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A Systematic Review and Meta-Analysis of Predictors of Response to Trauma-Focused Psychotherapy for Posttraumatic Stress Disorder

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Although trauma-focused psychotherapy (T-F psychotherapy) is the treatment of choice for posttraumatic stress disorder (PTSD), up to one half of patients do not respond to this treatment. Attempts to improve response to T-F psychotherapy have focused on augmenting fear extinction-based factors. Here, a systematic and meta-analytic review of predictors of T-F psychotherapy outcome was conducted with the goal of using an aggregate data-driven approach to elucidate baseline factors associated with treatment outcome. There were 114 studies that met inclusion criteria ($N = 61,970$; $M_{age} = 40.1$ years; 40.1% female). There were 237 effect sizes across 24 meta-analytic categories. Poorer treatment response is associated with lower pretreatment levels of activation of fear-related brain regions, psychophysiological reactivity to fear provocation, trauma-related cognitions, anger, depression, high-risk alleles of genes linked to fear, lower levels of executive control, and social support. A range of other factors also predicted poorer responses including being male, non-Caucasian, older in age, early trauma occurrence, more trauma experience, history of combat trauma, as well as comorbid sleep, pain, poor quality life, and alcohol abuse difficulties. This review provides one potential explanation for the limited success of T-F psychotherapy augmentation strategies that have focused only on fear circuity mechanisms at the exclusion of other factors. Here, poor response relating to predictors of early trauma onset and comorbidity are consistent with clinical presentations of complex PTSD, which may suggest T-F psychotherapy is less effective for this condition. This collective evidence suggests that clinicians should consider a tailored approach that targets potential barriers to successful treatment response.

Public Significance Statement

This meta-analysis of predictors of treatment response to trauma-focused psychotherapy for posttraumatic stress disorder (PTSD) indicates that there is a broad array of factors that contribute to how a person with PTSD responds to this treatment. Although many of these findings are correlational rather than causal in nature, and they were considered in isolation rather than in combination, these findings suggest there is a need to consider a broader range of candidates to augment treatment response. Clinicians may improve treatment response by addressing these potential barriers to optimal therapy outcome.

Keywords: posttraumatic stress disorder, trauma-focused psychotherapy, treatment response, predictor, meta-analysis

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continued

Posttraumatic stress disorder (PTSD) affects up to 5% of people and contributes to marked psychological, health, and social impairment (Stein et al., 2014). PTSD is characterized by a recurrent sense of reliving the trauma in the form of subjectively compelling intrusive memories, nightmares, and flashbacks that lead one to believe they are repeatedly experiencing the fear and threat as if it were occurring in the here-and-now. These memory symptoms typically prompt avoidance of reminders of the trauma as a way of alleviating the distress triggered by the memories. In addition, the PTSD sufferer can experience a range of alterations in normal mood and thoughts, and typically experience heightened arousal in the form of sleep disturbance, anger outbursts, or being hypervigilant to future potential threats (American Psychiatric Association, 2013). There is much evidence that trauma-focused psychotherapy (T-F psychotherapy) is the frontline treatment for PTSD, with most treatment guidelines recommending T-F psychotherapy over pharmacotherapy interventions (Foa et al., 2009; National Institute of Clinical Excellence, 2005). T-F psychotherapy is a descriptor for a range of treatments that share a number of common elements. These therapies are typically defined as trauma-focused because they focus on revisiting memories of the trauma, processing the associated emotions, and typically attempt to alter excessively negative thoughts about the trauma and one's self via some form of cognitive restructuring therapy. Variants of this approach include prolonged exposure, which focuses on repeated reliving of the trauma memory followed by discussion of one's experience of this exercise (Foa & Meadows, 1997). In this treatment, a person is asked to recount their trauma in an engaging manner for at least 30 min, and this process is repeated over numerous sessions, with the expectation that distress reduces as the person learns that the memories are no longer threatening to them. Cognitive processing therapy is another variant, and places greater emphasis on correcting appraisals of the trauma memory and one's role in the trauma (Resick et al., 2017). This therapy traditionally directs a person to write an account of their traumatic experience but therapy then focuses on altering exaggerated beliefs that are held about themselves and their world, often focusing on shame, blame, and guilt. Narrative exposure therapy was developed specifically for refugees and includes prolonged exposure to traumatic memories interspersed with contextualizing these events with positive experiences (Schauer et al., 2011). Over several sessions, the person is directed to create a "lifeline" which often involves a physical creation using physical props (such as a rope or drawing), and they are asked to place symbolic props (such as a flower for a positive event and a stone for a traumatic event) along this lifeline. In this format, the person conducts exposure to their trauma memories, as well as considering how these can be considered in relation to their positive experiences. Eye movement desensitization and reprocessing also comprises reliving of the trauma memory; however, this approach has the distinctive feature of asking the person to experience saccadic eye movements by following a moving visual stimulus; this process is intended to promote neural processes that facilitate integration of new information into the reconsolidation of the trauma memories (Shapiro, 2002). Finally, cognitive therapy initially directs the person to relive the trauma memory but then the focus of therapy is on altering maladaptive interpretations of the trauma and its aftermath (Ehlers et al., 2005). Although proponents of these different treatments often emphasize distinctive features of their approach, there is convergent opinion that they involve the common elements of processing trauma memories and restructuring excessively negative thoughts (Schnyder et al., 2016).

Despite agreement that T-F psychotherapy is the frontline treatment for PTSD, between one third to one half of PTSD patients do not optimally respond to these treatments and continue to display persistent PTSD symptoms (Bradley et al., 2005; Loerinc et al., 2015; Schottenbauer et al., 2008). To address this treatment gap, research has endeavored to augment treatment gains through use of a range of strategies. Most attempts to augment T-F psychotherapy have been driven by purported mechanisms underpinning this therapy, and this has predominantly focused on fear conditioning models (Ressler et al., 2022). This perspective proposes that at the time of trauma exposure the release of stress hormones results in strong associative learning between the fear response and stimuli present during the time of the trauma (Pitman et al., 2012). Subsequent exposure to these stimuli triggers fear reactions, including re-experiencing and arousal symptoms that in turn lend to ongoing avoidance behaviors. For example, a firefighter may develop PTSD after rushing into a burning building to save trapped people, and fear conditioning processes could result in strong associations being formed between the experience of fear and stimuli such as the smell of smoke or burning material. When the firefighter is subsequently exposed to stimuli that are reminiscent of these events, such as smelling the aroma of cooking meat at a barbeque, memories of the trauma may be triggered and experienced as flashbacks. When repeated exposure to these reminders of the trauma occur in a safe environment, new learning can occur where initially conditioned cues (e.g., aroma of cooked meat) become associated with safety, and in turn inhibit the original learned fear association (this process is termed "extinction learning"). On the basis of this model, there is now much known about the neurocircuitry of fear learning and extinction (Ressler et al., 2022). First, the amygdala and hippocampus have been implicated in fear learning insofar as associations between fear and other conditioned stimuli occur in the amygdala, and the hippocampus is responsible for registering the context surrounding traumatic memories (LeDoux, 2000). Central to our understanding of PTSD is that this fear is overgeneralized in that people do not discriminate between safe and unsafe contexts (Pitman et al., 2012). Essential for successful extinction learning is the capacity for efficient hippocampal functioning that can allow memory for learning that safety cues are generalized across contexts. In addition, prefrontal regions are

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also involved in the inhibition of fear-related behaviors. Specifically, the anterior cingulate cortex is recruited during extinction learning and is important for downregulation of emotions. Further, the insula is also relevant in this model because it monitors internal states, is part of the salience network that is alert to threats, and involves fear expression (Greco & Liberzon, 2016). In other words, there are known neural networks involved in context processing, fear processing, and top-down regulation are important for effective extinction processes.

