Yes
- 0 = No, another college or university, Specify:
- 5. Years of education including kindergarten:
- 6. What is your current legal marital status?
 - 1 = Single
 - 2 = Married (or in a Civil Commitment or Marriage-Like Relationship)
 - 3 =Separated
 - 4 = Divorced
 - 5 = Widowed
- 7. [For those who are not married:] Are you currently in a romantic relationship?
 - 0 = No
 - 1 = Yes
- 8. Are you Latino, Hispanic, or of Spanish origin?
 - 0 = No
 - 1 = Yes

- 9. Which of the following best describes you? (You may check more than one.)
 - 1 = A frican American/Black
 - 2 = American Indian/Native American/Alaskan Native Specify: _ 3 = Asian/Pacific Islander

 - Specify: _____
 - 4 = White5 = Other
 - Specify:
- 10. YOUR average yearly income if you support yourself or your parents' average yearly income if they support you (please check one).

\$0-\$5,000	\$40,000-\$50,000
\$5,000-\$10,000	\$50,000-\$60,000
\$10,000-\$20,000	\$60,000-\$70,000
\$20,000-\$30,000	\$70,000+
\$30,000-\$40,000	

11. What is your occupation?

Alcohol Consumption Questions

1)	1) Taste of beverage: I found the beverage to taste (circle one):											
	a.	(1) Ve	ery unp	leasant	(2) Ur	pleasar	nt (3) Ple	easant	(4) Ve	ery plea	sant	
	b.	(1) Ve	ery bad		(2) Ba	d	(3) G	ood	(4) Ve	ery good	ł	
2)	 How intoxicated do you feel RIGHT NOW on a scale from 0 to 11, with 0 = Not drun at all, 8 = Drunk as I have ever been, and 11 = More drunk than I have ever been. 											
	0	1	2	3	4	5	6	7	8	9	10	11
3)	3) Please rate how much impairment the alcohol you drank has caused on a scale from 0 to 10, where 0 is <i>no impairment</i> , 5 is <i>moderate impairment</i> , and 10 is <i>strong impairment</i> .											
	0	1	2	3	4	5	6	7	8	9	10	
4)	Do yo	u believ	ve you d	consume	ed alcoh	ol? (cir	cle one))				

Yes No

5) [If yes]: Do you believe you consumed a low dose or high dose of alcohol? Low dose (1-2 drinks) High dose (3-4 drinks)

Cognitive Processing Questionnaire, Adapted for a film (Halligan, Clark and Ehlers, 2002)

Below are a number of statements that describe the experiences people sometimes have while watching a stressful film. Rate the extent to which the following statements apply to your experiences while watching the film. There are no right or wrong answers to these questions; we are interested in your personal experience.

Please rate the extent to which each of the following statements applies to your experiences DURING THE FILM by circling the most appropriate number.

WHILE WATCHING THE FILM	Not at all	A little	Moderately	Strongly	Very Strongly
1. My thinking was very clear, not muddled.	1	2	3	4	5
2. My mind was filled with immediate impressions and reactions.*	1	2	3	4	5
3. I could not really take everything in.*	1	2	3	4	5
4. I did not fully understand what is going on.*	1	2	3	4	5
5. I thought ahead.	1	2	3	4	5
6. In my mind, I talked myself through what was happening in the film.	1	2	3	4	5
 It was like a stream of unconnected impressions following each other.* 	1	2	3	4	5
8. I didn't think clearly.*	1	2	3	4	5
9. There were so many sensations I could not put everything together.*	1	2	3	4	5
10. I had a clear impression of how one thing followed from another.	1	2	3	4	5
11. I got confused.*	1	2	3	4	5
12. I thought about what was happening.	1	2	3	4	5
13. My mind was fully occupied with what I saw, heard, smelled and felt.*	1	2	3	4	5
14. I was aware of very immediate things, just going from moment to moment.*	1	2	3	4	5
15. It all became a bit of a blur.*	1	2	3	4	5
16. My mind was racing but my thoughts were disjointed.*	1	2	3	4	5
17. I stayed focused.	1	2	3	4	5

(* Denotes data-driven processing items; other items conceptual processing)

Post-Film Questions

1.	Have ye	ou ever	seen t	his filn	n before	e? No	Yes				
2.	What p	ercenta	ge of t	he Frer	nch dial	ogue di	id you ι	understa	and?	_%	
3.	How di 0 Not at all			you fin 3		-		7	8	9	10 Very Much
4.	How m 0 None at all			2	ı pay to 4			7	8	9	10 Total Attention
5.	How er 0 Not at all	00	2			ene? 5	6	7	8	9	10 Very Much

Free Recall

Now, please take some time to think about what you saw and heard in the other participant room where you watched the film clip. Picture the room and think back to where you were sitting. Try to remember details about the environment, objects in the room, the tasks you completed, and the film clip.

