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A Systematic Review of the Self-Medication Hypothesis in the Context of Posttraumatic Stress Disorder and Comorbid Problematic Alcohol Use

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Abstract

Posttraumatic stress disorder (PTSD) and alcohol use disorder (AUD) commonly co-occur and are associated with many negative public health outcomes. There are several etiological models that explain the overlap between PTSD and AUD, including shared genetic risk and phenotypic causality, but the predominant model of etiologic association is the drinking-to-cope self-medication model. Although the self-medication model is conceptually appealing and has been widely accepted within the literature examining alcohol use and anxiety (e.g., PTSD) phenotypes, the findings are inconsistent and there is a lack of rigorous empirical evidence in support of this model. This review, which was, to our knowledge, the first systematic review of the self-medication model in relation to PTSD to date, aimed to synthesize the current literature on the association between PTSD and problematic alcohol use within the context of the self-medication model. In total, 24 studies met the inclusion criteria for the review and assessed the self-medication hypothesis using a variety of measurement instruments and data analytic approaches, such as mediation, moderation, and regression. Overall, the included studies provide evidence for the self-medication hypothesis but are limited in rigor due to methodological limitations. These limitations, which include issues with the operationalization (or lack thereof) of trauma-related drinking to cope, are discussed, and directions for future research are presented.

Posttraumatic stress disorder (PTSD) and alcohol use disorder (AUD) are prevalent, with lifetime general population estimates of PTSD ranging from 0.5% to 14.5%, depending on trauma timing and type (Koenen, Ratanatharathorn, Bromet, Karam, & Stein, 2018) and AUD at around 29% (Grant et al., 2015). Further, PTSD and AUD frequently co-occur, with comorbidity estimates ranging from approximately 30% to 50% (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Pietrzak, Goldstein, Southwick, & Grant, 2011). There is an even wider range of estimates for comorbid PTSD and alcohol misuse more broadly; for example,

in a review, Debell and colleagues (2014) reported rates of 9.8% to 61.3%.; Debell et al., 2014). Various adverse consequences have been associated with PTSD–AUD comorbidity, including higher levels of PTSD symptom severity, increased use of services, and poorer treatment prognosis compared to noncomorbid cases (Blanco et al., 2013; Ipser, Wilson, Akindipe, Sager, & Stein, 2015; Shorter, Hsieh, & Kosten, 2015). Given the high prevalence and associated public health impact of comorbid PTSD–AUD, theoretical and empirical work has led to the application of numerous etiologic models to explain the mechanisms that underlie this comorbidity. The purpose of the present paper is to provide a detailed review of studies that have investigated this comorbidity via the lens of the self-medication hypothesis, which is the model that has received the most empirical attention to date (Khantzian, 1997).

There are four proposed pathways to explain PTSD–AUD comorbidity. First, the PTSD susceptibility model proposes that substance use may increase susceptibility to PTSD, either through social (e.g., increased exposure to trauma) or physiological (e.g., impaired homeostatic response to stressors) pathways (Chilcoat & Breslau, 1998; Danovitch, 2016). For example, alcohol intoxication may increase the likelihood of trauma exposure, thus indirectly increasing the risk for the development of PTSD (Schumm & Chard, 2012). Second, the “common factors model” posits that shared factors, such as genetic influence, early environmental experiences, and personality traits, may contribute to an increased risk for and maintenance of both substance use and PTSD (Danovitch, 2016). Indeed, twin models have shown that upwards of 30% of the genetic vulnerability to PTSD is shared with AUD (Sartor et al., 2011; Xian et al., 2000). Third, the mutual maintenance model suggests that although there are several proposed independent pathways to explain the common co-occurrence of PTSD and AUD, it is notable that these various pathways are not mutually exclusive and instead likely constitute a combination of risk and maintenance processes that explain PTSD–AUD comorbidity (Stewart, Pihl, Conrod, & Dongier, 1998). In other words, the mutual maintenance model is characterized by the fact that AUD may exacerbate symptoms of PTSD and vice versa (Danovitch, 2016; Schumm & Chard, 2012; Stewart et al., 1998).

