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Posttraumatic Stress Disorder, Drinking to Cope, and Harmful Alcohol Use: A Multivariate Meta-Analysis of the Self-Medication Hypothesis

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Matthew T. Luciano[a,b], Samuel F. Acuff[c], Cecilia C. Olin[c], Rivian K. Lewin[c], Justin C. Strickland[d], Meghan E. McDevitt-Murphy[c], & James G. Murphy[c]

[a]San Diego State University, San Diego, CA 92104, United States

[b]San Diego VA Healthcare System, San Diego, CA, 92161, United States

[c]The University of Memphis, Memphis, TN 38152, United States

[d]Johns Hopkins University, Baltimore, MD 21218, United States

Corresponding Author (Matthew Luciano, PhD):

Address: 3350 La Jolla Village Dr., San Diego, CA 92161, United States

E-mail Address: Matt.T.Luciano@gmail.com

Phone: 607-229-2384

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Abstract

The association between posttraumatic stress disorder (PTSD) and harmful alcohol use has often been explained through the self-medication hypothesis via coping-related drinking motives. However, the magnitude of the indirect effect of PTSD on harmful alcohol use through coping motives is unclear. This paper aggregated this indirect effect using a meta-analytic structural equation modeling approach and explored moderators that influenced the indirect effect. We identified articles from PsycINFO, PubMed/MEDLINE, and PROQUEST (through 22 June 2021) containing measures of (1) PTSD symptoms, (2) coping-related drinking, and (3) harmful alcohol use. Thirty-four studies yielding 69 effect sizes were included (mean N = 387.26 participants; median N = 312; range = 42 – 1,896; aggregate sample n = 15,086). Coping motives mediated the relation between PTSD and harmful alcohol use, accounting for 80% of the variance in the total effect. Moderating variables and evidence of publication bias were also found. Findings suggest that coping-related drinking is a strong mediator in the relation between PTSD and harmful alcohol use, and that the strength of the indirect effect is meaningfully influenced by measurement approach, sample characteristics, and study design. Additional longitudinal and multivariate studies are needed to establish directionality and account for additional variance.

Keywords: Posttraumatic stress disorder; Alcohol; Coping motives; Self-medication; Negative reinforcement; Meta-analysis

General Scientific Summary: Coping-related drinking can help explain the relationship between PTSD and harmful alcohol use. However, the degree to which coping-related drinking explains this relationship depends on the measurement approach, sample characteristics, and study design.

Posttraumatic stress disorder, drinking to cope, and harmful alcohol use: A multivariate meta-analysis of the self-medication hypothesis

Research has demonstrated that strong relations exist between posttraumatic stress disorder (PTSD) and several aspects of harmful alcohol use, including consumption and frequency of use, alcohol-related problems, and alcohol use disorder (Berenz et al., 2017; Debell et al., 2014). Though sample characteristics and measurement approaches can influence prevalence estimates, large national samples have found that approximately 35% (Kessler et al., 1995) to 40% (Kessler et al., 2005) of individuals with PTSD have a comorbid alcohol use disorder (AUD). Compared to those with PTSD or AUD alone, individuals with this comorbidity often have a more severe symptom expression of both conditions (Blanco et al., 2013), poorer levels of psychosocial functioning (Straus et al., 2018), and a higher risk for suicide (Norman et al., 2018). In addition to the psychological ramifications of harmful alcohol use, the societal cost of excessive consumption is significant, including a 249 billion dollar estimate for the U.S. government in 2010 (Sacks et al., 2015). Thus, the pervasiveness, severity, heavy disease burden, and high societal cost of this comorbidity highlights the importance of understanding its etiology. By studying the relationship between PTSD and harmful alcohol use, researchers may work towards developing treatment approaches and prevention efforts for those with this comorbidity.

Self-medication hypothesis

Though several prominent theories exist that explain the relation between PTSD and harmful alcohol use (e.g., the shared vulnerability hypothesis, high-risk hypothesis), the most commonly studied is the self-medication hypothesis (Khantzian, 1997). The self-medication hypothesis suggests that psychopathology is accompanied by aversive

internal states that individuals are motivated to reduce. In behavioral terms, the anxiolytic effect of alcohol use reduces aversive internal experiences associated with PTSD, thus a pattern of alcohol use may develop through negative reinforcement (Blume et al., 2000). Self-medicated drinking is frequently reported among individuals with PTSD, as evidenced by data from the National Epidemiologic Survey on Alcohol and Related Conditions. Findings from this survey indicated that approximately 15% of individuals with PTSD have consumed alcohol to improve their negative mood related to a stress reaction, though this percentage may be understated since individuals are not always aware of what motivates their behavior. Self-medicated drinking was also associated with lower mental health-related quality of life when compared to those who did not self-medicate through alcohol (Leeies et al., 2010). Further, drinking alcohol to improve one's mood is associated with a more hazardous pattern of alcohol use which can lead to dependence (McHugh & McBride, 2020; Crum et al., 2013). Thus, the high prevalence and consequential nature of self-medicated drinking deserves focused attention.

A number of experimental studies provide compelling evidence for the self-medication hypothesis as it relates to PTSD and alcohol use. In one such study, researchers presented either a personalized trauma cue or a neutral imagery cue to individuals with comorbid PTSD and alcohol dependence (as measured by DSM-IV criteria). Findings indicated that those exposed to trauma-related imagery cues reported higher alcohol craving than the neutrally cued group (Coffey et al., 2002). This, along with other experimental studies (for review, see Snelleman et al., 2014), provide empirical support for the relevance of self-medication by demonstrating how trauma reminders lead to increased alcohol craving, which heightens the probability of drinking.