Please take the next 10 minutes to write out all you can remember about what you saw and heard in the other participant room where you watched the film clip. Please write down any detail you can remember, even if it might seem trivial. There is no limit to the amount of text you can provide.

The researcher will notify you when time is up, though you can also choose to end the task and complete the survey at any time.

Coding of Free Recall

Instructions provided to study personnel:

- 1. Code the number of objects/people mentioned. Any mention of an object (regardless of the term used) should be included. For example, if a participant stated there was a "veil" in the room, you would indicate, YES, the "curtain" was mentioned.
- 2. For each object that is mentioned, code the number of accurate details. For each person that is mentioned, code the number of accurate details and actions. Only count each action once (assign it to one subject). Only consider observable details (not judgments or inferences). Determine accuracy by referring to the relevant photos and the film clip.
- 3. For people in the film, locations/possessions should only be considered a detail when not referring to a separate film-object.
- 4. Determine whether there was any inaccurate information reported. Count up and indicate the total number of inaccurate units.
- 5. If you have other questions or are unsure how to code something, make a note to Anna in the text box at the end of the survey. (If you say you were unsure whether to code something, be sure to indicate whether you counted it or not in the response you submitted.)
- 6. Do NOT code any of the following:
 - a. Emotional reactions, judgments, personal anecdotes, or other editorializing
 - b. Tasks completed as part of the study
 - c. Grammatical or spelling errors
 - d. Organization of the response

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RA coding:
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Alcohol, Memories, & Emotions Study RA Coding of Free Recall Task

ROOM

Indicate whether each object is mentioned in the text response. If it is mentioned, indicate the number of accurate details or descriptive pieces of information (i.e., adjectives, physical location) mentioned. Refer to the (paper) photos to see where each item is in the room.

Object	м	entione	17	Number of details	List details
1. Curtain			Yes	uvuno	List details
2. Trash bin					
3. Paint can					
4. Coat rack					
5. Umbrella					
6. Heart rate strap					
7. Cloth					
8. Tripod		$\frac{10}{10}$			
9. Bookcase					
10. Lamp					
		$\frac{10}{10}$ 0			
11. Mug		$\frac{10}{10}$			
12. Highlighter/pens					
13. Book(s)					
14. Student directory			Yes		
15. Clipboard/pad of paper (if both, detail=1)		o o	Yes		
16. Cup		10 O			
17. Anchor		10 O			
18. Phonebook(s)	O N	lo O	Yes		
19. Boombox	ON	lo O	Yes		
20. Paperclip	O N	lo O	Yes		
21. Tree	O N	o ol	Yes		
22. Butterfly	O N	lo O	Yes		
23. (Second) trash bin	O N	O of	Yes		
24. Desk	O N	lo O	Yes		
25. Chair (not pictured)	O N	o ol	Yes		
26. Computer tower	O N	o ol	Yes		
27. Tissues	O N	o ol	Yes		
28. Light(s)	O N	lo O	Yes		
29. Speaker(s)		lo O	Yes		
30. Laptop		lo O			
31. Cord(s)		lo O			
32. Webcam		lo O			
33. Mouse					
34. Mousepad					
35. Intercom					
36. Hand sanitizer		lo O			
37. Mirror					
				number of details for each p	hoto separately (38a-d below). These should
38. Photo(s)	O N	Jo O	Yes		
38a. Elephant photo		$\frac{10}{10}$ $\frac{10}{0}$			
38b. Mountain photo					
38c. Flower photo		$\frac{10}{10}$ $\frac{10}{0}$	Yes		
		$\frac{10}{10}$ $\frac{10}{0}$	Yes		
38d. Bird photo		U U	i es		

Film- People

Indicate whether each PERSON from the film mentioned in the text response. For each person mentioned, indicate the following:

- The number of accurate DETAILS or descriptive pieces of information (e.g., adjectives, clothing ON the person, descriptive features)
- The number of accurate ACTIONS (i.e., verbs) committed by the person