Fourth, and one of the most widely accepted explanations for high comorbidity rates between PTSD and AUD, is the self-medication hypothesis, a causal model that postulates that individuals with PTSD are more prone to developing problematic drinking behaviors due to a tendency to drink to cope with negative internal experiences (Khantzian, 1997). Consistent with learning theory, the use of alcohol to temporarily alleviate PTSD symptoms acts as a cogent short-term negative reinforcer. Therefore, repeated and increased use of alcohol to obtain the learned and desired effects of temporarily mitigating PTSD symptomatology increases the likelihood of developing more severe forms of alcohol use (e.g., AUD; Schumm & Chard, 2012). Prior research has indicated that this pattern of reinforcement may occur at a subconscious or conscious level. For instance, the negative reinforcement model of drinking, posited by Baker, Piper, McCarthy, Majeskie, and Fiore (2004), would suggest that, through repeated cycles of PTSD symptoms and alcohol use, individuals learn to detect interoceptive cues of low-level PTSD symptoms preconsciousness, thereby prompting drinking. Conversely, at high levels of negative affect, individuals may become conscious of their affect, increasing cognitive biases toward response options that have efficiently ameliorated negative affect in the past (e.g., alcohol use) and thereby

becoming inclined to reflexively drink when they experience high levels of PTSD symptoms (Baker et al., 2004).

In addition to an increased risk for AUD, the use of alcohol to avoid trauma-related responses may also exacerbate or prolong PTSD symptomatology by preventing habituation of traumatic memories (Stewart et al., 1998), which is consistent with evidence for bidirectional causality (Berenz et al., 2017) and the mutual maintenance model. Furthermore, evidence suggests that consuming alcohol may also interfere with the processing of trauma memories needed for recovery from PTSD on a neurobiological level. For example, both animal and human experimental studies have demonstrated adverse effects of alcohol on fear conditioning and memory consolidation, two processes highly implicated in theories of PTSD development (Kaysen, Bedard-Gilligan, & Stappenbeck, 2017; Tipps, Raybuck, & Lattal, 2014). Taken together, the evidence suggests that models of comorbidity are not mutually exclusive nor are they unidirectional, and it underscores the clinical significance of the self-medication model such that drinking to cope with PTSD symptoms may lead to or exacerbate extant AUD, chronic PTSD, and comorbid PTSD–AUD, which have all been linked to critical public health outcomes, as previously described. Therefore, an empirically substantiated understanding of this model is important.

Evidence from different lines of research may serve to support the tenants of the self-medication hypothesis. First, PTSD onset typically precedes AUD onset (Kessler et al., 1995), and longitudinal studies have found associations between PTSD symptom onset and alcohol use onset following trauma exposure (Bremner, Southwick, Darnell, & Charney, 1996). Further, individuals with PTSD are more likely to endorse drinking to cope with general negative affect compared to individuals without PTSD (Waldrop, Back, Verduin, & Brady, 2007). Preliminary support also indicates that coping-oriented drinking may mediate the association between PTSD and problem drinking (O'Hare & Sherrer, 2011). Additionally, clinical laboratory studies have demonstrated that compared to those without PTSD, individuals with PTSD are more likely to use alcohol or other substances after experiencing diminished appetitive functioning (i.e., anhedonia, emotional numbing symptoms) and when they are exposed to trauma-related cues (Kashdan, Elhai, & Frueh, 2006; Ouimette, Coolhart, Funderburk, Wade, & Brown, 2007; Waldrop et al., 2007). Similarly, reminders of a traumatic event have been shown to cause alcohol or drug cravings in individuals with PTSD (Coffey et al., 2002). In a recent ecological momentary assessment (EMA) study in which researchers monitored PTSD symptoms and alcohol use, the results showed that an increase in PTSD symptoms from a participant's average PTSD symptom level was associated with an increase in drinking within a 3-hr window, further supporting the self-medication hypothesis (Possemato et al., 2015).

Multiple methodologic designs, such as EMA and clinical laboratory studies, have been used to study the self-medication hypothesis. However, with regard to the literature involving human samples, the most frequently used design includes the self-report of drinking to cope and/or alcohol expectancies (e.g., McDevitt-Murphy, Fields, Monahan, & Bracken, 2015; Stappenbeck, Bedard-Gilligan, Lee, & Kaysen, 2013). To date, this literature has not been subjected to a systematic review. The primary aim of the present review was to provide a critical synthesis of the existing literature on the theoretical assumption most commonly

used to explain PTSD–AUD comorbidity (i.e., the drinking-to-cope self-medication model) and to apply said synthesis to inform recommendations for the field moving forward, particularly with regard to methodology. Methodological inconsistencies and gaps in the existing literature are highlighted and used to identify important steps to be taken should the self-medication model continue to inform both research and clinical practice.

