Efficacy and Possible Mechanisms of Change in Written Exposure Therapy in Undergraduates with PtSS: A Randomized Trial of Wet Compared to Trauma-Focused Expressive Writing

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ABSTRACT

EFFICACY AND POSSIBLE MECHANISMS OF CHANGE IN WRITTEN EXPOSURE THERAPY IN UNDERGRADUATES WITH PTSS: A RANDOMIZED TRIAL OF WET COMPARED TO TRAUMA-FOCUSED EXPRESSIVE WRITING

Robyn A. Ellis, Ph.D.
Department of Psychology
Northern Illinois University, 2022
Holly Orcutt, Director

Trauma exposure is common, with a 90% lifetime endorsement rate in adults in the United States and a majority of first-year students reporting exposure to trauma prior to college. A minority of individuals go on to develop PTSD, with higher rates on college campuses (9-12.4%) than lifetime estimates (7%). Existing evidence-based practices for PTSD have demonstrated efficacy in reducing PTSD symptoms (PTSS), but critiques of evidence-based psychotherapies (EBPs) for PTSD include high levels of drop-out and barriers to access. Written exposure therapy (WET) is a brief promising intervention for PTSS with a growing literature of evidence suggesting efficacy with smaller dropout-rates and long-term treatment gains. There is little known about the mechanisms of action in WET the efficacy of the protocol alone (i.e., studies done outside of the supervision of the treatment founders). The current study sought to extend the scope of inquiry on WET through examining efficacy and possible mechanisms of change in WET compared to the protocol WET was derived from (expressive writing [EW]). Sample included nontreatment-seeking trauma-exposed undergraduates with elevated PTSS. Results suggested both WET and EW were associated with decreases in PTSS and depression symptoms. Contrary to expectations, no group differences in outcomes were detected. Differences between WET and EW in proposed mechanisms emerged, specifically evidence of
extinction processes occurring in WET alone. Further, both emotion regulation and posttraumatic cognitions demonstrated a dose-response relationship with changes in PTSS, but temporal precedence of proposed mechanisms could not be established due to limitations in statistical approach. Findings are interpreted in context with recent investigations into mechanisms of WET, adding to the call for more innovation in proposed change agent as well as greater examination of process-based metrics of interventions for posttraumatic recovery.
EFFICACY AND POSSIBLE MECHANISMS OF CHANGE IN WRITTEN EXPOSURE THERAPY IN UNDERGRADUATES WITH PTSS: A RANDOMIZED TRIAL OF WET COMPARED TO TRAUMA-FOCUSED EXPRESSIVE WRITING

BY
ROBYN A. ELLIS

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A DISSERTATION SUBMITTED TO THE GRADUATE SCHOOL IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE DOCTOR OF PHILOSOPHY

DEPARTMENT OF PSYCHOLOGY

Doctoral Director:
Holly Orcutt
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This project would not have been possible without the support of so many. First, I would like to thank my participants. Without you this project would never have gotten off the ground. I am in awe of your willingness to be vulnerable and the resiliency I was privileged to witness throughout our work together. Collecting data during a global pandemic was challenging, but there were many days your writing was a much-needed reminder of why I am passionate about this work.

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Thank you to my friends and family for your support through the ups and downs of this project, and especially the bad jokes about acronyms. Thank you for listening to me rant and rave
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DEDICATION

To the strong women in my family who have endured exceptional circumstances and chose love and family; your strength has been with me every step of the way
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Exposure to trauma is both a global problem as well as a more localized problem for higher education. Trauma exposure is common, with 90% of individuals reporting lifetime exposure to an A1-criterion traumatic experience (i.e., actual or threatened death, serious injury, or sexual violence) over their lifetime, but the vast majority do not develop posttraumatic stress disorder (PTSD; Kilpatrick et al., 2013). In fact, only about 7% of those exposed will go on to develop PTSD. Trauma exposure rates are similar on college campuses in incoming college freshman classes. One study of trauma exposure and PTSD rates in first-year college students found that 52.2% reported lifetime exposure to trauma (Boyraz et al., 2016) and another found 66% lifetime exposure rates in female undergraduates (Read et al., 2011). In addition, the prevalence of PTSD rates in freshman undergraduates appears to be comparable, if not higher, than population prevalence rates (i.e., 7%; Kilpatrick et al., 2013), with rates estimated at 9% (Read et al., 2011) and 12.4% (Boyraz et al., 2016). Given the rates of traumatic exposure and PTSD on college campuses, PTSD is a relevant concern for higher education institutions.

The prevalence of PTSD on university campuses may be of concern due to the potentially detrimental consequences of PTSD on the functioning of students. PTSD is associated with other psychiatric comorbidities such as depression and substance use (e.g., Brady et al., 2000), as well as poor physical health outcomes such as greater pain, more medical conditions, and lower self-reported health-related quality of life (Atwoli et al., 2015). Of particular relevance to academic
institutions, PTSD has also been linked with negative academic outcomes such as lower GPA and academic drop-out (Bachrach & Read, 2012; Boyraz et al., 2016). It appears that PTSD influences not only the mental and physical health of undergraduate students but may be an important factor in the decision to leave the university without having completed a degree program (Boyraz et al., 2016). Thus, understanding the development and maintenance of PTSD, as well as relevant interventions for PTSD, may be of importance for this population.

Development and Maintenance of PTSD

There are many theories that seek to explain the development and maintenance of PTSD due to the curious discrepancy between those who are exposed to trauma and the minority of individuals who subsequently develop PTSD. Theoretical consensus indicates PTSD results from a failure to recover from exposure to trauma, as the symptoms of PTSD are largely considered natural in the immediate wake of a trauma but tend to dissipate for the vast majority (e.g., Bonanno & Mancini, 2012; Pietrzak et al., 2014). Current definitions of PTSD characterize the disorder by four types of symptoms: re-experiencing symptoms of the traumatic event (e.g., intrusive memories, nightmares); hyperarousal (e.g., hypervigilance, enhanced startle); changes in cognitions and emotions regarding the self, others, and the world (e.g., “the world is completely dangerous”); and avoidance of reminders of the trauma both internally (e.g., thoughts, feelings) and externally (e.g., people, places, situations; American Psychiatric Association, 2013).

Confounding an understanding of this diagnostic label, presentations of PTSD symptoms can vary significantly, with 636,120 possible combinations of these symptom clusters that meet diagnostic criteria (Galatzer-Levy & Bryant, 2013). Thus, many theories have emerged to help
better elucidate this heterogeneous disorder. While a review of all theories of PTSD is outside the scope of this proposal, two of the well-established theories, cognitive theory (Ehlers & Clark, 2000) and emotion processing theory (Rauch & Foa, 2006), will be reviewed. Both theories have different emphases in terms of the factors affecting development and maintenance of PTSD, and as such, it is possible that each theory explains part of the etiology of PTSD.

Cognitive Theory

The cognitive theory of PTSD (Ehlers & Clark, 2000) argues that PTSD arises after a traumatic event due to two factors: (1) appraisals of the traumatic event or what happened as a result of the event and (2) individual factors leading to poor organization of and integration of the trauma memory into other autobiographical memories. In the appraisal of the event, Ehlers and Clark (2000) argue that those who develop PTSD do not identify the traumatic event as time limited, such that the perception of threat is ongoing. This appraisal is argued to come from overgeneralizing appraisals (e.g., “the world is completely dangerous”) as well as appraisals regarding how the individual acted during the event (e.g., “I should have fought back more”) and appraisals of the trauma sequelae, including the development of symptomatology (e.g., “I am losing control”). These appraisals are associated with a host of negative emotions that may reinforce the perception of continued threat in the absence of actual threat. Further, the nature of the traumatic memory being poorly integrated into other autobiographical memories leads to re-experiencing aspects of the trauma, along with its associated appraisals and emotions, thus reinforcing the perception of ongoing threat. The strategies an individual utilizes to cope with these re-experiencing symptoms may inhibit change in either the appraisals or nature of the traumatic memory. For example, the veteran who feels uncomfortable in crowds may believe
they are always dangerous and thus avoid crowded places, inhibiting the updating of this appraisal (Ehlers & Clark, 2000). Therefore, to treat PTSD according to the cognitive theory of PTSD, the appraisals must be updated, and maladaptive coping strategies must be modified to aid in the elaboration of the trauma memory.

There is empirical evidence that supports the cognitive theory of PTSD (Ehlers & Clark, 2000). While a comprehensive review of the literature is outside the scope of this chapter, there have been several prospective studies that provide compelling evidence for Ehlers and Clark’s (2000) theory. In one prospective study of recent (i.e., within the past 4 months) physical and sexual assault survivors ($N = 57$), PTSD symptoms, maladaptive coping, and cognitive factors, such as peritraumatic cognitions (e.g., I mentally gave up) and negative appraisals of peritraumatic reactions, were measured at six and nine-month follow-ups (Dunmore et al., 2001). Their results identified peritraumatic cognitions, negative appraisals of initial symptomatology and other’s responses, avoidant coping, and perception of unrepairable change predicted PTSD symptoms prospectively at both six- and nine-month follow-ups while controlling for gender and severity of assault (Dunmore et al., 2001). A more recent study recruited survivors of violent assault or motor-vehicle accident from the ER ($N = 828$) to prospectively test Ehlers and Clark’s (2000) model utilizing path analysis (Beierl et al., 2019). Their model included several waves of measurement, and results support the main tenets of cognitive theory. Specifically, negative appraisals ($\beta = .39$), trauma memory disorganization ($\beta = .31$), and avoidant coping ($\beta = .12, .16$, for safety behaviors and cognitive avoidance, respectively) at one-month posttrauma.

---

1 Waves of measurement: (1) peritraumatic processing while in the ER or up to 10 days after the event; (2) appraisals of the event, traumatic memory disorganization, avoidant coping (i.e., safety behaviors), and cognitive avoidance coping strategies (e.g., rumination, thought suppression) at one month following the trauma; (3) and measurement of PTSD symptoms at two-week and six-month follow-ups.
prospectively predicted PTSD symptoms at 6 months with good model fit ($\chi^2[11] = 14.74$, CFI = 1.00, RMSEA = 0.02 [0.00–0.05], SRMR = 0.02) and explained 52% of the variance in the model (Beierl et al., 2019). Further, initial PTSD symptoms at two weeks did not explain more variance in the model once included, bolstering the support for cognitive factors in the development of PTSD.

Ehlers and Clark (2000) offer a cognitive framework for understanding how an individual may develop PTSD as well as how those symptoms are maintained despite efforts to cope. The empirical evidence supports Ehlers and Clark’s (2000) argument that negative appraisals, memory disjointedness, and efforts to cope contribute significantly to the development and maintenance of PTSD (e.g., Beierl et al., 2019). Although these findings are methodologically rigorous and compelling, it is unclear if they are the only factors that may explain the development and maintenance of symptoms or may represent a handful of important factors in the etiology of PTSD. As such, another prevailing theory of PTSD with sustained empirical support may offer an alternative perspective on processes that enable the development and maintenance of symptoms.

**Emotion Processing Theory**

Emotion processing theory (EPT; Foa & Kozak, 1986) builds on Lang’s (1979) bioinformational theory of fear. The bioinformational theory of fear posits that fear is represented as a memory structure that includes the associated stimuli and responses that are related to that fear (e.g., running away, increased heart rate) as an adaptive mechanism to identify danger or threat; when one element of the network is activated it triggers the whole fear network, therefore alerting the individual to the perceived danger (Lang, 1979). Emotional
processing theory further suggests that anxiety disorders have underlying pathological fear networks associated with them. Pathological fear structures are manifested similarly to the fear structures described above but are highly sensitive to activation, are difficult to change, and the associated elements contained in the structure may not reflect reality (e.g., an association between men with brown hair and danger). For PTSD, EPT proposes that after a trauma, beliefs that the world is completely dangerous and the self is unable to cope underlie the development of PTSD symptoms and pathological fear structures. Thus, in treating PTSD, according to EPT, in order to modify the pathological fear structure, the structure must be activated and then contradictory, realistic information must be integrated into the fear structure to replace the pathological associations (Foa & Kozak, 1986).

Unlike cognitive theory for PTSD, empirical investigations into EPT are hindered by the unobservable theorized processes which it is posited to function through (e.g., activation of the fear network). Foa and Kozak (1986) identified three observable suggestions as proxies for the unobservable phenomena: initial fear activation (IFA), within-session habituation (WSH) of the fear response, and between-sessions habituation (BSH) of the fear response. A recent meta-analysis ($N=21$) examined the literature on these three phenomena in the context of exposure therapy to evaluate the support for EPT (Rupp et al., 2017). The results of their meta-analysis suggest significant moderate relationships between BSH and treatment outcomes ($b_0=.35, p < .01$), as well as WSH and treatment outcomes, although this relationship was trending towards significance ($b_0=.54, p = .09$), and no relationship between IFA and treatment outcomes (Rupp et al., 2017). The authors conclude that these findings offer highly limited support for EPT, further suggesting the mechanism of action may not be habituation, but rather, habituation may be a side-effect of other mechanisms operating in exposure.
While the empirical evidence may be limited, the literature may further be restricted in studying the etiology of PTSD through the lens of EPT due to the nature of the unobservable phenomena. Given the overlap between cognitive theory and EPT, some of the empirical literature supporting cognitive theory may also support aspects of EPT. Specifically, Beierl and colleagues’ (2019) findings that trauma memory disorganization and avoidant coping prospectively predict PTSD symptoms support EPT, as EPT argues the trauma memories’ disorganized nature is one of the factors that leads to the development of PTSD, and the avoidance maintains the fear network. Given the shared factors, it may be that EPT and cognitive theory are not theoretically competing, but rather place emphasis on factors differently. Future research is needed to critically evaluate both theories within a prospective design before stronger conclusions can be made on the etiology of PTSD.

One metric of the aforementioned theories may be their utility in furthering understanding of the development of PTSD, but another may include informing how to best intervene if symptoms fail to remit. Many treatments for PTSD have been developed and empirically tested, but the frontline treatments for PTSD with the strongest evidence base are rooted in EPT and cognitive theory for PTSD.

Evidence-Based Treatment for PTSD

Across the Department of Veteran’s Affairs/Department of Defense, the International Society for Traumatic Stress Studies, and the American Psychological Association, prolonged exposure (PE) and cognitive processing therapy (CPT) are two of the treatments with strongest recommendations for the treatment of PTSD based on the empirical evidence (American Psychological Association, 2017; Berliner et al., 2019; Department of Veteran’s Affairs and
Department of Defense, 2017). These PTSD treatments have been identified as having substantial empirical support and consistent efficacy across multiple studies, populations, and time. As such, only these “gold-standard” treatments will be reviewed for the purposes of this proposal.

**Frontline Treatments for PTSD**

**Prolonged Exposure**

Developed by Edna Foa, Elizabeth Hembree, and Barbara Rothbaum (2007), PE is a primarily exposure-based treatment for PTSD based in EPT. Prolonged exposure involves two types of exposures: *in vivo* exposures and *imaginal* exposures, completed over the course of 9-12 weeks. The in vivo exposures are completed outside of session and involve systematically approaching people, places, and situations that are relatively low risk but have been avoided nonetheless because they are reminders of the traumatic experience and/or seem “dangerous” (e.g., being in a crowd). The imaginal exposures involve repeated verbal recitation of the trauma memory in the present tense within session for 45-60 minutes out of the 90-minute session. The imaginal exposure is recorded, and daily listening is assigned for homework to promote habituation to the traumatic memory. Within the framework of EPT, these exposures function to modify the fear structure by activating it through the exposure (i.e., inducing fear) and introducing corrective information, consequently breaking erroneous associations between stimuli (e.g., certain smells, loud sounds) and danger or fear (Foa & Kozak, 1986; Rauch & Foa, 2006).

PE has been designated a frontline or “gold-standard” treatment for PTSD due to the substantial literature that supports its efficacy (e.g., Kline et al., 2018; Powers et al., 2010; Watts
et al., 2013). Powers and colleagues (2010) conducted a meta-analysis specifically on the efficacy of PE for the treatment of PTSD. Their review included 13 clinical trials, with a total of 658 participants, and offered strong support for PE. Specifically, compared to control groups, PE demonstrated a large effect on reducing PTSD at immediate posttreatment (Hedge’s $g = 1.08$) as well as a medium to large effect (Hedge’s $g = 0.68$) at follow-up (ranging from one month to 12 months). Additionally, similar results were found for secondary outcomes such as depression and anxiety (Powers et al., 2010). Similarly, a recent meta-analysis of 32 randomized control trials (RCTs; $N = 3,399$), found significant effects from pretreatment to follow-up (i.e., 6, 12, and 20 months) in the active psychotherapy conditions, which included PE. Further, this meta-analysis found that exposure-based treatment demonstrated greater effect sizes compared to other active psychotherapy in the posttreatment to follow-up period ($d = 0.27$, $d = .05$, respectively), despite no significant differences found pre- to posttreatment (Kline et al., 2018). The literature base for the efficacy of PE is strong, but it also has identified limitations, such as high levels of drop-out (e.g., Hembree et al., 2003; Imel et al., 2013), as well as concerns regarding access to providers who offer PE or other evidence-based psychotherapies (e.g., Yasinski & Rauch, 2018). These limitations are not unique to PE and will be discussed in depth later. While PE is one of the “gold-standard” treatments, many individuals may prefer to examine their thoughts and beliefs over enacting change focused on their behavior; for those, cognitive processing therapy may be preferred.

**Cognitive Processing Therapy**

Cognitive processing therapy (CPT) was initially developed in 1992 by Patricia Resick and Monica K. Schnicke to treat women who had been sexually assaulted (Resick & Schnicke,
In its original format, the treatment consisted of twelve 60-minute weekly sessions, primarily focused on challenging trauma-relevant cognitions that are maladaptive within the themes of safety, trust, intimacy, power/control, and esteem. These cognitions are identified as assimilated thoughts (i.e., thoughts about the trauma that are distorted, such as “I should have prevented the car accident”) and over-accommodated thoughts (i.e., beliefs about the self, the world, or other people that are extreme due to the traumatic exposure, such as “No one can be trusted”). In addition, treatment is bookended by a written impact statement of how the trauma has affected the individual within the five themes previously listed, as well as their beliefs as to why the trauma occurred. Individuals also complete two trauma narratives, initially termed “written exposures,” that were theorized to function similarly to the trauma narrative exposures seen in PE. In the updated version of CPT (Resick et al., 2017), the written exposures are considered optional and are termed “written accounts.” This shift in language reflects the assertion that the accounts are missing necessary components of exposure (e.g., repetition of the account and habituation), and therefore the accounts are utilized for the identification of additional maladaptive beliefs rather than an exposure to the trauma memory (Resick et al., 2017). This change was informed by a dismantling study of CPT, the results of which indicated the written narratives were not necessary for the reduction of PTSD symptoms (i.e., no significant differences in treatment outcome were found between those who did and did not write the accounts; Resick et al., 2008). The current protocol that includes the written accounts is identified as CPT +A. For the purposes of this review, given the relative recency of the change from CPT to CPT +A, all of the empirical studies will be discussed together.

Similar to PE, CPT has a strong literature base indicating its efficacy in treating symptoms of PTSD (see Asmundson et al., 2019; Kline et al., 2018; Watts et al., 2013, for
reviews). A recent meta-analysis of studies examining the efficacy of CPT for adults with PTSD included 11 studies (N = 1,130) and indicated CPT decreased PTSD symptoms posttreatment 89% more effectively than inactive controls (Asmundson et al., 2019); similar findings were found for non-PTSD outcomes, including depression, with CPT outperforming inactive controls by 84%. Further, the treatment gains appeared to last, with those having completed CPT reporting less PTSD and non-PTSD symptomatology with moderate to large effects (Hedge’s g = 0.90) at follow-up, ranging from one to 12 months across studies. Similar to findings from other meta-analyses that examine empirically supported treatment for PTSD, there were no significant differences between CPT and other active controls (i.e., dialogical exposure therapy, memory-specific training, PE, or written exposure therapy) at follow-up, but CPT appeared to outperform active controls at immediate posttreatment with a small to moderate effect, but this analysis only included four studies (Asmundson et al., 2019). As previously reported, Kline and colleagues (2018) also included CPT in their meta-analysis, providing further support for the long-term efficacy of CPT. Watts and colleagues (2013) also found large effects for the efficacy of CPT in the treatment of PTSD in another meta-analytic review of treatments for PTSD, including psychotherapy as well as psychopharmacological interventions. Despite sufficient literature supporting the efficacy of these frontline treatments for reducing PTSD symptomatology, there are documented concerns related to their dissemination and efficacy, such as high levels of drop-out (Hembree et al., 2003; Imel et al., 2013; Najavits, 2015) and limited access (Osei-Bonsu et al., 2017; Rauch & Rothbaum, 2016; Yasinski & Rauch, 2018).
Problems with EBPs for PTSD

The extant literature supports that the majority of individuals who engage in evidence-based psychotherapies (EBPs) for PTSD demonstrate substantial treatment gains, suggesting effective psychotherapies exist for PTSD (e.g., Asmundson et al., 2019; Powers et al., 2010). While the development of effective treatment is a critical step in intervening for individuals with PTSD, treatment efficacy is only relevant if individuals have access to providers who offer EBPs for PTSD and are able to complete the treatment as empirically evaluated. Concerns regarding high levels of drop-out and limited access to EBPS for PTSD pose significant problems for the extant EBPs for PTSD, and thus, calls for innovation in the PTSD treatment literature to address these issues have been made, including brief interventions to increase engagement and use of technology to improve access in rural areas (i.e., telehealth; Yasinski & Rauch, 2018).

Drop-Out

Drop-out is a major concern for trauma-focused EBPs for PTSD, with several meta-analyses suggesting drop-out rates hovering around 20% (Hembree et al., 2003; Imel et al., 2013) and more recent evidence suggesting slightly higher rates around 30% (Berke et al., 2019). In their meta-analytic review of 25 studies, Hembree and colleagues (2003) sought to examine whether drop-out rates in exposure-based treatment were higher than in other treatments for PTSD. Their review compared four active treatment types: exposure alone (e.g., PE), stress inoculation training or cognitive therapy (e.g., CPT), exposure plus cognitive therapy, or eye movement desensitization reprocessing (EMDR). Control conditions included relaxation training, supportive counseling, hypnotherapy, psychodynamic therapy, biofeedback, and
waitlist. Controlling for number of sessions, Hembree and colleagues found significant differences in drop-out rates were found between the active and control conditions ($\chi^2 [4, N = 1,582] = 14.77, p < .01$), but no significant differences in drop-out rates were found between the active conditions. Average drop-out rates ranged from 18.9% (EMDR) to 26.9% (exposure plus cognitive therapy) for all active treatment conditions (Hembree et al., 2003).

Building on Hembree and colleagues (2003), Imel et al. (2013) sought to further investigate drop-out rates in active PTSD treatment using 42 studies that directly compared two interventions. The mean drop-out rate across 1,850 patients was 18.28%, with substantial heterogeneity in the reported drop-out rates across studies (Imel et al., 2013). Follow-up analyses suggested that group-based treatment increased drop-out rates by 12% ($b = .12, p = .009$), and length of treatment also increased drop-out rates; specifically, for each added session, drop-out rates increased 1% ($b = .01, p = .009$). In line with the findings of Hembree and colleagues (2003), there were no statistically significant differences in drop-out rates across the active treatments. Further, having a trauma focus did not significantly impact drop-out rates among the active treatments, but when compared to present-centered treatments (PCT), trauma-focused treatments demonstrated significantly greater drop-out rates ($LOR = 0.70, p = .009$). Taken together, the results of these meta-analyses suggest that drop-out rates for trauma-focused psychotherapy are high, about 20%, but are comparable across trauma-focused treatment approaches.

More recent research on drop-out rates in trauma-focused treatment for PTSD appears to be in line with the previously reported meta-analytic reviews, with a slightly higher rate of drop-out. In a review of three RCTs for active-duty military service members, comparing variations of
CPT, PCT, and PE\(^2\), Berke and colleagues (2019) identified an overall drop-out rate of 30.7%, with a mean number of 5.53 sessions attended by those who dropped out (SD = 2.93). In addition, consistent with the findings of Imel et al. (2013), trauma-focused treatments demonstrated higher drop-out than PCT, and group-based treatment demonstrated lower attendance rates (Berke et al., 2019). Thus, it appears more recent evidence reinforces the findings of the last decade suggesting that about a quarter of individuals who initiate trauma-focused psychotherapy will discontinue treatment prematurely.

In an effort to examine whether individuals are dropping out due to treatment gains (i.e., leaving treatment because they feel better), Berke and colleagues (2019) examined PTSD symptoms between those who dropped out versus completed treatment. Their results suggest treatment completers were more likely to see symptom improvement \((d = 0.49, p < .001)\) and were less likely to see stagnation or worsening of symptoms over the course of treatment \((d = 0.35, p < .01)\). Further, there were not statistically significant differences between individuals who dropped out of treatment early (before session 5) and late (after session 6) in PTSD symptom change, and 75.1% of those who did not complete the final session of treatment either experienced no change in symptoms or worsened (Berke et al., 2019). Based on these results, it appears those who drop out of treatment are not doing so because of early treatment gains, further highlighting the problem of drop-out from existing trauma-focused treatments and bolstering the call for innovation in trauma-focused psychotherapies (e.g., Rauch & Rothbaum, 2016), but the literature is mixed. Szafranski et al. (2017) found in a combined sample from two

\(^2\) The three RCTs compared in Berke et al. (2019) included comparisons of: (1) CPT and PCT (Resick et al., 2015); (2) CPT delivered in two, 90-minute group sessions weekly and typically delivered CPT (i.e., 60 minutes weekly; Resick, Wachen, et al., 2017); and (3) 10 individual 90-minute sessions of PE delivered over 8 weeks, 10 individual 90-minute sessions of PCT delivered over 8 weeks, and 10 individual 90-minutes sessions of PE delivered over 2 weeks (Foa et al., 2018).
RCTs of CPT \((N = 321)\) that 37.74\% of those who dropped out of treatment reported clinically significant change in PTSD symptoms, and 35.85\% were considered to have ended in a “good state” (i.e., no significant impairment or low scores on PTSD symptom measures; Szafranski et al., 2017). These results are limited to CPT but also indicate more research is needed to determine why individuals prematurely leave treatment. While the drop-out rates for trauma-focused psychotherapy are high, access to therapists who are able (and willing) to provide evidence-based treatment for PTSD also poses a significant barrier to the treatment of PTSD.

**Access**

Reports from the nation-wide dissemination within the Veteran’s Administration (VA) hospital system indicate that in 2009, 96\% of VA facilities reported offering CPT or PE, with 72\% reporting that they provide both (Karlin et al., 2010). These numbers are promising but do not appear to tell the whole story, as estimates of veterans engaged in PE or CPT within the VA have been reported to be as low as 6\% (Watts et al., 2014), with other estimates of 13\% (Lu et al., 2016) and 33.9\% in a residential setting (Shiner et al., 2018). While substantial training is required to provide CPT and PE, the VA has devoted much effort to training providers within the context of their dissemination efforts, making the small percentage of veteran’s engaged in EBPs for PTSD particularly concerning.