					Number		Number	
Person		Men	tioned	d?	of details	List details	of actions	List actions
1. Main woman	0	No	0	Yes				
2. Perpetrator	0	No	0	Yes				
3. Woman (who provided directions)	0	No	0	Yes				
4. Person (who escaped perpetrator's first attack)	0	No	0	Yes				
5. Bystander	0	No	0	Yes				
6. Other people (walking around on the street)	0	No	0	Yes				

Film- Objects

Indicate whether each object from the film is mentioned in the text response. If it is mentioned, indicate the number of accurate details or descriptive pieces of information (i.e., adjectives, physical location) mentioned.

			Number of	
Object	Mentior	ned?	details	List details
1. Door/building (that the woman left)	O No	O Yes		
2. Plant in building	O No	O Yes		
3. Street	O No	O Yes		
4. Cars/bus/trucks/motorcycle	O No	O Yes		
5. Street lamps	O No	O Yes		
6. Sidewalk	O No	O Yes		
7. Railing	O No	O Yes		
8. "Passage" sign	O No	O Yes		
9. Stairs	O No	O Yes		
10. Tunnel	O No	O Yes		
10a. Ground/floor of the tunnel	O No	O Yes		
10b. Walls of the tunnel	O No	O Yes		
10c. Lighting in the tunnel	O No	O Yes		
11. Trash (in the tunnel)	O No	O Yes		
12. Graffiti	O No	O Yes		
13. Knife	O No	O Yes		
14. Woman's bag	O No	O Yes		
15. Woman's coat	O No	O Yes		
16. Drugs	O No	O Yes		
17. Male nudity	O No	O Yes		
18. France/French language	O No	O Yes		
19. Time of day (night/dark out)	O No	O Yes		

Refer to the paper copies of photos and the film. Was there any inaccurate information?

O No

• Yes; specify:

Number of inaccurate units:

If you have any other notes to Anna about this entry, enter them here:

Daily Diary Instructions

Definition:

• Film-Related Intrusive Memories:

- Thoughts or images <u>related to the film</u> that pop into your mind spontaneously, out of the blue, without a clear reminder and without making an effort to think about something.
- Can take the form of words and phrases ("verbal thoughts")
- Can take the form of mental pictures ("images") in your minds eye, which can include any of the five senses (including sounds)
- Can be fleeting or longer-lasting

Surveys:

• "Single Intrusive Memory"

- Includes questions about a single intrusive memory
- Can be completed at any time

• "Daily Diary"

- Includes questions about memories, alcohol use, and sexual activity in the past day
- Each experience should only be reported once. If you already reported a memory on the "Single Intrusive Memory" survey, this experience should not be reported on the Daily Diary.
- Alarm will sound every day at 8pm; reminder at 10pm
- Window to complete the survey closes at 1am
- Compensation is based on completing this survey
 - \$5 for each Daily Diary completed
 - \$10 bonus for completing all 7 days

Intrusive Memory Diary

Do you recall having any dreams in the past day with content related to the film?

Yes No

Below are some questions about intrusive memories. We define *intrusive memories* as a memory of the film that pops into your mind spontaneously, out of the blue, without deliberately thinking about it. This could be a fleeting memory or more vivid.

What goes through our minds can either take the form of words and phrases ("verbal thoughts"), or it can be like mental pictures ("images") in your minds eye. Although mental "images" often take the form of pictures they can actually include any of the five senses, so you can imagine sounds too. Please record all intrusive memories of the film you viewed.

In addition to any experiences you reported on the "Single Intrusive Memory" survey.... did you have any other film-related intrusive memories today (or since you completed the last diary)?

Yes No

[If Yes:]

1. About how many intrusive memories did you have?

[For each intrusion:]

- 2. Briefly describe the content of the intrusive memory.
- 3. What was the approximate time of the intrusive memory?
- 4. What was happening just prior to the intrusive memory?
- 5. How distressing was the intrusive memory?
 - a. Not at all
 - b. Slightly
 - c. Moderately
 - d. Very
 - e. Extremely
- 6. How would you best describe this intrusive memory?
 - a. A thought
 - b. An image
 - c. A combination of a thought and image
- 7. [If this was an image:] How vivid was this memory from 1 (*no image at all*) to 7 (*as clear as normal vision*)?