Method

Procedure

Search and selection criteria.—Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA; Moher, Liberati, Tetzlaff, & Altman, 2009) guidelines were used as a guide for the conduct of the systematic review. A comprehensive literature search was conducted using electronic psychological and medical databases (i.e., PubMed and PsycINFO). Search terms included in the final searches were as follows: Search 1: [(PTSD OR posttraumatic stress disorder OR traumatic stress) AND (alcohol OR drinking OR drink) AND (“self-medication” OR “self medication”)]; Search 2: [(PTSD OR posttraumatic stress disorder OR traumatic stress) AND (alcohol) AND (“self-medication” OR “self medication”) AND (cope OR drink* to cope OR coping-oriented OR coping)]; Search 3: [(PTSD OR posttraumatic stress disorder OR traumatic stress) AND (alcohol) AND (cope OR drink* to cope OR coping-oriented OR coping)]; and Search 4: [(PTSD OR posttraumatic stress disorder OR traumatic stress) AND (alcohol OR drinking OR drink) AND (tension reduction OR tension-reduction)]. Searches 2 through 4 were conducted separately because when the supplementary items were added to the original search terms (Search 1), the results were too restrictive, $k_s = 8, 1$, and 1 , respectively. The initial search was conducted in April 2017. Additional searches conducted in May 2018 and February 2019 revealed one and three new citations, respectively.

Screening of Search Results

Records produced by the search terms in both PubMed and PsycINFO were identified and screened, and 194 records were reviewed for possible inclusion. A total of 84 duplicates were removed: 10 duplicates were found between the four literature searches conducted within PubMed, an additional 10 duplicates were found between the four searches conducted in PsycINFO, and an additional 64 duplicates were found between the combined PubMed and PsycINFO searches. This resulted in a total of 110 articles identified for potential inclusion in the systematic review. Ten additional articles were identified as potentially eligible for inclusion through a manual search of existing self-medication review articles; this resulted in 120 potentially eligible records. From there, articles were included if they (a) assessed for PTSD diagnosis or trauma-related symptoms, (b) assessed coping-oriented drinking motives, tension-reduction, or alcohol-related expectancies, and (c) examined alcohol consumption, alcohol-related problems, and/or drinking motives as an outcome. Relevant dissertations and book chapters were included for consideration. See Supplementary Figure 1 for a flow diagram.

There were 24 that studies met inclusion criteria and were thus included in the research synthesis (see Supplementary Table 1 for an overview of included studies). Notably, multiple

articles that did not examine coping-related drinking motives or for which the outcome was not alcohol-related (e.g., review articles or examples of the self-medication model involving other substances) were considered for discussion to further synthesize the literature and to serve as exemplars of the operational and methodological inconsistencies and limitations across studies examining the self-medication model.

A supplementary literature search was conducted in February 2019 to confirm that no prior systematic reviews of the PTSD self-medication literature had been published to date. Those search terms were as follows: [(“self medication” OR “self-medication”) AND (systematic review) AND PTSD]. This search did not result in any papers. Therefore, to our knowledge, no systematic review has yet to be conducted that examines the self-medication model to understand the relationship between PTSD and alcohol use—or any substance use, for that matter—making this, to our knowledge, the first systematic review of its kind.

Results

Study Characteristics

Supplementary Table 1 presents the details of the 24 studies included in the review. Most of the studies assessed a range or combination of generalized drinking motives, $k = 19$; tension-reduction strategies, $k = 5$; and/or alcohol expectancies more broadly, $k = 5$, to explore self-medication. See Table 1 for sample items from measures used in the studies. Most studies that met inclusion criteria were cross-sectional in design, $k = 21$. Two studies were microlongitudinal in design (i.e., daily diary, EMA; Simpson, Stappenbeck, Luterek, Lehavot, & Kaysen, 2014; Taylor, 2010) and one was longitudinal in design (Beseler, Aharonovich, & Hasin, 2011). Additional study characteristics, such as sample size and sample demographics, are provided in Supplementary Table 1.

Evidence for the Self-Medication Hypothesis

A synthesis of results from the included studies provides general support for the self-medication hypothesis across a variety of methodological approaches.

Longitudinal findings.—Of the 24 studies that met eligibility criteria for the present review, only one was longitudinal in design. Beseler and colleagues (2011) found that drinking to cope with negative affect, as measured a decade before the September 11th, 2001, terror attacks in the United States (9/11), predicted alcohol consumption 1 week after 9/11 but not 16 weeks after. Beseler and colleagues also tested multiple moderation models but failed to find any significant three-way interactions between drinking motives, proximity to the World Trade Center attack site, or lifetime alcohol dependence in predicting alcohol consumption, which was contrary to the authors' study hypotheses. Notably, because preliminary analyses failed to show an association between PTSD and alcohol-related outcomes, no measure of PTSD or traumatic stress was included in the final analyses.