Furthermore, community settings, unlike the VA system, lack the structure to accurately estimate widespread access to EBPs, but there is some evidence to suggest access to trained providers outside the VA is limited. In a sample of licensed therapists in Wyoming \((N= 51)\), a majority of whom were master’s level \((n = 31, 60.8\%)\), 98\% reported using cognitive-behavioral approaches for the treatment of anxiety-based disorders, but less than half (48.9\%) reported
using exposure for the treatment of PTSD (Hipol & Deacon, 2013). In a stratified sample of licensed mental health providers in Texas (n = 463), over two-thirds reported treating veterans with PTSD, but only about half (47.52%) reported prior training in CPT, PE, or other EBPs for PTSD (i.e., EMDR or SIT) and only 45.14% reported using an EBP for the treatment of PTSD (Finley et al., 2018). Specific to the use of PE and CPT, estimates are lower, with only 23.33% reporting use of CPT and 12.10% using PE, despite the vast majority (81.66%) indicating positive attitudes towards outcomes for EBPs for PTSD and 50% indicating awareness of clinical practice guidelines (Finley et al., 2018). These findings are consistent with another recent study utilizing a stratified sample of licensed mental health providers in Texas and Vermont who report treating patients with PTSD at least occasionally. This study found that use of CPT was generally greater than PE for both psychologists and master’s-level clinicians, but less than 20% of providers reported being trained in or using a guideline-recommended treatment (i.e., EBP) for PTSD consistently (Finley et al., 2019). These numbers illustrate a serious problem with current EBPs for PTSD, with the majority of community providers lacking the necessary training or, despite having training, inconsistently providing the recommended treatments for PTSD.

**Summary**

The evidence suggests that while EBPs for PTSD, including PE and CPT, have a strong empirical base to support their widespread use, even within VA settings with more instrumental support for training, they are underutilized. Furthermore, utilization is even worse in community settings. Adding to the implementation issues, drop-out rates are consistently high, with evidence suggesting that even if an individual has a provider willing to use an EBP for PTSD, there is a significant chance that individual may drop out of treatment prematurely. As a result of these
identified EBP shortcomings, there has been an emphasis on innovation in evidence-based practice in recent years, with a particular emphasis on the development of internet-based or self-guided care as well as minimal-contact psychotherapies that may increase access to care due to less training and burden on patient and clinician (Yasinski & Rauch, 2018). More research is needed to further investigate the efficacy of these options.

Mechanisms of Change in PTSD Treatment

To further innovate existing EBPs and pursue novel approaches, an understanding of why the current treatments for PTSD work, or the mechanisms that produce change in treatment, is necessary. While there is a large body of research devoted to examining mechanisms of change, how “mechanism” is defined is central to evaluating potential mechanisms and their potential utility. Kazdin (2007) offers a conceptual framework for the evaluation of proposed mechanisms and the methodological approaches necessary to identify and test these mechanisms. Given the relative inconsistency in definitions across the literature in discussion of mechanisms of change in psychotherapy, Kazdin’s (2007) operationalization of mechanisms, mediators, and moderators will be utilized.

Mediators are defined as variables or factors that statistically account for the relationship between an independent variable and dependent variable, and as such, mediators may serve to identify potential mechanisms but may not be mechanisms in and of themselves. A mechanism of psychotherapy is defined as the steps, processes, or events that occur to produce change in psychotherapy (Kazdin, 2007). In other words, a mechanism explains why the treatment causes the change in symptoms and is more specific than mediators of treatment outcomes. In order to identify a mechanism of treatment, Kazdin (2007) reports multiple criteria are necessary,
including: (1) strong associations between intervention and the mediator; (2) specificity of relationship between intervention, mediator, and outcome; (3) consistency of results and replication; (4) experimental manipulation; (5) timeline for temporal relevance (i.e., mediator must precede outcome); (6) gradient (i.e., dose-related response, such that more of the mediator results in greater effect); and (7) plausibility (i.e., theoretical backing). Informed by Kazdin’s (2007) conceptual framework, the empirical literature on mechanisms in PTSD treatment will be reviewed.

**Hope/Hopelessness**

Hope, or the inverse – hopelessness, has been discussed as a potential “mechanism” by which treatment for PTSD may reduce PTSD symptoms. Hope has been shown to predict symptoms of PTSD as well as to change over the course of EBPs for PTSD (see Gallagher, 2017, for review). The empirical evidence demonstrates fulfillment of several of Kazdin’s requirements for a mechanism, such as strong associations between potential mechanism and outcome, as well as some consistency in empirical findings. For example, in one study of change in hopelessness over the course of PE and CPT, changes in hopelessness partially mediated the relationship between treatment modality and change in PTSD treatment, with greater effects for CPT ($R^2 = .25$; Gallagher & Resick, 2012). The authors interpreted these results to suggest change in hopelessness may be a potential mechanism of change in PTSD symptoms for CPT. In accordance with Kazdin (2007), it appears the empirical support for hope as a mechanism of change is missing several of the specific criteria, including a lack of specificity as well as gradient and experimental manipulation, suggesting that hope may be a mediator of treatment response, but not a mechanism of change. Further, there is some evidence to suggest that hope at
the midpoint of treatment is a better predictor of PTSD symptoms than vice versa, suggesting changes in hope may precede changes in PTSD symptoms (Gallagher & Resick, 2012), further suggesting a mediational effect. Yet, more research is necessary to elucidate the potential mechanistic status of hope in PTSD treatment.

**Neuroticism**

Gallagher (2017) identified neuroticism as a potential mechanism of change in PTSD treatments, but the evidence presented may be better conceptualized as a moderator of treatment effects. Moderators of treatment outcome are defined as factors that influence the direction or magnitude of the relationship between an independent variable and a dependent variable, such that moderators may also identify areas where mediators or mechanisms may be at play to produce different effects at the moderator level. Neuroticism has been shown to be related to PTSD and has been identified as a potential risk factor for the development of PTSD, but many features necessary to determine neuroticism as a mechanism of change are missing. It is clear that neuroticism is related to PTSD symptomatology (see Gallagher, 2017), but the empirical evidence does not include experimental manipulation, nor does it specify neuroticism as a necessary means for treatment gains, evidence that would point to neuroticism as a mechanism. More research is necessary in order to better understand how neuroticism may impact treatment outcomes, as well as identifying whether neuroticism’s relationship with PTSD and treatment outcomes is the result of other treatment mechanisms (i.e., the mechanism of action is also changing neuroticism concurrently with PTSD symptomatology).
Emotion regulation has demonstrated strong associations with PTSD severity and has been shown to be malleable (McLean & Foa, 2017). Gallagher (2017) identifies emotion regulation as a transdiagnostic mechanism, citing several meta-analytic reviews which identify emotion regulation as an important risk factor as well as a maintaining factor of PTSD (e.g., Aldao & Nolen-Hoeksema, 2010). Further, empirical evidence suggests emotion regulation predicts PTSD symptom improvement in the context of CBT for PTSD with small to moderate effects (partial $\eta^2 = .07$; Hinton et al., 2009; Sharma-Patel & Brown, 2016), providing some support for emotion regulation as a mediator of treatment response. Given the understanding of how emotion regulation impacts day-to-day functioning, as well as the preponderance of negative emotions and avoidance-based behaviors inherent to PTSD, it is plausible that changes in emotion regulation serve as a mechanism through which PTSD treatment functions. Thus, according to Kazdin (2007), emotion regulation currently holds many of the criteria needed to be identified as a possible mechanism, including strong associations with PTSD and PTSD treatment outcomes (McLean & Foa, 2017), consistency and replication in the literature (e.g., Aldao & Nolen-Hoeksema, 2010), experimental manipulation (Sharma-Patel & Brown, 2016), and plausibility (Gallagher, 2017). However, future research is necessary to determine whether emotion regulation demonstrates the remaining criteria to reach mechanism status, including temporal precedence in changes in emotion regulation and PTSD symptoms, specificity of emotion regulation in concurrent examination with other mechanisms, and a dose-response relationship with PTSD symptoms.

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3 Effect size reported in Sharma-Patel and Brown (2016); no effect size reported for Hinton et al. (2009).
Cognitions/Cognitive Reappraisal

Proposed by Gallagher (2017), cognitive reappraisal has promising empirical support for its role as a potential mechanism in PTSD treatment. Through the lens of both EPT (Rauch & Foa, 2006) and cognitive theory of PTSD (Ehlers & Clark, 2000), posttraumatic cognitions are central to the development of maintenance of PTSD. Further, PE and CPT both identify changes in thoughts as mechanisms through which the treatments function (Foa et al., 2007; Resick et al., 2017; Resick & Schnicke, 1992). A dismantling study of CPT, which ultimately led to the updated CPT protocol, identified that the cognitive restructuring component (i.e., development of cognitive reappraisal skills) alone produced greater symptom reduction over time ($F[2, 183] = 4.5, p = .01$) in comparison to a group that only completed written accounts (Hedge’s $g = 0.7$) and symptom reduction comparable to the group who received both cognitive restructuring and written accounts of the traumatic event (Hedge’s $g = 0.90$; Resick et al., 2008). This dismantling study suggests that cognitive reappraisal may have a dose-response relationship with PTSD symptoms, a necessary feature of treatment mechanisms as proposed by Kazdin (2007). In an unpublished dissertation, Barnes (2017) examined changes in written narratives across treatment in CPT and WET and findings also support cognitive reappraisal as a potential mechanism of change in treatment. Specifically, changes in accommodated thoughts were found to be a predictor of PTSD severity ($R^2 = 0.37$), and changes in accommodated thinking were greater in the CPT group despite no differences in overall PTSD severity across the groups (Barnes, 2017). These findings require replication but suggest specific types of reappraisal (i.e., accommodation) may be more important for change, given that assimilation and avoidance were controlled for in the models (Barnes, 2017).
The criteria for timeline also have limited support for cognitive reappraisal as a mechanism of treatment change for PTSD. Zalta and colleagues (2014) examined changes in posttraumatic cognitions in a sample of adult females completing PE. Given their methodology of measuring both PTSD symptoms and cognitions at each session, analyses revealed changes in cognitions predicted PTSD symptom change with medium to large-effect ($d = 0.66$), but the reverse was no-significant (Zalta et al., 2014). Similar results were found by Kleim and colleagues (2013) in a study examining changes in cognitions as a mechanism of change in trauma-focused CBT utilizing latent growth modeling ($\chi^2 [196] = 255.88$, $p < .01$, CFI = .99, RMSEA = .03, SRMR = .03), with those reporting greater declines in posttraumatic cognitions demonstrating greater decline in PTSS ($r_{\text{slope PTCI, slope PDS}} = .78$, $p < .01$), but the reverse was non-significant. Thus, it appears that there is preliminary evidence that suggests the timeline for changes in cognitions is consistent for the conceptualization of cognitive appraisal as a mechanism of change (Kleim et al., 2013; Zalta et al., 2014). In addition, meta-analytic reviews have identified that cognitive reappraisal skills are strongly associated with PTSD symptomatology (Aldao & Nolen-Hoeksema, 2010), but are frequently studied in isolation of other potential mechanisms, and as such, cognitive reappraisal lacks the necessary specificity to determine its status as a mechanism of change. Further, studies examining cognitive changes as a mechanism with other potential mechanisms of change are lacking, limiting the clarity and confidence at which one can identify cognitive reappraisal as a true mechanism of change in PTSD treatment.

**Habituation**

Habituation is defined as the reduction in subjective distress over repeated exposure to the distressing stimuli without engaging in avoidance tactics. Habituation has been identified as a
potential mechanism of change in exposure-based treatment for PTSD. According to EPT (Rauch & Foa, 2006), in order to modify the fear structure and thus decrease PTSD symptomatology, the fear structure must be activated, and change occurs through habituation to the feared stimuli while incorporating new information into the fear structure to update it. This notion has been investigated in treatment studies of PE (Bluett et al., 2014; Sripada & Rauch, 2015; Sripada et al., 2016).

Many reviews of the literature suggest that between-sessions habituation is more important for PTSD symptom reduction than within-session habituation (Sripada et al., 2016). Bluett and colleagues (2014) examined changes in subjective distress in patients \((n = 116)\) receiving PE. Results suggested that despite the majority of patients not demonstrating reliable change in distress, those who did report reliable reduction in their level of distress reported greater reduction in PTSD severity \(F[1,80] = 6.20, p = .02, \text{Hedge's } g = 0.55\). The findings of Sripada and Rauch (2015) also support the findings of Bluett and colleagues (2014) with the use of hierarchical linear modeling; results suggest symptom change pre- to posttreatment predicted the slope of subjective distress ratings across treatment sessions (i.e., between-sessions-habituation).

These empirical studies identify a strong association between treatment outcome and habituation, but the temporal sequencing of these constructs is unclear due to methodological or statistical constraints. In addition, the extant literature suggests that habituation may function as a dose response, with individuals who demonstrate greater habituation reporting greater reductions in PTSD symptoms (Bluett et al., 2014). The specificity of habituation as a potential mechanism requires further investigation, but preliminary evidence suggests that, while controlling for hopelessness, changes in subjective distress predicts changes in PTSD symptoms in those
engaged in PE (Gallagher & Resick, 2012). More research is needed to investigate the consistency of these relationships, as well as specificity and temporal sequencing of treatment gains, before habituation can be identified consistently as a mechanism of change.

**Summary of Potential Mechanisms**

In summary, potential mechanisms have been reviewed within the context of Kazdin’s (2007) criteria for mechanisms of treatment, but very few have sufficient empirical support for their identification as “mechanisms” rather than mediators of treatment response. The three mechanisms that have the most empirical support and meet the majority of Kazdin’s criteria include emotion regulation, cognitive reappraisal, and between-sessions habituation. The status of the literature may reflect an accurate perception of potential mechanisms, but these mechanisms may have garnered the most attention due to their posited role in the “gold-standard” treatments for PTSD. Gallagher (2017) also identified several other promising potential mechanisms (e.g., experiential avoidance) but concluded that these potential mechanisms did not have enough empirical evidence to evaluate them at the time of the study. More research is clearly necessary to continue to evaluate current candidate mechanisms as well as identify other potential mechanisms by which the efficacious treatments function. One way by which these mechanisms may be evaluated in the future is through the use of specific methodological recommendations of Kazdin (2007) including the incorporation of measuring outcome and mechanisms at all time points, as well as evaluating multiple mechanisms at one time.
Expressive Writing

While not listed on the VA/Department of Defense or the ISTSS guidelines as a frontline treatment for PTSD, expressive writing (EW) has demonstrated widespread physical and mental health benefits, including symptom reduction in PTSD (e.g., see Sloan et al., 2015, for review). The standard EW paradigm includes writing for 15 to 30 minutes each day for three to five consecutive days (Pennebaker, 1997). Using this standard framework, numerous studies have found positive physical and psychological health benefits for those who engage in expressive writing tasks (e.g., Frattaroli, 2006; Sloan et al., 2015; Smyth, 1998). Several meta-analyses have been conducted on the effects of expressive writing tasks for a variety of psychological and physical health outcomes (Frattaroli, 2006; Pavlacic et al., 2018; Smyth, 1998).

Smyth (1998) conducted a meta-analysis on the effect of the EW on general psychological outcomes and potential moderators. Their review included 13 randomized studies wherein the experimental group wrote about a traumatic topic and the control group was instructed to write about a neutral topic. This meta-analysis also investigated potential moderators, including population characteristics (i.e., type, gender, age), “dose” of intervention (i.e., duration of writing session, time between sessions, number of sessions), writing content (i.e., write about past or current traumatic events, recency of the traumatic event), and outcome type (i.e., psychological, physiological, general functioning and health behaviors). Overall, Smyth’s (1998) findings suggest written disclosure significantly improves functioning, with a moderate effect (weighted $d = .47$). For all outcomes except health behaviors, written disclosure appeared to be efficacious, with small to medium improvement in self-reported general

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4 This is also commonly referred to as the Pennebaker task or emotional disclosure.
functioning \((d = .33)\) and medium to large effects for physical \((d = .68)\) and psychological health \((d = .66)\). In terms of moderators, student populations reported greater overall effect sizes than nonstudents, samples with more male participants reported greater effect sizes overall, but gender did not moderate psychological or physiological outcome effect sizes, and similarly, greater spacing between writing sessions was found to be associated with greater overall effect sizes, but no differences in psychological or physiological effect sizes. Thus, this meta-analysis provides support for written disclosure as an intervention that improves overall functioning across several physical and psychological health outcomes (Smyth, 1998).

Building off the findings of Smyth (1998), Frisina and colleagues (2004) sought to investigate the effects of emotional disclosure on physical and psychological health outcomes in a psychiatric population. Their meta-analysis included nine experimental studies that utilized a written disclosure paradigm and included some qualitative measurement of health (i.e., mental, physical, general, and health behaviors) in a physically ill or psychiatric population. Consistent with the findings of Smyth (1998), but with a smaller effect, their overall findings suggest written disclosure significantly improves functioning \((d = .19)\). Diverging from Smyth (1998), Frisina and colleagues (2004) found no significant changes for psychological health, but they did find significant improvements in physical health outcomes \((d = .21)\), which may suggest differential effects of written disclosure in individuals with more severe physical or psychological problems.

The most recent meta-analysis on the effects of experimental disclosure broadly included 146 articles with a variety of conditions (Frattaroli, 2006). The results of this meta-analysis suggest that written disclosure produces positive outcomes, with small effects on average \((d = .075)\). It should be noted that this review indicated substantial heterogeneity in reported effect
sizes across the studies included, suggesting that the literature on expressive writing is highly mixed and noncohesive. As such, Frattaroli (2006) also identified several moderators that help identify factors that increased effect sizes. Studies that included individuals with greater physical health problems demonstrated greater effect sizes for self-reported health outcomes, which Frattoli interpreted as likely due to samples with healthy participants experiencing floor effects. Further, college samples reported smaller effect sizes for psychological outcomes. Environmental factors appear to influence the effect sizes for psychological outcomes, with both writing at home and increased privacy demonstrating increased effect sizes for psychological health. In terms of methodological moderators, having more disclosure sessions, more temporally recent topics of disclosure, detailed instructions, and follow-up periods of less than one-month also appear to increase effect sizes in studies of written disclosure. Of particular importance to the current proposal, Frattaroli (2006) also examined the role of trauma exposure, finding that utilizing trauma-exposed samples was associated with greater effect sizes ($r = .24$), suggesting trauma-exposed individuals may benefit particularly from written disclosure.

Taken together, the results of these meta-analyses suggest expressive writing produces consistent improvement in overall functioning, with some mixed evidence regarding effects on psychological outcomes (Frattaroli, 2006; Frisina et al., 2004; Smyth, 1998). In addition, it appears that more disclosure leads to greater effects, and timing of disclosure sessions may impact effect sizes, with mixed findings for more time leading to greater outcomes. Further, severity of symptomatology, as well as trauma exposure, appears to influence the efficacy of expressive writing. As such, the effects of expressive writing for individuals struggling with PTSD symptomatology has been investigated alongside the question of whether writing about traumatic experiences is beneficial.
Expressive Writing for PTSD

Given the demonstrated benefit of expressive writing on overall health, as well as mixed evidence for the benefits for psychological health, the literature has examined the effects, and tolerability, of expressive writing for those who have experienced trauma. Frattaroli (2006) results indicated that effect sizes for trauma-exposed samples were associated with greater effect sizes for more general outcomes (e.g., physical health, psychological health) but do not report on the tolerability of writing about traumatic experiences for those indicating a trauma history. In a study of veterans enrolled in residential treatment for chronic PTSD, Hernandez and colleagues (2005) found that veterans rated expressive writing groups as at least moderately valuable regardless of education level, other engagement in writing activities, PTSD symptom severity, and concerns about writing prior to start of expressive writing group. Further, in a study including 27 ICU nurses, Mealer and colleagues (2014) found that across writing topics that included challenges at work, feeling incapacitated, feeling conflicted, and rumination on sensitive topics, nurses reported that they would have rather focused their writing on only traumatic experiences. Thus, it appears that writing about traumatic events is tolerable as well as sometimes preferred over other expressive writing topics.

Fortunately, given the relative preference and subjective value reported for writing about traumatic experiences, the literature suggests that expressive writing also may reduce PTSD symptoms (Kloss & Lisman, 2002; Pavlacic et al., 2018; Sloan et al., 2015). In their review of the literature on narrative writing protocols for the reduction of PTSD symptoms, Sloan and colleagues (2015) reported evidence for the use of written disclosure. At the time of their review, only seven studies had been published utilizing a PTSD or probable PTSD population, and of
those, only three asked the participants to write about their trauma (Gidron et al., 1996; Possemato et al., 2011; Smyth et al., 2008). Across these studies, low drop-out rates were identified (i.e., 0 to 7%), but no significant improvements in PTSD symptoms were found (Sloan et al., 2015). Given this discrepancy with the literature on written disclosure broadly improving functioning, Sloan and colleagues suggested that these results may be related to dose of writing, as the studies that did include a trauma-focused writing prompt included three 20-minute writing sessions. This interpretation is supported by the results of Sloan and colleagues (2011), who found no significant differences between the trauma-focused and neutral writing topic groups in PTSD symptoms after three 20-minute writing sessions; they also found differences in physiological and self-reported arousal between the groups, suggesting that the trauma-focused group were more emotionally engaged and therefore, in accordance with EPT, began the process of fear network activation, but the dose was not sufficient to see changes in PTSD symptoms. This study led Sloan and colleagues to embark on a series of studies modifying the expressive writing task for PTSD ultimately resulting in the development of a new treatment for PTSD: written exposure therapy (WET; Sloan & Marx, 2019).

**Written Exposure Therapy (WET)**

**Development of WET**

Written exposure therapy (Sloan & Marx, 2019) was developed following a series of studies conducted by Sloan and colleagues intended to investigate written disclosure paradigms in relation to physiological reactivity/emotional arousal (Epstein et al., 2005; Sloan et al., 2005, 2011) and manipulations to the written instructions (Sloan et al., 2007). Epstein and colleagues
(2005) examined the role of written disclosure, gender, time, and heart-rate reactivity on depressive, stress, and anxiety symptoms and also conducted a linguistic examination of the participants’ writing. Their results suggest written disclosure reduces physical complaints, depression, stress, and anxiety symptoms in undergraduate students (n = 94) compared to a control condition asked to write about their day. The writing sessions were conducted on three consecutive days, 20-minute writing sessions per day, and outcome measures were collected at one-month follow-up. No gender differences were found in depressive, stress, or anxiety symptoms over time, but a main effect for gender was found in the linguistic analysis, such that women used more insight or causality language in their writing compared to men (F[1, 91] = 7.44, p < .01), but the effect was small (r\text{effect size} = 0.08); no Gender x Condition interaction was significant (Epstein et al., 2005). In terms of physiological reactivity, the disclosure group demonstrated greater heart-rate reactivity at the first writing session (F[2,83] = 4.31, p < .05, r\text{effect size} = 0.22), and heart-rate reactivity at the first writing session was correlated with changes in depression (r = 0.23, p < .05) and physical health complaints (r = 0.28, p < .05). The results of this study further support the literature implicating written disclosure in reduction of psychological symptoms as well as indicate the influence of physiological reactivity in the reduction of these symptoms (Epstein et al., 2005).

Building off the findings of Epstein and colleagues (2005), Sloan and colleagues (2005) examined the effects of emotional arousal in written disclosure comparing trauma-related written disclosure protocols in a sample of trauma-exposed undergraduate students (n = 79). In this study, both subjective and objective measures of emotional arousal were collected from participants randomized into three writing conditions: repeated trauma disclosure (i.e., writing about the same trauma each session), different trauma disclosure (i.e., writing about different
traumatic experiences each session), and control (i.e., writing about trivial everyday events). Consistent with Epstein and colleagues (2005), writing sessions were each 20 minutes, completed over three consecutive days. Outcome measures, including PTSD symptoms, depression, physical symptoms, and number of sick days, were assessed at baseline, four and eight weeks postintervention. Compared to the other two groups, the repeated trauma disclosure group demonstrated a clinically significant decrease in PTSD symptom severity ($F(4, 75) = 8.07, p < .01, r_{\text{effect size}} = 0.87$) and depression ($F[4, 75] = 7.02, p < .01, r_{\text{effect size}} = 0.80$) at both four- and eight-week follow-up; similar findings were also found for physical health complaints and number of sick days, suggesting the repeated trauma disclosure was most efficacious in reducing symptomatology across several domains of functioning (Sloan et al., 2005).

Further, while both trauma groups (i.e., repeated single-trauma disclosure and different-trauma disclosure) showed significant habituation from the first to last session (based on salivary cortisol), the different-trauma disclosure group did not significantly differ from the control group on outcome measures, providing evidence that writing about the same traumatic event multiple times may be necessary to obtain significant change in symptomatology (Sloan et al., 2005). One explanation for the discrepancy between habituation and changes in outcomes between the repeated and different trauma groups may be due to the level of physiological reactivity at the first session. Greater physiological reactivity at the first session was related to PTSD symptoms ($r = -.42, p <.05$), although differences between the repeated and different trauma groups approached significance in physiological arousal at session one ($p = .06$). Similarly, self-reported arousal at session one was significantly related to changes in PTSD and depression symptoms ($r = .39, p <.05$; $r = .40, p <.05$, respectively), and self-reported unpleasantness at session one was associated with reductions in PTSD severity ($r = .42, p <.05$). These findings point to
physiological and self-reported arousal as important factors in treatment response, and also provide evidence for the use of repeated trauma disclosure of a singular event over allowing individuals to vary in choosing a traumatic event for their writing each day (Sloan et al., 2005).

While the results of Sloan and colleagues (2005) provide compelling evidence for the use of repeated disclosure of a single traumatic event, their study was not conducted within a sample of individuals with elevated PTSD symptoms. Thus, Sloan and colleagues (2007) sought to examine whether written disclosure instructions had any effect on psychopathology in a sample of undergraduates \((n = 82)\) with moderate PTSD symptomatology. In this study, three writing conditions were evaluated: emotional expression (i.e., with as much emotion as possible), insight and cognitive assimilation (i.e., meaning of the event and how the event has impacted their life), and control (i.e., factual account of their day). Writing sessions were conducted over three days, each writing session lasting 20 minutes; follow-up sessions were conducted one month after the final writing session. The emotional expression group demonstrated significant improvement in comparison to a control group in both PTSD and depression symptoms \((F[3,81] = 14.03, p < .01)\), as well as a significant reduction in physical health complaints compared to both the control group and the insight group \((F[3,81] = 16.01, p < .01)\). Similar to the results of Sloan and colleagues (2005), heart-rate activity in the emotional expression group was greater at the first session compared to both other groups. Changes in heart rate between the first and last sessions were found for both the emotional expression and insight groups \((t(27) = 4.52, p < .001, r_{\text{effect size}} = .65; t(27) = 4.52, p < .001, r_{\text{effect size}} = .65\), respectively), suggesting habituation to the disclosure task over time. Further evidence for habituation was found for both active groups, with change in HR score decreasing over time for both the emotional expression and insight groups. In terms of self-reported arousal and unpleasantness, similar patterns emerged, with the
emotional expression group reporting the greatest unpleasantness and arousal compared to both other groups ($F(2, 79) = 56.23, p < .001, r_{\text{effect size}} = .64; F(2, 79) = 39.50, p < .001, r_{\text{effect size}} = .58$, respectively) and the insight group reporting greater unpleasantness and arousal compared to the control group ($p < .01$). These findings, along with the findings of Sloan and colleagues (2005), suggest self-report of arousal is consistent with physiological arousal, and written disclosure instructions impact the effects of the intervention on psychopathology, such that greater emotional engagement predicts greater change in symptoms of PTSD and depression (Sloan et al., 2007).