Trauma Memory Questionnaire, Adapted for a film (Halligan, Michael, Clark & Ehlers, 2003)

The following questions relate to the ways in which people sometimes describe their MEMORIES OF THE STRESSFUL FILM. Please rate the extent to which these statements apply to YOUR MEMORIES OF THE FILM by circling the appropriate number. If the statement is not true for you, please circle 'not at all.' There are no right and no wrong answers to these questions.

SINCE THE FILM	Not at all	A little	Moderately	Strongly	Very strongly
1. I feel that my memory for the film is incomplete.	0	1	2	3	4
2. There are periods of time during the film that I cannot account for.	0	1	2	3	4
3. I have trouble remembering the order in which things happened during the film.	0	1	2	3	4
4. My memory of the film is muddled.	0	1	2	3	4
5. I cannot get what happened during the film straight in my mind.	0	1	2	3	4
6. Many different things trigger memories of the film.	0	1	2	3	4
7. I experience feelings similar to those I had during the film even when I am not thinking of it.	0	1	2	3	4
8. I am reminded of the film for no apparent reason.	0	1	2	3	4
9. I find myself unexpectedly remembering the film.	0	1	2	3	4
10. My memories of the film consist of vivid images.	0	1	2	3	4
11. I experience strong emotions when remembering the film.	0	1	2	3	4
12. The feelings I had during the film keep coming back to me.	0	1	2	3	4
13. When I remember the film it is like I am watching it again, here and now	0	1	2	3	4

Compute mean scores.

Items 1 to 5: disorganization of trauma memory

Items 6 to 13: intrusion items

APPENDIX B: University of Nebraska – Lincoln Institutional Review Board Approval Letter



July 14, 2014

Anna Jaffe Department of Psychology 238 BURN, UNL, 68588-0308

David DiLillo Department of Psychology 216 BURN, UNL, 68588-0308

IRB Number: 20140714402FB Project ID: 14402 Project Title: Effects of Peritraumatic Alcohol Intoxication on Intrusive Memories

Dear Anna:

This letter is to officially notify you of the approval of your project by the Institutional Review Board (IRB) for the Protection of Human Subjects. It is the Board's opinion that you have provided adequate safeguards for the rights and welfare of the participants in this study based on the information provided. Your proposal is in compliance with this institution's Federal Wide Assurance 00002258 and the DHHS Regulations for the Protection of Human Subjects (45 CFR 46).

Date of Full Board review: 06/05/2014

You are authorized to implement this study as of the Date of Final Approval: 07/14/2014. This approval is Valid Until: 06/04/2015.

We wish to remind you that the principal investigator is responsible for reporting to this Board any of the following events within 48 hours of the event:

* Any serious event (including on-site and off-site adverse events, injuries, side effects, deaths, or other problems) which in the opinion of the local investigator was unanticipated, involved risk to subjects or others, and was possibly related to the research procedures;

* Any serious accidental or unintentional change to the IRB-approved protocol that involves risk or has the potential to recur;

* Any publication in the literature, safety monitoring report, interim result or other finding that indicates an unexpected change to the risk/benefit ratio of the research;

* Any breach in confidentiality or compromise in data privacy related to the subject or others; or

* Any complaint of a subject that indicates an unanticipated risk or that cannot be resolved by the research staff.

For projects which continue beyond one year from the starting date, the IRB will request continuing review and update of the research project. Your study will be due for continuing review as indicated above. The investigator must also advise the Board when this study is finished or discontinued by completing the enclosed Protocol Final Report form and returning it to the Institutional Review Board.

If you have any questions, please contact the IRB office at 472-6965.

Sincerely,

Julia Torquati, Ph.D. Chair for the IRB



APPENDIX C: Informed Consent Form





Department of Psychology

INFORMED CONSENT FORM Alcohol, Memories, and Emotions

Purpose of the Research:

You are invited to participate in a research study investigating how alcohol use affects memories and emotional responding. You were invited to participate because you are a female between the ages of 21 and 30, and are a social drinker.

Some of the questions may ask about sensitive information. Anyone in this study can choose to stop at any time for any reason, opt-out of any portion of the study, or choose not to participate at all.

Procedures:

If you agree to participate, the experimental portion of the study will take about 1.5 hours to complete in the first lab session. However, if you are assigned to the alcohol condition, you must stay at the location of the study until you reach a breath alcohol concentration (BrAC) of .03% and pass a field sobriety test. The average sobriety period will be approximately 5 hours, though this could be longer in some people. You must remain in the lab until two separate readings on the breathalyzer indicate a level of .03% or lower and you pass a field sobriety test.