Self-medication is often operationalized with measures of coping-related drinking motives (also known as "drinking-to-cope motives" or "coping motives"). For example, Cooper (1994) developed the Drinking Motives Questionnaire using a four-factor motivational model of drinking. In addition to coping motives, Cooper created scales for enhancement, social, and conformity motives. The coping motives subscale combined questions on negative valance (e.g., negative reinforcement) with internal source (e.g., the internal emotional experience that one hopes to influence by drinking). Research has since shown a fairly consistent relationship between PTSD and coping-related drinking. For example, one study demonstrated that veterans with PTSD reported higher scores on scales measuring drinking to cope when compared to trauma-exposed veterans who did not meet criteria for PTSD (McDevitt-Murphy et al., 2015). Further, moderation analyses exploring the influence of coping motives on the PTSD-alcohol use association suggest that a 1-unit increase in PTSD severity was associated with a 35% increase in alcohol consumption among participants who endorsed a high degree of coping-related drinking. This is compared to a 10% increase in alcohol consumption for those who were low on coping motives (Simpson et al., 2014).

Several studies have suggested that coping motives for drinking may mediate the relationship between PTSD and harmful alcohol use across various trauma-exposed populations. Among veterans, coping motives have been shown to mediate the relation between PTSD and alcohol consequences (Miller et al., 2017), hazardous drinking (McDevitt-Murphy et al., 2017), alcohol consumption, frequency of use, and heavy drinking (McCabe et al., 2019). In college student samples, coping motives also mediated the relation between PTSD and risky drinking (Aarstad-Marin & Boyraz, 2017) and

between alcohol use and problems (Hawn, Bountress et al., 2020). Additionally, Hawn, Bountress and colleagues (2020) found that a more specific measure of "trauma-related drinking to cope" partially mediated the relation between PTSD and alcohol use problems among college students. These analyses have also been conducted in samples of sexual assault survivors (Ullman et al., 2005), firefighters (Tomaka et al., 2017), and adults with a serious mental illness (O'Hare & Sherrer, 2011) and yielded similar findings.

Heterogeneity of self-medication effects

Despite strong support for the self-medication model, studies have reported heterogeneity in the strength of associations between PTSD, alcohol use, and coping motives. Correlations between PTSD severity and coping-related drinking motives range from .21 to .64 (Luciano et al., 2019; O'Hare & Sherrer, 2011). Similarly, there is a wide range of correlations between coping-related drinking and alcohol-related variables in the literature. Such variables include number of alcoholic drinks (r = .21-.42; McCabe et al., 2019; McDevitt-Murphy et al., 2017), frequency of consumption (r = .21-.67; Cloutier et al., 2018; McDevitt-Murphy et al., 2017), alcohol related problems (r =.20-.56; Eddinger et al., 2019; Stappenbeck et al., 2013), and risky drinking (r =.34-.64; Marshall-Berenz et al., 2011; Woolman et al., 2015). There is also variation in the size of the indirect effect for coping motives on the association between PTSD and alcohol use. Hawn, Bountress and colleagues (2020) found that trauma-related drinking to cope accounted for more than 87% of the variability in the relation between PTSD and alcohol use problems among a sample of college students. Meanwhile, drinking to cope accounted for roughly 31% of the variance of the total effect between PTSD symptoms and risky drinking in a different sample of trauma-exposed college students (Aarstad-Martin & Boyraz, 2017)

and between 45 and 52% between PTSD symptoms and problem drinking in a sample of first responders (Tomaka et al., 2017).

These disparate effect sizes are likely influenced by a number of factors. For example research from large epidemiological studies have shown that male sex, White race, and younger age were all factors that influenced self-medicated drinking (Bolton et al., 2006). Outside of demographic characteristics, measurement approaches may also be important moderators that influence the indirect effect of coping in the PTSD-alcohol misuse relation; yet, there has been little research studying their influence. Measurement variables include conceptualization of constructs (e.g., alcohol use consequences vs. consumption vs. hazardous drinking), measurement type (e.g., interview vs. questionnaire), and study design (e.g., longitudinal vs. cross-sectional). Further, study population (e.g., adults vs. adolescents) may also play a meaningful role in explaining variance. Yet, these moderating influences have not been systematically explored.

Though a large number of studies have critically evaluated the self-medication model, conceptual issues may warrant further exploration into this framework. For example, a recent systematic review summarized 24 studies that tested the self-medication framework in samples that measured PTSD and alcohol-related variables (Hawn, Cusack, et al., 2020). The authors concluded that, while there is research support for the self-medication hypothesis, this literature is impeded by variability in study design, inconsistency of statistical covariates, disparate measurement approaches of alcohol use and coping motives, as well as the possibility of a "file drawer effect" in which authors or journals tend not to publish statistically insignificant findings. Further, Hawn and colleagues found that fewer than half of the studies they included tested self-

medication using mediation analyses, despite the fact that it is an explanatory framework that would best be tested with such a statistical approach. If tested, these studies may be associated with little to no indirect effect. Given these limitations in the research, it may be beneficial to aggregate data across multiple studies to better determine the degree to which self-medication influences the PTSD and harmful alcohol use relationship, as well as examine potential bias in the literature. If the aggregated indirect effect is small or non-significant, it may support the need for alternative theoretical models and treatment approaches that focus less on coping skills.