On the basis of the evidence that extinction is a key process in learning new associations that overcome previously learnt fear associations, this model has been applied to explain many treatments for a range of anxiety conditions based on extinction processes. Commonly termed "exposure therapy," this component of T-F psychotherapy involves integration of new corrective information during emotional processing, wherein an individual repeatedly approaches their memories and situational reminders of the trauma in a way that teaches them that they do not result in harm (Lebois et al., 2019). In this way, it is thought that successful response to T-F psychotherapy involves extinction learning that the previously conditioned fear memories are no longer threatening, and this leads to reduction of PTSD symptoms.

Target of Extinction-Related Factors

On the premise that extinction is a primary mechanism underpinning exposure therapy strategies, most attempts to improve treatment response of T-F psychotherapy have modulated extinction-related processes. To do this, such attempts have typically targeted the neurocircuitry underpinning fear memories by administering pharmacological agents known to modulate extinction. To this end, a number of neurotransmitter systems that are critically involved in modulating the strength of fear memories in key regions of the brain detailed above have been targeted (Lebois et al., 2019). On the premise that administering pharmacological agents in close proximity to exposure therapy may consequently modulate critical neural pathways implicated in extinction learning, a number of avenues have been explored. For example, one approach has involved modulation of the glutamatergic system, namely the neurotransmitter glutamate that is involved in the cellular mechanisms underpinning general learning and memory. Acting upon this system has been thought to be important for extinction learning (Royer & Paré, 2002). To this end, several studies have used d-cycloserine (DCS), which is a common antibiotic traditionally used to treat tuberculosis. DCS has been applied to exposure therapy because it is a partial glutamatergic agonist that acts on N-methyl-D-aspartate-type receptors, which is one of the key signaling pathways of this system. Preclinical studies have shown that administering DCS to rats in association with extinction learning enhances the extinction memory (Richardson et al., 2004). Despite the promise of DCS to enhance exposure therapy, there are mixed findings in terms of DCS enhancing the benefits of T-F psychotherapy (de Kleine et al., 2012; Difede et al., 2014; Litz et al., 2012; Rothbaum et al., 2014), which accords with other studies in anxiety disorders that the effect of DCS on exposure therapies is modest (Mataix-Cols et al., 2017). Another approach that has been used to augment T-F psychotherapy is targeting the major stressor systems including the hypothalamic–pituitary–adrenal axis response (which culminates in glucocorticoid release, or cortisol in humans) and the noradrenergic system (involving secretion of epinephrine and norepinephrine), both of which mediate extinction-related plasticity (Roozendaal, Okuda, de Quervain, et al., 2006; Roozendaal, Okuda, Van der Zee, et al., 2006). Consistent with these mechanisms, norepinephrine infusions following extinction may facilitate extinction retention (Mueller et al., 2008). Similarly, glucocorticoid administration facilitates extinction memory retention in animals (Cai et al., 2006). Building on this work, a pilot study reported that hydrocortisone can augment T-F psychotherapy treatment (Yehuda et al., 2015); this effect, however, was not replicated in a full randomized controlled trial (RCT; Lehrner et al., 2021). Yohimbine has also been used to augment treatment because this alkaloid can elevate norepinephrine levels, which can result in stronger engagement with emotions (Meyerbroeker et al., 2011). On the premise that enhanced emotional involvement can promote greater extinction learning, one study of T-F psychotherapy administered yohimbine in association with exposure therapy (Tuerk et al., 2018); however, this study found no effect for yohimbine at follow-up assessment. Other augmentation attempts have used brain stimulation techniques, including transcranial direct current stimulation and transcranial magnetic stimulation, because they have the potential to promote activation in prefrontal neural regions, which as discussed above, are pivotal for extinction learning. Despite the theoretical promise of these approaches, they currently lack adequate evidence (Lebois et al., 2019). Finally, intensive exercise is another approach that has been used because brief bouts of exercise modulate key stress hormones including noradrenaline and glucocorticoids in addition to activation of brain-derived neurotrophic factor, which is a key growth factor that regulates neural plasticity (Winter et al., 2007), and more specifically extinction learning and memory (Andero & Ressler, 2012). Brief bouts of exercise improves extinction retention in both animals (Siette et al., 2014) and humans (Keyan & Bryant, 2019). Consistent with this preclinical evidence, there is early evidence that brief intensive exercise following exposure to trauma memories can augment trauma-focused cognitive behavioral therapy (Bryant, Dawson, et al., 2023). Overall, however, despite these numerous attempts to augment T-F psychotherapy, no specific strategy has emerged as being particularly strong (Mataix-Cols et al., 2017).

Identifying Factors That Predict Treatment Response

One approach to identify potential candidates for augmenting T-F psychotherapy is to focus on factors that are predictive of treatment response. In this context, there are a number of factors related to fear extinction that have been shown to predict treatment response following exposure-based therapies. For example, heightened arousal as measured by higher blood pressure, heart rate, and startle responses in response to stress provocations (such as trauma reminders) before exposure has been associated with better PTSD outcomes (Norrholm et al., 2016). Greater improvements in PTSD severity have been associated with coinciding changes in better glucocorticoid receptor sensitivity and reductions in heart rate reactivity (Rabe et al., 2006; Yehuda et al., 2015). Further, when exposed to fear-related stimuli prior to treatment, more regulated functioning in key brain regions implicated in fear extinction (e.g., greater ventromedial prefrontal cortical activation and larger rostral anterior cingulate cortex) have been associated with improved PTSD outcomes (Bryant, Felmingham, Kemp, et al., 2008; Bryant, Felmingham, Whitford, et al., 2008; Fonzo et al., 2017). Taken together, these findings suggest that greater arousal reflects more engagement with trauma-

related emotions, which allows for deeper learning via therapy that reminders of the trauma are no longer threatening. Interestingly, some studies have investigated whether carriers of risk alleles of genes modulating fear expression and related PTSD risk may be associated with T-F psychotherapy response (Bryant et al., 2010; Felmingham et al., 2013). It has been suggested that these individuals are prone to strong anxiety and fear conditioning and may have difficulty in engaging in extinction processes during T-F psychotherapy, and thus display attenuated response rates (Bryant et al., 2010).

In addition to these extinction-based factors predicting treatment response, there is also evidence of a broad array of other factors not directly related to fear that are also associated with T-F psychotherapy outcomes. These include a range of variables that are potentially malleable and may be targeted to modulate treatment response. It is not uncommon for individuals with PTSD to present with co-occurring conditions such as depression, substance abuse, suicidality, and physical conditions (Brady et al., 2000). Comorbid depression can impede T-F psychotherapy response (Buhmann et al., 2018; Stein et al., 2012). It is thought that depression can impede treatment response because depression can involve entrenched negative beliefs as well as low energy and motivation that can impede treatment progress (Stein et al., 2012). Other studies have found that pretreatment depression may promote better treatment response, which has been interpreted as more severely depressed patients "catching up" with other patients in the course of therapy as their depression improves in association with improved PTSD (Rizvi et al., 2009). Additionally, problematic alcohol use has been associated with poorer treatment outcomes, arguably because alcohol abuse contributes to treatment dropout and poor compliance (van Minnen et al., 2002; Zang et al., 2019). Further, the presence of other comorbid disorders such as generalized anxiety may also contribute to reduced treatment response because T-F psychotherapy does not address more general anxiety symptoms with strategies that are known to target these specific problems (Allan et al., 2017). While not a psychiatric comorbid condition, pretreatment levels of pain can impede treatment response because pain can be highly distracting and limits the extent to which patients can focus on and utilize therapy strategies (Sripada et al., 2019).