The study will take place in the 501 Building on the campus of UNL. Today, you will participate in the following procedures described below.

Screening Procedures

First, you will be asked to blow into a breathalyzer in order to ensure sobriety. If you have a positive BrAC test, you will be given an opportunity to reschedule the study for another time. You will then be asked to complete a urine pregnancy test. If your test is positive, then you will not be able to participate in the rest of the study due to the harmful effects of alcohol consumption on fetuses. Also if you are nursing, you will not be able to participate in the study. For safety reasons, if you are less than six feet tall, you must be less than 250 pounds to participate; if you are over six feet tall, you must be less than 300 pounds to participate.

Following the BrAC and pregnancy tests, you will be asked to review the answers of your phone screen. After this, you will be asked some questions about past potential head injuries. If you are eligible, you will be asked to proceed to the next part of the study.

Experimental Procedures

First, you will be asked to complete several questionnaires. These will ask questions about your demographics, past experiences, and your emotions and behaviors.

Next, you will be randomly assigned to a high dose alcohol condition, a low dose alcohol condition, or a no-alcohol condition. If you are in the high dose alcohol condition you will be asked to drink the equivalent of three to four alcoholic drinks. If you are in the low dose alcohol condition, you will be asked to drink the equivalent of one to two alcoholic drinks. In either condition, the exact number of drinks you receive may vary depending on your weight.



Next, you will be asked to place a strap around your chest, which will be used to measure your heart rate. You may be asked to place this under your clothes to ensure accurate assessment of your heart rate. You will be given privacy to do so.

You will also be asked to view a potentially distressing film clip depicting a sexual assault. You can stop this film (and end your participation) at any time using the space bar on the keyboard. Your facial reactions to the film will be video-recorded. These recordings will not be released and your name will not be attached to them.

After completion of the lab session, you will be asked to complete a few questions on your smartphone about memories, substance use, and sexual activities once a day for 7 days. These questions are expected to take 5-10 minutes. You will receive daily reminders to complete these questions.

In one week, you will be asked to return to the lab to respond to additional questions and discuss your reactions to the study. This lab session is expected to last about one hour. You will receive a reminder call, text, or email prior to this session.

Risks and/or Discomforts:

It is possible that you might experience some discomfort or distress when answering questions about your emotions and past experiences, or when viewing the stressful film. You may refuse to answer the questions or stop at any time without penalty and for any reason.

Small to moderate doses of alcohol consumption may sometimes be associated with nausea, vomiting, headache, sadness, and mildly disinhibited behavior. There are also safety risks associated with allowing you to leave a study in a state of intoxication. For these reasons, the following are required for you to consent to the study. Specifically, if you consume alcohol, you agree to:

Stay at the location of the study until you reach a BAC of .03% and pass a field sobriety test.

(initial) You will not be allowed to leave until two separate readings on the breathalyzer indicate a level of .03% or lower and you pass a field sobriety test.

Either have a friend pick you up from the study location or take a taxi that the study will (initial) provide.

Refrain from consumption of alcohol or other drugs for 24 hours and to not operate dangerous (initial) equipment for 12 hours.

Despite all the precautions described above, there is still a small chance that you will have a negative physiological reaction following alcohol consumption. In the case of a non-emergency physiological reaction, you will be assisted in calling the University Health Center if you are a UNL student. If needed, you will be escorted to the campus Health Center. If you are not a UNL student, you will be assisted in calling ______ (please list preferred medical facility). If needed, ______ (please list family member or friend) will be called at ______ (phone number) to escort you to the medical facility listed above. If the physiological reaction is more serious or urgent, medical services will be called using 911. In the unlikely event that medical care is needed, you will be responsible for paying for any medical treatment received.

If you wish to stop your participation in the study at any time, you may do so without harming your relationship with the researcher or with the university. In the event of emotional distress or similar problems resulting from participation in the study, psychological treatment is available at the UNL

Participant Initials



Psychological Consultation Center, (402) 472-2351, 325 Burnett Hall or the University Counseling and Psychological Services for UNL students, 15th & U Streets, (402) 472-7450.

Benefits:

Although there are no known direct benefits to you, this project may provide researchers with a better understanding of how alcohol affects memories and emotional processes.