Present Study Aims

This investigation aggregated data from peer-reviewed/published studies and unpublished thesis/dissertations manuscripts to systematically examine the relationship between PTSD, coping-related drinking, and harmful alcohol use. Correlations were extracted from manuscripts and were the analytic basis for conducting path analyses. Given significant heterogeneity in effect sizes in the literature, the first aim of the meta-analysis was to aggregate the effect sizes of available studies that report on all three of these variables. More specifically, we compiled effect sizes for the direct effect of PTSD on harmful alcohol use and the indirect effect of PTSD on harmful alcohol use through coping motives. Based on previous mediation studies and a recent systematic review, we hypothesized that there would be a significant indirect effect through coping-related drinking. The second aim of this study was to explain the heterogeneity of effect sizes by exploring moderators. We examined the potential moderators of population type, sex, race, study drinking criteria, PTSD assessment method, PTSD DSM definition, drinking

variable, and data type. Finally, the third aim of this investigation was to examine evidence of a publication bias and file-drawer effect on this area of study.

Methods

Study Selection and Data Coding

We identified peer-reviewed studies using PubMed and PsycINFO. We also searched abstracts in two PROQUEST Dissertation/Thesis databases (Humanities and Social Sciences Collection; Sciences and Engineering Collection). Searches were conducted to June 22, 2021 using the following Boolean search terms: (PTSD OR trauma OR posttraumatic stress OR posttraumatic stress disorder) AND (coping OR coping motives OR drinking motives OR drinking to cope OR drinking motives questionnaire OR reasons for drinking questionnaire) AND (drinking OR alcohol). We also performed searches replacing PTSD terms with "depression" and "anxiety" in order to find studies where PTSD was not the primary variable but may have been included in the analyses. Reviewers also performed forward searches. Of the three constructs of interest (PTSD, coping, and harmful alcohol use), the coping motives construct is the narrowest and was thought to return the most eligible studies without returning studies that were irrelevant to our manuscript. Thus, several widely cited articles on coping motives were selected for the forward search (Cooper, 1994; Cooper et al., 1995; O'Hare, 1997).

Studies that met the following criteria were included in the meta-analysis: (1) study used human subjects; (2) study was written in English; (3) study reported on PTSD severity, coping motives, and harmful alcohol use; (4) study used a non-experimental approach, or data was collected before the experimental manipulation occurred. No inclusion restrictions were made based on timing, population, or setting.

Two of the authors performed all of the searches. After removing duplicates, the same authors independently screened the remaining titles and abstracts to identify articles that were clearly eligible or clearly ineligible. Discrepancies at this stage were resolved through consensus by both reviewers. Eligible manuscripts were then reviewed in full and studies deemed ineligible were given a brief justification for removal. Finally, the authors checked reference lists of any recent systematic reviews or meta-analyses related to coping motives, PTSD, and harmful alcohol use. Figure 1 depicts the study selection procedure, which followed the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) standards.

Four of the study authors and one masters-level research assistant were involved in coding the final list of studies using a manual developed by the second author. All coders were trained on two cases before breaking into teams of two that would double code studies. Discrepancies in data entry were discussed during a meeting of all coders to ensure accurate transcription of the data. The percent agreement between coders was 88%, which is considered acceptable by conventional standards of interpretation. Agreement was based on all entered data (e.g., correlations, means, and moderation categories). Applying a probability of .5, we arrived at a Kappa of .75, which reflects excellent agreement (Shweta et al., 2015).

If data could not be extracted from an eligible study, then missing data were requested by email to the corresponding authors of the study. Authors who did not reply to the initial email were sent at least two additional reminder emails over the course of several weeks. Unavailable data occurred because corresponding authors declined to provide the data, could not provide data because they no longer had access to the file, or

did not respond to multiple data requests. A subgroup of studies were eligible for inclusion, however, important datapoints (e.g., correlations) from these studies were unavailable upon request.

Meta-analytic Approach

This study employed meta-analytic structural equation modeling (MASEM) using a two-stage structural equation modeling (TSSEM) approach (Cheung, 2019a; Cheung & Chan, 2005). All analyses were conducted on the correlation matrices including the full bivariate relationships between PTSD, coping motives, and harmful drinking variables.

First, violations of independence were addressed by aggregating non-independent correlations from individual studies into a single pooled correlation matrix. This resulted in 35 independent correlation matrices from the 34 included manuscripts (i.e., one manuscript included two independent samples; Cloutier et al., 2018). One study (Jeffery & Mattiko, 2016) had more than double the number of participants (N = 11,751) than any other individual study included herein. When including this study, there was an average sample size of 768 (median = 312; range = 42 – 11,751; aggregate sample = 26,879). Analyses of influential case using a leave-one-out jackknife approach suggested this study served as an influential case. For this reason, we excluded this study from the primary results described below. When included in the omnibus model, few differences were observed. Our result continued to be significant, and only the strength of the PTSD to coping pathway slightly weakened (.41 vs. .42).

Next, an omnibus model was tested using the TSSEM approach. In Stage 1 analyses, a random effects model was used to generate a pooled correlation matrix. Heterogeneity was quantified using the Q statistic and I^2 values. In Stage 2 analyses, the

pooled correlation matrix was used to fit the structural equation model using a weighted least square (WLS) estimation process. The tested model included the following three asymmetric paths: 1) PTSD to harmful drinking, 2) PTSD to coping motives, and 3) coping motives to harmful drinking. The model also included variance terms on the coping motives and harmful alcohol use variables. The indirect pathway from PTSD to harmful alcohol use through coping motives was computed using estimates from the other two path terms (i.e., PTSD to harmful alcohol use and coping motives to harmful alcohol use). Values were summarized using point estimates and the corresponding 95% likelihood-based confidence intervals (as recommended for metaSEM and discussed in Cheung & Cheung 2016).