There are other potentially malleable factors that impede treatment response. Compromised executive functioning has been shown to limit treatment response, arguably because the capacity to engage regulatory cognitive skills to utilize therapy strategies and manage aversive emotional states is important for therapy success (Crocker et al., 2018; Nijdam, de Vries, et al., 2015). Trauma-related cognitions, primarily involving exaggerated negative thinking patterns, is predictive of poor treatment outcome (Cooper et al., 2017; Holliday et al., 2018; Zalta et al., 2014), which accords with cognitive models of PTSD that a pessimistic cognitive style that tends to interpret events in an overly negative manner can impede treatment response (Ehlers et al., 1998). This finding is consistent with the pattern of a sense of external locus of control predicting poor treatment response because this attitude involves the sense that one has limited control over one's outcomes (Böttche et al., 2016; Livanou et al., 2002). Avoidant coping and higher dissociation levels also predict poor treatment response (Leiner et al., 2012; Resick et al., 2012). Dissociative responses are a form of avoidance that involve minimization of emotional discomfort, and so these factors can limit engagement with trauma memories and related emotions, thereby impeding extinction processes. Other affective states including anger (Lloyd et al., 2014; Miles et al., 2015) and elevated shame and guilt (Øktedalen et al., 2015) predict worse outcomes. It has been noted that these emotions may not respond to extinction processes because they are not fear-based, and so may not be responsive to exposure therapy (Foa et al., 1995; Litz et al., 2009). Additionally, different forms of social support may influence treatment outcomes. Specifically, positive support from others has been found to improve PTSD outcomes (Shnaider et al., 2017; Thrasher et al., 2010), arguably because positive social support is a very strong buffer against stress and promotes strong psychological well-being via a number of mechanisms (Bryant, 2023). In addition, modification in partners' behaviors with attempts to reduce a loved one's distress has been found to improve treatment outcomes (Fredman et al., 2016). Conversely, low levels of social support, including high expressed emotion from close others, predicts poorer treatment outcomes (Tarrier et al., 2000). It is relevant to note that the aforementioned malleable factors, including depression, appraisals, alcohol abuse, comorbid conditions, anger, guilt, pain, and social interactions, can be modified by targeted interventions (Cuijpers et al., 2014; Ehlers et al., 2014; O'Shea et al., 2017; Wang et al., 2023).

A number of demographic characteristics relating to the patient and their traumatic event has been associated with PTSD outcomes following treatment. For example, females are more likely to respond positively to T-F psychotherapy (Stenmark et al., 2014; Tarrier et al., 2000), possibly because hormonal factors such as estradiol may contribute to females having better memory for emotional events (Felmingham & Bryant, 2012; Segal & Cahill, 2009). Older people tend to respond more poorly to T-F psychotherapy (Litz et al., 2009), which may be attributed to chronicity of the PTSD or to diminished cognitive abilities that allow older people to use therapy strategies optimally (Dinnen et al., 2015). Poorer treatment response has also been noted in people with lower socioeconomic status (Wachen et al., 2014), and this may be attributed to treatment response being influenced by people having to also manage financial, housing, and employment stressors. In terms of trauma-related factors, experiencing trauma during childhood has been shown to be associated with poorer treatment response (Karatzias et al., 2019). This trend has been attributed to childhood abuse survivors with PTSD having difficulties in regulating emotions, and therefore not successfully benefiting to exposure-based therapies (Cloitre et al., 2002). Poorer response has also been noted in people who develop PTSD following sexual assault or combat trauma (Markowitz et al., 2017; Zandberg et al., 2016), which has been linked to the interpersonal nature of these traumatic events leading to more complex problems involving issues of blame, shame, guilt, or anger that may not be amenable to extinction-based strategies such as exposure therapy (Litz et al., 2009).

The Present Review

It is apparent that there are a diverse range of potential predictors of response to T-F psychotherapy. This is perhaps not surprising considering that PTSD is a highly heterogeneous condition that can comprise fear and nonfear-related emotional states, associated cognitive and behavioral features, and significant comorbidity (Bryant, Galatzer-Levy, & Hadzi-Pavlovic, 2023; Elhai & Palmieri, 2011; Galatzer-Levy & Bryant, 2013). In the context of the

accumulating evidence suggesting that attempts to augment T-F psychotherapies have been only modestly successful, it is possible that one reason for the limited success of these strategies has been the strong focus on extinction-based mechanisms when there are many other potential factors that can influence treatment response. Adopting a more data-driven approach of factors that predict T-F psychotherapy response may shed light on optimal candidates that should be tested to augment treatment outcomes. To this end, we aimed to conduct a comprehensive systematic review and meta-analysis of studies of T-F psychotherapy trials that reported baseline factors that influenced PTSD outcome. We conducted separate meta-analyses on factors assessed at baseline that were associated with subsequent treatment outcomes. Given this data-driven approach, a priori decisions relating to the number of meta-analyses and nature of predictor categorizations were not made at the outset. Additionally, secondary exploratory moderator analyses were conducted (as appropriate) to assess the impact of study design, and sample characteristics in influencing the magnitude of the factors' associations with treatment outcomes.

Method

Transparency and Openness

The study protocol was preregistered at the International Prospective Register of Systematic Reviews (registration number CRD42020162112). Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 reporting guidelines were used for this article (Supplemental Table S4). The data and coding manual used in this review have been deposited and can be viewed at Open Science Framework (https://osf.io/uy9j2/?view_only=c0c422ebe55342b4bb010b9e9f5921b0).

Search Strategies

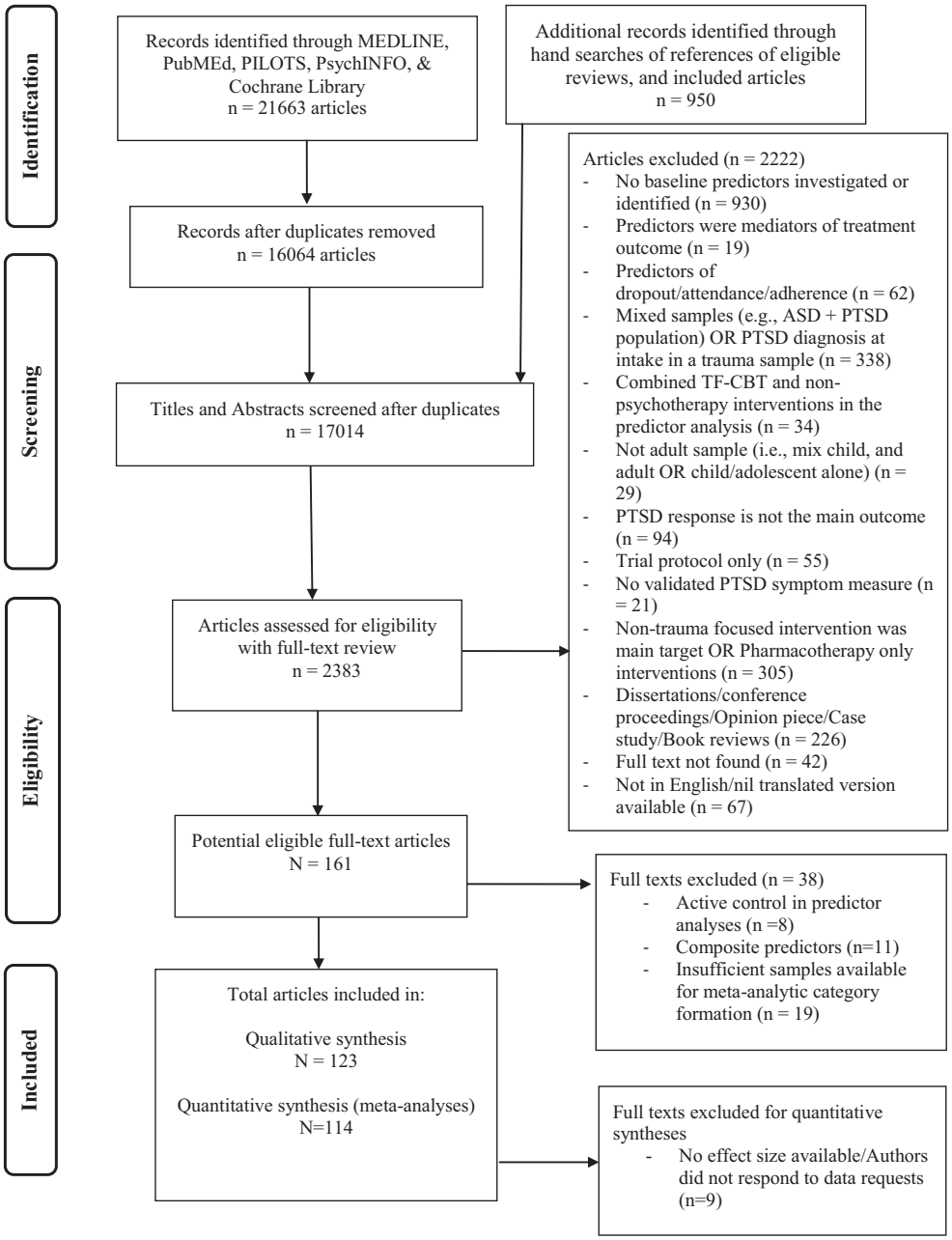
Electronic database searches were conducted across three separate cycles. Database searches were conducted on PubMed (includes MEDLINE), APA PsycInfo, PTSDpubs (formerly Published International Literature on Traumatic Stress), and Cochrane library using the following combination of terms: (Random OR trial OR RCT OR controlled OR allocate OR assign OR open) AND ("Psychotherapy"[Mesh] OR "Counseling"[Mesh] OR "Acceptance and Commitment Therapy"[Mesh] OR "Mindfulness"[Mesh] OR "stress inoculation" [Mesh] OR "treatment*"[tiab] OR "psychotherap*"[tiab] OR "psychoeduc*"[tiab] OR "psycho educ*"[tiab] OR "stress manag*"[tiab] OR "stress reduc*"[tiab] OR "self monitoring"[tiab] OR "counsel*"[tiab] OR "multicomponent"[tiab] OR "multi component"[tiab] OR "relaxation"[tiab] OR "mindfulness"[tiab] OR "meditati*"[tiab] OR "cbt"[tiab] OR "cognitive-behav*" [tiab] OR "cognitivebehav*" [tiab] OR "cognitive*" [tiab] OR "behavio*" [tiab]OR "emotional regulati*"[tiab] OR "emotion regulati*"[tiab] OR "emotion focused"[tiab] OR "problem solving"[tiab] OR "behavioural activation"[tiab] OR "behavioral activation"[tiab] OR "psycho analy*"[tiab] OR "self directed"[tiab] OR "self help"[tiab] OR "psychodynamic*" [tiab] OR "stress management*" [tiab]) AND ("PTSD" [MeSh] OR "posttraumatic*" [MeSh] OR "post-traumatic*" [Mesh] OR "stress disorder" [Mesh]). In the initial search, any form of clinical trial of psychotherapy for PTSD was sourced with the intention to ensure a thorough and comprehensive search of all available studies on T-F psychotherapy from inception to October 2022. A second database search including the above-mentioned search terms as well as the search terms "predictor" OR "moderator" was conducted to capture a thorough search of trials identifying moderators of T-F psychotherapy outcome. Finally, three additional "hand search" strategies were conducted to obtain relevant studies that were missed in the original database searches. First, reference lists of all eligible articles were searched. Second, reference lists of literature reviews, systematic reviews and meta-analyses of PTSD treatment interventions that were generated from the database searches above were screened for eligibility. Third, major journals that publish articles on PTSD were hand searched from inception to October 2022, including *Journal of Traumatic Stress*, *European Journal of Psychotraumatology*, and *Psychological Trauma.* The specific search terms for the range of databases searched are listed in the Supplemental Materials.

