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PTSD and Alcohol Associations Among Trauma-Exposed Women: Critical Questions for the Field

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The article by Langdon and colleagues (2016) provides an excellent review of the complex interrelationships between sexual victimization, PTSD, and alcohol use among women. In many ways their review highlights how the field is like the proverb of the blind men describing the elephant; what you see depends on what part of the elephant you are holding. The man holding the elephant's trunk is going to describe something different than the man holding the elephant's tail. The associations among trauma exposure, PTSD, and alcohol use are similar to this proverb in that the associations are likely to change depending on the timing and context of alcohol use, whether we are assessing women immediately post-trauma or months to years post-trauma after more chronic patterns have likely developed, and based on the methodologies used to examine these associations. In addition, the field must move to testing potential explanatory mechanisms to refine or even move beyond widely accepted but overly broad theories, such as self-medication, to better inform prevention and intervention efforts. In this commentary, we highlight several questions that, if addressed, would help move the field forward.

What is the Impact of Alcohol Use During Trauma?

As highlighted in the review by Langdon and colleagues (2016), it is well documented that alcohol use by the perpetrator, victim, or both is common and that alcohol use in general increases risk for sexual assault. However, it is less well understood *why* alcohol increases vulnerability and *how* alcohol influences outcomes following trauma exposure. The question of why alcohol increases vulnerability for assault is complicated. The “high risk model” discussed by the authors of this review is often posited as a theoretical explanation, suggesting that using substances, including alcohol, increases the likelihood of experiencing trauma. However, the empirical evidence for this theory is limited (Jacobsen, Southwick, & Kosten, 2001) and the theory itself does not speculate on explanatory mechanisms of how alcohol use might impact an individual's experience of potentially traumatic events. For example, the experimental literature demonstrates a relationship between alcohol use and a reduced ability to accurately detect threat cues in potentially dangerous situations and a reduced ability to use effective resistance strategies in escalating assault situations. Similarly alcohol myopia theory suggests that intoxication would narrow one's attentional focus to only the most salient cues, which may preclude attending to potential threat cues by the victim or attending to cues of lack of sexual interest from the perspective of the perpetrator.

Similarly, experimental research on perpetrators has compellingly shown that alcohol is often used as a tactic to obtain unwanted sex, either by impairing a woman's ability to consent or resist or by aiding in selecting a potential assault victim. As the blame for assault lays firmly on the perpetrator, a better understanding of the relationship between alcohol and assault among perpetrators, especially in more naturalistic settings, would help us to develop interventions aimed at decreasing perpetrator behavior. Moreover, research on the contexts where these assaults occur, and on the use of alcohol in these contexts, may help us refine bystander interventions to reduce sexual assault.

Similarly a burgeoning literature suggests that alcohol use during assault has implications for coping and recovery following assault, but the mechanisms of how this unfolds are just becoming understood. For example, animal and experimental studies have demonstrated effects of alcohol on memory with detrimental effects of alcohol on fear conditioning and memory consolidation, two processes highly implicated in theories of PTSD development (Tipps, Raybuck, & Lattal, 2014). Well controlled naturalistic research exploring these processes would be invaluable to advancing our understanding of the complicated interplay between trauma exposure, substance use, and PTSD development. Factors such as dose and timing of alcohol use, developmental stage of the victim, prior alcohol use history, and context of the assault, also need to be examined in terms of their impact on alcohol's effects on trauma recovery.