We performed moderator analyses using multigroup analysis with the TSSEM approach (Jak & Cheung, 2020). See Table 1 for operational definitions of moderators. A fixed effects approach was used for the moderator analyses given challenges with model convergence likely related to the reduced sample sizes within moderator groupings (see similar issues raised by simulation studies in Jak & Cheung, 2018). We first tested a model with all pathways unconstrained across the moderator variable and examined the model fit indices. Next, we constrained all paths to be equal and examined the change in fit based on chi-square differences from the unconstrained to the constrained model to determine significant difference across unconstrained and constrained models. Moderators included study population, sex and racial composition of studies, study drinking inclusion criteria (any level of drinking vs. binge drinking), PTSD assessment method (questionnaire vs. interview), PTSD DSM definition, drinking variable used, longitudinal vs. cross-sectional design, and peer-reviewed vs grey literature. All

moderators, excluding the harmful drinking variable (e.g., consumption, problems, hazardous drinking, heavy episodic drinking), were conducted on the pooled study correlation matrices. Analysis of the harmful drinking variable as a moderator was conducted on all correlation matrices recorded given that several studies included multiple drinking variables.

To examine the effects of single studies on the aggregate effect size, we used a one study removed procedure. We report the jackknife, which is the average omnibus effect size, and range of those effect sizes using this one study removed approach. To our knowledge, no validated or established methods for assessing publication bias with the TSSEM approach exist. However, we developed a method for assessing publication bias similar to the fail-safe *N* procedure. We created a study case that contained (1) the median number of participants; (2) the aggregate effect size for the PTSD and coping motives association; (3) the aggregate effect size for the PTSD drinking variable association; (4) an effect size of zero for the coping motives and drinking variable association. This allowed us to examine a model in which the total effect remained the same while the indirect effect equaled zero. We then systematically added this study until the aggregate indirect effect size was reduced to half the size of the aggregate effect found in our omnibus model.

Transparency and Openness

All analyses were conducted using the *metaSEM* package in *R* Statistical Analysis (Cheung, 2019b). This review was pre-registered in PROSPERO (CRD42019125450). This article represents the first of three manuscripts from a larger meta-analysis project that aims to quantify the relations between internalizing pathology, coping motives, and

alcohol use. The analyses for the current study were not pre-registered. All code and raw data files to replicate these analyses are available at https://osf.io/3djtr/?view_only=9b4351ae2bcb46d8a21b48b8009c9cb7.

Results

Meta-analytical Sample Characteristics

Included studies (k = 34 studies, n = 69 effect sizes; listed in supplemental materials) had an average sample size of 387.26 (median = 312; range = 42 – 1,896; aggregate sample = 15,086). On average, samples were 47.18% female (range = 1.9% - 100%), 62.73% White (range = 28% - 94%), 20.65% Black (range = 1.2% – 50.7%), and 29.00 years old (range = 15.6 – 44.7).

A majority of effect sizes were cross-sectional (n = 58), though some represented longitudinal effects (n = 11). Among the longitudinal studies, PTSD and coping were often assessed at the same time period (n = 6), as were coping and harmful alcohol use (n = 2). However, to be consistent with the self-medication hypothesis, PTSD was always assessed before the harmful alcohol use variable. The average time between PTSD and coping was 1.27 months. The average time between coping and alcohol measurement was 2.56 months. The average time between PTSD and harmful alcohol use was 4.11 months.

Studies used a range of measures to assess PTSD severity dimensionally, including the PTSD Checklist (DSM-IV n = 20; DSM-5 n = 17), the PTSD Symptom Scale – Interview (n = 1), the Clinician Administered PTSD Scale (DSM-IV n = 4; DSM-5 n = 11), the Trauma Symptom Checklist (n = 3), the Post-traumatic Diagnostic Scale (n = 6), the 4-item PTSD Checklist Military version (n = 4), and the Child PTSD Symptom Scale (n = 3). PTSD data was primarily obtained through self-report questionnaires (n =

52), although almost a quarter of the studies used clinical interviews (n = 17). Over half of studies assessed the DSM-IV definition of PTSD (n = 41); the remaining studies assessed the DSM-5 definition of PTSD (n = 28). The vast majority of studies examined PTSD over the past month (n = 58); however, a handful of studies also assessed PTSD over one week (n = 1), two weeks (n = 1), one year (n = 1), lifetime (n = 1), or some other undefined timeframe (n = 7).

Studies primarily assessed coping motives with iterations of the Drinking Motives Questionnaire (n = 64). Studies using the Drinking Motives Questionnaire used either the original iteration (n = 15) or the revised version (n = 49). The coping motives subscale of the Drinking Motives Questionnaire-Revised can be disaggregated into drinking to cope with depression (e.g., "I drink to turn off negative thoughts about things in my life") or drinking to cope with anxiety (e.g., "I drink because it helps me when I am feeling nervous"). Although most studies used the "general" coping motives subscale which consolidates items referring to depression and anxiety-related motives (n = 48), some cases utilized the depression-specific (n = 9) or anxiety-specific (n = 7) subscales. One case combined items from the DMQ general coping motives subscale with items from the Hilton Drinking Behavior Questionnaire. In addition to the DMQ, a 2-item alcohol-specific brief COPE (n = 2), a trauma-related drinking to cope measure (n = 1), and the Drinking Contexts Scale (n = 1) were also used. Most studies did not specify the timeframe for their measure of coping motives (n = 56), while other studies used a one-month (n = 4) or one-year (n = 3) timeframe.