Inclusion Criteria

Each article was subjected to the following inclusion criteria to guide the selection of studies for meta-analytic review. First, articles had to be a full-text report of a treatment study written or translation available in the English language and published in a peer-reviewed journal. Second, articles needed to include a sample where all participants were exposed to a traumatic event as defined by, *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, *DSM-III*, *DSM-IV,* or *DSM-5* Criterion A (American Psychiatric Association, 1980, 1994, 2013). Third, trial participants had to be assessed for PTSD symptoms at least 1 month after exposure to the index traumatic event, where primary acute stress disorder and/or mixed PTSD/acute stress disorder samples were excluded. Specifically, trial participants had to meet criteria for full- or subthreshold PTSD at intake, where the latter was defined as the presence of a Criterion A traumatic event and re-experiencing symptoms with at least one of avoidance, or hyperarousal symptom cluster being endorsed. Fourth, research articles had to report on adult-only samples where participants were 18 years of age or older, although the traumatic event may have occurred at any point in the lifespan. This criterion was selected as differential presentation of posttraumatic stress reactions across the lifespan (i.e., child and adolescent vs. adult samples) could influence response to T-F psychotherapy in divergent ways (Kaminer et al., 2005). Fifth, any form of treatment trial for PTSD (e.g., RCT, uncontrolled single-group design trial such as outpatient programs, nonrandomized clinical trial) was included. This criterion was selected to ensure an expansive search of all relevant treatment trials for PTSD. Sixth, PTSD outcome had to be assessed using either a validated clinician-rated or self-report measure of PTSD severity. Finally, T-F psychotherapy included any clinical intervention with the primary aim of reducing PTSD symptoms via target of trauma-related content including traumatic memories, interpretations, and associated emotions and the management of related stressors such as confrontation of trauma-related activities and situations. Interventions delivered in individual and group format, via virtual reality or telehealth/internet modalities were included. Interventions targeting only select PTSD symptoms in isolation (e.g., posttraumatic nightmares) were excluded.

Figure 1

Flowchart: Identification and Selection of Articles



Note. ASD = acute stress disorder; PTSD = posttraumatic stress disorder; TF-CBT = trauma-focused cognitive behavioral therapy.

Selection of Studies

Figure 1 summarizes the study selection process and exclusion criteria. A total of 21,663 records were identified through database searches. Following removal of duplicates, the titles and abstracts of 17,014 articles were screened according to the inclusion and exclusion criteria. At this review stage, 2,383 articles were identified as requiring full-text review for inclusion. Following exclusion of 2,222 articles (see Figure 1, for reasons), 123 articles were finalized for inclusion in the systematic review and 114 in the meta-analyses. Three independent raters reviewed articles and discrepancies in inclusion/exclusion decisions were decided by the first and last authors reviewing these articles a second time against the inclusion/exclusion criteria. Timing of predictors were restricted to baseline or pretreatment factors. Observed predictor analyses that assessed

PTSD treatment outcome in exclusively T-F psychotherapy conditions or via comparison of T-F psychotherapy conditions with inactive control conditions (e.g., waitlist control, supportive counseling) were included. These inactive conditions were coded as either passive or nonspecific; passive control conditions differed from nonspecific conditions in that the former did not involve any form of therapy (e.g., psychoeducation only), whereas the latter involved non-T-F psychotherapy conditions (e.g., present centered therapy, psychotropics). Articles consisting of predictor analyses that combined active and control conditions (e.g., mixed active controls such as present centered therapy and drug therapy groups with T-F psychotherapy samples) and/or involved comparison of two or more active T-F psychotherapy conditions (e.g., cognitive processing therapy vs. cognitive processing therapy and Behavioral Activation) were excluded ($n = 8$). Articles that examined a composite of predictors such as unique patient profiles in predicting PTSD treatment outcome were excluded ($n = 11$), as individual variables within the composite could not be meaningfully interpreted for the purpose of the current analysis. Finally, when the required data to evaluate the inclusion/exclusion criteria were not detailed in a given article, this information was requested via email from authors. Similarly, when an effect size was not available or data necessary to calculate an effect size was not provided, we contacted study authors to request the relevant data. See Supplemental Table S1 for a detailed description of the different reasons for exclusion of full texts.