Microlongitudinal findings.—Although the results of the systematic review revealed only one longitudinal study, two included studies (Simpson et al., 2014; Taylor, 2011) applied daily time-series analyses, each collecting daily monitoring assessments over a span

of 7 days. Simpson and colleagues (2014) used time-series analyses to assess whether PTSD symptoms would predict later increases in alcohol use (i.e., the self-medication hypothesis) and whether alcohol use would predict later increases in PTSD symptoms (i.e., the mutual maintenance hypothesis). Their findings demonstrated support for the self-medication hypothesis such that elevated PTSD symptoms predicted more same-day and next-day alcohol use. The findings did not support the mutual maintenance model. Additionally, the authors found that coping and enhancement drinking motives significantly moderated the relation between PTSD and same-day, but not next-day, alcohol use such that among individuals who endorsed higher levels of coping drinking motives, an increase in PTSD symptoms was related to a larger increase in same-day alcohol use as compared to individuals who endorsed low levels of coping drinking motives.

In contrast, Taylor (2011) failed to demonstrate support for the self-medication hypothesis. Contrary to the author's hypotheses, the study results demonstrated a negative association between PTSD symptoms and alcohol consumption, indicating that higher levels of PTSD symptoms predicted lower levels of next-day alcohol use. Additionally, the results did not provide support for the predictive effect of perceived coping via substance use or daily traumatic stress on past 24-hr alcohol use (Taylor, 2010).

Cross-sectional findings.

Mediation studies.—There were 11 included studies that tested mediation models to assess the self-medication model, none of which used longitudinal data. Eight out of these 11 studies found support for the self-medication model (Blumenthal, Leen-Feldner, Knapp, Badour, & Boals, 2015; Grayson & Nolen-Hoeksema, 2005; Kaysen et al., 2007; McCabe, Mohr, Hammer, & Carlson, 2018; Tomaka, Magoc, Morales-Monks, & Reyes, 2017; Ullman, Filipas, Townsend, & Starzynski, 2005; Ullman, Releya, Peter Hagene, & Vasquez, 2013; Vujanovic, Bonn-Miller, & Marlatt, 2011). A majority of the included cross-sectional mediation studies investigated coping motives as a mediator (Delker & Freyd, 2014; Grayson & Nolen-Hoeksema, 2005; Kaysen et al., 2007; McCabe, Mohr, Hammer, & Carlson, 2018; Tomaka, Magoc, Morales-Monks, & Reyes, 2017; Tuliao, Jaffe, & McChargue, 2016; Ullman et al., 2013; Vujanovic et al., 2011), whereas the remaining studies investigated alcohol expectancies (Blumenthal et al., 2015; Ullman et al., 2005; Vik, Islam-Zwart, & Ruge, 2008). Of note, one study that did not find support for mediation (Vik et al., 2008) assessed the mediation of the association between sexual assault exposure, not PTSD, and alcohol use, whereas the majority of studies that demonstrated significant findings assessed the mediation of PTSD symptoms and alcohol use (Kaysen et al., 2007; McCabe et al., 2018; Tomaka et al., 2017; Ullman et al., 2005, 2013), which is more consistent with the self-medication hypothesis. That said, both Delker and Freyed (2014) and Tuliao and colleagues (2016) tested the mediating effect of (a) coping motives and (b) relaxation and tension-reduction expectancies, respectively, on the relation between PTSD and substance and/or alcohol use and did not find evidence for a significant mediating effect. However, in the case of Delker and Freyed (2014), PTSD was assessed in the context of a unique trauma type (i.e., betrayal), and a latent variable of substance use was used as the dependent variable, which included drug use and, therefore, was not specific to alcohol.

Moderation studies.—In addition to the study by Beseler and colleagues (2011), which incorporated longitudinal data, six studies used cross-sectional data to test moderation models (Hruska & Delahanty, 2012; Peters, Khondkarya, & Sullivan, 2012; Schaumberg et al., 2015; Simpson et al., 2014; Stappenbeck et al., 2013; Vik et al., 2008). Four of these six studies reported significant results, which were evenly split between studies that investigated the role coping motives (Simpson et al., 2014; Stappenbeck et al., 2013) and tension-reduction expectancies (Hruska & Delahanty, 2012; Schaumberg et al., 2015) in the association between PTSD symptoms and alcohol use and/or problems. In contrast, Peters, Khondkarya, and Sullivan (2012) did not find support for alcohol use expectancies as a moderator of the association between interpersonal violence exposure and the number of days of alcohol use or alcohol problems. Perhaps, then, there is a different relation between alcohol-related expectancies and PTSD symptoms as compared to the relation between alcohol-related expectancies and interpersonal violence exposure. That said, Vik and colleagues (2008) tested whether PTSD symptom-specific alcohol expectancies moderated the association between PTSD symptom severity and alcohol use and did not find support for this relation. Taken together, the results from these studies are mixed, likely due to inconsistencies between studies regarding variable inclusion (e.g., trauma type, measure of coping, alcohol use outcome).