Meta-analytic Results

Stage 1 (i.e., pooled correlation matrix) meta-analytic results revealed significant associations between PTSD and coping motives (k = 34; r = .42; p < .001), PTSD and measures of harmful alcohol use (k = 34; r = .20; p < .001), and coping motives and harmful alcohol use (k = 34; r = .41; p < .001). The omnibus multivariate meta-analytic model, which reports effect sizes after accounting for other pathways in the model, is included in Table 1. The omnibus model does not account for the possible influence of moderator variables. The results suggested that coping motives accounted for most of the variance between PTSD and measures of alcohol use, such that the confidence intervals for the PTSD and drinking effect crossed zero.

There was significant heterogeneity of effect across studies (Q = 650.64; pQ < .001; I^2 = 0.77 [PSTD to Coping motives], 0.75 [PTSD to Alcohol Use], 0.90 [Coping motives to Alcohol Use]), which justified examination of study moderators. Results of the meta-analytic moderator analyses are reported in Table 1. Moderator analyses represent univariate comparison, and do not account for other moderating variables.

Several population characteristics meaningfully influenced the indirect effect of coping. For example, studies of military/first-responders had the strongest indirect effect for coping. Further, studies with a lower percentage of females exhibited larger indirect effects. However, there was no difference in self-medicated drinking based on the racial make-up of the individual samples.

Measurement approaches also acted as moderators of the indirect effect. Studies requiring binge drinking episodes for eligibility had smaller indirect effects compared to studies without an explicit drinking inclusion criterion. Studies using clinical interviews to assess PTSD reported smaller effects sizes compared to questionnaire-based measures.

Interestingly, studies using the DSM-5 definition of PTSD reported a stronger indirect effect compared to studies using the DSM-IV definition of PTSD. Finally, indirect effects were strongest for studies examining hazardous drinking (assessed with the AUDIT).

We also noted that the grey literature (i.e., unpublished thesis/dissertations studies) was associated with a weaker indirect effect as compared to published studies, and that studies using a longitudinal design reported a weaker indirect effect compared to studies that used a cross-sectional design.

Jackknife analysis indicated that no one study was impacting results substantially (jackknife mean and range for the omnibus indirect effect: 0.165 [0.161 - 0.170]. The results of our modified fail-safe *N* suggested that we would need 22 studies with a null zero order association between coping motives and the drinking variable for the effect size to be reduced by 50%.

Discussion

Coping-Related Drinking as a Mediator of the PTSD-Harmful Alcohol Use Relation

The current meta-analytic review aggregated data from 34 manuscripts resulting in 69 unique cases. The goal was to test the influence of coping-related drinking motives in the association between PTSD and harmful alcohol use. Results demonstrate that high PTSD severity was related to high levels of self-reported coping motives for drinking, which was related to a more harmful pattern of alcohol use. Based on findings from the overall aggregated analysis, coping-related drinking accounted for 80% of the total effect between PTSD and harmful alcohol use. Despite significant heterogeneity in the literature, the pooled analyses supported the mediating influence of coping-related drinking in this relationship.

These results align with findings from a number of studies that have also found that coping-related drinking mediated the relation between PTSD and a variety of alcohol use outcomes (e.g., Aarstad-Martin & Boyraz, 2017; Hawn, Bountress et al., 2020; McDevitt-Murphy et al., 2017). Consistent with the self-medication hypothesis, the findings from this study continue to underline the importance of coping motives as a significant and powerful influence in the PTSD and harmful alcohol use relationship.

Though this meta-analysis provides additional support for the self-medication hypothesis, it by no means establishes coping motives as the definitive causal mechanism between these two variables. Establishing coping motives as a mechanism would necessitate the temporal precedence of PTSD, which only a limited number of studies have currently examined. Furthermore, though coping-related drinking accounts for a large amount of the variance in the total effect, it is still noteworthy that 20% of the variance remains unaccounted for. Thus, while coping-related drinking is one important piece of the comorbidity puzzle, findings from this meta-analytic review suggest that other pieces are still needed to complete the larger picture and inform a more complete understanding of this relationship. This may include facets of other well-established models of comorbidity including the high-risk hypothesis (suggesting that pre-trauma substance use problems increase risk for trauma exposure; Windle, 1994), the susceptibility hypothesis (suggesting that pre-trauma substance use places an individual at risk for developing PTSD; Chilcoat & Breslau, 1998), and the shared-vulnerability hypothesis (suggesting that the association between PTSD symptoms and alcohol problems is attributable to shared risk factors; McLeod et al., 2001). Exploring these

other models will be important, especially since at least one longitudinal study has found partial support for the high-risk hypothesis (Haller & Chassin, 2014).

Because behavior is often the result of multiple motives acting on an individual concurrently, it is also possible that other motives from the motivational model of alcohol use may also help explain variability in the PTSD-harmful alcohol use relation. These could include internal positive reinforcement (drinking to enhance pleasurable feelings), external positive reinforcement (celebratory drinking in social situations), and external negative reinforcement (drinking to avoid disapproval from peers; Cooper, 1994). Because the studies included in our aggregated analyses largely looked at internal negative reinforcement (drinking to cope with distressing internal states) in isolation, the present analyses cannot establish if coping motives outperform other reasons for drinking.

In terms of clinical implications, these findings suggest that coping-related drinking may be an especially worthy target in the treatment of those with PTSD and harmful patterns of drinking. In fact, research suggests that individuals with comorbid PTSD and substance use disorder who receive skill-based (Boden et al., 2014) and trauma-focused (Zang et al., 2017) psychotherapy show reductions in avoidant coping (which may include drinking-to-cope). In these studies, reductions in avoidant coping occurred alongside reductions in alcohol use and PTSD symptoms, suggesting that the development of active coping strategies helps reduce reliance on drinking as a coping mechanism.