Coding Procedure

A standardized coding system was developed and applied to organize the coding process for each eligible article. A series of (a) study characteristics, (b) predictor analysis quality ratings, and (c) risk of bias (ROB) assessments were coded for use in moderator analyses. The first author and two trained graduate research assistants coded all articles where any discrepancies in coding were resolved by the first author. Type of study design was coded as two-group design including quasi- or fully RCT, single-group design involving open trials, and uncontrolled trials involving residential outpatient specialized programs. This coding method was chosen to simply descriptively characterize varying study designs included in this article. To account for bias inherent across these designs (e.g., predictors identified from the T-F-psychotherapy condition in a quasi- vs. fully randomized trial), separate predictor bias ratings were calculated (detailed further below). The *DSM* definition of PTSD used to deduce diagnosis at intake in a given clinical trial was also coded, as part of descriptive statistics. Dichotomous study characteristics included: sample population (civilian, refugee, or military); type of trauma (combat or war, acts of terror/mass violence or interpersonal violence, mixed traumas including accident and medical illness, and natural disaster); developmental timing of index trauma (adulthood, childhood or mixed); type of PTSD instrument (self-report vs. clinical report); and timing of PTSD outcome assessment coded as during treatment (mid treatment), immediately after treatment (post), and follow-up (from 1 month and up to 5 years since completion of treatment). Coding of continuous study characteristics included sample size, mean age of participants, and proportion of female participants. If a study provided both intent-to-treat and treatment completer sample specific predictor analyses, then the former was preferentially coded.

For all effect sizes, five quality items that would potentially introduce bias in the observed study-specific predictor analyses were coded. These included (a) the internal reliability of the PTSD instrument >.7 (Yes [1] vs. No/Not reported [0]); (b) internal reliability of the predictor instrument >.7 for self-report measures (Yes [1] vs. No/Not reported [0]); (c) the amount of score-level (and not study-level) missing data for the predictor or PTSD instrument <20% (Yes [1] vs. No/Not reported [0]); (d) whether authors used an appropriate statistical method for handling score-level missing data (Yes [1] if there was no missing data, list-wise deletion for less than 10% missing data, multiple imputation for more than 10% missing data, data were assessed to be missing at random for two-group design studies vs. No/Not reported, list-wise deletion for more than 10% missing data, missing imputation including last observation carried forward, data not missing at random [0]); and (e) percent of study sample retained at each PTSD outcome time point for the predictor analysis (80%–100% retained [2], 50%–79% retained [1], less than 50% retained or unknown [0]). A measure of predictor analysis quality for observed effect sizes was deduced by adding these five quality items detailed above. In terms of neuroimaging, psychophysiological, and genetic analyses, predictor analysis quality relating to the predictor instrument was not assessed using the aforementioned metrics. Instead, these were assessed as having a satisfactory quality if relevant studies used specific standardized paradigms that were conducted in accordance with required standards pertinent to those domains.

Methodological Quality

To assess for the level of ROB at the study design level, eligible studies were assessed using predefined criteria based on the Cochrane ROB tool for randomized and nonrandomized trials (Higgins et al., 2011) combined with the Downs and Black checklist (Downs & Black, 1998) for items specific to uncontrolled designs that were not captured in the Cochrane guidelines. This combined approach focused on five key domains: (a) reporting, confounding, and external validity bias; (b) adherence to interventions, incomplete outcome data, and blinding of outcome assessment; (c) internal validity, attrition and missing outcome data related bias; (d) measurement of outcomes and detection bias; and (e) selective reporting and related bias. Lack of blinding (of participants and investigators) was considered of minor concern, as this is generally difficult to achieve in psychological intervention studies. A graduate research assistant independently coded each study for ROB in accordance with the above guidelines. ROB for each criterion was rated as high, medium or unclear, or low risk (Higgins et al., 2011). Interrater reliability was assessed by having a second blind rater code 25% of randomly selected studies. There was substantial agreement between the two raters ($\kappa = 0.68$, $p < .0001$). ROB results are presented in Supplemental Figure S1.

Meta-Analytic Procedure

To allow predictors to be subjected to meta-analytic study, all eligible predictor studies were categorized in a data-driven manner that were agreed upon by raters after initial perusal of predictor types. A meta-analysis was performed if a given baseline factor was

identified in at least three independent research articles (studies excluded; $n = 19$). The included predictor categories (and subtypes subsumed within) were: (a) patient-related demographic factors (i.e., age, gender, race, and education); (b) pretreatment PTSD severity; (c) trauma-related demographic factors (i.e., "childhood- vs. adult-onset," "trauma load," "duration since the trauma," "service-related trauma."); (d) depression; (e) trauma-related cognitions; (f) anger difficulties; (g) emotion regulation; (h) social support; (i) executive function; (j) fear biology ("fear-neural" factors that involved neural activation in brain regions implicated in fear, "fear-physiological reactivity," and "fear-genetic risk alleles"); (k) other clinical factors related to general functioning (i.e., "problems with pain," "service-related disability," "sleep issues," and "quality of life"); and (l) psychiatric comorbidities (i.e., "problematic alcohol use," and "personality disorder screen"); note that comorbid depression is in its own category because of the strong evidence that it has high comorbidity with PTSD (Rytwinski et al., 2013). Subcategories subsumed within predictor category types resulted in a total of 24 meta-analyses (see Supplemental Table S2).

Effect sizes were computed based on the information provided in the research articles and/or obtained from authors. A range of statistics were used across studies to quantify the relationship between baseline factors and T-F psychotherapy outcome; these included standardized and unstandardized β values, *t*-statistics from one- and two-group analyses, and *F* statistics from two-group one-way analyses of variance. A minority of studies dichotomized PTSD response (e.g., responder vs. nonresponder) and provided means and standard deviations for these groups with follow-up *t* or *F* statistics. Others reported a bivariate or partial correlation to depict the role of a given baseline factor being associated with outcomes. For the current purpose, we calculated a bivariate correlation (*r*) and its 95% confidence interval between a given predictor category and PTSD severity following T-F psychotherapy. The magnitude of the correlation was interpreted as small (0.10), medium (0.30), or large (0.5; Cohen, 1992). For studies in which these data were not directly reported, correlations were derived from *t* values or *F* ratios. To this end, a small percentage of studies ($n = 6$) could not be quantified when it was considered erroneous to convert some of the reported statistics to correlations (e.g., unstandardized β coefficients without reporting of standard errors). If multiple separate time points (e.g., post vs. follow-up) and report formats (e.g., self- vs. clinician report) were assessed in one study, all eligible effect sizes were coded. If, however, multiple methods were used to assess PTSD severity for a given time point (e.g., percentage improvement and pre- to post-PTSD symptom change), then an aggregate effect size was coded. Similarly, when multiple effect sizes for a given baseline measure were reported in the one study, then an aggregate effect size was coded. For studies in which both the total scores and subscale scores were reported for a given intended meta-analysis (e.g., total trauma cognitions vs. mistrust only cognitions), only the total score was extracted for effect size calculations. Effect sizes were coded such that higher levels of a given predictor category (e.g., higher trauma-related negative beliefs in the cognition category) represented higher levels of PTSD following treatment. In this instance, the expected relationship between trauma-related cognitions and PTSD was positive. Using this category as an example, if articles reported effect sizes in which negative beliefs was represented by lower scores, the reported effect size was reversed. After requesting the required information from a total of 10 studies (that did not report the necessary information for effect size calculation), we received effect sizes from one study (Fonzo et al., 2017).

All analyses were conducted using the Comprehensive Meta-Analysis software package, Version 3 (Borenstein et al., 2011). Separate meta-analyses for 24 predictor categories, including relevant moderator analyses were performed using random effects models. Random effects models are generalizable and conservative in that they allow for random differences between studies insofar as methodology, samples and other study characteristics (Lipsey & Wilson, 2001). Fisher's *Z* transformation was applied, 95% confidence intervals were computed for the combined effect sizes in each predictor category, and they were then converted to bivariate correlations for ease of reporting. As the assumption of independence for effect sizes was not met for some studies (e.g., self-report and clinical report for a given time point), an adjusted effect size was calculated in this instance. Further, heterogeneity of effect sizes was separately examined for each predictor category using the Q, I^2, and T^2 statistics. The Q statistic evaluated the significance of heterogeneity; however, as this statistic provides a sum and therefore is sensitive to the number of studies (Cuijpers, 2016), the I^2- and T^2 statistics were together considered to evaluate whether there was sufficient variability of samples around the 24 effect sizes to test for potential moderation. Specifically, I^2 tells us the *proportion* of variance in observed effects that reflects true effects (rather than sample error); whereas the T^2 statistic provides an *estimate* of the variation of true effect sizes (Borenstein et al., 2017). Finally, publication bias was assessed via visual inspection and symmetry of separate funnel plots using Egger's test of the intercept (Egger et al., 1997). This scatterplot depicts the estimated predictor effect size against the measure of the study size (or precision). Here, larger (and more precise studies) appear near the combined effect size at the top of the plot, whereas smaller (and less precise studies) will display a wider distribution at the bottom of the plot. The absence of publication bias will be depicted by symmetrically distributed effects on both sides of the combined effect size line, and an intercept that is not significantly different from zero. Thus, a significant Egger's test was followed up with Duval and Tweedie's trim-and-fill procedure (Duval & Tweedie, 2000). Adjusted effect sizes and confidence intervals that account for missing studies based on asymmetry of the funnel plot are provided by this method.