Next, Sloan and colleagues investigated the efficacy of a trauma-focused written emotional disclosure as an intervention in a sample of undergraduates who met diagnostic criteria for PTSD (Sloan et al., 2011). Undergraduate students who met criteria for PTSD ($n = 42$) were randomized into either the written emotional disclosure group (WED; i.e., writing about same traumatic experience with emotions) or control group (i.e., factual account of how they spent their day devoid of emotion). Measures of PTSD, depression, heart rate (i.e., objective physiological arousal), and self-reported ratings of physiological arousal were collected. A linguistic analysis was also conducted as a manipulation check for percentage of emotion (e.g., sad, afraid) and cognitive insight words (e.g., think, because) utilized in the writings; the manipulation appeared successful with participants in the WED condition reporting greater negative emotion words and insight words on average compared to the control group. Consistent across many studies of written disclosure, writing sessions lasted 20 minutes and were conducted on three consecutive days (Sloan et al., 2011).

The results of Sloan and colleagues (2011) diverged from the extant literature on written disclosure. That is, no significant effects for condition were found for PTSD symptoms or
depression. Further, there was a significant effect for time, such that all participants (regardless of condition) saw improvements in PTSD symptoms from baseline to one-month follow-up ($F[1,40] = 25.18, p < .01, r_{effect size} = .61$), suggesting, despite differences in language, the intervention appeared equivalent to a factual account of the participant’s day. Although no differences were found between groups on symptomatology, Sloan and colleagues found a significant main effect of condition for physiological arousal, with the WED condition demonstrating greater physiological arousal at session one compared to the control condition ($F[1,41] = 4.28, p < .05, r_{effect size} = .31$). Also inconsistent with prior findings, no differences were found in reductions in heart rate from first to last session for either condition, suggesting participants may not be habituating to the increased physiological arousal over time in the WED group; no changes in the control group were hypothesized. In terms of self-reported arousal, findings indicated individuals in the WED condition felt greater unpleasantness ($F(1,41) = 20.51, p < .01, r_{effect size} = .58$) and arousal ($F(1,41) = 22.87, p < .01, r_{effect size} = .60$) compared to the control group.

Given the pattern of findings in relation to arousal at the first session, both self-reported arousal and heart rate, and previous findings on the relationship between emotional arousal and reductions in symptomatology (Epstein et al., 2005; Sloan et al., 2005, 2007), it is surprising that no differences in symptomatology were found between the active and control condition in Sloan et al. (2011). For this reason, Sloan et al. (2011) posited that given their sample meets full diagnostic criteria for PTSD, three consecutive days of 20-minute written disclosure may not be sufficient to see expected treatment gains in a population with more severe psychopathology. In addition, Sloan and colleagues identified the lack of psychoeducation and treatment rationale as a potential reason for their nonsignificant differences between their groups (Sloan et al., 2011),
foreshadowing changes made to the WED protocol to create written exposure therapy (Sloan & Marx, 2019).

Following the Sloan et al. (2011) study, Sloan and colleagues made substantial changes to the WED protocol and began referring to their protocol as “written exposure therapy,” arguing that the changes differentiated the protocol from previously utilized expressive writing and written disclosure tasks. These changes included expanding the writing sessions to 30 minutes and adjusting the frequency of writing sessions to weekly sessions for five weeks to increase the “dose” of treatment based on the findings of Sloan et al. (2011). Further psychoeducation about PTSD and rationale for the benefits of writing about traumatic experiences was added to the protocol. Based on their findings suggestive of habituation as a predictor of treatment response (Epstein et al., 2005; Sloan et al., 2005, 2007), Sloan and colleagues concluded that writing about the traumatic experience may function as an exposure, and thus written exposure therapy (WET) was coined to allow for further empirical evaluation of the protocol.

**Empirical Evidence for WET**

Between the development of WET and the proposal for the current study, there were five published studies examining the effects of WET in four unique samples (Mealer et al., 2014; Sloan et al., 2012, 2013, 2018; Thompson-Hollands et al., 2018). The first empirical test of WET was conducted by Sloan and colleagues as an RCT in a sample of adults with a primary diagnosis of PTSD related to a motor-vehicle accident. Participants \( n = 46 \) were randomized into either WET or a waitlist control condition. Diagnostic status of participants was identified using the Clinician-Administered PTSD Scale (CAPS; Weathers et al., 2001), and participants reported on their subjective arousal and unpleasantness after each writing session. In addition,
measures of treatment credibility and satisfaction with treatment were collected. Outcome assessments were completed at baseline and 18 weeks postbaseline for all participants, as well as six months posttreatment for those in the WET condition. Results of the multilevel model included a significant Time x Condition interaction such that those in the WET condition reported greater reductions in their PTSD symptoms over time ($B = 17.96$, 95% CI[-13.04, -22.89]) with a medium to large effect ($r = .46$) compared to the waitlist control. Said differently, participants in the WET condition showed statistically significantly greater symptom reduction than the waitlist group. At both 6- and 18-week follow-up, fewer participants in the WET condition met diagnostic criteria for PTSD compared to the control condition ($\chi^2 = 37.66, p < .01$), with only 5% of participants in the WET condition meeting criteria at 6-week follow-up and 0% at 18 weeks, compared to 88% and 67% of participants in the control group at 6- and 18-week follow-up, respectively. Participants in the WET condition also reported significant decreases in their self-reported negative affect ($t[19] = 8.89, p < .01$, $r_{\text{effect size}} = .89$) and arousal ($t[19] = 5.22, p < .01$, $r_{\text{effect size}} = .77$). In addition, the drop-out rate for the WET condition was 9% ($n = 2$); one participant reported dropping out due to symptom improvement (Sloan et al., 2012). These results suggest that Sloan and colleagues (2011) may have been correct in their assertion that a higher dose of writing may be necessary for individuals with greater PTSS severity to see significant symptom reduction.

While WET appeared to be efficacious in reducing PTSD symptomatology in adults with motor-vehicle accident-related PTSD (Sloan et al., 2012), Sloan and colleagues next sought to investigate the efficacy of WET in a veteran population (Sloan et al., 2013). Sloan and colleagues (2013) examined changes in CAPS scores after engagement in WET in a sample of seven veterans at pretreatment, posttreatment, and three-month follow-up. Index traumas for the
sample were majority combat related, but one veteran indicated a military sexual trauma as their index event. In this study, only one veteran dropped out due to beliefs that the treatment could not help him. Results suggested clinically significant improvements in PTSD severity for the majority of participants (57%) at immediate posttreatment, with almost all participants (85.7%) indicating significant improvements by three-month follow-up. Further, five of the seven participants (71.4%) did not meet diagnostic criteria for PTSD following engagement in WET; authors note that the two participants who continued to meet criteria posttreatment had the most severe PTSD symptoms at the start of treatment (CAPS scores of 90 and 88). While their sample size was small, Sloan and colleagues’ (2013) findings provide preliminary evidence for the efficacy of WET in a veteran population, but a larger sample and more stringent comparison is necessary to determine if WET offers an efficacious alternative to established EBPs for PTSD. Since the proposal of the current study, data on the rollout of WET in the VA healthcare system has emerged offering greater support for WET in the treatment of PTSD in veterans (LoSavio et al., 2021). Utilizing intent to treat (ITT) analysis of 227 Veterans representing 24 VA sites, WET demonstrated significant reductions in PTSD symptoms (M_PCL-5 = 12.13; d = .86), depression symptoms (M_PHQ-9 = 2.55; d = .47) and functional impairment (M_B-IPF = 8.29; d = .36) from pre- to posttreatment (LoSavio et al., 2021).

Recognizing the need for WET to be validated on a larger scale with a more stringent comparison, Sloan and colleagues conducted a direct comparison of WET with CPT (Sloan et al., 2018). In a PTSD-positive sample of 126 treatment-seeking adults with heterogeneous trauma histories, participants were randomized into either the WET or CPT+A conditions. Assessments of PTSD symptoms utilizing the CAPS-5 were completed at 6, 12, 24 and 36 weeks postbaseline. No significant differences in sample characteristics were found across the two
conditions, including age, income, gender, veteran status, ethnicity, or PTSD symptom severity at baseline. Analyses of drop-out indicated participants were significantly more likely to drop out of the CPT condition prematurely ($\chi^2 = 12.84, p < .01$), with 31.7% of the sample dropping out prior to session six and a total rate of 39.7% drop-out overall for the CPT group, compared to 6.3% total drop-out in the WET group (Sloan et al., 2018).

While the participants in the WET group were less likely to drop out compared to the CPT group, no differences were found in symptom reduction between the groups. Specifically, both conditions demonstrated significant reductions in their PTSD symptoms over time ($B = -2.33$, SE=0.35, $t = -6.68$, $p < .01$; $B = -3.43$, SE=0.44, $t = -7.70$, $p < .01$, for WET and CPT, respectively). Further, no significant differences in CAPS severity scores were found between the WET and CPT groups at any time point; the CPT group showed a larger effect in CAPS score at the 24-week follow-up ($d = 0.29$), but this effect was not statistically significant nor maintained at the 36-week follow-up. In addition, in both groups, less than half the participants ($<48\%$) met criteria for PTSD at the 24- and 36-week follow-ups. This study was the first to directly compare WET to an extant EBP for PTSD, the results of which suggest WET is equally efficacious to CPT for the treatment of PTSD, with significantly less drop-out (Sloan et al., 2018).

Given the relative noninferiority of WET in comparison to CPT in maintaining short-term treatment gains in PTSD symptoms (Sloan et al., 2018), an investigation of long-term outcomes and other symptomatology was warranted. Thompson-Hollands and colleagues (2018) investigated the long-term effects of WET in comparison to CPT on PTSD and depression symptoms utilizing the same sample from the original noninferiority trial of WET (Sloan et al., 2018). Utilizing multilevel modeling (MLM), Thompson-Hollands et al. examined PTSD
symptom severity at 60 weeks posttreatment initiation and found WET maintained noninferiority status in comparison to CPT. Said differently, there were no differences found in the amount of PTSD symptom reduction between the WET and CPT groups 60 weeks post-treatment initiation. Further, less than 30% of the sample met criteria for PTSD at 60 weeks, and there were no significant differences between the groups in likelihood of meeting criteria at 60-week follow-up ($\chi^2 = 0.36, p = 0.55$). Discrepant from the finding related to PTSD symptoms, a significant Time x Condition effect was found for depressive symptoms ($b = -0.70, SE = 0.33, t = -2.13, p = 0.034$), such that depression symptoms declined more rapidly in the CPT group compared to the WET group, but the difference was small at 60 weeks ($d = 0.19$). Although there were differences in depression symptoms, further analysis of the noninferiority data suggest that depression is not a moderator of treatment outcome (Thompson-Hollands et al., 2019). In fact, the only moderator of treatment outcome found was full scale IQ (FSIQ), but this was only the case for the CPT condition, suggesting CPT may be more effective with those with higher FSIQs, but FSIQ does not appear to affect treatment outcomes in WET (Marx, Thompson-Hollands, et al., 2021: Thompson-Hollands et al., 2019). Although promising, more research is necessary to disentangle for whom treatments may be more effective.

Most recently, in light of the COVID-19 pandemic, considerations have been made to the provision of WET via telehealth. Worley and colleagues (2020) reported on a virtual facilitated collaborative program to train clinicians in WET within the VA healthcare system that was underway at the COVID-19 pandemic onset. Results prior to COVID-19 suggested a drop-out rate of 20.8%; after the onset of COVID-19, drop-out rate was 27% ($n = 31$) of the 115 enrolled, of whom slightly under half ($n = 15$) reported dropping out due to factors related to telehealth (e.g., technical problems, personal preference) or factors related to COVID-19 (e.g., childcare).
Further, the pandemic also appeared to impact treatment fidelity to weekly sessions, such that more than one week elapsed between treatment sessions due to both clinician (e.g., childcare, illness) and patient (e.g., technological access, illness) factors (Worley et al., 2020). Of note, data from the VA rollout of WET suggest the overall drop-out rate was 25.27%, but those who were completing treatment via telehealth were significantly more likely to complete treatment (21.3% drop-out rate for telehealth vs. 34% for in person), with the majority of patients completing treatment within the manualized five sessions, but 28.01% (n = 58) patients who completed treatment required more than five sessions (LoSavio et al., 2021).

In sum, with the growing empirical evidence, WET appears to be a promising intervention for the treatment of PTSD (Sloan et al., 2012, 2013, 2018) with long-term, sustained treatment gains (Thompson-Hollands et al., 2018). There is also limited empirical evidence for WET in the reduction of depression symptoms (Thompson-Hollands et al., 2018), but more research is needed to enhance understanding of the efficacy of WET as well as potential outcomes other than PTSD symptomatology.

Proposed Mechanisms of Change in WET

While WET appears to be a promising new treatment for PTSD, there is limited empirical insight into why WET works or the mechanisms that drive treatment response. Sloan and Marx (2004) identified three potential mechanisms by which written disclosure tasks are proposed to function: emotional inhibition, cognitive adaptation, and emotional engagement/exposure. Like the other mechanisms reviewed in this proposal, the proposed mechanisms of WET will be evaluated through the lens of Kazdin’s (2007) criteria for a mechanism.
Emotional Inhibition

Sloan and Marx (2004) indicated at the time of their review that there was little empirical support for the role of emotional inhibition (i.e., restriction of negative affective states) as a mechanism for change in written disclosure paradigms. They suggest that the empirical literature on emotion regulation suggests that the suppression of emotions increases sympathetic activation with downstream consequences on physical and psychological outcomes. The previously reviewed literature on emotion regulation and PTSD treatment implicates emotion regulation as a mediator and potential mechanism of change (e.g., McLean & Foa, 2017). Further, a more recent review of the emotional disclosure literature showed similar outcomes for cognitive-behavioral writing, which includes both expressive writing and components of CBT (i.e., exposure and cognitive restructuring), compared to CBT for PTSD (see Pascoe, 2016, for review). Therefore, it may be that what is discussed as emotional inhibition could be conceptualized as operating through other mechanisms, such as emotion regulation, habituation (as the mechanism in exposure) or cognitive reappraisal. Thus, given the relative lack of empirical support for emotional inhibition at this time, as well as potential other mechanisms identified throughout the empirical literature, it also appears emotional inhibition lacks sufficient criteria to be considered a mechanism (Kazdin, 2007).

Cognitive Adaptation

As with other treatments for PTSD, cognitive adaptation or reappraisal has been implicated as a potential mechanism of change for WET (Sloan & Marx, 2004). Sloan and Marx describe cognitive adaptation as working to reconcile the incongruence between information
learned through traumatic experiences and internal models. Thus, through writing, an individual is able to structure, organize, and integrate the traumatic memory, which may not have happened spontaneously. In the context of WET, this is theorized to occur through the repeated traumatic accounts as well as explicit instruction to evaluate how the event has impacted one’s life.

The extant literature on cognitive adaptation may support cognitive reappraisal as a mechanism by which WET functions (e.g., Kleim et al., 2013; Zalta et al., 2014). There is some empirical evidence that writing about traumatic events is associated with a decrease in intrusive thoughts, which is also associated with increases in working memory, but the relationship is correlational (Sloan & Marx, 2004). In a study of written disclosure manipulating the organizational component of cognitive adaptation, three groups of undergraduate students (n = 116) wrote one of the following: a trauma narrative, a fragmented trauma narrative (e.g., lists), or a narrative on a trivial topic (Smyth et al., 2001). Results of this study suggest the trauma narrative group was less likely to report illness-related restriction of activities than the other groups (F[2,107]= 8.17, p <.01) but were also more likely to engage in avoidant thinking (F[2,107]= 3.70, p <.05). The authors interpreted the increase in avoidance but reduction in illness-related restriction as potentially due to the “dose” being insufficient (Smyth et al., 2001). While the results of Smyth and colleagues (2001) offer some potential support for cognitive adaptation as a mechanism, their study lacks specificity and associations with PTSD symptomatology, which are the first two criteria for mechanisms laid out by Kazdin (2007).

Offering evidence for the missing criteria, Wisco and colleagues (2013) examined cognitive emotion regulation strategies in the sample from the motor-vehicle accident RCT of WET (n = 46). They found a significant interaction between positive reappraisal and condition (β = .38, t (36) = 4.24, p <.01) as well as an interaction between putting-in-perspective (i.e.,
attempts to place the traumatic event in context of one’s whole life) and condition (β = .34, \( t (36) = 2.85, p < .01 \)) in predicting changes in PTSD symptoms over time, such that higher use of these strategies was associated with greater decreases in PTSD symptoms in the waitlist group. Curiously, for those in the WET group, greater use of positive reappraisal was associated with smaller changes in PTSD symptoms (Wisco et al., 2013). These results establish a strong association between cognitive reappraisal and PTSD symptoms but appear to point to a moderation effect as a better description of the nature of this relationship, thus limiting the support for cognitive adaptation as a mechanism of change in WET.

Further complicating a discussion of cognitive changes, Barnes (2017) utilized the data from the noninferiority trial of WET (Sloan et al., 2018), whose results may offer support for cognitive reappraisal as a candidate mechanism. While the following results reflect an unpublished dissertation, they offer a methodologically rigorous approach to examining changes in cognitions through use of an established qualitative coding scheme (CHANGE coding; Hayes et al., 2007) and therefore may offer relevant insights, despite lacking peer-review status. Both impact statements and two narratives were collected from the CPT group, and four narratives were collected from the WET group. All writing was coded for multimodal trauma network activation in four domains: cognitive (perceptions of self, others, and the world), emotional, somatic, and behavioral; in addition, writing was coded for avoidance and three other cognitive variables. The three cognitive variables included assimilation, defined as changes to the trauma memory to fit pre-existing beliefs (e.g., I am to blame because of what I was wearing); accommodation, defined as integration of information from the traumatic experience into the belief system in an adaptive way (e.g., Some people cannot be trusted, but not everyone is
untrustworthy); and overgeneralization, defined as changing the belief system to reflect what happened during or as a result of the trauma (e.g., All men are dangerous).

Controlling for word count and detail, no main effects for assimilation or overgeneralization were found, but an interaction effect was found between condition and overgeneralization such that only those in the CPT group demonstrated significant reductions in overgeneralization ($F[1, 79] = 5.78, p = 0.02$). In addition, both groups showed significant increases in accommodation over the course of treatment, but similar to overgeneralization, there was a significant interaction with condition such that the CPT group reported larger increases in accommodation over time ($F[1, 79] = 23.39, p < .01$). Despite significant interaction effects, there were no differences between conditions in predictive strength of accommodation, despite greater accommodation predicting greater reduction in PTSD symptoms at 12-week follow-up (Barnes, 2017). Taken together, Barnes (2017) suggests accommodation plays a role in PTSD symptom reduction in WET. Their findings offer greater specificity than in previously described studies of cognitive changes as a mechanism of change, given the concurrent examination of other potential mechanisms (e.g., avoidance, trauma network activation). Largely missing is a discussion of the temporal relationship between changes in cognitions and PTSD symptoms, and thus, due to methodological limitations, conclusions cannot be drawn.

Given the limited empirical data in the extant literature on cognitive reappraisal in WET as a potential mechanism, more research is necessary to better understand the nature of the relationship. Further, in line with Kazdin (2007) and informed by Barnes’s (2017) results, cognitive reappraisal may be a promising avenue in the investigation of mechanisms of change in WET as it has demonstrated some of the criteria for a mechanism, with two notable deficits: temporal sequencing and replication, deficits which future research should consider in methodology, specifically.
Exposure/Habituation

Sloan and Ma (2004) also identified exposure as a potential mechanism by which written disclosure may function. Given the description of conditioned responding by Sloan and Marx (2004), a more accurate term for the proposed mechanism is “habituation.” Sloan and colleagues argue that the written disclosure paradigm may function as an exposure to the traumatic memory, providing corrective information and extinction of the unconditioned stimulus (i.e., the trauma) and the conditioned stimulus (i.e., trauma memory) producing reductions in intrusions and avoidance (Sloan & Marx, 2004). As previously reviewed, there is a substantial literature implicating habituation as a potential mechanism of trauma-focused psychotherapy (e.g., Bluett et al., 2014; Sripada et al., 2016), but Sloan and Marx (2004) invoke extinction processes as the proposed mechanism. The goal of exposure is extinction, whereas the process by which extinction is achieved is through habituation; thus, habituation appears to be the more accurate terminology in reference to identifying mechanisms of treatment response (Abramowitz, 2013).

The limited literature on mechanisms of change in WET may also support habituation as a candidate mechanism of change. There is only one study published, to the author’s knowledge, which investigates habituation processes in the context of WET (Wisco et al., 2016). Wisco and colleagues (2016) examined WET compared to waitlist control while also collecting self-reported and physiological arousal (i.e., interbeat-interval cardiac activity). Their results suggested participants who demonstrated greater reductions in their PTSD symptoms also reported significant differences in self-reported arousal between sessions \( B = -0.01, t(19) = 3.0, p < .01, pr = .57 \). Similarly, individuals who reported greater physiological arousal at the first writing session demonstrated greatest reduction in PTSS \( B = -0.31, t(20) = -2.58, p = .02, pr = \)
These results provide evidence for a dose-response relationship between habituation and PTSD symptom reduction, but much like the extant literature on habituation as a mechanism in treatment, this study lacks the methodological requirements to determine temporal relationships between habituation and PTSD symptom reduction. Future research is necessary to replicate these findings as well as demonstrate temporal precedence of habituation before habituation can be identified as a mechanism with confidence in accordance with Kazdin’s (2007) guidelines.

While the direct measurement of emotional engagement found in Wisco and colleagues (2016) is methodologically preferred, Barnes (2017) also offers some preliminary evidence for habituation as a mechanism for WET utilizing post hoc coding of the written narratives for emotional engagement. Based in EPT, the trauma network activation serves as the first step in extinction and therefore theoretically should precede any habituation processes (Rauch & Foa, 2006). Results of Barnes (2017) suggest multimodal network activation, coding described previously, was associated with greater decreases in CAPS scores ($\beta = -.29, p = .05$), but based on moderation analyses, only in the WET condition ($\beta = .28, p = .05$). These results may suggest some specificity in trauma network activation (i.e., a proxy for habituation) as related to, and a potential mechanism of, WET compared to CPT. These results are also replicated in the peer-reviewed publication using the same data suggesting negative emotion expression in the initial trauma narrative predicted sudden gains (i.e., rapid stable decline in symptoms) in WET and CPT (Sloan, Thompson-Holland, et al., 2021).

**Summary**

Altogether, the limited literature on WET appears to mirror the larger extant literature on mechanisms in trauma-focused treatment, pointing to cognitive reappraisal and habituation as
garnering the most empirical support as potential mechanisms of treatment. The WET literature requires more research on potential mechanisms to fulfill the consistency and replication requirement of the mechanism framework laid out by Kazdin (2007). In addition, methodologies including measurement of proposed mechanisms and outcomes at all time points to determine temporal sequencing and evaluation of multiple mechanisms are lacking in the WET literature. Of note, it is encouraging that the WET literature appears to mirror the findings of the extant literature on possible mechanisms of change, but it is unclear if this is a self-fulfilling prophecy, with the mechanisms historically being investigated informing the WET literature’s examination of mechanisms, or if these investigations reflect the current status of theory. These assertions may also not be mutually exclusive, thus a call for more research is emphasized based on these and other limitations of the extant literature.

In support of these assertions, since the proposal of the current study, a more rigorous examination of the proposed mechanisms of action in WET has been published utilizing the data from the noninferiority trial of WET compared to CPT (Lee et al., 2021). Lee and colleagues examined temporal sequencing of PTSD symptoms and proposed mechanisms (i.e., extinction and cognitive reappraisal) through measuring within- and between-sessions changes in emotional arousal, valence, and posttraumatic cognitions at each WET session and at 6-, 12-, 24-, 36-, and 60-week follow-ups. Findings suggested that between-sessions postwriting change in emotional valence preceded changes in PTSD symptoms but was not associated with treatment outcomes. Further, changes in posttraumatic cognitions did not temporally precede changes in PTSD but were correlated and appeared to change in concert with PTSD symptoms. As such, authors concluded that the proposed mechanisms may be correlates rather than mechanisms of action in
WET (Lee et al., 2021). Thus, the call for more research into the mechanisms and more novelty in examination of mechanisms is bolstered.

**Limitations of the WET Literature**

While there has been substantial work done in the past decade to develop and empirically validate WET as a treatment for PTSD, there are several limitations to the literature as it stands to date. One of the largest limitations of the WET literature at the time of the current study’s proposal was that almost all the literature in the development and empirical testing of WET has been conducted under the supervision of Denise Sloan, Ph.D., the lead investigator in creating the WET protocol (Epstein et al., 2005; Sloan et al., 2013; Sloan & Marx, 2004, 2019; Sloan et al., 2005, 2007, 2011, 2012, 2015; Thompson-Hollands et al., 2018, 2019; Wisco et al., 2013, 2016). At the time, there had been only one study examining the efficacy of WET without the involvement of Dr. Sloan (Gallagher et al., 2018), which did not find significant decreases in PTSD symptoms; in fact, they found slight increases in PTSS in a sample of Chinese cancer survivors, which was interpreted to be due to cultural differences. Since the proposal of the current study, two other studies have been published on the protocol without Sloan (Andrews et al., 2021; Park et al., 2021); these will be detailed later. The Gallagher et al. (2018) study, in conjunction with the severely limited literature on WET completed without Sloan’s involvement, illustrates the need for more research completed by other investigators to identify whether WET is efficacious using only the established protocol. Further, in all of the studies done by Sloan and colleagues, both doctoral and master’s-level clinicians were utilized. Given the problem of access to EBPs for PTSD, it may be beneficial for WET, given the proponent of not needing to
be provided by a doctoral-level clinician, to be studied within the context of only master’s-level providers.

Another significant limitation of the WET literature is the samples utilized to examine efficacy. The vast majority of the research on WET has been conducted on treatment-seeking adults with PTSD in the Boston area (Sloan et al., 2012, 2018; Thompson-Hollands et al., 2018, 2019); one exception is the study utilizing a small veteran sample (Sloan et al., 2013). In fact, no study on WET has utilized an undergraduate sample since the written disclosure paradigm was investigated in the development of WET. Research on varied types of samples is needed to better understand if WET is efficacious in other populations in which PTSD is prevalent. In addition, while the literature on WET is promising, with consistent evidence that WET works to reduce PTSD symptomatology, the majority of research on the efficacy of WET utilizes the same two datasets: (1) the noninferiority trial conducted by Sloan and colleagues (2018) and (2) the RCT of WET using motor-vehicle accident survivors. To illustrate this point, the following studies utilized the noninferiority trial data (not including the original study): Barnes (2017), Shiner et al. (2018), Thompson-Hollands et al. (2019), and the following studies utilized the RCT of WET using motor-vehicle accident survivors: Wisco et al. (2013, 2016). Taken together, these studies make up almost the entirety of the WET literature, and while valuable insights have been provided, additional samples are needed to increase confidence in generalizability of WET.