Factors Influencing the Mediating Effect of Coping Motives

The results from this study also uncovered factors contributing to heterogeneity in the size of the indirect effect. For example, differences in the strength of the indirect effect were related to population type such that military and first responders had a significantly stronger indirect effect for coping-related drinking as compared to general adult and adolescent/emerging adult samples. One possible explanation of these findings is that some factors associated with military, such as a pervasive drinking culture, make alcohol use the preferred coping mechanism in the face of trauma (Ames et al., 2007). However, it should also be noted that the general adult group was fairly diverse and included studies of sexual assault survivors, persons with severe mental illness, and those with multiple traumas. Thus, the heterogeneity within these derived sample categories may obscure findings and covariance may also exist between population type and other moderators examined in our analysis.

Our analysis also suggests that coping-related drinking accounted for 45% of the variance of the total effect among studies that used binge drinking as an inclusion criterion. This was significantly less than those studies with less restrictive drinking inclusion criterion (83% variance in the total effect). This may be due to a range restriction in the alcohol variable that artificially reduced the overall contribution of coping-related drinking in the total effect of the model. It may also be the case that samples of only binge drinkers are consuming alcohol for a number of complex reasons that are not experienced by samples with mixed levels of drinking. For example, one factor that could differentiate a sample of binge drinkers from a broader sample of drinkers is the experience of alcohol withdrawal, which is more likely to occur in

individuals with an overall heavier pattern of drinking and may motive individuals to continue using alcohol so that they avoid physiological symptoms.

Coping motives accounted for 95% of the variance in the total effect when studies included questionnaire-based measures of PTSD, which was significantly greater than studies that used interview methods of assessing PTSD (in which coping motives accounted for 67% of the variability in the total effect). It is unclear why questionnaire-based measures of PTSD were associated with a stronger self-medication effect compared to an interview approach. Diagnostic interviews (e.g., CAPS-5) are often referred to as the "gold-standard" in assessment since interviewers have the opportunity to probe individuals for deeper understanding and ask clarifying questions. The strong external validity of interview-based assessment, coupled with our findings that these types of assessments are associated with weaker indirect effects, may suggest that coping-related drinking does not account for as much variance between PTSD and alcohol use as the aggregated overall model suggests (since the omnibus model largely includes questionnaire-based measures of PTSD symptoms). This finding may also reflect other differences such as setting, sample, and base-rate of harmful drinking among participants who receive this measure. Yet, it may also be the case that questionnaire-based measures of both coping motives and PTSD symptoms show a greater degree of correlation due to shared method variance (see Campbell & Fiske, 1959).

Further, assessment measures corresponding to DSM-5 criteria for PTSD were associated with greater indirect effects than DSM-IV based measures of PTSD. It may be that some symptoms within that newer conceptualization aligns better with the self-medication model. For example, the DSM-5 PTSD criteria include a question about

"reckless or self-destructive behavior" that was not included in DSM-IV. This question could align with self-medication if individuals view drinking in response to trauma as reckless or self-destructive, thus inflating the effect for DSM-5 criteria.

Significant differences were also found among four drinking constructs, such that assessing hazardous drinking resulted in the strongest indirect effect. One possible explanation for this finding is that self-medication operates differently between consumption, consequences, and drinking behavior. The hazardous drinking construct, however, reflects all three of these domains to some degree. Because hazardous drinking reflects so many areas of drinking, it may allow coping-related drinking to account for more variance. Of course, sample bias may also help to explain this finding, since researchers are more likely to include measures of hazardous drinking symptoms in adult samples and those with more symptom severity.

Because the self-medication hypothesis is dependent on the temporal relationships between PTSD and harmful drinking, we were especially interested in testing longitudinal design (vs. cross-sectional design) as a potential moderator. We found that coping motives mediate the PTSD and harmful drinking relation in longitudinal studies, bolstering our confidence in the self-medication hypothesis. Yet, longitudinal studies were associated with a significantly smaller indirect effect than cross-sectional studies. In fact, coping motives accounted for 95% of the variance in the total effect for cross-sectional studies compared to 64% in longitudinal studies.

Potential Bias in the Literature

In order to understand bias in this literature, this study first examined the moderating influence of unpublished "grey" literature vs. peer-reviewed published

studies. Findings from this moderation analysis suggest that indirect effects are stronger for peer-reviewed articles, though the mediating influence of coping is still present among the thesis/dissertation studies sampled here. The smaller mediating effect from the grey literature that we sampled, however, may suggest that there is a file-drawer effect among published studies that include PTSD, coping motives, and harmful alcohol use.

Additionally, we found evidence for a file-drawer effect via the one study removed analysis. Results suggest that 22 studies would have to exist with null findings in order to reduce the size of the indirect effect by 50%. Given the number of studies in the field that include these common variables, it seems likely that well over 22 null studies may exist to taper the reported effect.

Strengths and Limitations

The present investigation has several noteworthy methodological strengths. First, our study screened over 14,300 articles related to PTSD, coping-related drinking, and/or different forms of harmful alcohol use. Thus, we conducted a sweeping review of the current state of science on this topic. Second, we utilized a novel and statistically advanced data analytic approach to aggregate indirect effects. To our knowledge, this approach has been seldom used in the field, though it is clearly an important tool for answering critical questions about etiology. Third, we included both peer-reviewed published articles as well as unpublished "grey literature" to shed light on the possibility of a file-drawer effect.