We next examined the role of methodological characteristics in influencing a given baseline category association with PTSD outcome. These characteristics were explored as two separate metaregression analyses. First, study design or selection characteristics involving predictor analysis quality ratings, ROB, and number of therapy sessions were explored together in a multivariate metaregression. Predictor categories with fewer number of studies were assessed for bivariate associations with study design characteristics first. If bias variables were significant, these were retained as a covariate for subsequent metaregression analyses involving study sample characteristics of interest. Specifically, if sufficient heterogeneity was present (based upon Q, I^2, and T^2 statistics), study sample characteristics including age, percent female, sample type, trauma type, and trauma timing characteristics were assessed for bivariate associations via separate metaregressions first.

Significant bivariate moderators were then simultaneously added into a multivariate metaregression. This two-step approach was taken with study sample moderation tests to accommodate the low number of studies in a number of predictor categories, and thereby attempt to ensure reliable moderator analyses. Last, sensitivity analyses were performed on a minority of studies that recruited patients with subthreshold (vs. full) PTSD at intake, and we endeavored to explore whether this difference in intake criterion significantly influenced the results in any given category.

Results

Characteristics of the Studies

There were 17,014 articles screened, 2,383 articles required full-text review, and 123 research articles comprising of 114 studies were finally included (Figure 1; see Supplement 1, Table S3, for a full list). Of these, nine studies could not be quantitively analyzed as the authors of these research articles did not respond to data requests or could not provide the relevant information requested, and as such these research articles have been qualitatively detailed under the relevant treatment predictor categories. There were 237 observed effect sizes across 24 treatment predictor categories. There were 57 randomized controlled or controlled clinical trials (RCT or CCT), and 57 uncontrolled clinical trials, comprising 61,970 participants (mean age 40.14 ± 8.02 years; females, 40.1%). Studies assessed civilian (46.5%), refugee or torture survivor samples (7%), and military and veteran samples (46.5%). Primary index traumas that were the target of treatment interventions included 40% combat/war, 7% acts of terror/mass violence, 17% interpersonal violence, 3% accident, natural disaster or medical illness, and 33% mixed traumas across studies. Among studies that reported timing of primary index trauma, 68.4% assessed individuals with traumas that occurred in adulthood, 4.4% in childhood, and 18.4% were a combination of both timings; timing of the primary index trauma could not be categorized for 8.8% of the total number of studies. The *DSM-IV* definition was most frequently used to diagnose PTSD at intake (80.7%), followed by the *DSM-5* (8.8%), *DSM-III* (3.5%), *International Classification of Diseases–10* criteria (3.5%), and a combination of *DSM-III, IV, and 5* (3.5%) definitions. Most common measures of PTSD were the Clinician-Administered PTSD Scale (34%), PTSD Checklist (29%), and Posttraumatic Diagnostic Scale and PTSD Scale-Self-Report (13%). Less commonly used measures included the Impact of Events Scale, Harvard Trauma Questionnaire, Mississippi Scale for PTSD, and Posttraumatic Symptom Scale. The majority of studies comprised seven–11 treatment sessions (66%). Table 1 details descriptive characteristics of the studies. Observed effect sizes from the 114 samples were derived from statistical analyses comprising the T-F psychotherapy only (83%) and including comparisons with a passive (6%), or nonspecific (11%) control condition. Given the purpose of the meta-analysis was to determine the magnitude of effect of predictors of T-F psychotherapy, observed effect sizes from analyses involving an active control condition were excluded because these studies compared variations of T-F psychotherapy or other evidence-based therapy; the effect sizes provided by these studies did not permit calculation of the effect of T-F psychotherapy outcome itself (see Figure 1).

Association Between Baseline Characteristics and Trauma-Focused Treatment Outcome

Table 2 provides details on the baseline factors that comprised each predictor category. Supplemental Table S2 details information relating to the descriptors of each predictor category. Table 3 presents the results for all 24 meta-analyses including within-category effect sizes, heterogeneity, and relevant moderation tests. Figure 2 depicts a forest plot of the 24 predictor categories. Supplemental 2 details funnel plots relating to publication bias included observed and imputed estimates (after accounting for bias). (https://osf.io/uy9j2/?view_only=c0c422ebe55342b4bb010b9e9f5921b0).

Fear Biology Factors

Neural Provocation. Brain regions defined as being involved in the fear circuitry network included the amygdala, hippocampus, insula, anterior cingulate, and frontal gyrus. Six studies assessed the relationship of neural activation in response to provocation tasks conducted prior to treatment commencing in these target regions and subsequent treatment outcome. Increased activation was associated with better PTSD outcomes ($r = -0.44$; 95% CI [−0.28, −0.58]; $Z = 4.98$, $p < .0001$). One study that could not be quantified similarly identified that greater activation in the anterior insula and modulation in the prefrontal regions during an emotion appraisal task at baseline was associated with PTSD improvement (Duval et al., 2020). Heterogeneity analyses were not significant (n.s.), $Q(5) = 7.14$, $p > .05$, $I^2 = 30.01$, $\tau^2 = 0.02$. Thus, moderator analyses with study sample characteristics were precluded. Egger's test of the intercept was not significant, $t(4) = 0.52$, $p > .05$, indicating no publication bias.

Physiological Reactivity. Physiological arousal in response to fear provocation stimuli prior to T-F psychotherapy was assessed in seven studies implicating cortisol reactivity and cortisol awakening response, heart rate reactivity, and blood pressure changes. Higher physiological reactivity at baseline was associated with better PTSD treatment outcome ($r = -0.46$; 95% CI [−0.71, −0.11]; $Z = 2.49$, $p < .015$). Heterogeneity tests suggested that 86% true between-study variance, $Q(6) = 44.22$, $p < .0001$, $I^2 = 86.43$, $\tau^2 = 0.23$, indicating that moderator analyses were appropriate. Egger's test of the intercept was significant, $t(5) = 3.31$, $p < .05$. Trim-and-fill analyses revealed two studies were missing to the right of the mean, but the adjusted correlation did not substantially differ from the observed value ($r = -0.34$; 95% CI [−.59, −0.02]). Thus, the potential impact of publication bias was minimal.

Genetic Polymorphisms. Across three studies, risk alleles of genes modulating fear expression (and PTSD) of the FK506 binding protein 5 or brain-derived neurotrophic factor genes were associated with poorer outcomes ($r = 0.49$; 95% CI [0.31, 0.64]; $Z = 4.83$, $p < .0001$). Heterogeneity analyses were not significant, precluding that further moderation tests, $Q(2) = 2.93$, $p > .05$, $\tau^2 = 0.01$. Egger's test of the intercept was not significant, $t(1) = 10.51$, $p > .05$.