Other studies.—Finally, six studies used statistical approaches aside from mediation or moderation, including hierarchical regressions, logistic regressions, and correlation analyses. Results among these studies were generally consistent, demonstrating, broadly, that coping motives (Cloutier et al., 2018; Dixon, Leen-Feldner, Ham, Feldner, & Lewis, 2009; Eddinger, Humiston, Sutton, Jobe-Shields, & Williams, 2018; McDevitt-Murphy et al., 2015; Sheerin et al., 2016) and tension-reduction expectancies (Creech & Borsari, 2014) were positively associated with both alcohol use and alcohol-related problems and PTSD.

Discussion

The present systematic review aimed to provide a critical synthesis of the existing literature on the drinking-to-cope self-medication hypothesis with regard to comorbid PTSD and alcohol use, specifically, and to use the findings to inform future directions in the field. Overall, the literature provides support for the self-medication hypothesis across a variety of methodological approaches such that drinking-related coping motives and tension-reduction expectancies were broadly found to influence the association between PTSD and alcohol use. However, it should be noted that a majority of the included studies reported positive findings, which could potentially support the presence of a “file drawer” effect, which suggests that studies that did not produce significant findings (i.e., did not find support for the self-medication hypothesis) were less likely to be published than those that did.

By and large, the included studies assessed a range of generalized drinking motives, tension-reduction strategies, or alcohol expectancies to infer trauma-specific drinking-to-cope rather than to assess trauma-related drinking-to-cope specifically; this demonstrates a notable lack of measures that explicitly assess trauma- or PTSD-specific drinking-to-cope motives. Moreover, we found methodological inconsistencies across studies that we believe may have

implications for the self-medication literature as a whole. Each of these topics is discussed in turn.

Despite its popularity, a systematic review of the literature revealed a lack of rigorous empirical evidence in support of the self-medication model, which is likely due to several important methodological considerations. Such considerations, deduced from this systematic review, include the lack of specific operationalization of trauma-specific drinking-to-cope, lack of longitudinal study designs and mediational analyses to account for the temporal and causal assumptions underlying the self-medication model, heterogeneity in alcohol assessment, and failure to account for potential confounding variables, such as sample selection (e.g., sex, race/ethnicity), trauma type and timing, and other commonly comorbid internalizing disorders, such as depression and anxiety.

With the exception of the PTSD-Alcohol Expectancies Questionnaire (P-AEQ), which assesses expectations of the effects of alcohol on PTSD symptoms and was only used in two of the 24 included studies, the present systematic review of the literature revealed that the self-medication model is frequently postulated to explain PTSD–AUD comorbidity in the absence of any explicit validated measure of trauma-specific drinking-to-cope. The prolific use of measures assessing general drinking-to-cope motives (e.g., the Coping subscale of the Drinking Motives Questionnaire [DMQ-Cope]) to infer trauma-related drinking creates an illusion in the literature that drinking-to-cope motives are synonymous with drinking to cope with PTSD symptoms specifically; however, in practice, drinking to cope is assessed with regard to negative internal experiences more generally. For example, a sample item on the DMQ-Cope asks respondents if they use alcohol “to cheer you up when you’re in a bad mood” (Cooper, 1994). To date, it appears that no studies have explicitly examined the extent, in terms of frequency and quantity, that drinking-to-cope relates to PTSD symptoms specifically, making this a critical void to fill.

In addition to this critical lack of assessment of drinking to cope with trauma-specific symptoms, only a few longitudinal published longitudinal studies have investigated the temporal precedence of PTSD before AUD, a basic assumption of the self-medication model. If alcohol use among individuals with PTSD represents attempts at self-medication of PTSD symptoms, then it stands that problematic alcohol use should develop following trauma exposure and the emergence of PTSD symptoms (Stewart et al., 1998). Thus, to truly test the theory that PTSD increases the subsequent risk for AUD (i.e., self-medication), longitudinal research designs are warranted. However, according to a recent review published by Langdon and colleagues (2017), and confirmed through the present systematic review, only three studies to date have prospectively examined the effects of trauma exposure and PTSD on subsequent alcohol use (Breslau, Davis, & Schultz, 2003; Read et al., 2012; Testa, Livingston, & Hoffman, 2007). Notably, none of these studies met the inclusion criteria for the present systematic review because they did not include any assessment of drinking-to-cope. Of the 24 studies that did meet the current review’s eligibility criteria, only one was longitudinal in design (Beseler et al., 2011). However, because preliminary analyses failed to show an association between PTSD and alcohol-related outcomes, Beseler and colleagues (2011) did not include any measure of PTSD or traumatic stress in their final analyses.