Since the proposal of the current study there have been several open pilot studies published utilizing WET in diverse populations (Andrews et al., 2021; Park et al., 2021). Andrews and colleagues (2021) reported on findings from a pilot study examining the efficacy of WET translated into Spanish in a Latinx immigrants (n = 20). Findings suggested significant, clinically meaningful reductions in PTSD (M = 17.06) and depression symptoms (M = 6.25),
with all but one treatment completer reporting symptoms beneath clinical thresholds for PTSD (Andrews et al., 2021). In a sample of 25 Korean patients diagnosed with PTSD, Park and colleagues (2021) found significant reductions in CAPS-5 scores, self-reported PTSD symptoms, and depression and significant improvement in global functioning pre/posttreatment. Further, at six-week follow-up, over half of study participants (60.9%) no longer met criteria for PTSD (Park et al., 2021). While these studies suggest promising results, their sample sizes are small and more research is necessary to evaluate the generalizability of WET.

Methodological limitations outside of sample characteristics also have limited the WET literature. With the exception of the noninferiority trial (Sloan et al., 2018), WET has not been tested with an active control group. While the noninferiority trial of WET versus CPT is compelling and the literature using a waitlist control demonstrates that WET works better than no treatment (Sloan et al., 2013), the literature would benefit from more active control conditions to demonstrate that WET is (a) a distinct treatment from the expressive writing from which it was developed and (b) its treatment effect is not driven by miscellaneous other factors, or common factors such as meeting with someone weekly. Both of these goals can be achieved through the use of more active control group designs. Of note, since the proposal of the current study, four study designs have been published indicating more rigorous testing of WET against other active psychotherapies, and with modifications, others are planned, specifically testing WET against CPT in active military (Sloan et al., 2020), WET + mindfulness vs. WET alone in China (Li et al., 2021), noninferiority trial of WET vs. PE (Sloan, Marx, et al., 2021), and an RCT of WET + crisis response planning for reduction in suicide risk (Marx, Fina, et al., 2021).

Finally, the creators of WET indicate that it works through mechanisms such as habituation and cognitive changes, but there is limited research investigating its mechanisms of
change. While there have been three published studies (Lee et al., 2021; Wisco et al., 2013; 2016), and one unpublished dissertation (Barnes, 2017) that have explicitly set out to investigate why changes in PTSD symptoms occur in the context of WET, more research is necessary to replicate their findings and identify other potential mechanisms of change using methodologies consistent with Kazdin’s (2007) recommendations.

Overall, more research is necessary to increase understanding of whether WET works and – if it works – for whom and why it appears to be efficacious. Thus, the current study sought to add to the literature by addressing gaps in the WET literature to address the broader problems of access and drop-out in the treatment of PTSD as a whole.

The Current Study

The current study sought to address several gaps in the literature and to answer three main research questions: (1) is WET efficacious in nontreatment-seeking undergraduates with elevated PTSD symptoms, henceforth referred to as PTSS; (2) is WET more efficacious than an expressive writing task focused on trauma; and finally, (3) what mechanisms of change are operating in WET? The current study aimed to address these questions through the examination of WET compared to a trauma-focused expressive writing task in undergraduates with elevated PTSS while measuring potential mechanisms and outcomes at all seven time points.

In reference to the first question, as reviewed previously, WET has not been examined in an undergraduate population because the studies developing WET, and the majority of the studies demonstrating the efficacy of WET, have been treatment-seeking adults in the greater Boston area. Thus, the current study offers the WET literature a novel sample to examine efficacy as well as potential support for generalizability of the treatment. Further, the current
study was the only examination of WET, to the author’s knowledge, that did not involve supervision or input by the primary investigator of the WET protocol, Denise Sloan, Ph.D., as well as the only study to be delivered solely by a master’s-level clinician, both criteria representing important factors in determining feasibility of dissemination.

Second, the current study sought to examine whether WET is distinct in efficacy from the written expressive task from which it was developed. WET has been examined against primarily waitlist control groups, excluding the noninferiority trial, and has not been examined directly against an expressive writing task. To determine the uniqueness of WET, distinct from the Pennebaker task and its adaptations, direct comparison is needed. Thus, the current study was the first to directly test WET against a trauma-focused expressive writing task and examine changes in both PTSD and depression symptoms.

Finally, the third research question seeks to identify and test potential mechanisms of change in WET utilizing a method recommended by Kazdin (2007) to identify candidate mechanisms more accurately. Informed by both the WET literature and the PTSD treatment literature broadly, emotion regulation, cognitive reappraisal, and habituation appear to have the most empirical support and to fulfill the greatest number of Kazdin’s (2007) criteria for candidate mechanisms, and as such, these mechanisms were examined as potential mechanisms of change for WET. To address methodological gaps in the mechanism literature, including examination of a single mechanism and lack of outcome measurement outside pre/post design, the current study measured both candidate mechanisms and outcome (PTSS) at every time point, allowing for examination of the temporal relationships among the variables, per the recommendations of Kazdin (2007).
Hypotheses

See Appendix A for illustrative examples of equations used in the model-building process.

**Hypothesis 1. Symptom Reduction**

Based on the literature indicating WET and expressive writing (EW) are effective at improving both depression and PTSS, it was expected that all participants would show a statistically significant within-subject reduction in PTSS and depression from pre- to postintervention regardless of condition.

**Hypothesis 2. WET Superior to EW**

Based on the findings of Sloan and colleagues (2011) supporting the assertion that psychoeducation and treatment rationale may be necessary for populations with more severe psychopathology, as well as the extant empirical literature supporting the efficacy of WET to reduce PTSD and depression symptoms with large effects (e.g., Sloan et al., 2012, 2018) compared to the smaller effects of EW (e.g., Frattaroli, 2006), the following hypotheses were advanced:

2a. Participants in the WET condition would see statistically significant reductions in their PTSS compared to EW condition, controlling for the passage of time.

2b. Participants in the WET condition would see statistically significant reductions in their depression compared to EW condition, controlling for the passage of time.
2c. Replicating the findings of Sloan et al. (2011) and Sloan et al. (2005), participants in the WET condition will report statistically greater arousal at session one compared to EW condition.

**Hypothesis 3. Mechanism of Change: Habituation**

To examine associations between changes in the proposed mechanisms and changes in PTSS, as well as differences between conditions in those associations, multilevel linear models (MLM) were built for each mechanism in line with Wisco et al. (2016), with the addition of interaction terms to examine differences across groups (details to follow).

3a. Based on the extant literature on between-sessions habituation in PTSD treatment (e.g., Sripada et al., 2016) and to replicate the findings of Wisco and colleagues (2016) in their study of WET, it was expected that between-sessions change (BSC) in self-reported arousal would be positively associated with change in PTSS, controlling for the effect of time.

3b. Consistent with Sloan et al. (2011), it was expected that those in the WET condition would demonstrate a stronger association between BSC and change in PTSS compared to EW, controlling for the effect of time.

3c. Given the consistent finding that BSC is more relevant to symptom reduction in PTSD treatment compared to within-session change (WSC; e.g., Bluett et al., 2014; Sripada et al., 2016), no hypothesis was made about the relationship between WSC and treatment outcomes, but the relationship between WSC self-reported arousal and change in PTSS, controlling for the effect of time, was tested.
Hypothesis 4. Mechanism of Change: Emotion Regulation

Based on the extant literature on possible mechanisms of change in PTSD treatments suggesting emotion regulation as a mediator of treatment response (e.g., Hinton et al., 2009; Sharma-Patel & Brown, 2016), as well as the literature identifying emotion regulation as a maintaining factor of PTSD symptoms (e.g., Aldao & Nolen-Hoeksema, 2010), the following were expected:

4a. It was expected that change in emotion regulation would be positively associated with change in PTSS, controlling for the effect of time.

4b. Consistent with Sloan et al. (2011), it was expected that those in the WET condition would demonstrate a stronger association between emotion regulation change and reduction in PTSS compared to EW, controlling for the effect of time.

Hypothesis 5. Mechanism of Change: Cognitive Reappraisal

Based on findings demonstrating greater cognitive emotion regulation strategies (Wisco et al., 2013) and increased accommodation (Barnes, 2017) are positively related to changes in PTSD symptoms in WET, the following hypotheses were made:

5a. It was expected that change in posttraumatic cognitions would be positively associated with change in PTSS, controlling for the effect of time.

5b. Consistent with Sloan et al. (2011), it was expected that those in the WET condition would demonstrate a stronger association between change in posttraumatic cognitions and change in PTSS compared to EW, controlling for the effect of time.
CHAPTER 2

METHODS

Participants

Data were collected and screened for eligibility from 224 undergraduate students enrolled in the Introductory Psychology (PSYC 102) course at a large midwestern university during the Fall 2020 and Spring 2021 semesters. Inclusion criteria required that participants must be at least 18 years old, report elevated PTSS related to an associated Criterion A traumatic event at mass testing and/or the baseline survey (more detail to follow), and own a smartphone. Exclusion criteria included self-reported concurrent trauma-focused treatment at the time of enrollment in the proposed study. Of the 224 screened, 43 undergraduates were deemed eligible and invited to participate in the current study. The final sample of enrolled participants included 33 undergraduates (M<sub>age</sub> = 18.76 years, SD = 0.936, range 18-21), 15 participants in EW and 18 participants in the WET condition. See Figure 1 for a flow chart of recruitment, enrollment, randomization, and retention. In line with intent-to-treat analysis guidelines, detailed later, only participants who completed at least two lab sessions were included in primary analyses (N = 30).

Participants were mostly female (75.8%; n = 25), with one participant identifying as nonbinary. A majority of the sample identified as White (63.6%), followed by Black or African

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1 Data collection originally began in Spring 2020, with 50 participants screened, 9 participants eligible and 4 participants enrolled in the study. Due to the COVID-19 pandemic, data collection had to be halted in March 2020. Due to the transition to online procedures, detailed later, these participants are not included in the current sample.
Figure 1: Recruitment, enrollment, and retention flow.
American (15.2%; n = 5), “Other” (12.1%; n = 4), American Indian or Alaskan Native, (6.1%; n = 2); 3% preferred not to respond. Regarding ethnicity, most participants indicated they were not of Hispanic/Latino descent (60.6%; n = 20), and one participant preferred not to respond. Participants reported considerable trauma exposure, with a mean of 8.88 (SD = 3.88) types of traumatic events endorsed on the Life Events Checklist, of which on average 2.97 (SD = 1.61) were direct exposure. On average, participants also reported clinically meaningful PTSS (M_{PCL-5} = 37.15, SD = 13.78, range 20-68) and depression symptoms (M_{PHQ-9} = 13.06, SD = 5.78) at baseline assessment. Of note, at baseline in the full sample 57.6% (n = 19) reported symptoms above clinical cut offs of whom, 73.68% were randomized into the WET condition (n =14). In the ITT sample, at baseline 53.33% (n = 16) reported symptoms above clinical cut-offs, of whom 68.75% (n = 11) were in the WET condition.

**Power Analysis**

For the MLM analyses, the extant literature has identified 30 clusters of data as a rule of thumb for estimates of fixed-effect standard errors (see McNeish & Stapleton, 2016, for review). In addition, for Hypothesis 1, dependent t tests were utilized, so a power analysis was conducted to inform sample size for the current proposal in conjunction with the necessary sample size for MLM analyses. Given the only effect size reported to date for WET comes from the original RCT (Cohen’s d = 1.03; Sloan et al., 2012), this effect size was first examined for the proposed dependent t tests, with error probability = .05 and power = .80. This power analysis resulted in a total sample size needed of N = 9. Given the methodological differences between the original RCT and the proposed study (i.e., inactive versus active control), it was expected smaller effects would be found, such that a medium effect was then examined, resulting in a sample of n = 27.
Taking both types of analyses into account, as well as considering the increase in power that has been identified through increasing sample size for MLM analyses (Scherbaum & Ferreter, 2008), the current study appears to be adequately powered with 30 participants.

Measures

**Trauma Exposure**

The Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013) is a 17-item self-report questionnaire on experiences of trauma across the lifespan. Participants are asked to identify for each event if (1) it happened to them, (2) they witnessed it happen to someone else, (3) they learned about it happening to someone close to them, (3) were exposed as part of their job (e.g., police, first responder), (4) not sure, or (5) does not apply to them. The first 16 items represent DSM-5 Criterion A traumatic events that involve threatened or actual death, serious injury, or sexual violence (American Psychiatric Association, 2013), and the last item is labeled “Other stressful experience.” Follow-up questions designed by Weathers and colleagues (2013) include a narrative description of the worst event as well as whether the event included threatened or actual death, serious injury, or sexual violence.

As it is typically administered, participants are asked to identify their “worst event” from those reported on the first part of the measure; this event serves as the subject for the follow-up questions, as well as the index event for the companion measure, PTSD Checklist for DSM-5 (PCL-5). Bardeen and Benfer (2019) compared the traditional “worst event” methodology of the LEC-5 to a modified methodology such that all endorsed items on the LEC were followed by the follow-up questions and the PCL-5. Their results identified problems with utilizing the “worst
event” method. Roughly 70% of individuals who identified a non-Criterion A traumatic event as their “worst event” also reported experiencing a Criterion A trauma (coined the secondary Criterion A group), and there were no significant differences in PTSD severity for the “worst event” and secondary Criterion A trauma groups (Bardeen & Benfer, 2019). Thus, it appears that utilizing the “worst event” method is not sufficient for identifying individuals suffering with PTSD symptomatology connected to a Criterion A event, it may also cause inadvertent removal of trauma-exposed participants.

To balance feasibility with methodological rigor, in the current study participants were asked to rank order their endorsed events in terms of how bothered they are by them, and answer the follow-up questions and then complete the PCL-5 for their top three events. This methodology was proposed by Bardeen and Benfer (2019) as a methodology needing further empirical testing but may balance the burden of their tested methodology (i.e., completing follow-up questions and the PCL-5 for all endorsed items) with the identified problems of the “worst-event” method for assessing trauma history. For the current study, the LEC-5 was administered at baseline (T1) and postintervention (T7). Of note, the majority of the sample (72.7%; \(n = 24\)) identified a Criterion A event as their worst event with accompanying elevated PTSS, followed by 15.2% \((n = 5)\) reporting an A1 criteria event with PTSS as their third worst event, and 12.1% \((n = 4)\) reporting an A1 criteria event with PTSS ranked second.

There is no published psychometric data on the LEC-5 to date on samples in the United States, but the National Center for PTSD states that due to the relatively minor changes from the LEC for DSM-IV to the LEC for DSM-5, the psychometric properties of the DSM-IV version are likely to represent that of the LEC-5. In its initial validation study, the LEC demonstrated adequate psychometric properties in both undergraduate \((n = 108)\) and veteran \((n = 131)\) samples.
(Gray et al., 2004). Test-retest correlation for direct exposure was significant, demonstrating a strong association between responses on first and second administration seven days later ($r = .82, p < .01$). The LEC also demonstrated convergent validity with an established measure of traumatic exposure (i.e., Traumatic Life Events Questionnaire [TLEQ]), with a total scale correlation of $r = -.55, p < .01$; of note, the item “sudden, unexpected death of a loved one” appeared to be problematic, with low convergence with the TLEQ (kappa = .38), but this does not pose a problem for the current study due to this item being changed in the updated LEC-5. Given the induced latent structure of a checklist (i.e., inducing the latent structure of “trauma exposure”), studies have examined the associations between the LEC and measures of related constructs to trauma (e.g., PTSD via PCL [$r = -.48, p < .01$], the Modified PTSD Symptom Scale [$r = -.44, p < .01$], and the Mississippi Scale for Combat-Related PTSD [$r = -.33, p < .05$]; Gray et al., 2004); it is unclear whether a sum score of a checklist’s relationship to such outcomes speaks to the psychometric strength of the measure.

The most recent psychometric study on the LEC, an update to DSM-5 in Poland, supports the documented psychometric properties of the LEC (Rzeszutek et al., 2018). High interclass correlation coefficients were found in a two-week test-retest design, with all coefficients significant and greater than .93 and kappas greater than .60, suggesting strong temporal stability. These results may not be generalizable to a United States sample but offer preliminary support for the LEC-5 psychometric properties as comparable to that of the original LEC.

**PTSD Symptoms**

The PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015) is a 20-item self-report measure of posttraumatic stress symptoms. Participants respond to items describing symptoms of
PTSD based on how bothered they have been by the symptom on a scale of 0 (Not at all) to 4 (Extremely). All items are summed for a total score ranging from 0 to 80, with a cut-off score of 31-33 for probable PTSD (Bovin et al., 2016; Weathers et al., 2013). The PCL-5 offers two time frames for assessment: past week and past month. The current study utilized both the “past month” and “past week” versions of the PCL-5. The past-month version was administered at baseline (T1) and at mass testing for screening of individuals with elevated PTSS. A cut-off of 20 was utilized for elevated PTSS in the undergraduate sample, as participants are required to either experience all symptoms mildly or at least one symptom at moderate severity to obtain this score. The PCL-5 past week was administered at all time points (T1-T7), consistent with the recommendations of Kazdin (2007) for studies seeking to investigate potential mechanisms of change in treatment.

Supporting the use of the cut-off of 20 for elevated PTSS in the undergraduate sample, pilot data from the Fall 2019 semester indicates a high level of PTSS in the PSYC 102 population as well as those who self-selected into the study. Although all participants could take the PCL-5 regardless of trauma history at mass testing, only those who endorsed a Criterion A traumatic experience are reported here. A total of 588 undergraduates completed mass testing during the Fall 2019 semester. Out of 269 participants who completed the PCL-5, 109 indicated a Criterion A trauma, agreed to follow-up contact, and scored at least a 20 on the PCL-5. Of those 109, about 61% indicated experiencing at least moderate levels of intrusive memories, 44% endorsed nightmares at a moderate level, 81.7% indicated at least moderate levels of avoidance of internal trauma cues (i.e., thoughts, feelings, memories), and 58.7% indicated avoiding external trauma cues (i.e., people, places, situations). Further, of the 109 participants who met eligibility based on the PCL-5, only 5.5% of the sample reported less than six symptoms at less
than a moderate severity. Thus, it appears those who are reporting elevations on the PCL of at least a 20 are reporting at least moderately elevated symptoms on more than a few items. In addition, of those who enrolled in the pilot test of the current study ($n = 5$), the mean PCL-5 score at baseline was 39.8 ($SD = 12.70$), well above probable PTSD cut-offs; all but one enrolled participant reported a PCL-5 above a 31-33.

Scores from the PCL-5 have demonstrated strong psychometric properties across several studies (see Blevins et al., 2015). In its initial validation study, scores from the PCL-5 were validated on two samples of trauma-exposed undergraduates (Blevins et al., 2015). The PCL-5 demonstrated strong internal consistency in both samples ($\alpha = .94, .95$), one-week test-retest reliability ($r = .82$), as well as high convergent validity with other PTSD measures ($r$ ranging from .84 to .86). Evidence of divergent validity was also demonstrated with moderate correlations between the PCL-5 and measures of depression ($r = .60$) and weaker correlations with measures of antisocial personality ($r = .39$) and mania ($r = .31$); a similar pattern of correlations emerged in the second sample of undergraduate students. Confirmatory factor analyses suggested mixed results based on evaluation of fit indices with the four-factor model. Specifically, in the first sample, the model demonstrated reasonable fit on misfit indices (SRMR = .07, RMSEA = .08) but poorer fit on goodness-of-fit indices (CFI = .86, TLI = .84). The second sample CFA demonstrated better fit with the four-factor model, with adequate fit on at least one goodness-of-fit index (CFI = .91, TLI = .89) and reasonable fit on misfit indices (SRMR = .05, RMSEA = .07; Blevins et al., 2015).

Similar psychometric properties have been found in studies utilizing veteran samples (Bovin et al., 2016; Wortmann et al., 2016). Wortmann and colleagues (2016) investigated the PCL-5 in a sample of 912 veterans, finding high internal consistency ($\alpha = .91$); good convergent
validity with measures of PTSD \((r\text{ ranging .68 to .87})\), depression \((r = 0.64)\), and anxiety \((r = 0.61)\); and divergent validity with measures including alcohol use \((r = 0.10)\) and anger \((r = 0.33)\).

Bovin and colleagues (2016) also demonstrated high internal consistency \((\alpha = 0.96)\), test-retest reliability \((r = 0.84)\), and good convergent validity with measures of PTSD \((r = 0.87)\), depression \((r = 0.74)\), and generalized anxiety \((r = 0.67)\), as well as good divergent validity with measures of alcohol abuse \((r = 0.14)\) and antisocial traits \((r = 0.08)\). Taken together, the PCL-5 has demonstrated strong psychometric properties for the measurement of PTSD symptomatology. In the current study, the PCL-5 past-month version demonstrated good internal consistency at both baseline \((T1)\) and follow-up \((T7)\) for all three ranked traumas \((\alpha = 0.86-0.96)\). The PCL-5 past-week version demonstrated similarly high internal consistency across all time points \((\alpha = 0.88-0.96)\).

**Depression Symptoms**

The Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001) is a commonly utilized nine-item self-report measure of symptoms of depression based on the DSM-IV criteria for depressive disorders. While the DSM has been updated since the DSM-IV, no significant changes were made to the criteria for major depressive disorder in the DSM-5 (Uher et al., 2014). Further, the PHQ-9 represents one of the most widely used measures of depression in clinical settings (El-Den et al., 2018; Maurer, 2012), with a Web of Science citation count of 14,039, of which 9,462 citations come from the last five years (i.e., 2016 – current). The measure represents the depression module from the full Patient Health Questionnaire (Spitzer et al., 1999), a three-page self-report measure of eight DSM-IV diagnoses. On the PHQ-9, participants rate the frequency at which they experienced each symptom of depression over the past two
weeks on a scale of 0 (not at all) to 3 (nearly every day). Responses are summed to create a total score ranging from 0 to 27; total scores 5-9 indicate minimal depression, 10-14 indicate moderate depression, 15-19 indicate moderately severe, and greater than or equal to 20 indicates severe depression. The PHQ-9 was administered at all time points (T1-T7), but the time frame for consideration was modified to “past week” for the lab portion of the study (T2-T6) to increase specificity of measurement for the evaluation of changes in depression on a week-to-week basis, as participants were attending weekly sessions. This change has been made in at least one other study to the author’s knowledge (Held et al., 2020) but was utilized in a case study, so aggregate psychometric properties related to this change are unknown.

Scores from the PHQ-9 have demonstrated strong psychometric properties for a brief self-report measure with a vast literature examining its psychometric properties. In the original validation study, the PHQ-9 demonstrated high internal consistency in both the primary care and OBGYN settings ($\alpha = .87$ and $\alpha = .89$, respectively; Kroenke et al., 2001). The PHQ-9 also demonstrated good test-retest reliability over 48 hours ($r = .84$). Criterion validity was established through comparison of the PHQ-9 with interviews with mental health professionals; results suggested good criterion validity with sensitivity of .95 for diagnosing depression. Construct validity was demonstrated through strong associations between increasing PHQ-9 scores and decreasing functioning in mental health ($r = .73$), general health ($r = .55$), social functioning ($r = .52$), role functioning ($r = .43$), physical functioning ($r = .37$), and pain ($r = .33$; Kroenke et al., 2001). In a study using a nonclinical sample, the PHQ-9 was found to be strongly significantly associated with scores on other validated measures of depression (e.g., with the BDI $r = .73$, $p < .01$), and a similar pattern of associations between the PHQ-9 and domains of functioning was also found (Martin et al., 2006). A more recent examination of the PHQ-9
utilizing two samples including RCTs and a clinical sample demonstrated good test-retest reliability over four weeks ($r = .79$), as well as high internal consistency ($\alpha = .83$) and good convergent validity with another measure of distress ($r = .70$ and .71, for both samples; Staples et al., 2019). Overall, the PHQ-9 has demonstrated strong psychometric properties for use in both clinical and nonclinical settings for the identification of depressive symptomatology. In the current study, the PHQ-9 demonstrated high internal consistency for both the past two weeks administration at baseline and follow-up ($\alpha = .87$ and .83 for T1 and T7, respectively) as well as for the past-week administrations at T2-T5 ($\alpha = .83$-.89).

### Emotion Regulation

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a multi-dimensional, self-report measure of emotion regulation. Participants evaluate the frequency at which each of the 36 statements apply to them, using a 5-point scale ranging from 1 (almost never) to 5 (almost always). The DERS has six subscales: nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity. A total score can be calculated as the sum of all 36 items (with Items 1, 2, 6, 7, 8, 10, 17, 20, 22, 24, and 34 reverse scored), subscale scores can also be calculated as the sum of the items included in the subscale. Higher scores indicate greater emotion regulation difficulty. There have been several short forms of the DERS developed, including a 16-item (DER-16; Bjureberg et al., 2016) and two 18-item versions (DER-SF, Kaufman et al., 2016; DDER-18, Victor & Klonsky, 2016). For the purposes of the current study, the full-scale DERS (Gratz & Roemer, 2004) was administered at baseline (T1) and postintervention (T7), and the DER-16 (Bjureberg et al., 2016) was
administered at all time points (T1-T7), as emotion regulation is proposed as a potential mechanism of treatment. Only the total score were utilized for analyses in the current study.

The DERS is a widely used measure of emotion regulation, and as such, there is a vast literature examining its psychometric properties. In the original development and validation study, the six subscales were developed through factor analysis, accounting for 55.68% of the total variance. The measure demonstrated good internal consistency for both the total score ($\alpha = .93$) and for each of the subscales with alphas ranging from .80 (Awareness) to .89 (Goals).

Construct validity was established through significant correlations with the Negative Mood Regulation Scale (Catanzaro & Mearns, 1990) in the overall score ($r = -.69$) as well as for all the subscales, with correlations ranging from -.34 (Awareness) to -.69 (Strategies). This factor structure has been supported by some studies (e.g., Weinberg & Klonsky, 2009), but many studies have demonstrated poor model fit, suggesting improving model fit with a five-factor model by eliminating the awareness factor (e.g., Bardeen et al., 2012; Fowler et al., 2014; Lee et al., 2016). Other modifications to the DERS have been proposed, such as eliminating reverse scoring through item modification, which resulted in a five-factor solution with Awareness and Clarity items loading onto a single factor, high internal consistency ($\alpha = .97$), and strong associations with the NMR scale ($r = -.74$; Bardeen et al., 2016). Despite concern around the factor structure of the DERS, the total score appears consistently to be related to psychopathology including PTSD (e.g., Ehring & Quack, 2010).

The DERS-16 was examined in both a clinical sample ($n = 96$) and two community samples ($n = 102$ and 482). The DERS-16 short form demonstrated high internal consistency ($\alpha = -.92$) and test-retest reliability over a mean of 8.32 days ($ICC = .85, p < .01$), as well as construct validity in relation to the full-scale DERS in a clinical sample, with all subscales across
the measures significantly correlated and no significant differences found between the measures. In addition, the patterns of relationships between the DERS and DERS-16 with measures for convergent validity (i.e., other emotion regulation, psychiatric symptoms) and divergent validity (i.e., emotional intensity, amplification, observation of internal states) were comparable (Bjureberg et al., 2016).