Alcohol exerts effects on physiological arousal and responding, which is particularly important during the fight or flight response that occurs during trauma exposure. Alcohol has known anxiolytic properties and thus may reduce victim fear responses during a sexual assault. How these decreased fear sensations might impact PTSD development remains unknown. Similarly, alcohol use likely interacts with perceptions of the assault and the meaning that the individual makes of the experience. Lack of congruence between the emotional and cognitive experience of a sexual assault and the decreased perceptions of threat and physiological fear may make it more difficult for victims to make sense of their responses. As discussed in Langston (2016), prospective research has found that alcohol use during the assault predicts lower initial PTSD symptoms but also a slower rate of recovery among individuals who were intoxicated during assault. This suggests that alcohol use during assault interferes with recovery following trauma exposure, although the mechanisms responsible have not yet been identified. Animal studies suggest that alcohol's dose dependent effects on memory and fear learning may be one possible mechanism. In these studies, low doses of alcohol enhance fear learning, suggesting that low levels of alcohol consumed prior to a trauma may increase likelihood of developing PTSD, whereas high doses impair fear learning, suggesting that high levels of alcohol consumed prior to a trauma may have some protective effects (Tipp et al., 2014). However, studies of alcohol's effects on trauma recovery in humans have not examined dose effects on fear conditioning to examine whether there is a differential effect based on level of intoxication during a trauma. Cognitions are another possible mechanism for differential effects of alcohol on sexual assault outcomes. In some studies alcohol use during the assault may actually be protective to the extent that the individual attributes blame for the experience outside themselves and to the specific context and behaviors that occurred during the assault. In this case, when the individual engages in what is termed "behavioral self-blame" they are less likely to develop

PTSD symptoms following assault. Taken together this suggests many unanswered questions about how alcohol use during assault predicts PTSD development and recovery and that memory formation, anxiety reactions, and meaning are all potential important factors worthy of further study.

How Does The Association Between Alcohol Use and PTSD Change Over Time?

When considering the associations between trauma exposure, PTSD symptoms, and alcohol use it is also important to consider how recently the trauma occurred. Different relationships may be observed in acute versus chronic stages of PTSD and alcohol use development. PTSD may influence various stages of alcohol use in different ways including a shift from occasional to regular use, escalation from regular use to dependence, and changes from cessation to relapse. Models may have better explanatory power for different stages of development of comorbidity. Theories such as self-medication and mutual maintenance (discussed by Langston et al 2016) have not generally addressed whether mechanisms such as negative reinforcement or physiological effects of alcohol (e.g. physiological cravings or withdrawal) differ for an individual based on chronicity of the associations.

Little attention to date has been paid to the associations between PTSD and alcohol use in the early stages following trauma exposure. A self-medication model would presume that increases in PTSD and distress would lead to increases in drinking, which would then reduce symptoms through a process of negative reinforcement. However, this model does not explicitly incorporate prior learning, such as drinking patterns, drinking identity, and prior trauma exposure, which would be likely to influence the development of these associations. This also includes research on the impact of alcohol use during trauma exposure on later drinking behavior, which to date is limited. In cross-sectional and prospective studies individuals who report alcohol use during trauma exposure report greater alcohol use post assault particularly to cope with trauma-related symptoms. Although this makes sense from the perspective that individuals who were drinking during the assault are likely prone to drinking behavior independent of trauma exposure, consuming alcohol could be a cue associated with the assault that should therefore be avoided according to widely accepted theories of PTSD development. This does not seem to be the case for many women who have experienced alcohol-involved sexual assaults which is perplexing and so far has not been a topic of empirical study. There is a clear need for further refinement of theoretical models to address this phenomenon, as well as prospective research, including longitudinal and daily-level studies to better understand complex relationships between pre-trauma behaviors, PTSD, and drinking behavior in the immediate aftermath of trauma and the establishment of ongoing maladaptive behavior patterns.

When examining longer-term associations between ongoing PTSD and drinking, PTSD and alcohol use can also serve to maintain each other over time (as discussed in Langsdon et al., 2016). In support of the mutual maintenance model, alcohol consumption may serve as an avoidance behavior that over time prevents the individual with PTSD from recovering. Moreover, withdrawal symptoms themselves may exacerbate or mimic symptoms of PTSD.

For example, anxiety, sleep disturbance, and irritability are commonly associated with alcohol withdrawal and overlap with PTSD. These reciprocal relationships make it difficult to determine the order of effects. Although this mutual maintenance pattern has been supported in longitudinal studies, the support is less clear when examining daily processes among individuals with established chronic patterns of PTSD symptoms and alcohol use. Although there is limited research, mutual maintenance models did not hold up when examining proximal relationships between PTSD and drinking in a sample with chronic PTSD and alcohol dependence (Simpson et al., 2014). It is also unclear conceptually what the expected lag time would be between alcohol use (or cessation of use) and anticipated increase in PTSD symptoms, making empirical study of this relationship complicated.