Although the results of the systematic review revealed only one longitudinal study, two included studies (Simpson et al., 2014; Taylor, 2011) applied daily time-series analyses, each collecting daily monitoring assessments over the span of 7 days. Time-series analyses are similar to longitudinal analyses in that variables are examined at multiple times; however, whereas longitudinal data typically examine changes in variables over long periods of time (e.g., months or years) between multiple individuals, time-series information is a collection of observations for a single individual at multiple short and evenly spaced time intervals (e.g., every 24 hr for 7 days), and, typically, these observations are modeled as time-dependent in study analyses (Kazdin, 2003). It is interesting to note that the two studies that used time-series analyses reported conflicting results: One study provided support for the self-medication model (Simpson et al., 2014), whereas the other failed to demonstrate support for the self-medication model (Taylor, 2011). The stark contrast between these findings could likely be explained by a number of factors, including the use of disparate samples and methodology, the implications of which are discussed in more detail in the following sections.

The present systematic review revealed additional notable methodological concerns and inconsistencies that likely contribute to the current lack of substantiality of the self-medication model. For instance, provided that the self-medication model is inherently mediational by design, it would follow that the use of mediational analyses to test its validity is imperative. Surprisingly, however, of the 24 studies included in the systematic review, fewer than half included mediational analyses as part of their data analytic plan, and, when they did, it was not always with alcohol as the outcome and drinking to cope as the mediator, as the self-medication hypothesis would suggest.

Another notable threat to the internal validity of the self-medication model is the failure to standardize the operationalization of alcohol use as an outcome. This is a difficult feat given the vast heterogeneity of AUD, which presents symptoms on a continuum of severity that ranges from occasional binge drinking to chronic heavy drinking (Litten et al., 2015). In attempts to capture AUD heterogeneity, numerous validated measures have been created to reliably capture these various presentations and neurobiological research that are used to inform heuristic frameworks for personalized treatment. The inclusion of a plethora of alcohol phenotypes may potentially result in the increased credibility of the self-medication model but does pose a challenge with regard to synthesis of the literature. For example, the studies included in this review operationalized alcohol outcomes in several different ways. The frequency and/or quantity of alcohol consumption was the most commonly used assessment of alcohol use, with 41.6% ($k = 10$) of the included studies assessing the frequency or quantity of use within a specified time period, which ranged from drinks per day to drinks over a 3-month period. Alcohol use disorders and alcohol abuse symptoms were the second most commonly used method for assessing alcohol-related outcomes (37.5%; $k = 9$). However, the studies varied concerning which edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* was used for diagnostic criteria, creating further complications in that diagnostic criteria evolves with each version of the *DSM*. Finally, 20.8% ($k = 5$) of the included studies assessed alcohol use by measuring various negative consequences and problems related to drinking, such as issues of an interpersonal, occupational, and/or physical nature. In many ways, this variability in measurement is a

notable strength of the self-medication literature as it helps capture the heterogeneity of alcohol use and AUD. However, the lack of standardization between studies that claim to support the self-medication model makes the empirical certainty of the self-medication model difficult to determine.

A review of the literature also calls for consideration of the variation between sample selection criteria and subsequent sample descriptive characteristics, as these differences hold a number of methodological implications. The broad variation between studies with regard to ascertainment and sample constitution has both empirical strengths and weaknesses. For instance, the extension of this literature beyond common convenience samples (e.g., undergraduate populations) to more distinct populations, particularly those at an increased risk for both PTSD and AUD (e.g., veteran populations), is a strength of the current PTSD–AUD self-medication literature such that it increases external validity, or the extent to which the results of an investigation can be generalized, beyond the conditions of the experiment, to other populations, settings, and circumstances (Kazdin, 2003). The examination of these associations among community, college, veteran, inpatient, epidemiologic, and domestic violence populations allows for inferences to be made for a wider breadth of individuals and, consequently, may lead to more broad-reaching dissemination of prevention and intervention programs. The same strength could be said for the variability among other important factors, such as sex and race/ethnicity. However, as is commonly seen across various literature, the studies that used undergraduate samples were composed of disproportionately more female than male participants. Additionally, multiple studies ($k = 8$) used exclusively female samples. In some instances, this was likely because the assessed trauma exposure was sexual in nature, whereas in other instances, the choice to analyze an all-female sample was unexplained. This failure to examine both sexes equally is likely problematic given that the existence of significant sex differences regarding both PTSD and alcohol is well established in both the PTSD and alcohol literature (Goldstein, Dawson, Chou, & Grant, 2012; Koenen et al., 2018). Important sex discrepancies highlight the need for consideration of sex in both sample ascertainment and data analytic plans.