A recent study examining the full-scale DERS along with the published short forms (i.e., DERS-18, DERS-SR, and DERS-16) in a sample of treatment-seeking adults \( n = 427 \) found high internal consistency for total and all subscales of the DERS (alphas ranging between .86 and .94, with the exception of Awareness and Clarity \( \alpha = .82; \) Hallion et al., 2018). For the short forms, a similar pattern emerged, with Awareness and Clarity consistently demonstrating lower internal consistency and high internal consistency overall for all three short forms \( \alpha = .89, .94, \) and .89 for DERS-18, DERS-16, and DERS-SF, respectively). Further, analyses on incremental validity suggested the DERS-16 and DERS-18 were not inferior to the DERS in predicting anxiety and stress, with the DERS explaining a nonsignificant 2% or less of the variance above and beyond the DERS-18 and DERS-16 (Hallion et al., 2018). Given the relative consistency in the psychometric properties of the DERS and DERS-16, as well as the consistently high internal consistency in the DERS-16 compared to other short forms and estimated time of completion \( M_{\text{Time}} = 2.32 \) minutes; Bjureberg et al., 2016), the DERS-16 was utilized for week-to-week measurement of emotion regulation. In the current study, the DERS demonstrated high internal consistency for both the full measure (i.e., DERS-36) administered at T1 \( \alpha = .91 \) and T7 \( \alpha = .93 \) as well as for the DERS-16 administered at T2-T5 \( \alpha = .91-.93 \) and calculated from the full measure at T1 \( \alpha = .88 \) and T7 \( \alpha = .93 \).
Posttraumatic Cognitions

To assess degree of cognitive reappraisal, changes in posttraumatic cognitions were measured. The Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999) is a 33-item self-report measure of trauma-related thoughts or beliefs. Participants rate their agreement with each statement on a scale of 1 (totally disagree) to 7 (totally agree). There are three subscales on the PTCI: Negative Cognitions About the Self, Negative Cognitions About the World, and Self-Blame. A total score can be utilized, or subscale scores reflecting the sum of items in each subscale can be calculated, with greater scores reflecting greater negative posttraumatic cognitions. For the purposes of the current study, the PTCI was administered at all time points (T1-T7), and total score was utilized for analyses.

The PTCI was developed on a large (n = 600), diverse sample including treatment-seeking individuals, community members, and undergraduate students (Foa et al., 1999). The sample included both trauma-exposed and nontrauma-exposed individuals. High internal consistency was demonstrated for each factor (α = .86-.97) and for the total score (α = .97). Adequate test-retest reliability for total scores and subscales was also demonstrated for both a one-week retest (ρ = .74-.89) and three-week retest (ρ = .80-.85). Convergent validity for total score and subscale scores was also demonstrated through significant moderate to high correlations with measures of PTSD (r = .57-.79) and depression (r = .57-75). Significant differences on the PTCI were also found between individuals with and without a trauma history (χ² [2] = 242.79, p < .001), with those with a trauma history reporting higher scores on the PTCI. Further, the PTCI discriminated between those with and without PTSD with 86% accuracy,
suggesting the PTCI represents a measure of cognitions highly associated with PTSD (Foa et al., 1999).

More recent research also supports the psychometric properties of the PTCI found in the original psychometric study (Andreu et al., 2017; Beck et al., 2004). In a sample of motor-vehicle accident survivors, adequate internal consistency was found for the total score ($\alpha = .93$) and subscales ($\alpha = .81-.93$) as well as significant moderate to high correlations with measures of PTSD ($r = .50-.59$) and depression ($r = .73$; Beck et al., 2004). Discriminant validity was established through nonsignificant correlations with social desirability for all subscales except Self-Blame ($pr = -.17$). Discriminative validity was also replicated, with the PTCI correctly identifying 76% of the sample with or without PTSD (Beck et al., 2004). Similarly, in a sample of sexual assault survivors, the PTCI demonstrated adequate internal consistency for Negative Cognitions About Self and World ($\alpha = .82$ and .92) and also offered further support for concurrent and discriminate validity (Andreu et al., 2017). In the current study, the PTCI demonstrated excellent internal consistency at all time points ($\alpha = .92-.96$).

Emotional Arousal

For the current study, two measures of emotional arousal were utilized. The Self-Assessment Manikin (SAM; Bradley & Lang, 1994) is a nonlinguistic self-report measure of subjective arousal that has been designated one of the gold standards in the evaluation of emotional responses (e.g., Iturregui-Gallardo & Méndez-Ulrich, 2019). The measure consists of three sets of graphic characters depicting a range of responding for three dimensions of emotions: valence (i.e., positive to negative), arousal (i.e., high to low), and dominance (i.e., low to high); see Appendix B for the scales. Participants rate on a 9-point scale their current
emotional state for valence, arousal, and dominance. The current study utilized the arousal
domain of emotion responding, with higher scores indicating greater arousal.

Scores from the SAM have demonstrated strong psychometric properties. In the original
validation study utilizing the International Affective Picture System (IAPS; Lang et al., 1999),
the three factors of the SAM were identified and accounted for 59% of the total variance
(Bradley & Lang, 1994). Concurrent validity was demonstrated for the valence and arousal
dimensions, with high correlations ($r = .97$, $.94$, respectively) with the Semantic Differential
Scale (Mehrabian & Russell, 1974; i.e., another measure of valence, control, and arousal).
Further, the correlation between the arousal and valence domains was nonsignificant and small ($r$
$= -.09$), suggesting the domains reflect orthogonal constructs (Bradley & Lang, 1994). In another
psychometric evaluation of the SAM in a Persian sample, test-retest reliability over two weeks
ranged from $.55$ to $.78$, with adequate internal consistency for the arousal and valence
dimensions ($\alpha = .83$, $.89$, respectively; Ali et al., 2012).

Subjective units of distress (SUDS) were also collected to assess participants’ emotional
arousal during the written exposures. The Subjective Units of Distress Scale (SUDS; Wolpe &
Lazarus, 1966) is a self-reported rating of distress commonly used within the context of
exposures for both PTSD and anxiety-based disorders. Participants rate their level of distress in
the moment using a scale of 0 (complete relaxation) to 100 (maximum distress). For the current
study, participants were asked to rate their SUDS prior to the writing portion of the study and
immediately after the writing and also their peak SUDS, operationalized as the highest their
SUDS reached over the course of the writing exercise, postwriting. No psychometric properties
have been published on scores from the SUDS but it is a commonly utilized measure of distress
in many protocols for exposure (e.g., PE; Foa et al., 2007).
Conditions

Written Exposure Therapy

Written exposure therapy (Sloan & Marx, 2019) is a brief, five-week treatment for the treatment of PTSD. The first session takes approximately 60 minutes, and all subsequent sessions last about 40 minutes. At the first session, psychoeducation about common reactions to traumatic exposure and PTSD is presented followed by the first written exposure to an identified traumatic event. The written exposure lasts 30 minutes, with a 10-minute check-in at the end of the writing designed to elicit reactions to writing and instruct individuals to not avoid any thoughts, feelings, or images related to the trauma that may arise over the course of the week.

All subsequent sessions follow the same structure: a brief check-in and review of the prior week, written exposure, and brief check-in postwriting. The brief check-in at the start of the session is to evaluate whether the individual was able to resist avoidance and think about the traumatic event, as well as an opportunity for the therapist to provide feedback on the writing done the previous session. Feedback on the writing is given within the following domains: adherence to instructions, degree of trauma focus, and length. Following the check-in, therapists present the instructions for the writing task, answer questions, assess prewriting SUDS; and leave the individual to write for 30 minutes. A brief check-in is conducted postwriting, including the reporting of postwriting SUDS and instruction for the participant to resist avoidance of thoughts/feelings/images regarding the trauma over the course of the upcoming week. The writing instructions vary across sessions, with the emphasis in the first two writing sessions on writing a detailed trauma narrative, and starting in Session 3, adding in details regarding how the trauma has impacted the individual’s life (see Appendix C for narrative instructions). As reported
previously, the empirical support for WET suggests that it is an efficacious treatment for the reduction of PTSD symptoms in both veteran (Sloan et al., 2013), and nonveteran populations (Sloan et al., 2012, 2018).

**Expressive Writing (EW)**

An adapted version of the Pennebaker (1997) writing task with a focus on a single traumatic event was used as a comparison condition. To control for number of sessions and time writing in comparison to WET, all participants were asked to write for 30 minutes over the course of five weekly sessions. Participants were given the following prompt across all five sessions:

I would like for you to write about your very deepest thoughts and feelings about the trauma you identified as most distressing earlier in this session. In your writing, I’d like you to really let go and explore your very deepest emotions and thoughts. You might tie your topic to your relationships with others, including parents, lovers, friends, or relatives; to your past, present, or your future; or to who you have been, who you would like to be, or who you are now. All of your writing will be kept completely confidential. Don’t worry about spelling, sentence structure, or grammar. The only rule is that once you begin writing, continue to do so until your time is up.

Although the writing prompts are collected from participants and screened for any risk-related content, no feedback is given to the participant regarding the content or focus of their writing.

**Procedure**

**Recruitment and Baseline Measurement (T1)**

Participants were recruited through two avenues: mass testing and an open baseline survey on SONA. Students who reported a traumatic event on the Life Events Checklist (LEC-5; Weathers et al., 2013) and elevated PTSS on the PTSD Checklist for DSM-5 (PCL-5; Weathers...
et al., 2013), but indicated they are not in treatment related to their traumatic exposure on the mass testing survey, were contacted by the PSYC 102 coordinator (Dr. Michelle Lilly) via email regarding their eligibility to participate in the current study. The email outlined their eligibility and the procedures of the current study, including what they would be asked to do, how many sessions the study would take, as well as what information they would be asked to provide. The email also detailed that participation in the current study replaced their participation in 8 research credits for partial class credit if they chose to enroll. Participants were also informed that if they chose to enroll in the current study, they forfeited the option to complete other studies for credit and if they chose to discontinue, they would be required to complete the alternative written assignment to receive partial course credit. Of note, participation in the current study did not disqualify participation in other studies for extra credit. Participants were informed in this email that upon completion of the study they would be awarded full research credit (i.e., 8 credits). The email also contained a unique link to an online survey hosted by Qualtrics, which they completed in order to be enrolled in the current study.

Once participants completed the online survey, they were contacted by the researcher via email and scheduled for their first lab session. The Qualtrics baseline survey was also open for anyone to participate in and any participants who completed the survey and were deemed eligible (i.e., at least 18 years old, indicated a traumatic event on the LEC, elevated PTSS on the PCL-5, and indicated that they are not in trauma-focused treatment) were contacted by a research assistant to schedule their first lab session. Participants were randomly assigned to either the

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2 Exceptions to this rule included if participants demonstrated clinically significant increases in PTSS or depression symptoms, wherein they would be offered to discontinue the study without requirement to complete the alternative assignment. This change was agreed upon and approved by the IRB in response to a community mental health provider’s concern raised to the IRB in March 2021. Only one participant was eligible to opt out after the change was implemented and they chose to continue in the study.
WET or EW condition upon scheduling their first lab session; randomization was completed utilizing Microsoft Excel’s random number generator. To account for drop-out, additional condition slots were added to the end of the enrollment list as participants dropped out of the study (i.e., when a participant dropped out, another slot opened at the end of the list for the condition they were randomized.

**Lab Sessions (T2-T6)**

Participants were not considered enrolled in the current study until they completed the first lab session. At the first lab session, participants provided informed consent and credit structure was reiterated (see Appendix D for informed consent and debriefing). If participants chose to enroll in the current study, the research assistant scheduled the next four lab sessions weekly on the same day of the week. Enrollment at Session 1 for the study of the current proposal was 100% \( n = 33 \), such that all participants who attended the first lab session chose to enroll in the study after reviewing the credit structure and informed consent. The research assistant confirmed the index trauma (reported on the baseline survey) for which the individual was asked to use as the focus of their writing for the next five weeks. Participants then completed the prewriting measures. The research assistant provided the intervention for the appropriate condition (i.e., EW or WET), obtained the participant’s SUDS, and allowed the participant privacy to write for 30 minutes. After 30 minutes had elapsed, the experimenter returned and recorded the participant’s SUDS immediately postwriting and their reported peak SUDS rating. Participants then completed the Self-Assessment Manikin (SAM) and the experimenter read over the completed writing for any risk-related (i.e., suicidality, homicidal ideation) content that required further discussion. The remaining lab sessions (T3-T6) followed a similar protocol.
Participants arrived at the lab, completed presession measures, completed the writing assignment consistent with their condition, completed postwriting measures, and checked in with the experimenter.

**Follow-Up Postintervention (T7)**

Once participants completed lab Session 5, they were sent a link to a Qualtrics survey containing the follow-up measures. This survey was identical to that of the baseline for accuracy of pre/post measurement of symptoms. For the purposes of reducing attrition, all credits towards their PSYC 102 course were granted once participants had completed the final survey. That is, in order to receive their credit, participants had to complete the T7 survey. A few notable exceptions were made to this rule: one participant who contracted COVID was granted partial credit for their time, another participant who did not complete the last lab session due to a family emergency was granted partial credit, and one participant who was dropped from the study due to noncompliance was also granted partial credit; all exceptions were granted during the COVID-19 pandemic.

**Changes to Procedures Due to COVID-19 Pandemic**

Data collection for the current study began in February 2020 as proposed. Between February 4, 2020, and March 3, 2020, nine individuals were deemed eligible for the study; four of those participants were enrolled in the study having completed at least one lab session. On March 11, 2020, the World Health Organization declared COVID-19 a global pandemic. On March 13, 2020, the university closed campus and moved all course instruction online. By March 2, 2020, the IRB halted all in-person data collection and thus data collection for the
current study was halted. At the time of data collection being halted, two of the four participants enrolled had completed all lab sessions. As such, changes in the protocol were made such that the current study could be transitioned to an online format. Namely, all lab sessions were conducted via Microsoft Teams, wherein participants would go through the same study procedures as detailed above but would also be required to upload their writing to OneDrive for review of risk-related content. In addition, administration of the SAM changed such that the experimenter shared the image of the SAM and participants responded with the number associated with a figure that reflected their current emotional state (i.e., arousal, valence, and dominance). Beginning in fall 2020, all data collection for the current study restarted. The primary analyses reported in this document reflect only data collected online.

Preliminary Data from the In-Person Pilot Study

Participants

Participants in the pilot study included five undergraduate students (60% male) from the introductory psychology class at a large midwestern university. In terms of race and ethnicity, participants identified as 80% White (one participant identified as Asian or South Asian) and 20% (n = 1) identified as Hispanic. Mean age of participants was 19 years old (SD = .70) and the majority (n = 4) identified as either a freshman or sophomore in college. The sample reported considerable trauma exposure, with 7.2 (SD = 3.49) traumatic events endorsed on the LEC on average and a reported mean of 2.6 (SD = 1.52) directly experienced events. Most commonly endorsed directly experienced traumatic events included transportation accident (80%; n = 4), physical assault (60%; n = 3), and sexual assault (40%; n = 2). At baseline participants reported
elevated PTSS on the PCL-5 (M = 39.80, SD = 12.70), with significant variation in baseline
PTSS, ranging from a total score of 25 to 60. The most commonly identified index event for the
pilot study was sexual assault (40%; n = 2). In addition, at baseline, participants reported
elevated depression symptoms, with a mean PHQ-9 score of 18.40 (SD = 3.97), suggesting
moderate to severe levels of depression in the current sample.

**Enrollment and Retention**

As previously outlined, participants were not enrolled in the current study until they
completed lab Session 1 (T2). Enrollment for the pilot study was 100% (n = 5), meaning that all
participants who attended the lab session chose to enroll in the pilot study. Participants were
randomly assigned to either the WET or EW conditions upon signing up for the first lab session;
three participants were assigned to the WET condition and two were assigned to the EW
condition. No significant differences were found between the groups on PTSS severity (t[3] =
0.13, p = .93, d = 0.15) or number of traumas on the LEC (t[3] = .13, p = .93, d = 0.15) at
baseline. Retention was high; only one participant dropped out of the study. The participant who
dropped out was enrolled in the EW condition, but no evidence suggested their drop-out was
related to study content. All other participants completed the five in-lab sessions as well as the
pre- and postintervention surveys, suggesting a multitime-point study, as proposed, is feasible in
this population.

**Preliminary Results**

Given that data are only available for one of the EW participants due to drop-out, no
comparisons between the groups will be reported. Instead, within-subject comparisons of PTSD
and depression symptoms will be reported only for the WET group (n = 3). The pilot study utilized the PCL-5 past-month version for the pre/post surveys,\textsuperscript{3} which may confound estimates of PTSS severity given that the time frame includes the majority of the study. Therefore, the PCL-5 weekly version administered at the final lab session (T6) was utilized in analyses of changes in PTSS pre to postintervention.

All participants in the WET condition demonstrated decreases in their PTSS and depression symptoms over the course of treatment. The mean PCL-5 score at T6 was 16.33 (SD = 21.46) and the mean PHQ-9 score was 8.67 (SD = 10.26). Two paired-samples $t$ tests were carried out to examine changes in PTSS and depression symptoms pre- to postintervention for the WET condition. Results indicate participants reported significant reductions in their PCL-5 score, $t(2) = 7.47, p = .02$, with a large effect ($d = 4.30$) and an average reduction of 24 points (SD = 5.57) on the PCL-5. Similarly, participants in the WET group also demonstrated reductions in their PHQ-9 total score, but this decrease was marginally significant, $t(2) = 3.11, p = .09, d = 1.79$. Given the small sample size, these results should be interpreted with caution, but they do offer some preliminary support for the proposed study and hypotheses.

**Data Analytic Plan**

Preliminary data analyses included examination of missing data patterns for all proposed analyses. Randomization checks were also conducted to examine any differences between conditions on demographic or outcome measures preintervention (T1 & T2). Independent samples $t$ tests were utilized for continuous variables; chi-square analyses were conducted for the

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\textsuperscript{3} The proposal utilized the past-month version of the PCL-5 only for inclusion criteria.
categorical variable for the randomization checks. For all analyses, with the exception of \( t \) tests for Hypothesis 1 and Hypothesis 2c, multilevel modeling (MLM) was conducted utilizing HLM 8 because it is the most appropriate analysis for longitudinal data nested within clusters (i.e., individuals with multiple data points; Nezlek, 2008; Scherbaum & Ferreter, 2008). Data were organized into two levels, with the repeated-measure outcome (level 1) nested within individuals (level 2).
CHAPTER 3

RESULTS

Data Screening and Preliminary Analyses

Model assumptions and other data requirements for all analyses (i.e., multilevel models and $t$ tests) were examined. In regard to the $t$-test assumptions, assumptions were met. However, for Hypothesis 1, outliers were evident, specifically for depression in both groups and PTSS in the EW group only. As such, for outcomes with significant outliers, analyses were conducted with and without outliers; both are reported in the respective sections below. No significant outliers were identified in self-reported arousal in Session 1 (i.e., DV for Hypothesis 2c).

Where violations of the multilevel model assumption of normality occurred, robust estimation of standard errors was implemented. In addition, any violations of the homogeneity of variance assumption in MLM resulted in fitting a more complex model allowing for heterogeneous level-1 variance and testing for significant changes in model fit; only the best fitting model was compared to the more complex model with heterogeneous level-1 variance. Model assumptions are reported for each model below. The interclass correlation (ICC) and design effect (DEFF) were calculated for each model to assess the extent of clustering within each individual. Table 1 shows correlations and descriptive statistics for study variables.
### Table 1

Descriptive Statistics and Bivariate/Biserial Correlations Between Study Variables in Full Sample (N = 33)

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<td>5. Baseline PCL-5 (month)</td>
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<td>.63**</td>
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<td>8. Baseline PTCI</td>
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<td>9. Pre/Post Lab PCL-5 (week)</td>
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<td>10. Pre/Post Lab PHQ-9 (week)</td>
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<td>-.20</td>
<td>-.41*</td>
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<td>.49**</td>
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N  | 28  | 28  | 28  | 28  | 28  | 27  | 27  | 27  | 27  | 28  |
Mean | 2.25 | 15.61 | 8.71 | 0.71 | 7.43 | 17.89 | 7.63 | 36.41 | 99  | 3.5  |
SD  | 4.19 | 21.79 | 8.00 | 2.34 | 13.39 | 16.87 | 4.82 | 13.49 | 36.02 | .75  |
Min | -7  | -12  | -9  | -4  | -34  | 0  | 0  | 17  | 33  | 3  |
Max | 14  | 81  | 24  | 6  | 35  | 80  | 19  | 70  | 155 | 6  |

Note. Condition 0 = EW, 1 = WET; Gender coded dichotomously 0 = Male, 1 = Female; Race coded dichotomously 0 = White, Non-White = 1; *p < .05 **p < .01; Pre/Post indicated Lab 5 score subtracted from Lab 1; Intervention duration = number of weeks between Lab 1 and Lab 5
Missing Data

Utilizing Little’s (1988) missing completely at random (MCAR) test, missing data were determined to be MCAR, \( \chi^2 (138) = 127.158, p = .736 \). The percentage of missing data, including all participants, was 9.34%. Upon examination, a large proportion of missing data was missing from participants who dropped out of the study after the first lab session \( (n = 3) \) and therefore did not provide any postintervention outcome data. After removing such cases, the percentage of missing data was found to be 2.99%.

As missing data were MCAR, complete case analysis (i.e., listwise deletion) is considered to be appropriate and unbiased (Bell et al., 2014; Li & Stuart, 2019), but it is best practice to utilize an intent-to-treat (ITT) approach in randomized trials, which require all randomized participants to be included in analyses (Gupta, 2011; White et al., 2012). A strict interpretation of the ITT guidelines from the 2001 Consolidated Standards of Reporting Trials (CONSORT) would prohibit the inclusion of participants without outcome data (Altman et al., 2001), but a more recent CONSORT update omits the term “ITT” and requires reporting of analysis sample size and whether analysis includes original groupings (Schulz et al., 2010). Given the limitations of the current study’s design and context of data collection (i.e., participation for partial course credit), outcome data for participants who dropped out of the current study were not available beyond the final lab session attended. Further, given timing of survey administration prior to provision of intervention at lab Session 1, only participants who completed two lab sessions would have provided postintervention outcome data. To balance power with best practice, all participants with at least partial outcome data (i.e., participants who completed more than one lab session) were included in the MLMs \( (N = 30) \). To address missing
data, full information maximum likelihood (FIML) estimator was utilized as it has been recommended as best practice to handle missing data in ITT analysis (Li & Stuart, 2019; White et al., 2012) and a recommended approach for MLMS (Grund et al., 2019).

Randomization Checks

Randomization checks were conducted on the ITT study sample. Table 2 provides results for all randomization checks. Overall, there were no significant differences between conditions in baseline measures of depression, PTSS, emotion regulation, posttraumatic cognitions, intervention duration (i.e., number of weeks between lab Session 1 and lab Session 5), age, gender, race, or ethnicity. While there was a significant correlation between condition and baseline levels of PTSS when the full sample was included ($r = .398, p< .05$), suggesting significantly greater baseline symptoms of PTSD in the WET condition, these differences were nonsignificant when the ITT sample was examined for randomization checks. While nonsignificant, the WET condition still demonstrated more severe PTSS and depression at baseline. Given the robust nature of MLM analyses controlling for within- and between-persons variance and examination of the slope of change in PTSS and depression, these nonsignificant differences in baseline symptoms did not change interpretation of outcomes.

Null Models

Null models (i.e., MLMS fit with only the outcome variable and no predictor variables) allow for the evaluation of data as appropriate for MLM analysis. Specifically, null models allow for examination of variance in outcome variables across level-1 (time-varying) and level-2 (person-level) variables. Table 3 includes null models fitted for all proposed analyses; the
Table 2

Randomization Checks in ITT Sample (n = 30)

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<tr>
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<th>WET (n = 15)</th>
<th></th>
<th>EW (n = 15)</th>
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<th>t</th>
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<td>M</td>
<td>SD</td>
<td>M</td>
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<td>5.56</td>
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<td>120.67</td>
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<td>73.3% (n = 11)</td>
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<td>33.3% (n = 5)</td>
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* p < .05

Random effect in all null models varied significantly (ps < .001). Further, fitting null models facilitates computation of statistics to evaluate the importance of clustering in the data—e.g., the interclass correlation coefficient (ICC) and design effect (DEFF). The ICC indicates the amount of between- and within-group variance due to clustering and the DEFF refers to the amount of standard error inflation due to clustering (Krull & MacKinnon, 2001; Maas & Hox, 2005; Nezlek, 2008). For both ICC and DEFF, larger values indicate greater impact of clustering in the data. For all outcomes, the null models provided ICCs above .20 and DEFFs above 2, suggesting clustering should not be ignored in the dataset (Table 3). Therefore, MLM was deemed an appropriate analysis for the current study’s data.
Table 3

Estimation of Null Model Random Effects, Interclass Correlations, and Design Effects

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<th>Outcome</th>
<th>SD</th>
<th>Variance Component</th>
<th>df</th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>ICC</th>
<th>DEFF</th>
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<td>4.50</td>
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<td>15.59</td>
<td>29</td>
<td>229.71</td>
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<td>4.29</td>
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<td>1.00</td>
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<td>576.9761</td>
<td>&lt;.001</td>
<td>.73</td>
<td>5.21</td>
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Note. ICC = Interclass correlation; DEF = Design effect; ITT sample ($n = 30$)

Model Building

All models were built with a forward-stepping procedure wherein each predictor was entered individually, building from the null model and starting with level-1 variables (Nezlek, 2008). This procedure allows for evaluation of each predictor’s significance and fit of the model. For each proposed predictor, an intercept-as-outcome model was fitted first, then predictors were added as cross-level interactions with time (level-1 variable), i.e., slopes-as-outcomes models. For each fitted model, a fixed slope was examined first and compared to a fitted random slope model for improved model fit. Only best fitting models for each step in model building are reported here. For all models, continuous predictors were grand mean centered with the exception of time, time was centered at the last lab session given the heterogeneity in completion of the T7 survey from lab Session 5 ($M_{\text{days}} = 2.11$, $SD_{\text{days}} = 3.54$, $\text{Range}_{\text{days}}$: 14).

Effect Sizes

While effect sizes are utilized to evaluate the strength of a significant effect, there is not agreement on how to calculate effect sizes in MLM. One method to evaluate local (i.e.,
individual predictor) effects in MLM is the proportion reduction in variance (PRV), which assesses the reduction in variance when a predictor is added to a model during model building (Peugh, 2010; Raudenbush & Bryk, 2001; Singer & Willett, 2003). To calculate the PRV, the following equation was utilized (Peugh, 2010):

\[ PRV = \frac{\text{var}_{\text{NoPredictor}} - \text{var}_{\text{Predictor}}}{\text{var}_{\text{NoPredictor}}} \]

In this equation, the \( \text{var}_{\text{NoPredictor}} \) refers to the variance of the model prior in model building that does not include the predictor of interest, and \( \text{var}_{\text{Predictor}} \) refers to the model including the predictor of interest. Given the inclusion of both level-1 and level-2 predictors, the variance used was relevant to the respective level of the predictor of interest (i.e., \( \sigma^2 \) for level-1 and \( \tau_{00} \) or \( \tau_{01} \) for level-2 predictors). While the current study calculated PRV, Nezlek (2008) warns effect sizes may not be an accurate representation of strength in MLM, as they are based on error variance rather than fixed parameters used in calculating effect sizes in other statistical analysis. Of note, it is possible to obtain a negative PRV value in models that contain level-2 predictors, but negative values are understood to occur when there is little variation in a predictor, and a negative PRV does not indicate a reduction in model fit (Roberts et al., 2010).