Self-medication has received more empirical attention than mutual maintenance. Although studies have increasingly examined self-medication in chronic samples, using either daily or longitudinal designs, we do not know how the daily associations between drinking and PTSD change over time. Based on this model, one would expect to see increases in daily-level cravings as these behaviors become more established, and stronger associations between PTSD on a given day and later alcohol use. We would expect to see movement from occasional to more regular use during this stage. Over time, chronic PTSD and long-standing alcohol consumption may become habitual, where drinking in response to PTSD is less prompted by distress and becomes a more automatic behavior. To drive theory refinement and the identification of causal mechanisms the field needs to consider the role of chronicity in predicting patterns of PTSD and drinking, pre-trauma drinking behavior, and prior trauma history, incorporating multiple methods including experimental research, and longitudinal event-level designs. Overall, both drinking behavior and PTSD symptoms are fluid and dynamic behaviors that are likely to change in relationship to time since trauma exposure, a factor that has been underemphasized in both theoretical and empirical models.

What are the Functional Mechanisms Underlying PTSD and Alcohol Associations?

There is a need within the field of trauma, PTSD, and alcohol use to identify potential mechanisms of action. Relatively little research has expanded upon self-medication theory to identify the *how's* and *why's* behind these associations and the research that has been conducted has relied overwhelmingly on self-report measures and cross-sectional designs, with a few notable exceptions. As pointed out by Langdon and colleagues (2016), emotion regulation, distress tolerance, and experiential avoidance are important areas that deserve further study. Further development of motivational models, as well as examining the role of PTSD on reward sensitivity are other areas that are worth investigating. Motivational models, predominantly focusing on the role of coping motives, is one arena that has received proportionally more research focus. In these models coping motives are typically treated as a static trait; however, the salience of drinking motives may actually vary based on internal and external factors such as mood or situational context. This may in turn influence decisions of whether or not to drink and how much to consume. Thus the role of context and focus on within-person variability in drinking motives would help to move the field forward.

Another area that has not been explored is the potential role of reward sensitivity in shaping PTSD and alcohol use behaviors and how this may influence other potential motivational processes (Koob, 2015). PTSD exhibits high levels of co-occurrence with depression, anhedonia, and loss of positive emotional experiences, and thus may be associated with deficits in reward functions. This may reduce the expectations of receiving rewards and result in lower satisfaction upon getting rewards. At the same time, anxiety can increase reactivity to substance cues, in that urges to drink increase, alcohol is perceived as more pleasant, and alcohol is seen as more rewarding following stress or negative mood induction. Individuals may be motivated to drink alcohol not only to cope with distress, but also to enhance positive emotions. Use of substances following higher stress-induced distress is associated with higher perceptions of those substances as more pleasurable or satisfying. Thus, PTSD itself may lead to higher enhancement motives for use (i.e., beliefs that alcohol increases positive mood or experience) which then may lead to escalation in use. It is possible that due to tolerance, the reduction in distress associated with alcohol consumption decreases over time, and thus coping motives may be more salient early in the development of PTSD and alcohol misuse, and enhancement motives may become more critical later in the process. It may also help better explain PTSD's role in predicting propensity to relapse, although this has also not yet been examined.

Concluding Remarks

Relationships between trauma exposure, PTSD and alcohol use are complex and likely vary by both trauma-related factors, such as whether or not alcohol was consumed during the trauma, and by how recently the trauma occurred. In addition, alcohol use patterns vary by what stage of alcohol use someone is in, from first initiation to addiction to cessation. Research on potential mechanisms is evolving but still is relatively narrowly focused on correlational associations rather than conceptually and theoretically based ones. The problem with this narrow focus is that potential mechanisms are also potential means by which to improve treatments and prevention efforts. By failing to better identify functional associations, the research cannot guide treatment development and refinement which is desperately needed for this population.

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