Other Clinical Factors

Executive Function. Across five studies, increased executive function including attentional control predicted better outcomes ($r = -0.29$; 95% CI [−0.39, −0.17]; $Z = -4.79$, $p < .0001$). One study

Table 1
Descriptive Characteristics for Meta-Analyses

Study design characteristic	*n*
Decade	
1990s	5
2000s	19
2010s	68
2020s	20
Country	
Australia	13
North America	67
United Kingdom	7
Denmark	7
France	1
Germany	2
Korea	1
Poland	1
Netherlands	13
Uganda	2
Trial type	
RCT	56
CCT	1
UCT	57
PTSD definition used	
DSM-III	4
DSM-IV	92
DSM-5	10
DSM-IV and DSM-III combined	1
DSM-IV and DSM-5 combined	3
ICD-10	4
PTSD measure used	
CAPS-III, IV, V	39
PSSI	6
PCL-M/-S-C	33
PDS/PSS-SR	15
IES/IES-R	3
HTQ	8
MPSS	4
Other (e.g., SI-PTSD)	4
Number of treatment sessions	
One—six sessions	7
Seven–11 sessions	74
12–20 sessions	23
20+ sessions	5
Not reported/unknown	5

Study sample characteristic	*n*/*M* (*SD*)
Mean age	40.14 (8.02)
Mean percent female	40.14 (34.17)
Sample type	
Civilian	53
Military/veteran	53
Refugee/torture survivor	8
Trauma timing	
Adulthood	78
Childhood	5
Mixed	21
Not specified	10
Trauma type	
Combat/war	45
Acts of terror/mass violence	8
Interpersonal violence	19
Mixed traumas (e.g., natural disaster, medical illness, accident)	41

(*table continues*)

Table 1 (*continued*)

Note. *n* = number of studies; RCT = randomized controlled trial; CCT = controlled clinical trial; UCT = uncontrolled clinical trial; PTSD = posttraumatic stress disorder; *DSM* = *Diagnostic and Statistical Manual of Mental Disorders*; *ICD* = *International Classification of Diseases*; CAPS = Clinician-Administered PTSD Scale; PSSI = PTSD Symptom Scale Interview; PCL-M = PTSD Checklist-Military (S = Specific, C = Civilian); PDS = Posttraumatic Diagnostic Scale; PSS-SR = PTSD Symptom Scale: Self-Report; IES = Impact of Events Scale (R = Revised); HTQ = Harvard Trauma Questionnaire; MPSS = Mississippi Scale for PTSD; SI-PTSD = Structured Interview for PTSD.

could not be quantified where the role of activation in executive function brain networks during an executive function task at baseline was associated with PTSD outcome (Falconer et al., 2013); the relationship was similarly negative. Heterogeneity analyses were not significant, $Q(5) = 7.54$, $p > .05$, $\tau^2 = 0.01$. Egger's test of the intercept was significant, $t(4) = 4.08$, $p < .05$. Trim-and-fill analyses revealed two studies were missing to the left of the mean, but the adjusted correlation did not substantially differ from the observed value ($r = -0.36$; 95% CI [−.47, −0.25]). Thus, the potential impact of publication bias was minimal.

Trauma-Related Cognition. Across 13 studies, trauma-related cognitive distortions prior to treatment predicted poorer outcomes ($r = 0.37$; 95% CI [0.21, 0.51]; $Z = 4.26$, $p < .0001$). Sensitivity analyses with one study removed (Owens et al., 2008) that utilized subthreshold (vs. full) PTSD diagnosis at intake, revealed comparable effects with a similar overall correlation ($r = 0.39$; 95% CI [0.21, 0.54]), suggesting that this study did not significantly influence the results. One study that was not quantified detailed that positive reappraisal at baseline was associated with worse PTSD outcome (Wisco et al., 2013). This study was omitted from the meta-analysis as it was dissimilar to the rest of the studies depicting the nature of negative trauma cognitions in influencing PTSD outcome. Heterogeneity levels indicated 88% true between-study variance, $Q(11) = 88.88$, $p < .0001$, $I^2 = 87.82$, $\tau^2 = 0.08$, indicating appropriate further moderation tests. Egger's test of the intercept was not significant, $t(10) = 0.94$, $p > .05$.

Anger. Difficulties with anger reported across seven studies was significantly associated with worse outcomes ($r = 0.31$; 95% CI [0.23, 0.38]; $Z = 7.54$, $p < .001$). Heterogeneity tests were not significant, $Q(6) = 1.9$, $p > .05$, $I^2 = 0.00$, $\tau^2 = 0.00$, precluding further moderation tests with sample specific moderators. Egger's test of the intercept was not significant, $t(5) = 2.25$, $p > .05$.

Emotion Regulation. Difficulties with emotion regulation across three studies did not relate to PTSD outcomes ($r = 0.20$; 95%CI [−0.26, 0.59]; $Z = 0.85$, $p > .05$).

Social Support. Across 16 studies, higher levels of social support (and lower levels of negative interpersonal and social reactions) predicted better outcomes ($r = -0.30$; 95% CI [−0.37,

Table 2
Baseline Predictor Categorizations

Descriptor	Meta-analytic category	Mean ES	*k*	$N_{studies}$	$N_{sample\ size}$
Fear biology factors	Neural provocation	−.443	26	6	192
	Physiological reactivity	−.462	11	7	195
	Risk alleles	.493	3	3	129
Other clinical factors	Executive function	−.285	13	5	392
	Trauma-related cognition	.371	27	13	1,157
	Anger	.309	8	7	475
	Emotion regulation	n.s.	4	3	163
	Social support	−.300	21	16	3,317
	Problems with pain	.148	6	5	3,556
	Service-related disability	.116	5	4	26,110
	Sleep issues	.452	4	4	349
	Quality of life	−.191	4	4	727
Demographic characteristics	Gender—Male	.120	9	7	27,025
	Education	n.s.	4	4	3,813
	Race—Caucasian/European	.159	8	6	6,578
	Older age	.279	6	5	1,537
Trauma-related characteristics and severity	Childhood trauma	.275	6	5	309
	Time since trauma	.548	3	3	67
	Trauma load	.369	3	3	386
	Service-related trauma	.329	5	4	1,124
	Pretreatment PTSD severity	.29	33	27	4,970
Psychiatric comorbidities	Depression	.418	18	16	1,680
	Problematic alcohol use	.189	7	6	24,620
	Personality disorder screen	.442	3	3	2,952

Note. ES = effect size; n.s. = not significant; PTSD = posttraumatic stress disorder.

−0.23]; $Z = -7.51$, $p < .0001$). Sensitivity analyses with one study removed (Price et al., 2013b) that utilized subthreshold (vs. full) PTSD diagnosis at intake did not significantly influence these results ($r = -0.31$; 95% CI [−0.39, −0.23]), suggesting that this study did not significantly influence the results. Heterogeneity tests revealed 72% true between-study variance, $Q(15) = 53.42$, $p < .0001$, $I^2 = 71.92$, $\tau^2 = 0.02$, suggesting follow-up moderation tests with sample characteristics were appropriate. A significant Egger's test of the intercept, $t(14) = 4.58$, $p < .005$, was followed up with trim-and-fill analyses. This revealed that six studies were missing to the right of the mean, with an adjusted correlation comparable to the observed value ($r = -0.22$; 95% CI [−0.29, −0.15]).

Service-Related Disability. The presence of service-related disability across four studies at baseline was associated with poorer PTSD treatment outcome ($r = 0.12$; 95% CI [0.06, 0.17]; $Z = 4.37$, $p < .0001$). One study that could not be quantified evidenced service connection and related disability to have similar positive relationship with PTSD outcome (Voelkel et al., 2015). True between-study variance was high, $Q(3) = 22.67$, $p < .0001$, $I^2 = 86.77$, $\tau^2 = 0.00$. Egger's test of the intercept was not significant, $t(2) = 0.41$, $p > .05$.

Problems With Pain. Pain-related interference in general functioning was associated with worse PTSD outcomes across five studies ($r = 0.15$; 95% CI [0.08, 0.21]; $Z = 4.31$, $p < .001$). Heterogeneity analyses were not significant, $Q(4) = 7.70$, $p > .05$, $I^2 = 48.07$, $\tau^2 = 0.00$. Egger's test of the intercept was significant, $t(3) = 5.33$, $p < .05$; trim-and-fill analyses revealed that two studies were missing to the left of the mean, where adjusted correlation was comparable to the observed value ($r = 0.12$; 95% CI [0.04, 0.17]).