Despite the strengths of this meta-analytic review, this study is not without limitations. First, while review of the literature was rigorous, it is possible that some appropriate studies were missed in the data extraction phase. This includes relevant

publications with missing data that could not be obtained by the authors. Because these studies could not provide missing data, it is impossible to know if they differed from the included studies in some meaningful way.

Second, because this study focused on coping-related drinking motives, we excluded other relevant mediators that could support the self-medication hypothesis (such as "avoidant" coping, "maladaptive" coping; alcohol expectancies; negative urgency; and emotion regulation). We also excluded measures of coping that assessed 'coping through substance abuse' more broadly since we could not be sure that the substance being used was alcohol.

Third, this analysis was limited by the scope of the current literature, which overwhelmingly utilizes cross-sectional data analyses. Though our meta-analysis found that coping-related drinking mediated PTSD-harmful alcohol relationships in both cross-sectional and longitudinal designs, we would be more confident in our findings if we could have included additional longitudinal studies. This is important to note, since the self-medication hypothesis conceptually depends both on the mediating influence of coping motives, and on PTSD preceding harmful alcohol use in time.

Finally, several analytic issues may impact our findings. For example, the effects of PTSD reported here may be better accounted for by co-occurring anxiety or depression (which should be the subject of future work). Further, we could not account for the impact of autocorrelation in our longitudinal studies since not measure was assessed at every timepoint. Additionally, we conducted univariate analyses to avoid over parametrizing our model. As a result, there is likely overlapping variance in our study that remains unaccounted for.

Future Directions

It is clear from this meta-analysis, systematic reviews (Hawn, Cusack et al., 2020), and narrative reviews on self-medication (Turner et al., 2018) that the literature lacks longitudinal studies exploring the temporal relationship between PTSD, coping-related drinking motives, and harmful alcohol use. As a result, researchers doing work in this area should focus more on longitudinal designs opposed to cross-sectional ones (which have already been overly focused on).

It is also clear from the moderator analyses we described that the effect of self-medication will vary widely based on multiple domains, and that there are likely to be subsets of individuals who do not drink to cope with their internal emotional states at all. For these individuals, other relevant theories such as the high-risk hypothesis or the shared vulnerability hypothesis may be more relevant. Thus, future studies may wish to take a more person-centered approach to systematically uncover different groups of individuals and the reasons that they drink.

Future research in this area should also consider using a more trauma-informed measure of coping-related drinking motives, such as the recently developed Trauma Related Drinking Questionnaire (TDR). This measure assesses the desire to drink in response to four PTSD symptom clusters. Psychometric research on the TDR suggests that it is a more specific measure than the DMQ coping subscale, whose questions focus on depression and anxiety more generally (Hawn, Aggen, et al., 2020).

We were unable to assess other potentially important moderators in this investigation. One such moderators is trauma-type. Trauma type was not included in our coding process since participants often report experiencing multiple criterion A traumas,

because some studies do not require the presence of a criterion A event, and because many studies do not report on what traumatic events were experience by a participant. Future researchers may wish to creatively account for this in their work.

Though we assessed sex and race as two independent moderators in our study using an unbiased tertiary cut, we also acknowledge that a more fine-grained analysis of how sex and race interact and influence coping-related drinking is warranted. For example, there are limits on what we can learn by distilling racial groups to White vs. Non-White given the importance of intersectionality and a host of unmeasured variables (including values, beliefs, customs, experience of racism, etc).

Conclusions

In summary, the present study suggests that the relationship between PTSD symptoms and harmful alcohol use is explained by coping-related drinking motives, yet variance still remains to be explained by additional theoretical approaches. Measurement approach, study design, and sample characteristics may result in differences among both the direct and indirect effects. Additional work is needed in the field to better understand this theory.

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Table 1. Omnibus Model and Moderator Analyses