Confidentiality:

Any identifying information (e.g., names) obtained during this study will be protected and will not be disclosed unless required by law or regulation. The responses you provide will be identified only by a randomly assigned participant identification number. Only study personnel will be able to link your name and the data you provide for the purpose of providing reminder texts and emails. These study personnel have agreed to a professional code of conduct that includes maintaining confidentiality.

Your name and social security number will also be required so that you can be paid for your participation in the study. However, your name and social security number, which will appear on a copy of the participant receipt, will be kept separate from all study data and will be delivered to the UNL Bursar for permanent storage.

Any paper data will be stored in a locked cabinet in the principal investigator's office and will be kept for five years after the study is complete. Any computerized data that you provide will be stored without any identifying information on a password-protected computer. Video recordings will be saved on a password-protected computer until researchers have recorded the relevant information from the videos. Only the researchers listed at the bottom of this form and study personnel will have access to your data. The knowledge gained from this study may be published in scientific journals or presented at scientific meetings, but it will be reported only as aggregate data.

Compensation:

You will receive \$10 per hour for participating in each lab session and \$5 for each daily questionnaire you complete (up to 7). If you complete all 7 daily questionnaires, you will earn a \$10 bonus. If you are excluded from the study after the in-person screening procedures, you will receive \$10. If you withdraw before completing all elements of the study, you will still receive compensation for your time, but not the \$10 bonus.

Opportunity to Ask Questions:

You may ask any questions concerning this research and have those questions answered before agreeing to participate in or during the study. You may contact the investigator, Anna Jaffe, M.A. at anytime (402-603-0244) to ask research-related questions. You may also contact the University of Nebraska – Lincoln Research Compliance Services at 402-472-6929 if you have any questions regarding your rights as a research participant.

Freedom to Withdraw:

Participation in this study is voluntary. You can refuse to participate or withdraw at any time without harming your relationship with the researchers or the University of Nebraska-Lincoln, or in any other way

Participant Initials



receive a penalty or loss of benefits to which you are otherwise entitled.

Consent, Right to Receive a Copy:

You are voluntarily making a decision whether or not to participate in this research study. Your signature certifies that you have decided to participate having read and understood the information presented. You will be given a copy of this consent form to keep.

 $\begin{tabular}{ll} \hline $(initial)$ Initial here if you agree to be video-recorded while viewing the stressful film. \end{tabular}$

Signature of Participant:

Name of Research Participant

Signature of Research Participant

Date

Date

Signature of Person Obtaining Consent

In my judgment, the participant is voluntarily and knowingly giving informed consent to participate in this research study.

Signature of Person Obtaining Consent

Names and Phone Numbers of Investigators Anna Jaffe, M.A., Principal Investigator David DiLillo, Ph.D., Secondary Investigator

(402) 603-0244 (402) 472-3297

Participant Initials

APPENDIX D: Debriefing Form

Alcohol, Memories, and Emotions Debriefing Form

Thank you for participating in the Alcohol, Memories, and Emotions study!

The goal of this study was to examine the influence of alcohol consumption on your emotional and cognitive responses to a stressful film, and how this influenced the formation of memories for this film.

Prior to viewing the stressful film, you either received an alcoholic beverage equivalent to approximately 3 to 4 mixed drinks at a bar, an alcoholic beverage equivalent to 1 to 2 mixed drinks, or a placebo beverage (the rim of the glass was sprayed with alcohol). Viewing the stressful film was meant to serve as an analogue for a traumatic event. While you were viewing the stressful film, we recorded your heart rate and emotional reactions to the film. We also asked you a number of questions about your experiences of intrusive memories. We are interested in whether the impact of alcohol consumption on intrusive memories is related to one's cognitive and emotional processing of stressful situations.

Because the study depends on people not knowing about the study prior to participating, we ask that you please refrain from discussing the study with others.

If you experience any distress after you leave the study, there are two mental health facilities you can contact: the UNL Psychological Consultation Center, 325 Burnett Hall, telephone (402) 472-2351, which offers affordable services based on a sliding fee scale; and the University Counseling and Psychological Services, 15th & U, telephone (402) 472-7450, which offers three free clinic visits to UNL students enrolled in more than seven credits.

If you have questions and/or concerns about this study, you may contact the principal investigator, Anna Jaffe, at (402) 603-0244 or anna.e.jaffe@gmail.com.

Thank you!