Further complicating a systematic comparison of results across studies that claim support for the PTSD–AUD self-medication model is the well-known and substantiated variability in outcomes related to trauma type and timing. Certainly, there is great variability between study designs in the PTSD–AUD self-medication literature, including differences in timing between trauma exposure and assessment of alcohol and PTSD. Traumas that are experienced during childhood, for example, have been consistently shown to result in more severe PTSD irrespective of cumulative trauma load and trauma-specific characteristics, such as trauma type and subjective perception of the event (Ogle, Rubin, & Siegler, 2013). Additionally, trauma type has been shown to influence the likelihood that an individual will develop PTSD such that interpersonal traumas, such as sexual and physical assault, are more likely to result in PTSD than accidental traumas or natural disaster–related traumas (Brewin, Andrews, & Valentine, 2000).

It is important to note that measures of trauma exposure can be highly variable, with assessments differing both across and within trauma types. This heterogeneity is both necessary and unavoidable as it provides a representative picture of the multiplex nature of

trauma exposure and PTSD, but it limits standardization in general. Despite substantiated effects of trauma type and timing on PTSD outcomes, only about one-third ($k = 8$) of the 24 studies identified for inclusion in the present review accounted for one or both of these potential confounds, let alone assessed for these considerations in their study design. Indeed, obtaining information about trauma type and timing history is time-intensive and arguably burdensome on the participant. However, trauma type and timing have considerable effects on PTSD and, most likely, alcohol use and are, therefore, crucial to consider when attempting to investigate the veracity of the self-medication model.

Finally, given the common comorbidity between PTSD and other internalizing disorders, such as anxiety and depression (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), as well as the notion that the drinking-to-cope self-medication model postulates that individuals may develop problematic drinking behaviors due to a tendency to drink to cope with negative internal experiences (Khantzian, 1997), it is plausible that individuals with PTSD may be drinking to cope with more than PTSD symptoms, making the frequent lack of consideration of these potential confounding variables noteworthy. As discussed, the field as a whole is prone to relying on the assumption that individuals with PTSD are using substances or alcohol to cope with their PTSD symptoms; however, individuals with PTSD may be drinking to cope with other internalizing phenotypes characterized by negative internal experiences, such as depression and anxiety. Indeed, like PTSD, both depression and anxiety commonly co-occur with AUD (Kessler, Chiu, Demier, Merikangas, & Walters, 2005). This comorbidity highlights the need for consideration of other phenotypes commonly comorbid with PTSD for which drinking may alleviate negative internal experiences, consistent with the self-medication model.

Given that the basic premise of the self-medication model is that PTSD influences the development of AUD via drinking to cope with unpleasant symptoms of PTSD, failure to explicitly test trauma-specific drinking-to-cope is an irrefutable gap in the current literature. Because trauma-specific drinking-to-cope may likely help explain the common co-occurrence between PTSD and AUD, further work is needed to create a gold standard measure for self-reported trauma-specific drinking-to-cope, which would serve not only to improve methodology by generating reliability and validity but would also improve treatment and prevention efforts by increasing the ability to target individuals with PTSD who may be at an increased risk for developing problematic alcohol use and AUD.

In addition to operationalizing trauma-specific drinking-to-cope, another methodological suggestion that would significantly increase the veracity of the commonly assumed self-medication model would be to increase the amount of longitudinal research investigating this model. Given the basic causal premise of the self-medication model, the use of longitudinal data to conduct mediational analyses would increase understanding with regard to if and how much variance in the association between PTSD and AUD is explained by trauma-specific drinking to cope, which would ultimately provide validated, empirical support for the model. Correlated mediation models, specifically, could allow for the examination of indirect effects of trauma-specific drinking-to-cope while accounting for more generalized drinking-to-cope measures, such as the DMQ (e.g., Hawn et al., 2020). Moreover, the inclusion of drinking-to-cope motives and alcohol expectancies into daily

diary and EMA study designs would enable the assessment of daily patterns of drinking-to-cope with symptoms of PTSD in real time. It is notable that only two of the 24 included studies used EMA or daily diary approaches, thereby excluding studies that have added meaningfully to the self-medication literature by assessing daily PTSD–alcohol use patterns, but not coping motives, from our review (e.g., Possemato et al., 2015).