	Path c′ (Indirect Effect)		Path a (PTSD to Coping)		Patch c (Direct Effect)		Path b (Coping to Drinking)	
Model/Moderator	Estimate	LL, UL	Estimate	LL, UL	Estimate	LL, UL	Estimate	LL, UL
Omnibus Model	0.16	0.14, 0.19	0.42	0.38, 0.45	0.04	-0.01, 0.09	0.40	0.34, 0.45
Population Type*†								
Adolescent/Emerging Adult (k=15)	0.18	0.17, 0.19	0.41	0.39, 0.43	0.03	0.002, 0.05	0.44	0.42, 0.46
General Adult (k=10)	0.16	0.14, 0.17	0.38	0.36, 0.41	0.07	0.05, 0.10	0.41	0.39, 0.44
Military/First Responder (k=9)	0.23	0.21, 0.24	0.46	0.44, 0.49	-0.06	-0.09, -0.03	0.49	0.46, 0.52
Sex (% female)* **								
<33% (k=9)	0.23	0.21, 0.24	0.46	0.44, 0.49	-0.06	-0.09, -0.03	0.49	0.46, 0.52
34-67% (k=13)	0.16	0.15, 0.18	0.38	0.35, 0.41	0.06	0.03, 0.10	0.43	0.39, 0.46
>67% (k=11)	0.17	0.16, 0.19	0.40	0.38, 0.42	0.04	0.02, 0.06	0.43	0.41, 0.45
Race (% White)								
<50% White (k=7)	0.17	0.16, 0.18	0.40	0.38, 0.42	0.05	0.02, 0.07	0.42	0.40, 0.45
50%+ White (k=24)	0.18	0.17, 0.19	0.42	0.40, 0.44	0.05	0.03, 0.08	0.43	0.41, 0.45
Study Drinking Criteria* **								
Any Drinking (k=16)	0.20	0.18, 0.21	0.45	0.43, 0.48	0.04	0.01, 0.06	0.44	0.41, 0.46
Binge Drinking (k=5)	0.10	0.07, 0.12	0.39	0.34, 0.44	0.12	0.06, 0.18	0.25	0.19, 0.31
PTSD Assessment Method**								
Questionnaire (k=27)	0.19	0.18, 0.20	0.42	0.41, 0.44	0.01	-0.004, 0.03	0.45	0.44, 0.47
Interview (k=7)	0.12	0.10, 0.16	0.35	0.29, 0.40	0.06	-0.004, 0.12	0.36	0.30, 0.42
PTSD DSM Definition* **								
DSM-IV (k=20)	0.16	0.14, 0.17	0.37	0.35, 0.39	0.04	0.02, 0.06	0.42	0.40, 0.44
DSM-5 (k=14)	0.23	0.21, 0.24	0.48	0.46, 0.49	-0.02	-0.04, 0.01	0.48	0.46, 0.50
Drinking Variable*†								
Quantity-Frequency (k=27)	0.16	0.15, 0.18	0.42	0.40, 0.44	-0.01	-0.03, 0.02	0.39	0.37, 0.41
Drinking Problems (k=16)	0.14	0.13, 0.15	0.38	0.36, 0.40	0.09	0.07, 0.11	0.37	0.35, 0.39
Hazardous Drinking (k=16)	0.23	0.22, 0.24	0.46	0.44, 0.48	-0.02	-0.04, -0.004	0.50	0.49, 0.52
HED Frequency (k=10)	0.15	0.13, 0.17	0.35	0.32, 0.39	-0.01	-0.05, 0.03	0.44	0.40, 0.47

Publication Type*								
Peer-Reviewed (k=30)	0.19	0.18, 0.20	0.41	0.40, 0.43	0.02	0.001, 0.03	0.45	0.43, 0.46
Grey Literature (k=4)	0.18	0.15, 0.21	0.45	0.41, 0.49	0.01	-0.05, 0.06	0.40	0.34, 0.45
Data Type***								
Longitudinal (k=5)	0.14	0.11, 0.17	0.40	0.35, 0.44	0.08	0.03, 0.14	0.35	0.30, 0.40
Cross-Sectional (k=29)	0.19	0.18, 0.20	0.42	0.41, 0.43	0.01	-0.01, 0.03	0.46	0.44, 0.47

Note. Asterisk next to the moderator analysis designation indicates a significant difference in model fit between categories in models that freely estimate effects compared to a model constraining all pathways to be equal across all models. † = All categories differed significantly from one another, LL = Lower Limit; UL = Upper Limit; PTSD = Posttraumatic Stress Disorder; DSM = Diagnostic and Statistical Manual; HED = Heavy Episodic Drinking; * p <.05, ** p <.01, *** p <.001

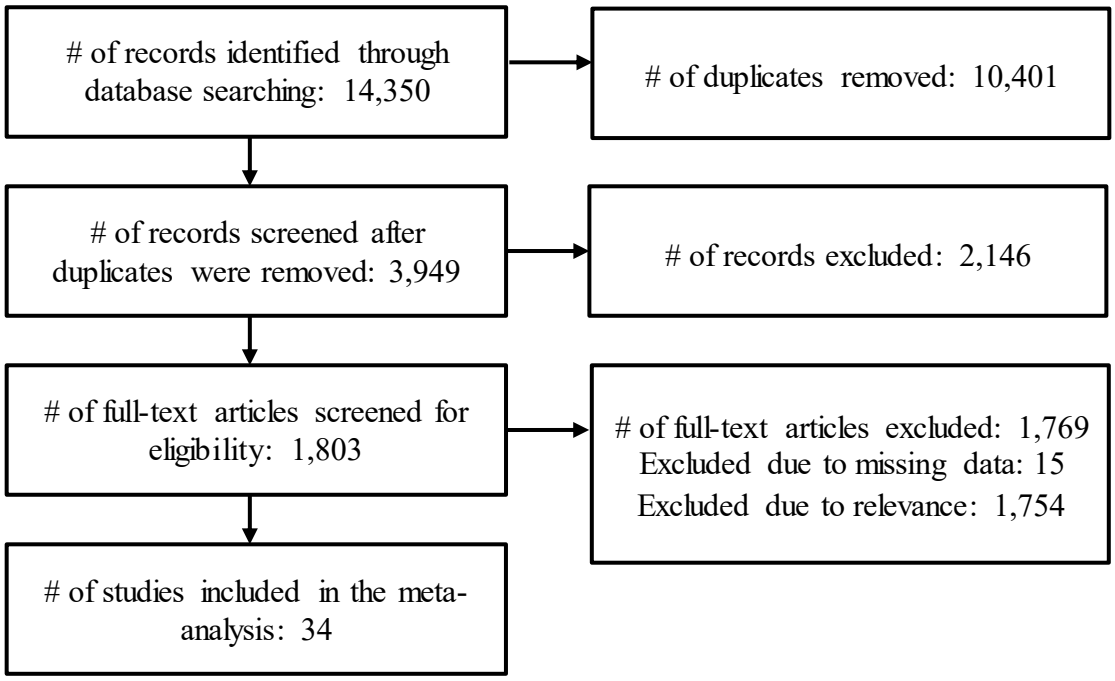

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) inclusion flow diagram.