**Primary Analyses**

**Hypothesis 1: Within-Subject Changes in Symptoms Pre/Postintervention**

Hypothesis 1 examined changes in PTSD and depression symptoms within-subjects from preintervention to postintervention. Four dependent \( t \) tests were conducted, one for each outcome (i.e., PTSD and depression) in each condition (i.e., WET or EW). Given the presence of significant outliers in PTSS for the EW condition, as well as significant outliers in depression for
both conditions, analyses with and without outliers included are reported; if results were consistent across analyses, only means with outliers are reported.

As hypothesized, both conditions demonstrated significant within-person reduction in PTSS from pre- to postintervention. For the WET group, a mean reduction of 16 points (SD = 11.02) on the PCL-5 was identified from T2 (M = 33.38, SD = 10.98) to T6 (M = 17.38, SD = 17.32), \(t(12) = 5.23, p < .001, g = 1.35\). For the EW group a significant reduction in PTSS was also found both with (\(t[14] = 4.17, p = .001, g = 1.02\)) and without outliers included in the analysis (\(t[12] = 6.036, p < .001, g = 1.55\)). From pre- (\(M = 24.33, SD = 13.87\)) to post-intervention (\(M = 15.80, SD = 13.97\)), a mean reduction of 8.53 (\(SD = 7.93\)) points was found.

Regarding within-subject change in depression, as expected, the WET condition demonstrated significant decreases in symptoms from pre- (\(M = 11.46, SD = 3.69\)) to postintervention (\(M = 8.23, SD = 4.76\)), both with outliers (\(t[12] = 2.473, p = .029, g = 0.64\)) and without outliers included (\(t[11] = 2.26, p = .045, d = 1.36\)). Average decrease in symptoms was 3.23 (\(SD = 4.71\)) and 2.33 (\(SD = 3.58\)) points with and without outliers, respectively. In the EW condition, a significant decrease in depression was only found when outliers were not included in the analysis (\(t[13] = 2.60, p = .022, g = 0.65\)), with a mean decrease of 2.00 (\(SD = 2.88\)) points on the PHQ-9; when outliers were included, no significant change in depression symptoms was found (\(t[14] = 1.497, p = .157, d = 0.80\)).

Hypothesis 2: WET vs. EW Symptom Reduction

Hypothesis 2a: Changes in PTSS

Hypothesis 2a examined changes in PTSS postintervention between the WET and EW conditions, controlling for the effects of time. A series of two-level MLMs were fitted using
robust estimation of standard errors because the assumption of level-1 residual normality was violated (see Table 4). First, time was entered into the model centered at T6 (i.e., time – 6) as a level-1 fixed covariate (Table 4, Model 1); the random slopes model demonstrated better model fit than the nonrandom slopes model ($p < .001$; Table 4, Model 2). The linear decrease in PTSS across time was statistically significant ($b = -1.76, p < .001, PRV = 68\%$); allowing slopes of PTSS across time to vary accounted for a 32\% reduction in error variance (Table 4, Model 2), suggesting heterogeneity in the slope of PTSS over time. Mean PCL-5 at the final lab session was 19.15 for the full sample.

Next, condition was added to the model as a predictor of Lab 5 PTSS. Results showed that the mean PTSS at the final lab session was not significantly different between conditions ($b = 5.81, p = 0.173, PRV = 4\%$; Table 4, Model 3a). With condition included in the model, the linear reduction in PTSS symptoms over time remained significant ($b = -1.75, p < .001$); the inclusion of condition in the model did improve overall model fit compared to the less complex model without this predictor ($p < .001$). Model 4 included a cross-level interaction between condition and time (Table 4), but contrary to hypothesis, the interaction was not statistically significant, suggesting that condition did not significantly impact the slope of PTSS over time ($b = 0.15, p = 0.894, PRV = 0\%$; Table 4, Model 4). Further, Model 4 did not significantly improve model fit ($p > .500$).

Further, as the assumption of homogeneity of level-1 variance was violated, a model allowing for heterogeneity in level-1 variance was fitted for the best fitting model (i.e., Model 3a). This model significantly improved model fit ($p < .001$) and replicated the results of Model 3a, with a significant linear reduction in PTSS over time ($b = -1.74, p < .001$) and no effect of condition on mean PTSS at T6 ($b = 6.47, p = .14$; Table 4, Model 3b). As such, Hypothesis 2a
Table 4

Model Building Predicting PTSS with Robust Standard Errors

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1 (Fixed Slope of Time)</th>
<th>Model 2 (Random Slope of Time)</th>
<th>Model 3a (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Predictors Estimate SE p PRV</td>
<td>Predictors Estimate SE p PRV</td>
<td>Predictors Estimate SE p PRV</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>18.54 2.35 &lt;.001</td>
<td>19.15 2.83 &lt;.001</td>
<td>16.26 3.41 &lt;.001</td>
</tr>
<tr>
<td>Time</td>
<td>-1.91 .31 &lt;.001 68%</td>
<td>-1.76 .53 .001 32%</td>
<td>-1.75 .53 .002</td>
</tr>
<tr>
<td>Condition</td>
<td></td>
<td></td>
<td>5.81 4.16 .173 4%</td>
</tr>
<tr>
<td>Time* Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance</td>
<td>1535.679</td>
<td>1509.515</td>
<td>1507.625</td>
</tr>
<tr>
<td>Model Comparison p-value</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

|                   | Model 3b (Heterogeneous L1 Variance) | Model 4 (Random Slope of Time)* |
|                   | Predictors Estimate SE p PRV        | Predictors Estimate SE p PRV    |
| (Intercept)       | 15.66 3.32 <.0001                  | 16.01 3.11 <.001               |
| Time              | -1.88 .5 <.001                    | -1.82 .47 <.001                |
| Time* Condition   | .15 1.08 .894 0%                  |                                 |
| Deviance          | 1488.03                          | 1507.61                        |
| Model Comparison p-value | <.001                 | >.50                            |

Note. *Designates random slopes model demonstrated better model fit via chi-square test p <.05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p– value reflects result of chi-square test comparing less complex (prior) model to the tested model; time reflects Lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30)
Hypothesis 2b: Changes in Depression

Hypothesis 2b examined changes in depression postintervention between the WET and EW conditions, controlling for the effects of time. A two-level MLM was fitted using robust standard error estimation to address the violation of the assumption of level-1 residual normality (see Table 5). First, time was entered as a fixed level-1 covariate centered at T6 (Table 5, Model 1). There was a significant linear effect of time such that mean depression scores reduced over time ($b = -0.72, p < .001$, $PRV = 19\%$), with a mean of 7.71 on the PHQ-9 at T6 (Table 5, Model 1). The random slopes model improved model fit ($p = .002$) and accounted for a 20% reduction in level-1 error variance (Table 5, Model 2), suggesting heterogeneity in the slope of depression symptoms over time.

Next, condition was entered as a level-2 predictor of mean depression symptoms at T6 (Table 5, Model 3a). Condition was a marginally significant predictor ($b = 2.77, p = .058$, $PRV = 4\%$) such that those in the WET condition demonstrated greater depressive symptoms at T6 (Table 5, Model 3a). Model fit was improved, as deviance reduced, but this reduction was marginally significant ($p = .056$). Condition was added as a cross-level interaction with time in Model 4. Contrary to predictions, condition did not significantly impact the slope of change in depression symptoms over time ($b = -0.28, p = .389$, $PRV = 1\%$; Table 5, Model 4), nor did the inclusion of this interaction term significantly improve model fit ($p = .241$).

As the assumption of homogeneity of level-1 variance was violated, a more complex model of Model 3a (i.e., best fitting model) was fitted with the allowance of level-1 variance to
Table 5

Model Building Predicting Depression with Robust Standard Errors

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1 (Fixed Slope of Time)</th>
<th>Model 2 (Random Slope of Time)</th>
<th>Model 3a (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Predictors</td>
<td>Estimates SE p PRV</td>
<td>Estimates SE p PRV</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>7.71 .86 &lt;.001</td>
<td>7.72 .86 &lt;.001</td>
<td>6.35 1.3 &lt;.001</td>
</tr>
<tr>
<td>Time</td>
<td>-.72 .16 &lt;.001 19%</td>
<td>-.72 .16 &lt;.001 20%</td>
<td>-.72 .16 &lt;.001</td>
</tr>
<tr>
<td>Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time*Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance</td>
<td>1114.702</td>
<td>1101.993</td>
<td>1098.425</td>
</tr>
<tr>
<td>Model comparison p-value</td>
<td>&lt;.001</td>
<td>.002</td>
<td>.056</td>
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</table>

<table>
<thead>
<tr>
<th>Model 3b (Heterogeneous L1 Variance)</th>
<th>Model 4 (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictors</td>
<td>Estimates SE p PRV</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>6.96 1.24 &lt;.001</td>
</tr>
<tr>
<td>Time</td>
<td>-.45 .12 &lt;.001</td>
</tr>
<tr>
<td>Condition</td>
<td>2.76 1.43 .064</td>
</tr>
<tr>
<td>Time*Condition</td>
<td>-.28 0.32 .389</td>
</tr>
<tr>
<td>Deviance</td>
<td>1085.85</td>
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<tr>
<td>Model comparison p-value</td>
<td>.001</td>
</tr>
</tbody>
</table>

Note. *Designates random slopes model demonstrated better model fit via chi-square test p < .05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p-value reflects result of chi-square test comparing less complex (prior) model to the tested model; time reflects lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30)
vary (Table 5, Model 3b). The inclusion of heterogeneous level-1 variance improved model fit ($p = .001$) and replicated the results of Model 3b, a significant linear reduction of depression symptoms over time ($b = -.45, p < .001$) and condition approaching significance as a predictor of mean depression at T6 ($b = 2.76, p = .064$), with those in the WET condition reporting greater depression symptoms. In sum, Hypothesis 2b was partially supported because a significant linear reduction in depression was found across time, but condition did not moderate this effect.

Hypothesis 2c: Session 1 Emotional Arousal

Hypothesis 2c examined differences in self-reported arousal at Session 1 across conditions. An independent-samples $t$ test was conducted to examine differences between the mean level of self-reported arousal across conditions at Session 1 utilizing postwriting SAM arousal scores. Contrary to expectations, results showed no significant differences between the WET condition ($M = 5.67, SD = 1.33$) and the EW condition ($M = 4.80, SD = 1.57$) in self-reported arousal at Session 1, $t(28) = -1.57, p = .127, g = -0.56$.

Hypothesis 3: Habituation as a Mechanism of Change

To examine Hypothesis 3, two two-level MLMs were fitted, one examining changes in postwriting emotional arousal over time (i.e., between-session change [BSC]) and another examining within-session change (WSC) over time. See Table 6 and Table 7 for fitted models using BSC and WSC as outcomes, respectively.
Table 6

Model Building Predicting Between-Session Change in Postwriting Arousal

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1 (Fixed Slope of Time)</th>
<th>Model 2 (Random Slope of Time)</th>
<th>Model 3 (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictors</td>
<td>Estimates SE p PRV</td>
<td>Estimates SE p PRV</td>
<td>Estimates SE p PRV</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>4.303 .26 &lt;.001</td>
<td>4.28 .34 &lt;.001</td>
<td>4.35 .36 &lt;.001</td>
</tr>
<tr>
<td>Time</td>
<td>-.19 .076 .013 5%</td>
<td>-.20 .10 .066 30%</td>
<td>-.19 .11 .091 .04 .02 .025 -11%</td>
</tr>
<tr>
<td>∆PTSS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time*∆PTSS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time*Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>∆PTSS *Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Deviance 532.12 508.35 487.37
Model comparison p-value .012 <.001 <.001

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 4 (Random Slope of Time)*</th>
<th>Model 5 (Random Slope of Time)</th>
<th>Model 6 (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictors</td>
<td>Estimates SE p PRV</td>
<td>Estimates SE p PRV</td>
<td>Estimates SE p PRV</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>4.38 .34 &lt;.001</td>
<td>4.46 .47 &lt;.001</td>
<td>4.95 .53 &lt;.001</td>
</tr>
<tr>
<td>Time</td>
<td>-.17 .10 .096</td>
<td>-.17 .10 .096</td>
<td>.01 .12 .932</td>
</tr>
<tr>
<td>∆PTSS</td>
<td>-.02 .03 .522</td>
<td>-.02 .03 .592</td>
<td>.01 .03 .853</td>
</tr>
<tr>
<td>Time*∆PTSS</td>
<td>-.02 .01 .028 21%</td>
<td>-.02 .01 .028</td>
<td>-.01 .01 .153</td>
</tr>
<tr>
<td>Condition</td>
<td>-.19 .03 .671 4%</td>
<td>.126 .61 .048</td>
<td>-.40 .19 .047 21%</td>
</tr>
<tr>
<td>Time*Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>∆PTSS *Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time*∆PTSS *Condition</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Deviance 483.22 483.067 479.32
Model comparison p-value .039 .50 .05

Table 6 continued on following page
Table 6 (continued)

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Estimates</th>
<th>SE</th>
<th>p</th>
<th>PRV</th>
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<tbody>
<tr>
<td>(Intercept)</td>
<td>4.94</td>
<td>.51</td>
<td>&lt;.001</td>
<td></td>
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<tr>
<td>Time</td>
<td>.01</td>
<td>.12</td>
<td>.932</td>
<td></td>
</tr>
<tr>
<td>∆PTSS</td>
<td>.05</td>
<td>.03</td>
<td>.178</td>
<td></td>
</tr>
<tr>
<td>Time*∆PTSS</td>
<td>-.01</td>
<td>.01</td>
<td>.153</td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>-1.3</td>
<td>.58</td>
<td>.035</td>
<td></td>
</tr>
<tr>
<td>Time*Condition</td>
<td>-.40</td>
<td>.19</td>
<td>.047</td>
<td></td>
</tr>
<tr>
<td>∆PTSS *Condition</td>
<td>-.07</td>
<td>.03</td>
<td>.032</td>
<td>6%</td>
</tr>
<tr>
<td>Time*∆PTSS *Condition</td>
<td>-.01</td>
<td>.02</td>
<td>.632</td>
<td>1%</td>
</tr>
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</table>

Model 7 (Random Slope of Time)*

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Estimates</th>
<th>SE</th>
<th>p</th>
<th>PRV</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Intercept)</td>
<td>4.94</td>
<td>.51</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>.01</td>
<td>.12</td>
<td>.932</td>
<td></td>
</tr>
<tr>
<td>∆PTSS</td>
<td>.05</td>
<td>.03</td>
<td>.178</td>
<td></td>
</tr>
<tr>
<td>Time*∆PTSS</td>
<td>-.01</td>
<td>.01</td>
<td>.153</td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>-1.3</td>
<td>.58</td>
<td>.035</td>
<td></td>
</tr>
<tr>
<td>Time*Condition</td>
<td>-.40</td>
<td>.19</td>
<td>.047</td>
<td></td>
</tr>
<tr>
<td>∆PTSS *Condition</td>
<td>-.07</td>
<td>.03</td>
<td>.032</td>
<td>6%</td>
</tr>
<tr>
<td>Time*∆PTSS *Condition</td>
<td>-.01</td>
<td>.02</td>
<td>.632</td>
<td>1%</td>
</tr>
</tbody>
</table>

Model 8 (Random Slope of Time)*

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Estimates</th>
<th>SE</th>
<th>p</th>
<th>PRV</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Intercept)</td>
<td>4.94</td>
<td>.51</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>.01</td>
<td>.12</td>
<td>.932</td>
<td></td>
</tr>
<tr>
<td>∆PTSS</td>
<td>.05</td>
<td>.03</td>
<td>.178</td>
<td></td>
</tr>
<tr>
<td>Time*∆PTSS</td>
<td>-.01</td>
<td>.01</td>
<td>.153</td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>-1.3</td>
<td>.58</td>
<td>.035</td>
<td></td>
</tr>
<tr>
<td>Time*Condition</td>
<td>-.40</td>
<td>.19</td>
<td>.047</td>
<td></td>
</tr>
<tr>
<td>∆PTSS *Condition</td>
<td>-.07</td>
<td>.03</td>
<td>.032</td>
<td>6%</td>
</tr>
<tr>
<td>Time*∆PTSS *Condition</td>
<td>-.01</td>
<td>.02</td>
<td>.632</td>
<td>1%</td>
</tr>
</tbody>
</table>

Note: *Designates random slopes model demonstrated better model fit via chi-square difference test p <.05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p-value reflects result of chi-square difference test comparing less complex (prior) model to the tested model; time reflects Lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30)
<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Fixed Slope of Time)</td>
<td>(Random Slope of Time)</td>
<td>(Random Slope of Time)*</td>
</tr>
<tr>
<td>Predictors</td>
<td>Estimate</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>8.35</td>
<td>1.39</td>
<td>&lt;.001</td>
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<tr>
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<tr>
<td>Time*ΔPTSS</td>
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<tr>
<td>Deviance</td>
<td>1061.92</td>
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<tr>
<td>Model Comparison p-value</td>
<td>&lt;.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Model 3b         | (Heterogeneous L1 Variance) | Model 4                    | (Random Slope of Time)*    |
| Predictors       | Estimate | SE     | p    | PRV            | Estimate | SE     | p    | PRV            |
| (Intercept)      | 8.58     | 1.41   | <.001|                | 8.37     | 1.38   | <.001|                |
| Time             | -1.51    | .59    | .016 |                | -1.63    | .57    | .008|                |
| ΔPTSS            | -.07     | .11    | .534 |                | -.06     | .14    | .667|                |
| Time*ΔPTSS       |          |        |      |                | .02      | .06    | .794| 0%              |
| Deviance         | 1010.61  |        |      |                | 1010.96  |        |      |                |
| Model Comparison p-value | >.50  |        |      |                | >.50     |        |      |                |

*Designates random slopes model demonstrated better model fit via chi-square difference test p < .05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p-value reflects result of chi-square difference test comparing less complex (prior) model to the tested model; time reflects Lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30)
Hypothesis 3a: BSC in Arousal

Hypothesis 3a examined the association between BSC in self-reported arousal and ΔPTSS (i.e., change in PTSS T1-T6), controlling for the effects of time. First, time was entered as a fixed level-1 covariate centered at T6 (Table 6, Model 1); a significant linear effect of time was found when the slope for time was fixed \((b = -0.19, p = .013, PRV = 5\%)\), but when a random slopes model was fitted, this effect was marginally significant \((b = -0.20, p = .066)\). The inclusion of random slopes for time accounted for a 30% reduction in error variance and improved model fit \((p < .001)\). Mean emotional arousal postwriting at T6 was 4.28 when all participants, regardless of condition, were considered.

Next, ΔPTSS was added as a level-2 predictor of emotional arousal at T6 (Table 6, Model 3). ΔPTSS was a significant predictor of T6 emotional arousal \((b = 0.04, p = .025, PRV = -11\%)\), such that greater ΔPTSS predicted higher emotional arousal at T6; of note, given the negative PRV, this effect is likely small. No linear effect of time was found \((b = -0.19, p = .091)\). Despite the small effect, the addition of ΔPTSS improved model fit \((p < .001)\). ΔPTSS was then added as a cross-level interaction with time (Table 6, Model 4). There was a significant cross-level interaction effect of PTSS on the association between time and BSC \((b = -.02, p = .028, PRV = 21\%)\), such that for those with greater decreases in their PTSS pre/postintervention demonstrated greater BSC in self-reported arousal over time (Table 6, Model 4). Model fit was improved by the inclusion of the ΔPTSS x Time cross-level interaction \((p = .039)\). Hypothesis 3a was supported as a significant positive association between change in PTSS and BSC in arousal was found, controlling for the passage of time.
Hypothesis 3b: Effect of Condition on BSC

Hypothesis 3b examined the effect of condition on the relationship between BSC and ΔPTSS. Building off Model 4 presented in Hypothesis 3a (Table 6), condition was added as a level-2 predictor of T6 emotional arousal but was not statistically significant ($b = -.19, p = .671$, PRV = 4%; Table 6, Model 5), nor did it improve model fit compared to the model without condition as a predictor ($p > .50$). Next, a cross-level interaction between condition and time was added to the model (Table 6, Model 6). There was a significant cross-level interaction effect of condition on the association between time and BSC ($b = -.40, p = .047$, PRV = 21%) such that those in the WET condition demonstrated greater BSC in self-reported arousal over time (Table 6, Model 4). In addition, condition significantly predicted T6 emotional arousal ($b = -1.26, p = .048$), suggesting those in the WET condition reported lower emotional arousal postwriting at T6. The addition of condition as a cross-level interaction term improved model fit ($p = .05$).

Next, the interaction between ΔPTSS x Condition was entered as a level-2 predictor of T6 emotional arousal (Table 6, Model 7). The effect was significant ($b = -0.07, p = .032$, PRV = 6%), suggesting that those in the EW condition who had higher change in PTSS demonstrated the highest emotional arousal postwriting at T6. However, model fit was not significantly improved with the addition of the ΔPTSS x Condition term ($p = .152$). Finally, a three-way cross-level interaction effect of the interaction of ΔPTSS x Condition on the relationship between time and BSC was added to the model (Table 6, Model 8) but did not predict BSC ($b = -0.01, p = .632$, PRV = 1%), nor did it improve model fit ($p > .50$). As such, Hypothesis 3b was supported as there was significant effect of condition on the relationship between time and BSC in
emotional arousal, and those in the WET condition reported lower postwriting emotional arousal at T6.

**Hypothesis 3c: WSC in Arousal**

Hypothesis 3c examined the effect of WSC in self-reported arousal and ΔPTSS, controlling for the effects of time. To account for the nonnormality of level-1 residuals, results are reported with robust standard errors (Table 7). Time was entered as a fixed level-1 covariate (Table 7, Model 1), demonstrating a significant linear negative effect of time on WSC ($b = -1.64$, $p = 0.004$, PRV = 10%); WSC at a T6 in time was 8.35 points. The random slopes model improved model fit ($p = .005$) and accounted for a 16% reduction in error variance (Table 7, Model 2). Next, ΔPTSS was entered as a level-2 predictor (Table 7, Model 3a). Results suggested ΔPTSS was not associated with WSC at T6 ($b = -0.07$, $p = .52$, PRV = -1%), but the addition of this predictor significantly improved model fit ($p <.001$). ΔPTSS was then added as a cross-level moderator of the relationship between time and WSC, but no significant cross-level ΔPTSS × Time effect was found ($b = 0.02$, $p = .794$, PRV = 0%), and model fit was also not significantly improved ($p >.50$).

As the assumption of homogeneity of level-1 variance was violated, Model 3a (i.e., the best fitting model) was fitted, but with the allowance of level-1 variance to vary (Table 7, Model 3b). The allowance of level-1 variance to vary did not significantly improve model fit ($p >.50$) but did replicate the results of Model B with a significant linear reduction of WSC in self-reported arousal over time ($b = -1.51$, $p = 0.016$) but no significant association between ΔPTSS and WSC at T6 ($b = -.07$, $p = 0.534$). As expected, no significant association between WSC in emotional arousal and change in PTSS over time was detected.
Hypothesis 4: Emotion Regulation as a Mechanism of Change

To examine Hypothesis 4, a series of two-level MLMs were fitted; see Table 7 for fitted models built in forward-stepping manner. Due to observed nonnormality, robust standard errors were estimated for all models. Further, because the assumption of level-1 homogeneity of variance was also violated, the best fitting model was compared to a model that allowed for heterogeneous level-1 variance (Table 7, Model 4b).

Hypothesis 4a: Association Between Changes in ER and PTSS

Hypothesis 4a examined the relationship between changes in ER and changes in PTSS, controlling for the effects of time. Time was entered as a fixed level-1 covariate, centered at T6 (Table 8, Model 1); a significant negative linear effect of time was found ($b = -1.76, p < .001, \text{PRV} = 37\%$). Mean emotion regulation difficulties at T6 were 37.02. The random slopes model of time on ER demonstrated significantly improved model fit ($p < .001$) and accounted for a 33% reduction in error variance (Table 8, Model 2). Next, $\Delta$PTSS was entered as a level-2 predictor of difficulties with ER at T6 (Table 7, Model 3). Results suggested those with a greater reduction in PTSS reported fewer difficulties with ER at T6 ($b = -1.74, p = .001, \text{PRV} = -14\%$). Said differently, greater improvement in PTSS was associated with better ER at T6. Model fit was improved through the addition of $\Delta$PTSS ($p < .001$).

Next, $\Delta$PTSS was then added as a cross-level moderator of the effect of time (Table 7, Model 4a). Consistent with hypotheses, there was a significant cross-level interaction effect of $\Delta$PTSS on the association between time and ER ($b = -0.14, p < 0.001, \text{PRV} = 71\%$), such that individuals with greater decreases in their PTSS pre/postintervention demonstrated greater decreases in
difficulties with emotion regulation over time (Table 8, Model 4a). Of note, with the inclusion of
ΔPTSS × Time cross-level interaction, the effect of ΔPTSS on T6 ER was non-significant ($b = -.08$, $p = .698$). Model fit was improved compared to Model 3 ($p < .001$).

**Hypothesis 4b: Effect of Condition on ER**

Hypothesis 4b examined the effect of condition on change in ER, controlling for the effects of time. Building upon the models presented in Hypothesis 4a, condition was entered as a level-2 of T6 difficulties in ER (Table 8, Model 5). Condition did not demonstrate a significant association with T6 ER ($b = 5.24$, $p = .18$, PRV = 6%), nor did the inclusion of condition improve model fit ($p = .195$). Next, a cross-level interaction effect of condition on the relationship between time and BSC was added (Table 8, Model 6). The cross-level interaction effect of condition on time was not significant ($b = 0.12$, $p = .815$, PRV = 0%), nor did the inclusion of this term improve model fit ($p > .50$). Model 7 represents the addition of ΔPTSS × Condition interaction as a predictor of T6 ER. Similarly, there was no significant interaction effect of ΔPTSS × Condition on T6 ER ($b = -.08$, $p = .838$), nor did model fit significantly improve ($p < .50$). Finally, a condition by ΔPTSS interaction term was entered into the model as a cross-level interaction on the relationship between time and ER. Contrary to Hypothesis 4b, there was no significant cross-level interaction found ($b = -0.06$, $p = 0.25$, PRV = 10%) nor did model fit improve ($p = .266$).