Additionally, the inclusion of relevant factors, such as race, ethnicity, trauma type, and trauma timing would substantially improve the PTSD–AUD self-medication literature and likely result in more consistent findings across studies. One explanation for the inconsistency of the current literature is that these crucial confounding variables are scarcely accounted for, resulting in skewed findings. This is exemplified by the commonly supported finding that only a percentage of individuals go on to develop problematic alcohol use following PTSD onset (Kilpatrick et al., 2003), indicating that there is likely an array of confounding risk factors necessary to explain the existence of alcohol abuse in association with PTSD. This restates the need for this association to be understood in the context of a multifactorial model. To that end, studies should extend additional risk factors to include other psychiatric conditions, particularly given that alcohol abuse is associated with other psychiatric disorders, such as depression and anxiety (Brown et al., 2001). Moreover, given evidence for a moderate overlap in genetic variance between PTSD and AUD (Sartor et al., 2011; Xian et al., 2000), genetically informed research regarding the self-medication model is warranted. Investigations into the shared genetic risk and biological underpinnings of comorbid PTSD and AUD would help to further elucidate common etiological pathways underlying PTSD, AUD, and intermediate trauma-specific drinking-to-cope, which is imperative to the development of effective prevention and treatment programs.

Despite the high prevalence of comorbid PTSD and AUD, there is an unsettling sparsity in empirically based treatment for comorbid PTSD–AUD. In fact, the results of a systematic review and meta-analysis of psychological interventions for comorbid PTSD and SUD in general demonstrated that the quality of evidence found in the extant literature is low and that high attrition rates exist across most treatment interventions (Roberts, Roberts, Jones, & Bisson, 2015). Similarly, a Veterans Affairs (VA) consensus panel (Department of Veterans Affairs, 2009) concluded that there is currently no “gold standard” treatment for comorbid PTSD–AUD, imploring that further research into and development of alternative approaches for treating these comorbid conditions be undertaken. In addition to informing much-needed evidence-based treatment modalities, increased understanding of the underlying mechanisms between PTSD and AUD (i.e., trauma-specific drinking-to-cope) would allow for early screening and identification of trauma-exposed individuals who are at risk for developing comorbid PTSD–AUD and, hopefully, help to ameliorate the current public health crisis.

Both high prevalence estimates (Grant et al., 2015; Koenen et al., 2018) and associated negative sequelae of comorbid PTSD–AUD (Blanco et al., 2013; Ipser et al., 2015; Read, Brown, & Kahler, 2004; Shorter et al., 2015) highlight the need for a better understanding of the PTSD–AUD comorbidity. To the best of our knowledge, this was the first systematic review of the self-medication model in relation to PTSD. The results of the systematic review suggest that the literature to date does, overall, provide evidence for the self-medication hypothesis but that empirically rigorous investigation of the hypothesis is lacking

due to numerous methodological limitations, as discussed previously. Future research that seeks to provide sound evidence for the self-medication hypothesis should focus on incorporating a measure to assess drinking to cope with trauma-specific symptoms, testing mediational hypotheses using longitudinal data, and comprehensively assessing and including key covariates in their analyses.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1**Summary of Measures Used in Studies Included in the Systematic Review**

Measure	<i>k</i>	Sample items
DMQ-Cope	10	Decide how frequently your own drinking is motivated by each of the reasons listed below. <ul style="list-style-type: none"> To forget your worries. To cheer you up when you are in a bad mood.
Modified DMQ-R	2	Decide how frequently your own drinking is motivated by each of the reasons listed below. <ul style="list-style-type: none"> Coping-Anxiety subscale: To relax; To reduce my anxiety Coping-Depression subscale: To cheer me up when I'm in a bad news; Because it helps when I am feeling depressed
P-AEQ	2	Positive subscale: After a few drinks, my flashbacks about past traumatic events would decrease. Negative subscale: After a few drinks, my flashbacks about past traumatic events would increase.
AEQ	1	Relaxation & Tension-Reduction subscale: Alcohol enables me to fall asleep more easily.
CEOA	4	Tension-Reduction subscale: After a few drinks of alcohol, I would be more likely to feel calm.
Brief COPE	4	Substance Use subscale: I've been using alcohol or other drugs to make myself feel better; I've been using alcohol or other drugs to help me to get through it.
Reasons for Drinking Scale	1	Coping with Negative Affect subscale: To feel better when down on myself; To avoid sadness.
Subjective measure of drinking to cope	1	Have you used drugs/alcohol for trauma-related problems?

Note. CEOA = Comprehensive Effects of Alcohol questionnaire; Modified DMQ-R = Modified Drinking Motives Questionnaire-Revised; DMQ-Cope = Drinking Motives Questionnaire, Coping subscale; AEQ = Alcohol Expectancy Questionnaire; (P-)AEQ = (PTSD-)Alcohol Expectancy Questionnaire.