As the assumption of homogeneity of level-1 variance was violated, Model 4a (i.e., best fitting model) was fitted with the allowance of level-1 variance to vary (Table 8, Model 4b) replicating the results of Model 4a with a significant linear reduction of difficulties with ER over
Table 8

Model Building Predicting Difficulties with Emotion Regulation with Robust Standard Errors

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1 (Fixed Slope of Time)</th>
<th>Model 2 (Random Slope of Time)</th>
<th>Model 3 (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 2 Predictor</td>
<td>Predictors Estimates SE  p   PRV</td>
<td>Predictors Estimates SE  p   PRV</td>
<td>Predictors Estimates SE  p   PRV</td>
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<tr>
<td>(Intercept)</td>
<td>37.02  2.27  &lt;.001</td>
<td>40.46  1.95  &lt;.001</td>
<td>39.59  2.21  &lt;.001</td>
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<tr>
<td>Time</td>
<td>-1.76  .36   &lt;.001  37%</td>
<td>-1.8   .35   &lt;.001  33%</td>
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<tr>
<td>ΔPTSS</td>
<td></td>
<td></td>
<td>-1.74  .36   &lt;.001  -14%</td>
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<td>Time*ΔPTSS</td>
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<td>1374.42</td>
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<td>Model comparison p-value</td>
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<td>&lt;.001</td>
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<table>
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<tr>
<th>Level 1 Predictor</th>
<th>Model 4a (Random Slope of Time)*</th>
<th>Model 4b (Heterogeneous L1 Variance)</th>
<th>Model 5 (Random Slope of Time)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 2 Predictor</td>
<td>Predictors Estimates SE  p   PRV</td>
<td>Predictors Estimates SE  p   PRV</td>
<td>Predictors Estimates SE  p   PRV</td>
</tr>
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<td>-1.64  .25   &lt;.001</td>
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Table 8 continued on following page
Table 8 (continued)

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<tr>
<th>Predictors</th>
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<th>Model 7 (Random Slope of Time)*</th>
<th>Model 8 (Random Slope of Time)*</th>
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<tr>
<td>(Intercept)</td>
<td>37.38 3.04 &lt;.001</td>
<td>37.38 3.02 &lt;.001</td>
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<td>Time</td>
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<td>-1.69 .33 &lt;.001</td>
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<td>-.24 .43 .582</td>
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<td>Time*∆PTSS *Condition</td>
<td>-.06 .05 .25 10%</td>
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Deviance 1281.77 1281.74 1280.5

Model comparison p-value >.50 >.50 .266

Note. *Designates random slopes model demonstrated better model fit via chi-square difference test p <.05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p– value reflects result of chi-square difference test comparing less complex (prior) model to the tested model; time reflects Lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30)
time ($b = -1.62, p < 0.001$) and a significant cross-level moderating effect of $\Delta$PTSS on difficulties with ER over time ($b = -0.14, p < .001$).

**Hypothesis 5: Cognitive Reappraisal as a Mechanism of Change**

To examine Hypothesis 5, a series of two-level MLMs was fitted; see Table 8 for fitted models built in forward-stepping manner. The assumption of normality of level-1 residuals was violated; as such, all model results are reported with robust standard errors. Further, the assumption of level-1 homogeneity of variance was also violated; as such, the best fitting model was compared to a more complex model that allowed for heterogeneous level-1 variance (Table 9, Model 4b).

**Hypothesis 5a: Association Between Changes in CR and PTSS**

Hypothesis 5a examined the relationship between changes in CR (operationalized as changes in posttraumatic cognitions) and $\Delta$PTSS, controlling for the effects of time. Time was entered as a fixed level-1 covariate centered at T6 (Table 9, Model 1); a significant negative linear effect of time was found ($b = -3.89, p < .001$, PRV= 23%). Mean total posttraumatic cognitions at T6 was 102.28. The random slopes model of time on CR demonstrated significantly improved model fit ($p < .001$) and accounted for a 35% reduction in error variance (Table 9, Model 2). Next, $\Delta$PTSS was entered as a level-2 predictor of posttraumatic cognitions at T6 (Table 8, Model 3). Results suggested $\Delta$PTSS was not associated with posttraumatic cognitions at T6 ($b = -.39, p = .457$, PRV= 3%). Model fit was improved through the addition of $\Delta$PTSS ($p < .001$). $\Delta$PTSS was then added as a cross-level moderator of the effect of time (Table 9, Model 4a). In line with Hypothesis 5a, there was a significant cross-level interaction effect of $\Delta$PTSS on
Table 9

Model Building Predicting Cognitive Reappraisal with Robust Standard Errors

<table>
<thead>
<tr>
<th>Level 1 Predictor</th>
<th>Model 1 (Fixed Slope of Time)</th>
<th>Model 2 (Random Slope of Time)</th>
<th>Model 3 (Random Slope of Time)*</th>
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<td></td>
<td>Estimates</td>
<td>SE</td>
<td>p</td>
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<td>5.98</td>
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<tr>
<td>Time</td>
<td>-3.89</td>
<td>.89</td>
<td>&lt;.001 23%</td>
</tr>
<tr>
<td>∆PTSS</td>
<td>-3.79</td>
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<td>&lt;.001 35%</td>
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<tr>
<td>Time*∆PTSS</td>
<td>-3.9</td>
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</tr>
<tr>
<td>Partial Deviance</td>
<td>9.8</td>
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<td>.001</td>
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Model comparison p-value <.001

<table>
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<tr>
<th>Level 2 Predictor</th>
<th>Model 4a (Heterogeneous L1 Variance)</th>
<th>Model 4b (Random Slope of Time)*</th>
<th>Model 5 (Random Slope of Time)*</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Estimates</td>
<td>SE</td>
<td>p</td>
</tr>
<tr>
<td>(Intercept)</td>
<td>101.95</td>
<td>5.99</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Time</td>
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<td>∆PTSS</td>
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Model comparison p-value .01

Table 9 continued on following page
Table 9 (continued)

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<tr>
<th>Predictors</th>
<th>Model 6 (Heterogeneous L1 Variance)</th>
<th>Model 7 (Random Slope of Time)*</th>
<th>Model 8 (Random Slope of Time)*</th>
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<tr>
<td>(Intercept)</td>
<td>101.81 5.94 &lt;.001</td>
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<tr>
<td>Time</td>
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<td>-5.01 .65 &lt;.001</td>
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<td>ΔPTSS</td>
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<td>-.27 .05 &lt;.001</td>
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<td>ΔPTSS *Condition</td>
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<td>Time*ΔPTSS *Condition</td>
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<tr>
<td>Model comparison p-value</td>
<td>.023 &gt;.50</td>
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<td>&gt;.50</td>
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</table>

Note. *Designates random slopes model demonstrated better model fit via chi-square difference test p < .05 compared to fixed slope of time model (not shown); PRV is reported in relation to the proportion of variance added from the previous model for each predictor, for Model 1 the PRV is in relation to the null model; Model comparison p– value reflects result of chi-square difference test comparing less complex (prior) model to the tested model; time reflects Lab Session 5 (T6), all other continuous predictors grand mean centered; ITT sample (n = 30).
the association between time and posttraumatic cognitions \((b = -0.22, p = .006, \text{PRV} = 27\% )\), such that individuals with greater decreases in their PTSS pre/postintervention demonstrated greater decreases in posttraumatic cognitions over time (Table 8, Model 4a). Of note, with the inclusion of \(\Delta\text{PTSS} \times \text{Time}\) cross-level interaction, the effect of \(\Delta\text{PTSS}\) on T6 posttraumatic cognition was marginally significant \((b = -1.08, p = .051)\), such that those with greater change in PTSS reported less posttraumatic cognitions at T6. Model fit also improved with the inclusion of the cross-level effect of \(\Delta\text{PTSS}\) on the relationships between time and posttraumatic cognitions \((p = .01)\).

**Hypothesis 5b: Effect of Condition on CR**

Hypothesis 5b examined the effect of condition on change in CR, controlling for the effects of time. Building upon the models presented in Hypothesis 5a, condition was entered as a level-2 predictor of T6 posttraumatic cognitions (Table 9, Model 5). Condition did not demonstrate a significant association with T6 posttraumatic cognitions \((b = 12.97, p = .193, \text{PRV} = 8\% )\), nor did the inclusion of condition improve model fit \((p = .279)\). Next, cross-level moderation of condition on the relationship between time and posttraumatic cognitions was added (Table 9, Model 6). The cross-level interaction effect of condition on time was not significant \((b = 2.47, p = .094, \text{PRV} = 10\% )\), nor did the inclusion of this term improve model fit \((p = .142)\).

Model 7 represents the addition of \(\Delta\text{PTSS} \times \text{Condition}\) interaction as a predictor of T6 posttraumatic cognitions. Similarly, there was no significant association between \(\Delta\text{PTSS} \times \text{Condition}\) on T6 posttraumatic cognitions \((b = -.65, p = .507, \text{PRV} = 2\% )\), nor did model fit improve significantly \((p > .50)\). Finally, a condition \(\times \Delta\text{PTSS}\) interaction term was entered into
the model as a cross-level effect on the relationship between time and CR; there was a cross-
level interaction found that was approaching significance ($b = -0.19$, $p = 0.06$, PRV = 6%).

Based on graphical representation, it appears that participants in both the WET and EW
conditions who reported greater ∆PTSS demonstrated a decline in posttraumatic cognitions, with
those in the EW condition demonstrating a greater decline. Given the interaction was marginally
significant, simple slopes were not probed. As such, findings provide some support for
Hypothesis 5c, suggesting that the slope of CR may be associated with the interaction between
condition and change in PTSS.

As the assumption of homogeneity of level-1 variance was violated, Model 4a (i.e., best
fitting model) was refitted with the allowance of level-1 variances to vary (Table 9, Model 4b).
Results replicated that of Model 4a with a significant linear reduction of posttraumatic cognitions
over time ($b = -3.97$, $p < 0.001$), a significant cross-level effect of ∆PTSS on posttraumatic
cognitions over time ($b = -0.23$, $p = .005$), and greater ∆PTSS predicting fewer posttraumatic
cognitions at T6 ($b = -1.10$, $p = .048$). Further, model fit was improved ($p = .023$).
The current study sought to examine the efficacy and possible mechanisms of change in WET for undergraduates with elevated PTSS, compared to a trauma-focused expressive writing task, with three main research questions: (1) is WET efficacious in reducing PTSS in non-treatment-seeking undergraduates with elevated PTSS; (2) is WET more efficacious than EW in this population; and (3) what mechanisms of change may be operating in WET? Due to the COVID-19 pandemic, these research questions were shifted to evaluate the provision of WET vs. EW via telehealth.

Hypotheses related to the first research question (i.e., Hypotheses 1 and 2a) were supported, suggesting WET is efficacious in reducing PTSS. Significant within-person change (Hypothesis 1) and linear reduction in PTSS when controlling for time (Hypothesis 2a) were demonstrated.

Hypotheses related to the second research question (i.e., Hypotheses 1 and 2) were partially supported. Specifically, both conditions demonstrated significant within-person reductions in PTSS (Hypothesis 1), but there were no significant differences in PTSS at T6 between conditions, nor did condition affect the slope of change in PTSS across time (Hypothesis 2a). Examining depression as an outcome, mixed results were found. Specifically, those in the WET condition demonstrated significant decreases in symptoms pre/postintervention, but the EW condition only demonstrated such effect with outliers excluded.
(Hypothesis 1). Further, utilizing MLM to examine differences across conditions, a difference between conditions in level of depression symptoms at T6 was approaching significance \((p = .064)\), such that those in the WET condition demonstrated greater depression symptoms immediately postintervention, controlling for the passage of time. Although differences were found at T6, condition did not moderate the relationship between time and slope of change in depression symptoms, suggesting depression symptoms decreased at comparable rates in both conditions. Further, contrary to hypotheses, there was not a significant difference in Session 1 emotional arousal detected between WET and EW.

For the third research question examining possible mechanisms of change in WET versus EW (i.e., Hypotheses 3-5), support for proposed mechanisms was mixed but consistent with recent findings, suggesting proposed mechanisms may be correlates of treatment outcomes rather than mechanisms of action (Lee et al., 2021). Hypothesis 3 examined BSC as a potential mechanism; however, findings supported BSC as a *correlate* of change in PTSS across conditions (Hypothesis 3a). Further, consistent with study hypotheses, differences between conditions were found. Specifically, those in the WET condition reported lower postwriting emotional arousal at T6 compared to EW. A cross-level interaction between condition and time was found such that those in the WET condition demonstrated reductions in BSC across time (Hypothesis 3b). Within-session change of peak emotional arousal did decrease over time but was not associated with changes in PTSS (Hypothesis 3c). Taken together, results suggest WET is more effective at targeting habituation and extinction processes than EW.

Hypotheses related to examining emotion regulation as a mechanism of change suggested linear reductions in difficulties with emotion regulation across time, and changes in emotion regulation were positively associated with changes in PTSS (Hypothesis 4a). No difference
between conditions was detected (Hypothesis 4b). Evidence was found for a relationship between cognitive reappraisal and changes in PTSS, such that those with greater changes in PTSS demonstrated greater changes in posttraumatic cognitions over time, with differences between conditions approaching significance. Specifically, those in the EW condition demonstrated greater improvement in posttraumatic cognitions across time, suggesting EW may be more effective at targeting cognitive reappraisal, although this finding was marginally significant and should be interpreted with caution.

Overall, findings suggest WET is associated with significant reductions in depression and PTSS in undergraduates, but EW may offer similar treatment outcomes. Regarding mechanisms of action, the current study offers support for habituation occurring in the context of WET, but not EW, which may be more strongly associated with changes in posttraumatic cognitions. Those who responded to intervention (i.e., greater changes in PTSS), regardless of condition, demonstrated improvement in PTSS, emotion regulation, and posttraumatic cognitions, suggesting both interventions have utility in improving mental health outcomes in the collegiate context.

Symptom Reduction in WET

The current study offers support for the burgeoning literature suggesting WET is an effective brief intervention for the reduction of PTSD symptoms. Consistent with hypotheses and the empirical evidence to date (Andrews et al., 2021; LoSavio et al., 2021; Park et al., 2021; Sloan et al., 2012, 2013, 2018; Thompson-Hollands et al., 2018), participants in the WET condition demonstrated significant reductions in PTSS pre- to postintervention, with a mean PCL-5 change of 16 points (SD=11.02). Of note, the mean PCL-5 score at baseline for
participants in the WET group was above probable PTSD thresholds at 33.38 ($SD = 10.98$), and mean symptoms postintervention dropped below probable PTSD cut-offs ($M = 17.38$, $SD = 17.31$). The current study’s change in PTSS across time was a larger effect ($g = 1.35; r = .57; PRV = 68\%$) than found in other studies of WET, but this may due to the small, high-functioning (i.e., enrolled in college) sample that also included participants with subthreshold symptoms. Specifically, in the original RCT of WET in a motor-vehicle accident survivor sample ($n = 46$), WET demonstrated a medium to large effect ($r = .46$; Sloan et al., 2012) using the CAPS-5, and in the noninferiority trial of WET ($n = 126$, also using the CAPS-5), a medium effect of reductions in PTSS was found from baseline to six-week follow-up ($d = .51$). Interestingly, for the latter trial, this effect grew over time from baseline to 12-week ($d = .82$), 24-week ($d = .97$), and 36-week ($d = 1.08$) follow-ups. Further, more recent investigations of WET report large effect sizes, including the VA rollout ($n = 227$, $d_{PCL-5} = .86$; Losavio et al., 2021), open pilot study with Latinx Spanish speakers ($n = 20$, $d_{PCL-IV-C} = 3.138$; Andrews et al., 2021), and patients in Korea ($n = 41$, $d_{PCL-5} = 1.23$, $d_{CAPS-5} = 1.98$; Park et al., 2021). In sum, the current study offers evidence for WET as an effective intervention for the reduction of PTSS in undergraduates, a novel sample in the extant literature to date, with a large effect comparable to other investigations of WET with similar sample sizes and studies using both more comprehensive assessment of PTSD symptomatology and larger treatment-seeking samples.

Further, the current study offers preliminary support for the provision of WET via telehealth. This study is only the second study to date that has investigated the efficacy of WET provided online. Losavio and colleagues (2021) reported on the VA rollout of WET, which included provision of WET via telehealth and in person. They reported that drop-out rates were lower in those who completed WET via telehealth but otherwise demonstrated comparable
treatment outcomes to those who were seen in person (Losavio et al., 2021). While not treatment seeking, the current study had a drop-out rate of 12.12% \((n = 4)\), all from within the WET condition. Of note, the baseline PCL-5 past-month score was higher in those who did not complete the study \((n = 5; M = 45.00)\) compared to those who did \((n = 28; M = 36.18)\), but given both groups’ means were above probable PTSD cut-offs, caution is urged in interpreting such differences as the reason for drop-out. Reported reasons for drop-out included dropping the course \((n = 1)\), recent death in the family \((n = 1)\), no reason given \((n = 1)\), and dropped from the study due to noncompliance \((n = 1)\). As such, even though drop-out rates were higher in the WET condition, caution is advised in interpreting this finding due to participants representing a nontreatment-seeking sample and reasons for drop-out not necessarily reflecting intolerability of the WET protocol. Of note, drop-out in the current study was lower than in the extant WET literature (~20-30%), but this may also be due to the small sample size. Taken together, the current study bolsters the call for more investigations into the provision of WET via telehealth, as results suggest WET via telehealth could reduce drop-out rates in PTSD treatment as well as increase access to mental health services due to its brevity and demonstrated efficacy online.

**Comparison of WET vs. EW**

**PTSS Outcomes**

Contrary to expectations and the extant literature suggesting smaller treatment effects of EW to address PTSS, those in the WET condition did not demonstrate greater statistically significant reductions in PTSS compared to the EW group. In the current study, both conditions demonstrated significant reductions in their PTSS, controlling for the passage of time, but there
were no statistically significant differences in the slope of PTSS change nor severity of PTSS postintervention. One explanation for the comparable findings across conditions may be due to the sample including subthreshold participants and therefore less severe symptoms overall. The literature on effectiveness of EW to reduce PTSS is mixed, with one review reporting no significant reductions in PTSS (see Sloan et al., 2015, for review) and another reporting small to medium effects when using DSM-IV assessment of PTSS (Pavlacic et al., 2018). In Sloan and colleagues’ (2015) review, they concluded that, for those with greater severity of symptoms, EW alone may not be enough, whereas Pavlacic and colleagues found larger effect sizes for EW when full diagnostic criteria were required in the study \( d = .55, k = 86 \) than when studies did not require PTSD \( d = .32; k = 16 \). Of note, the Pavlacic et al.’s review included three studies on WET (Sloan et al., 2012, 2013, 2018), which likely influenced larger effect sizes in the PTSD-required analyses, but they do not account for the overall effect found. Further, other sample characteristics may have influenced the lack of group differences in PTSS detected. Specifically, a recent meta-analysis of expressive writing for depression found that, while the overall effect size of EW for depression was small \( g = -.09 \), moderators that increased effect size included studies with more writing sessions, a specific topic, greater age, and greater proportion of female participants (Reinhold et al., 2018). Based on Reinhold et al.’s (2018) findings, the current study sample and design reflect many of the factors that increase the effect of EW (i.e., greater number of sessions, specificity, high proportion of female participants) and thus may account for the lack of group differences detected.

Given that one half of the current study’s sample reported PTSS at or above probable PTSD symptom thresholds \( n = 16 \), another possible explanation may be that trauma-exposed undergraduates are particularly responsive to EW with greater sessions, and thus EW may be
sufficient for the reduction of PTSS without the added components of WET. While the current study may be an outlier when compared to the literature broadly, with reductions in PTSS using EW with a large effect size ($g = 1.02$) pre/posttreatment and an average 8.53-point reduction on the PCL-5, it does correspond with the findings of Sloan et al. (2005). Specifically, Sloan et al. (2005) found a large effect ($n = 79, r = .87$) for trauma-focused EW in trauma-exposed undergraduates. Further, although Sloan and colleagues (2011) did not find significant differences between the EW group and control in reduction of PTSS in a sample of undergraduates with PTSD, they completed only three 20-minute writing sessions on consecutive days. Contrary to the conclusions of Sloan et al. (2011), it may be that for undergraduates with more severe symptomatology, more sessions are needed to see significant reductions in PTSS, and the added components in the development of WET (i.e., treatment rationale and psychoeducation) may not be necessary to see symptom reduction in this population. Further, contrary to the findings of Sloan and colleagues (2011), the current study did not find an increased level of self-reported emotional arousal in the WET condition compared to the EW condition, suggesting that differences between the protocols may be minimally related to emotional arousal (i.e., a reported difference between WET and other nontrauma-focused EW). Future research is needed to replicate the findings of the current study to determine if five sessions of trauma-focused writing is sufficient to produce significant reductions in PTSS in other samples – importantly, treatment-seeking and PTS-diagnosed samples.

**Depression Outcomes**

Similar results to PTSS were found regarding depression outcomes in the current study. Specifically, while overall there was a significant linear reduction in depression symptoms over
time, there were no differences found based on condition. The current study offered limited evidence that those in the WET condition reported greater depression symptoms at T6, but this finding should be interpreted with caution as it was approaching significance and explained little variance in the model ($p = .064$, PRV = 4%). Further, results of the within-subject $t$ test also suggest caution in interpretation, given that a significant difference in depression symptoms pre/posttreatment in the EW group was only found when outliers were included in the analysis. Taken in context with the significant reduction in depression symptoms across time in the MLM models, it may be that the outliers are masking a significant decrease in depression symptoms in the EW group when examined via $t$ test. Overall, more research is necessary to determine if there are differences in depression outcomes between WET and EW protocols.

In general, WET was not designed to target depression symptoms directly, but the current finding of an overall downward trend in depression symptoms is consistent with the limited research on WET and depression outcomes. Specifically, the current study found that time accounted for a modest proportion of variance in depression outcomes (PRV = 19%), with an additional 20% of model variance accounted for by allowing slopes of depression symptoms across time to vary, suggesting heterogeneity in changes in depression across time. In the noninferiority trial of WET versus CPT, similar findings were reported, with a significant linear reduction in depression across time in both conditions, but the WET group demonstrated a smaller effect ($d = .19$; Sloan et al., 2018). Examining depression outcomes in the VA rollout of WET, LoSavio and colleagues (2021) report reduction of depression symptoms with medium effect ($d = .47$). Taken together, the current study offers support for WET and EW for the reduction of depression symptoms, but further research is needed to determine differences between WET and other active treatment conditions.
Habituation as a Mechanism of Change

One of the current study’s strengths is the examination of mechanisms of action at all time points alongside outcomes. The current study offers mixed support for habituation (an observable outcome for extinction processes) as a potential mechanism of WET, providing evidence of between-sessions habituation in the WET condition but not the EW condition. Specifically, a significant Time × Condition interaction effect was found such that those in the WET condition reported greater decreases in their postwriting emotional arousal across time as well as lower postwriting emotional arousal at T6 compared to those in the EW group. Further, an interaction effect of treatment response (i.e., change in PTSS pre/posttreatment) and condition on emotional arousal was found, such that those in the WET group who demonstrated greater reductions in their PTSS reported the lowest postwriting emotional arousal at T6, and those in the EW group who demonstrated greater reductions in PTSS demonstrated the highest postwriting emotional arousal at T6. The proportion of variance this interaction explained was small (PRV = 6%), but it points to a potential difference in processes acting in WET versus EW. Specifically, while the WET group demonstrated between session habituation across time, there was little change in the EW group in postwriting emotional arousal across time. Further, changes in PTSS did not affect the slope of emotional arousal over time, nor did it predict T6 emotional arousal when condition was included as a moderator of the relationship between time and emotional arousal, suggesting that changes in PTSS symptoms may only be related to BSC based on condition. Overall, this provides evidence for habituation associated with changes in PTSS in WET but not in EW.
The current finding of habituation in the WET but not the EW group offers support for the consistency and replication requirements of Kazdin’s (2007) mechanism framework in conjunction with the limited extant literature on emotional arousal in WET. Wisco and colleagues (2016) compared WET to waitlist control and, like the current study, found a dose-response relationship between changes in PTSS and emotional arousal (self-reported and physiological arousal) between sessions. Further, Barnes (2017) found that initial multimodal trauma network activation coded in written narratives was associated with greater changes in PTSS, but only in the WET condition compared to CPT, suggesting emotional activation predicts treatment outcomes in WET. Initial activation has also been associated with sudden gains in WET (Sloan, Thompson-Hollands, et al., 2021). Interestingly, while the current study offers support for a relationship for between-sessions emotional arousal and changes in PTSS in WET, there were not differences in self-reported emotional arousal at Session 1 between conditions. This is in contrast to the findings of Wisco and colleagues (2016) and Sloan and colleagues (2005), but which may be explained by the use of a similar intervention comparison group, whereas both other studies utilized waitlist control and nontrauma-focused writing, respectively.

The current findings related to between-sessions habituation in WET are also in line with a recent rigorous evaluation of the mechanisms of WET that included examination of temporal precedence (Lee et al., 2021). Like the current study, Lee and colleagues found that postwriting emotional arousal decreased across time in the WET condition as well as that changes in self-reported emotional valence preceded changes in PTSS in WET but not CPT; these changes were not associated with treatment outcomes. Given the limitations of the current study’s statistical approach, temporal precedence cannot be established, but findings provide support for extinction processes acting in the context of WET, as compared to other active treatment groups. More
research is needed to replicate the findings of Lee and colleagues (2021) to satisfy Kazdin’s (2007) temporal precedence criterion and to continue to investigate the role of extinction in primarily exposure-based protocols.

The current study also adds to the large body of literature that suggests that BSC in emotional arousal is more relevant to symptom reduction in PTSS than WSC (Bluett et al., 2014; Sripada et al., 2016). Specifically, the current study found that WSC (i.e., peak SUDS - post SUDS) decreased linearly across time, but changes in PTSS were not associated with the slope of change across sessions, suggesting no relationship between participants’ reduction in SUDS within the writing session and treatment outcomes.

Emotion Regulation as a Mechanism of Change

Consistent with study hypotheses and the extant literature, emotion regulation abilities changed over the course of treatment and were associated with changes in PTSS, but no differences between conditions were detected. Specifically, emotion regulation improved over time and this relationship was moderated by treatment response, such that those who reported greater changes in PTSS pre/post treatment saw stronger decline in emotion regulation difficulties. Contrary to expectations, condition did not predict emotion regulation difficulties at T6, nor did condition moderate the relationship between time and emotion regulation difficulties. In line with the extant literature, changes in PTSS explained the majority of the remaining variance (PRV = 71%) through a moderation of the relationship between time and emotion regulation, suggesting a strong relationship between changes in emotion regulation and PTSS. Strong empirical evidence has linked emotion regulation with severity of PTSD symptoms (McLean & Foa, 2017), and emotion regulation has been found to predict improvements in CBT
(Hinton et al., 2009; Sharma-Patel & Brown, 2016). The current study adds to this literature by offering evidence for a dose-response relationship between changes in emotion regulation and PTSS – a Kazdin (2007) criteria for a mechanism.

While WET appeared to target changes in emotional arousal across time, suggesting participants may be demonstrating less experiential avoidance, the reporting of emotional arousal did not translate into differences in emotion regulation skills between groups, although both saw improvements. Emotion regulation difficulties have been identified as a transdiagnostic mechanism of psychopathology (e.g., Aldao & Nolen-Hoeksema, 2010; Gallager, 2017). As such, the lack of group differences in treatment conditions may be due to the transdiagnostic nature of emotion regulation difficulties. Said differently, emotion regulation is a risk factor for a broad swath of psychopathology, so it is unsurprising that EW, a transdiagnostic intervention with demonstrated efficacy in improving a range of mental health outcomes (Frattaroli, 2006; Pavlicic et al., 2018; Smyth, 1998), would also impact emotion regulation. Further, a recent study examining psychophysiological responding in real time during an expressive writing task suggested that EW related to a stressful or traumatic life event was associated with greater heart rate variability than the neutral control group, with large effect \( \eta^2_p = .22 \); Jacques et al., 2020). Heart rate variability has been conceptualized as a marker of emotion regulation processes occurring, with greater variability indicating greater flexibility in response. Thus, it is likely that for both conditions in the current study, with greater participant engagement (i.e., writing about the traumatic event, not avoiding) they were likely engaging in emotion regulation processes,

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1 Adopting language from Kazdin (2007), dose response as used in the current study is short-hand for discussing a gradient effect between levels of tested mechanisms and outcomes, rather than number of sessions. Another consideration in “dose” may be to examine the number of intervention sessions completed, as it is related to mechanisms and outcomes. Due to limited variation in number of sessions completed, this relationship was not tested.
and over time their skills may have been strengthened through repetition, although this hypothesis needs empirical testing. Future research is needed to further elucidate the relationship between emotion regulation in the context of WET and EW, and temporal precedence is necessary to determine if such changes are a correlate or mediator of changes in PTSS.

Cognitive Reappraisal as a Mechanism of Change

Similar to results of emotion regulation, hypotheses related to cognitive reappraisal as a mechanism of change in WET were partially supported. As expected, posttraumatic cognitions reduced over time and changes in PTSS pre/postintervention moderated this relationship such that those with greater response to intervention reported greater decline in posttraumatic cognitions (i.e., greater cognitive reappraisal) over time. Controlling for the passage of time and the Time × Condition interaction effect, change in PTSS was a marginally significant predictor of posttraumatic cognitions at T6 ($p = .051$), providing further evidence of a dose-response relationship between posttraumatic cognitions and overall PTSS.

These findings are in opposition to the results of Wisco and colleagues (2013), who found a significant interaction effect of positive reappraisal and putting-in-perspective with condition on changes in PTSS, suggesting a dose-response relationship between use of these strategies and improvement in PTSS only in the control condition, with positive reappraisal associated with attenuated PTSS treatment response in the WET group. A more recent investigation of changes in cognitions in WET suggests findings consistent with the current study; specifically, decreases in posttraumatic cognitions were positively associated with changes in PTSS across time, and greater reductions in posttraumatic cognitions were associated with greater reduction in PTSS across two active treatment conditions (i.e., WET and CPT; Lee
et al., 2021). Discrepant findings between the Wisco et al. (2013) study and both the current study and Lee and colleagues (2021) may be due to differences in measurement, with Wisco et al. (2013) using a measure of cognitive emotion regulation strategies and the current study and Lee et al. (2021) utilizing the same measure of posttraumatic cognitions. Importantly, Lee and colleagues (2021) found that changes in posttraumatic cognitions did not temporally precede changes in PTSS in WET and thus provided the first test to this author’s knowledge of the temporal precedence criterion required by Kazdin (2007). These findings require replication to meet consistency standards of Kazdin's (2007) protocol for evaluating mechanisms, but they do provide preliminary evidence that proposed mechanisms in WET, including cognitive changes, may be better conceptualized as correlates of treatment outcomes rather than change agents. The current study offers support for this hypothesis through establishing a dose-response relationship between changes in posttraumatic cognitions in line with Lee and colleagues (2021) in a novel sample, but it also suggests these changes may not be solely related to WET, with a marginally significant Condition × Time interaction effect suggesting greater changes in posttraumatic cognitions in the EW group. As such, more research is necessary to disentangle differences in correlates and change agents in WET and EW.

Limitations and Future Directions

The current study adds to the literature supporting the efficacy of WET in undergraduates with elevated PTSS and has a few important strengths and limitations. First, the current study is the first to test WET against the active treatment from which it was developed, offering support for the efficacy of WET but introducing questions regarding the distinctiveness of the protocol from EW in the current sample. Findings suggest that, at least in subthreshold samples, EW may
be sufficient to reduce PTSS; thus, future research should investigate the utility of the additional components added to the WET protocol (e.g., treatment rationale, psychoeducation). While the current methodology was a stringent test of the changes made to the WET protocol, without the inclusion of a no-treatment group, how PTSS may have changed over time naturally is unknown. It is possible some of the sample could have resolved PTSS symptoms unrelated to the study interventions, and as such, future tests of WET compared to active controls should consider including inactive control conditions.

Second, an important limitation of the current study is generalizability due to sample characteristics. Specifically, participants were nontreatment-seeking undergraduates, and two-thirds reported subthreshold symptoms of probable PTSD. It is possible that differences between WET and EW may have emerged in a sample meeting full diagnostic criteria for PTSD. Although elevated, but not diagnostic, levels of symptoms are often neglected in the context of intervention RCTs; the current findings suggest individuals may benefit from intervention when reported symptoms are below quasi-arbitrary cut-offs for categorical diagnoses. While non-treatment-seeking samples may represent lower symptom acuity or less functional impairment due to their status as nontreatment seeking, future research is necessary to determine if there are differences in intervention outcomes due to such factors, especially when evaluating interventions for a disorder characterized by avoidance. Further, it is unknown given the small sample size in the current study if results would generalize to a larger sample with subthreshold symptoms. Additionally, the use of the nontreatment-seeking sample offers a unique test of the WET protocol outside the context of a therapeutic relationship and thus limiting the effects of common factors on intervention outcomes. As such, finding large effects for within-person change is surprising given the known importance of common factors in psychotherapy outcomes.
Future research may consider examining whether these interventions are more effective in therapeutic contexts and consider how such factors enhance treatment components.

Further, completing the current study outside the context of a therapeutic relationship offers important implications related to scalability of brief treatments such as WET. Specifically, the current study provides evidence for WET and EW provided via telehealth by a master’s-level clinician in the context of the first year of a global pandemic. Currently, the gold-standard EBPs for PTSD require doctoral-level and/or other intensive training to provide treatment. The current study may suggest interventions such as WET or EW can be provided by a diverse set of providers utilizing telehealth and may not be limited to mental health specialists and/or mental health settings. Future research may seek to explore more diverse settings and professionals for the provision of WET. Such research may be increasingly important given the expanding gap between need and access to mental healthcare, as highlighted by the COVID-19 pandemic (e.g., Bojdani et al., 2020; Murphy et al., 2021).

Third, the current study relied on self-report questionnaires of study outcomes; thus, while measures were chosen based on evidence for psychometric validity and are in common use in similar studies, more error may have been introduced into findings due to reliance on self-report items. Further, the self-report measures used have demonstrated stability with high test-retest reliability and little psychometric data on sensitivity to change, which may have also influenced study findings. More comprehensive assessment of PTSD symptoms utilizing the CAPS-5 would increase confidence in results. Further, use of multimethod data collection, including behavioral, physiological, and self-report indicators of mechanisms of interest, would have allowed for greater specificity in understanding how each of these factors are implicated in
the reduction of symptoms and would have offered potentially greater objectivity than self-report measures can provide.

Lastly, while a strength of the current study is the methodology, including collection of mechanisms and outcomes at all study time points, a limitation of the statistical approach is that determining temporal precedence was outside the scope of the study at present. Further, while the study did collect both mechanisms and outcomes at all time points, given the results of Lee and colleagues’ (2021) analysis of mechanisms of change in WET and CPT, more data points and/or ecologically valid measurement of momentary processing (e.g., examination of emotional changes throughout the writing) may be needed to disentangle how change occurs in the context of these interventions. Overall, the current study offers support for the call for more rigorous testing of proposed mechanisms as well as innovation and exploration of other potential change agents that may not be represented in the current theory of posttraumatic recovery.

Conclusions

The current study sought to add to the growing literature on WET, offering promising outcomes through a rigorous comparison of WET to the protocol it was derived from (EW) while also aiming to examine the proposed mechanisms of change acting in WET. To the first aim, the current study offers support for the use of WET in an undergraduate sample for the reduction of PTSS and depression symptoms. Being the first known study of its kind to examine WET in undergraduate students, this is an important step in the validation of WET as a treatment modality, given the high rates of trauma and PTSD on college campuses. Undergraduates are often regarded as samples of convenience and rarely are utilized in RCTs. The current study offers an example of the importance of including such individuals in evaluations of treatment
outcomes given the high levels of trauma exposure (i.e., mean of eight Criterion A1 events) and PTSS in the current, young sample.

The second aim of the current study sought to examine WET head-to-head with its predecessor, EW. Hypotheses were not fully supported and group differences did not emerge in PTSS and depression outcomes; both conditions saw improvements in such symptoms, suggesting both may be effective options for the reduction of PTSS and depression in undergraduates. Additionally, this study offers further support for the utility of written disclosure paradigms and may inform the literature regarding sufficient dose of EW, given it is the first of its kind to examine five sessions of 30 minutes of EW. Overall, both interventions demonstrated efficacy in reducing PTSS and depression with large effects – welcome news in the context of the growing need for brief, effective interventions for PTSS.

Finally, the current study aimed to evaluate the proposed mechanisms of action in WET, with mixed results. While overall outcomes may not have differed between the conditions, findings related to proposed mechanisms revealed potentially important differences between the protocols. Most notably, the participants in the WET protocol demonstrated between-sessions habituation across intervention sessions, whereas the EW participants did not see significant linear changes in their emotional arousal. This finding offers support for extinction processes occurring in the context of WET but not EW. For both emotion regulation and cognitive reappraisal, a dose-response relationship with changes in PTSS was found across conditions, offering evidence for the validity of the association between these constructs and PTSS. However, lacking temporal precedence, it is possible changes in emotion regulation and posttraumatic cognitions are also outcomes of intervention due to as-yet unidentified change agents. Further, while not rising to statistical significance, a marginally significant difference in
condition on the slope of change in posttraumatic cognitions may offer support for EW as more effectively targeting these cognitions. More research is necessary to continue to reveal *why* effective interventions such as WET reduce PTSS, and a call for more innovation in future investigations is warranted.

Overall evidence from the current study is consistent evidence that WET works, as well as EW, in undergraduates with elevated PTSS, but what is being modified to result in such reductions remains largely unknown. The current study offers limited evidence that WET and EW may be distinct in the processes they evoke (e.g., extinction) and provokes interesting questions regarding potential differences in writing content and structure, given the substantial overlap in protocols across conditions. As the conceptualization and definition of PTSD evolves over time, intervention strategies will necessarily need to flex with the inclusion of new information. The current study offers a call for more emphasis on process-based metrics of treatment success, over decreases in practically defined diagnostic criteria, as there is clearly still much to be learned about how to most effectively assist individuals recovering from trauma than can be gleaned by examining symptom reduction alone.
REFERENCES


APPENDIX A

EXAMPLES OF MODEL-BUILDING MLM EQUATIONS
Examples of Model-Building MLM Equations

Predictor & Outcome Variables
*All continuous predictors were grand mean centered
- Time = Time point coded 0 (T1) to 6 (T7), centered on T6
- Condition = EW coded 0 and WET coded 1
- PTSD = PCL-5 total score
- ΔPTSS = Difference score of PCL-5 (T1) – (T6), greater scores indicate more symptom reduction
- ER = DERS-16 total score (emotion regulation)

Example A: Model Building Predicting PTSS (Hypothesis 2)

Null Model:
Level 1: PTSD = π₀ᵢ + eᵢ
Level 2: π₀ᵢ = β₀₀ + r₀ᵢ

Model 1:
Level 1: PTSD = π₀ᵢ + π₁ᵢ (Time) + eᵢ
Level 2: π₀ᵢ = β₀₀ + r₀ᵢ
π₁ᵢ = β₁₀

Model 2:
Level 1: PTSD = π₀ᵢ + π₁ᵢ (Time) + eᵢ
Level 2: π₀ᵢ = β₀₀ + r₀ᵢ
π₁ᵢ = β₁₀ + r₁ᵢ

Model 3a:
Level 1: PTSD = π₀ᵢ + π₁ᵢ (Time) + eᵢ
Level 2: π₀ᵢ = β₀₀ + β₀₁(Condition) + r₀ᵢ
π₁ᵢ = β₁₀ + r₁ᵢ

Model 3b:
Level 1: PTSD = π₀ᵢ + π₁ᵢ (Time) + eᵢ
Var(e) = σ² and log(σ²) = α₀ + α₁(Time)
Level 2: π₀ᵢ = β₀₀ + β₀₁(Condition) + r₀ᵢ
π₁ᵢ = β₁₀ + r₁ᵢ

Model 4:
Level 1: PTSD = π₀ᵢ + π₁ᵢ (Time) + eᵢ
Level 2: π₀ᵢ = β₀₀ + β₀₁(Condition) + r₀ᵢ
π₁ᵢ = β₁₀ + β₁₁(Condition) + r₁ᵢ
Example B: Model Building Predicting ER (Hypothesis 4)

Null Model:
Level 1: ER = π_{0l} + e_{li}
Level 2: π_{0l} = β_{00} + r_{0l}

Model 1:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + r_{0l}
π_{1l} = β_{10}

Model 2:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + r_{0l}
π_{1l} = β_{10} + r_{1l}

Model 3:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + r_{0l}
π_{1l} = β_{10} + r_{1l}

Model 4a:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + r_{1l}

Model 4b:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Var(e) = σ^2 and log(σ^2) = α_0 + α_1(Time)
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + r_{1l}

Model 5:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + β_{02}(Condition) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + r_{1l}

Model 6:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + β_{02}(Condition) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + β_{12}(Condition) + r_{1l}

Model 7:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + β_{02}(Condition) + β_{03}(Condition * ΔPTSS) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + β_{12}(Condition) + r_{1l}

Model 8:
Level 1: ER = π_{0l} + π_{1l} (Time) + e_{li}
Level 2: π_{0l} = β_{00} + β_{01}(ΔPTSS) + β_{02}(Condition) + β_{03}(Condition * ΔPTSS) + r_{0l}
π_{1l} = β_{10} + β_{11}(ΔPTSS) + β_{12}(Condition) + β_{13}(Condition * ΔPTSS) + r_{1l}
APPENDIX B

MEASURES
Demographics Questionnaire

1. What is your gender?
   a. Male
   b. Female
   c. Nonbinary (please specify): ________________________
   d. Prefer not to answer

2. What is your sex?
   a. Male
   b. Female
   c. Other (please specify)____________________
   d. Prefer not to answer

3. What is age? _______(2 digits)

4. What is your race?
   a. American Indian or Alaskan Native
   b. Asian or South-Asian
   c. Black or African American
   d. Native Hawaiian or Pacific Islander
   e. White
   f. Other (please specify) ________________________
   g. Prefer not to respond

5. What is your ethnicity?
   a. Hispanic, Latino
   b. Non-Hispanic/Latino

6. How many years of education have you completed?
   a. ______ Years of education
   b. Prefer not to respond

7. What year in school are you now?
   a. Freshman
   b. Sophomore
   c. Junior
   d. Senior
   e. Graduate
   f. Other (please specify) _________________
   g. Prefer not to respond
8. What is your sexual orientation?
   a. Heterosexual/straight
   b. Bisexual/pansexual
   c. Gay/Lesbian
   d. Something else (please specify): ____________
   e. Unsure
   f. Prefer not to respond

9. What is the highest level of education completed by your mother?
   a. Some high school
   b. High school diploma/GED
   c. Some college
   d. College degree
   e. Some graduate work
   f. Graduate/Doctorate degree
   g. Don’t know
   h. Prefer not to respond

10. What is the highest level of education completed by your father?
    a. Some high school
    b. High school diploma/GED
    c. Some college
    d. College degree
    e. Some graduate work
    f. Graduate/Doctorate degree
    g. Don’t know
    h. Prefer not to respond

11. Are you currently in counseling or therapy focused on a traumatic/stressful experience?
    a. Yes
    b. No
    c. No, but I have in the past
    d. Unsure
    e. Prefer not to respond
Life Events Checklist for DSM-5 (LEC-5; Weathers et al., 2013)

<table>
<thead>
<tr>
<th>Event</th>
<th>Happened to me</th>
<th>Witnessed it</th>
<th>Learned about it</th>
<th>Part of my job</th>
<th>Not Sure</th>
<th>Doesn't Apply</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Natural disaster (for example, flood, hurricane, tornado, earthquake)</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>2. Fire or explosion</td>
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<tr>
<td>3. Transportation accident (for example, car accident, boat accident, train wreck, plane crash)</td>
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<tr>
<td>4. Serious accident at work, home, or during recreational activity</td>
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</tr>
<tr>
<td>5. Exposure to toxic substance (for example, dangerous chemicals, radiation)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>6. Physical assault (for example, being attacked, hit, slapped, kicked, beaten up)</td>
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<tr>
<td>7. Assault with a weapon (for example, being shot, stabbed, threatened with a knife, gun, bomb)</td>
<td></td>
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<tr>
<td>8. Sexual assault (rape, attempted rape, made to perform any type of sexual act through force or threat of harm)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>9. Other unwanted or uncomfortable sexual experience</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>10. Combat or exposure to a war-zone (in the military or as a civilian)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Captivity (for example, being kidnapped, abducted, held hostage, prisoner of war)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Life-threatening illness or injury</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Severe human suffering</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>14. Sudden violent death (for example, homicide, suicide)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Sudden accidental death</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>16. Serious injury, harm, or death you caused to someone else</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17. Any other very stressful event or experience</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Be sure to consider your entire life (growing up as well as adulthood) as you go through the list of events.
PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015)

Part 3: Below is a list of problems that people sometimes have in response to a very stressful experience. Keeping your worst event in mind, please read each problem carefully and then circle one of the numbers to the right to indicate how much you have been bothered by that problem in the past month.

<table>
<thead>
<tr>
<th>In the past month, how much were you bothered by:</th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Repeated, disturbing, and unwanted memories of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. Repeated, disturbing dreams of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. Feeling very upset when something reminded you of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. Avoiding memories, thoughts, or feelings related to the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. Trouble remembering important parts of the stressful experience?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. Blaming yourself or someone else for the stressful experience or what happened after it?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. Having strong negative feelings such as fear, horror, anger, guilt, or shame?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. Loss of interest in activities that you used to enjoy?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. Feeling distant or cut off from other people?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. Irritable behavior, angry outbursts, or acting aggressively?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. Taking too many risks or doing things that could cause you harm?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. Being “superalert” or watchful or on guard?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. Feeling jumpy or easily startled?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. Having difficulty concentrating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. Trouble falling or staying asleep?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
### Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001)

Over the last 2 weeks, how often have you been bothered by any of the following problems? (use "✓" to indicate your answer)

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Little interest or pleasure in doing things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Feeling down, depressed, or hopeless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Trouble falling or staying asleep, or sleeping too much</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Feeling tired or having little energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Poor appetite or overeating</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Feeling bad about yourself—or that you are a failure or have let yourself or your family down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Trouble concentrating on things, such as reading the newspaper or watching television</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Moving or speaking so slowly that other people could have noticed. Or the opposite—being so fidgety or restless that you have been moving around a lot more than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Thoughts that you would be better off dead, or of hurting yourself</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004)

Please indicate how often the following statements apply to you by writing the appropriate number from the scale below on the line beside each item.

1) I am clear about my feelings.
2) I pay attention to how I feel.
3) I experience my emotions as overwhelming and out of control.
4) I have no idea how I am feeling.
5) I have difficulty making sense out of my feelings.
6) I am attentive to my feelings.
7) I know exactly how I am feeling.
8) I care about what I am feeling.
9) I am confused about how I feel.
10) When I'm upset, I acknowledge my emotions.
11) When I'm upset, I become angry with myself for feeling that way.
12) When I'm upset, I become embarrassed for feeling that way.
13) When I'm upset, I have difficulty getting work done.
14) When I'm upset, I become out of control.
15) When I'm upset, I believe that I will remain that way for a long time.
16) When I'm upset, I believe that I will end up feeling very depressed.
17) When I'm upset, I believe that my feelings are valid and important.
18) When I'm upset, I have difficulty focusing on other things.
19) When I'm upset, I feel out of control.
20) When I'm upset, I can still get things done.
21) When I'm upset, I feel ashamed at myself for feeling that way.
22) When I'm upset, I know that I can find a way to eventually feel better.
23) When I'm upset, I feel like I am weak.
24) When I'm upset, I feel like I can remain in control of my behaviors.
25) When I'm upset, I feel guilty for feeling that way.
26) When I'm upset, I have difficulty concentrating.
27) When I'm upset, I have difficulty controlling my behaviors.
28) When I'm upset, I believe there is nothing I can do to make myself feel better.
29) When I'm upset, I become irriated at myself for feeling that way.
30) When I'm upset, I start to feel very bad about myself.
31) When I'm upset, I believe that wallowing in it is all I can do.
32) When I'm upset, I lose control over my behavior.
33) When I'm upset, I have difficulty thinking about anything else.
34) When I'm upset, I take time to figure out what I'm really feeling.
35) When I'm upset, it takes me a long time to feel better.
36) When I'm upset, my emotions feel overwhelming.
Please indicate how often the following statements apply to you by writing the appropriate number from the scale above (1 – 5) in the box alongside each item.

1. I have difficulty making sense out of my feelings. [CLARITY*]
2. I am confused about how I feel. [CLARITY]
3. When I'm upset, I have difficulty getting work done. [GOALS]
4. When I'm upset, I become out of control. [IMPULSE]
5. When I'm upset, I believe that I will remain that way for a long time. [STRATEGIES]
6. When I'm upset, I believe that I'll end up feeling very depressed. [STRATEGIES]
7. When I'm upset, I have difficulty focusing on other things. [GOALS]
8. When I'm upset, I feel out of control. [IMPULSE]
9. When I'm upset, I feel ashamed with myself for feeling that way. [NONACCEPTANCE]
10. When I'm upset, I feel like I am weak. [NONACCEPTANCE]
11. When I'm upset, I have difficulty controlling my behaviors. [IMPULSE]
12. When I'm upset, I believe that there is nothing I can do to make myself feel better. [STRATEGIES]
13. When I'm upset, I become irritated with myself for feeling that way. [NONACCEPTANCE]
14. When I'm upset, I start to feel very bad about myself. [STRATEGIES]
15. When I'm upset, I have difficulty thinking about anything else. [GOALS]
16. When I'm upset, my emotions feel overwhelming. [STRATEGIES]
Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999)

We are interested in the kind of thoughts which you may have had after a traumatic experience. Below are a number of statements that may or may not be representative of your thinking.

Please read each statement carefully and tell us how much you AGREE or DISAGREE with each statement.

People react to traumatic events in many different ways. There are no right or wrong answers to these statements.

1. Totally disagree
2. Disagree very much
3. Disagree slightly
4. Neutral
5. Agree slightly
6. Agree very much
7. Totally agree

1. The event happened because of the way I acted.
2. I can't trust that I will do the right thing.
3. I am a weak person.
4. I will not be able to control my anger and do something terrible.
5. I can't deal with even the slightest upset.
6. I used to be a happy person but now I am always miserable.
7. People can't be trusted.
8. I have to be on guard all the time.
9. I feel dead inside.
10. You can never know who will harm you.
11. I have to be especially careful because you never know what can happen next.
12. I am inadequate.
13. I will not be able to control my emotions, and something terrible will happen.
14. If I think about the event, I will not be able to handle it.
15. The event happened to me because of the sort of person I am.
16. My emotions since the event mean that I am going crazy.
17. I will never be able to feel normal emotions again.
18. The world is a dangerous place.
19. Somebody else would have stopped the event from happening.
20. I have permanently changed for the worse.
21. I feel like an object, not like a person.
22. Somebody else would not have gotten into this situation.
23. I can't rely on other people.
24. I feel isolated and set apart from others.
25. I have no future.
26. I can't stop bad things from happening to me.
27. People are not what they seem.
28. My life has been destroyed by the trauma.
29. There is something wrong with me as a person.
30. My reactions since the event show that I am a lousy cop.
31. There is something about me that made the event happen.
32. I will not be able to tolerate my thoughts about the event and I will fall apart.
33. I feel like I don't know myself anymore.
34. You never know when something terrible will happen.
35. I can't rely on myself.
36. Nothing good can happen to me anymore.
The Self-Assessment Manakin (SAM; Bradley & Long, 1994)

Note. Scales are represented in each row: Valence, Arousal, and Dominance, in ascending order.
APPENDIX C

WET NARRATIVE INSTRUCTIONS
WET Narrative Instructions (Sloan & Marx, 2019)

Session 1 Instructions
Over the next five sessions I would like you to write about your trauma. Don’t worry about your spelling or grammar. I would like you to write about the details of the trauma as you remember it now—for example, how the trauma event happened and whether other people were involved. In writing about the details of the trauma, it is important to write about specifics of what happened and what you were feeling and thinking as the trauma was happening. Try to be as specific in recounting the details as possible. It is also important that you really let go and explore your very deepest emotions and thoughts about the trauma. You should also keep in mind that you have five sessions to write about this experience, so you don’t need to be concerned with completing your account of the trauma within today’s session. Just be sure to be as detailed about the trauma as possible and also to write about your thoughts and feelings as you remember them during (and immediately after) the trauma.

For your first writing session, I’d like you to write about the trauma starting at the beginning. For instance, you could begin with the moment you realized the trauma was about to happen. As you describe the trauma, it is important that you provide as many specific details as you can remember. For example, you might write about what you saw (e.g., headlights of the car approaching you, person approaching you), what you heard (e.g., car horn, screeching tires, person threatening you, explosion), or what you smelled (e.g., blood, burning rubber). In addition to writing about the details of the trauma, you should also be writing about your thoughts and feelings during the trauma as you remember it now. For example, you might have had the thought “I’m going to die,” “This can’t be happening,” or “I’m going to be raped.” And you might have felt terrified, frozen with fear, or angry at another person involved.

Remember, you don’t need to finish writing about the entire trauma in this session. Just focus on writing with as much detail as possible and include the thoughts and feelings you experienced during and immediately after the trauma. Remember, the trauma is not actually happening again, you are simply recounting it as you look back upon it now.

Session 2 Instructions
Today, I want you to continue to write about the trauma as you look back upon it now. If you feel that you didn’t get the chance to completely describe the trauma in the last writing session, then you can pick up where you left off. If you completed writing about the trauma event in the last session, please write about the entire trauma again. While you are describing the trauma, I really want you to delve into your very deepest feelings (e.g., fear, shock, sadness, anger) and thoughts (e.g., “Is this really happening?” “I’m going to die”). Also, remember to write about the details of the trauma. That is, describe the setting; the people involved; and what you saw, heard, and felt. Remember that you are writing about the trauma as you look back upon it now.

Session 3 Instructions
I want you to continue writing about the trauma event as you think about it today. If you have completed writing about the entire trauma you experienced, you can either write about the trauma again from the beginning or you can select a part of the trauma that is most upsetting to you and focus your writing on that specific part of the experience. In addition, I would also like
you to begin to write about how the traumatic experience has changed your life. For instance, you might write about whether or not the trauma has changed the way you view your life, the meaning of life, and how you relate to other people. Throughout your writing, I want you to really let go and write about your deepest thoughts and feelings.

Session 4 Instructions
I want you to continue to write about the trauma today. As with your writing in the last session, you can select a specific part of the trauma to write about; that is, the part of the trauma that was most upsetting to you. Today, I would also like you to write about how the trauma event has changed your life. You might write about whether the trauma has changed the way you view your life, the meaning of life, and how you relate to other people. Throughout the session I want you to really let go and write about your deepest thoughts and feelings.

Session 5 Instructions
Today is the last session. I want you to continue to write about your feelings and thoughts related to the traumatic event and how you believe this event has changed your life. Remember that this is the last day of writing and so you might want to try to wrap up your writing. For example, you might write about how the traumatic experience is related to your current life and your future. As with the other writing sessions, it is important for you to delve into your deepest emotions and thoughts throughout the session.