Sleep Issues. Problems with sleep was associated with poorer outcomes across four studies ($r = 0.45$; 95% CI [0.22, 0.64]; $Z = 3.58$, $p < .001$). Between-study heterogeneity was high, $Q(3) = 12.71$, $p < .01$, $I^2 = 76.39$, $\tau^2 = 0.05$. Egger's test was significant, $t(2) = 30.40$, $p < .05$; trim-and-fill analyses revealed that one study was missing to the right of the mean, where adjusted correlation was comparable to the observed value ($r = 0.38$; 95% CI [0.14, 0.58]).

Quality of Life. Higher quality of life at baseline was associated with better outcomes across four studies ($r = -0.19$; 95%CI [−0.31, −0.07]; $Z = -3.04$, $p < .01$). Between-study heterogeneity was not significant, $Q(3) = 6.33$, $p > .05$, $I^2 = 52.61$, $\tau^2 = 0.01$. Egger's test of the intercept was significant, $t(2) = 6.78$, $p < .05$; trim-and-fill analyses revealed that one study was missing to the right of the mean ($r = -0.16$; 95% CI [−0.28, −0.03]).

Demographic Characteristics

Gender. Across seven studies, males were more likely to have worse PTSD outcomes ($r = 0.12$; 95% CI [0.06, 0.18], $Z = 4.08$, $p < .0001$). One study that could not be quantified (Voelkel et al., 2015) identified a similar association between gender and PTSD outcome. Although heterogeneity tests revealed approximately 73% true between-study variance, $Q(6) = 21.42$, $p < .001$, $I^2 = 71.98$, $\tau^2 = 0.00$, an overlap of variables within this category with study sample characteristics precluded any meaningful moderator tests. Egger's test of the intercept was significant, $t(5) = 7.55$, $p < .001$. Trim-and-fill analyses revealed two studies were missing to the left of the mean, where adjusted correlation was comparable to the observed value ($r = 0.10$; 95% CI [0.03, 0.16]).

Table 3
Baseline Factors Association With PTSD Symptoms Following T-F Psychotherapy

Predictor variable	*k*	*N*	*r* [95% CI]	*Q* (*df*)	ΔT^2
			Fear biology factors		
Neural provocation					
Estimate	26	192	−0.44**** [−0.28, −0.58]	7.14 (5)[n.s.]	
Moderator tests			Coef.		
Study quality			−0.02 [−0.54, 0.50]		
ROB (low vs. medium)			0.04 [−1.50, 1.58]		
No. of sessions			[a]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Physiological reactivity					
Estimate	11	195	−0.46** [−0.71, −0.11]	44.22 (6)****	
Moderator tests			Coef.		
Study quality			−0.02 [−0.16, 0.13]		
ROB (ref high)			[a]		
No. of sessions			−0.09 [−0.71, 0.53]		
M_{age}			[c]		
% female			[c]		
Sample type			[c]		
Trauma type (ref combat/war)			[c]		
Trauma timing			[c]		
Genetic polymorphisms					
Estimate	3	129	0.49**** [0.64, 0.31]	2.93 (2)[n.s.]	
Moderator tests			Coef.		
Study quality			[b]		
ROB (ref high)			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type (ref combat/war)			[b]		
Trauma timing			[b]		
			Other clinical factors		
Executive function					
Estimate	13	392	−0.29**** [−0.39, −0.17]	7.54 (5)[n.s.]	
Moderator tests			Coef.		
Study quality			0.08 [−0.05, 0.20]		0.001
ROB (ref low)					0.01
Medium			0.09 [−0.24, 0.42]		
No. of sessions (ref seven–11 sessions)					0.001
12–20 sessions			0.15 [−0.09, 0.38]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Trauma-related cognition					
Estimate	27	1,157	0.37**** [0.21, 0.51]	88.88 (11)****	
Moderator tests			Coef.		
Study quality			−0.01 [−0.12, 0.11]		0.01
ROB (ref high)					0.01
Low			−0.29 [−0.98, 0.42]		
Medium			−0.44 [−1.15, 0.27]		
No. of sessions (ref one–six sessions)					0.04
Seven–11 sessions			−0.19 [−0.98, 0.61]		
12–20 sessions			−0.14 [−1.00, 0.71]		
20+ sessions			−0.11 [−1.00, 0.79]		
M_{age}			[c]		
% female			[c]		
Sample type			[c]		
Trauma type			[c]		
Trauma timing			[a]		

(*table continues*)

Table 3 (*continued*)

Predictor variable	*k*	*N*	*r* [95% CI]	*Q* (*df*)	ΔT^2
Anger					
Estimate	8	475	0.31*** [0.23, 0.38]	1.91 (6)[n.s.]	
Moderator tests			Coef.		
Study quality			−0.02 [−0.08, 0.04]		0.00
ROB (ref medium)					0.00
Low			−0.13 [−0.46, 0.20]		
No. of sessions (ref seven–11 sessions)					0.00
12–20 sessions			0.05 [−0.23, 0.26]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
# Social support					
Estimate	21	3,317	−0.30**** [−0.37, −0.23]	53.42 (15)****	
Moderator tests			Coef.		
Study quality			0.001 [−0.11, 0.11]		0.00
ROB (ref low)					0.00
Medium			−0.02 [−0.30, 0.25]		
No. of sessions (ref seven–11 sessions)					0.00
12–20 sessions			0.02 [−0.19, 0.25]		
20+ sessions			−0.12 [−0.57, 0.33]		
Not reported/unknown			0.15 [−0.23, 0.52]		
M_{age}			[c]		
% female			[c]		
Sample type			[c]		
Trauma type (ref combat/war)					
IPV/acts of mass terror			−0.20 [−0.59, 0.20]		
Mixed traumas			−0.25 [−0.56, −0.07]		
Trauma timing (ref adulthood)					
childhood			0.37 [−0.07, 0.81]		
mixed timing			0.08 [−0.26, 0.41]		
not specified			0.02 [−0.31, 0.35]		
Emotion regulation					
Estimate	4	163	0.20 [−0.26, 0.59][n.s.]		
Moderator tests					
Study quality			[d]		
ROB			[d]		
No. of sessions			[d]		
M_{age}			[d]		
% female			[d]		
Sample type			[d]		
Trauma type			[d]		
Trauma timing			[d]		
Service-related disability					
Estimate	5	26,110	0.12**** [0.06, 0.17]	22.67 (3)****	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Problems with pain					
Estimate	6	3,556	0.15*** [0.08, 0.21]	7.70 (4)[n.s]	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		

(*table continues*)

Table 3 (*continued*)

Predictor variable	*k*	*N*	*r* [95% CI]	*Q* (*df*)	ΔT^2
Sleep issues					
Estimate	4	349	0.45*** [0.22, 0.64]	12.71 (3)**	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Quality of life					
Estimate	4	727	−0.19** [−0.31, −0.07]	6.33 (3)[n.s]	
Moderator tests					
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
			Demographic characteristics		
Gender—male					
Estimate	9	27,025	0.12**** [0.06, 0.18]	21.42 (6)***	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Education					
Estimate	4	3,813	0.07[n.s.] [−0.30, 0.42]		
Moderator tests			Coef.		
Study quality			[d]		
ROB			[d]		
No. of sessions			[d]		
M_{age}			[d]		
% female			[d]		
Sample type			[d]		
Trauma type			[d]		
Trauma timing			[d]		
Race—Caucasian/European					
Estimate	8	6,578	0.16**** [0.09, 0.23]	42.59 (5)****	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Age					
Estimate	6	1,537	0.28**** [0.15, 0.40]	5.23 (3)[n.s.]	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		

(*table continues*)

Table 3 (*continued*)

Predictor variable	*k*	*N*	*r* [95% CI]	*Q* (*df*)	ΔT^2
Trauma type			[b]		
Trauma timing			[b]		
			Trauma-related characteristics and severity		
Childhood trauma					
Estimate	6	309	0.28**** [0.17, 0.38]	1.57 (4)[n.s.]	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Time since trauma					
Estimate	3	67	0.55**** [0.34, 0.70]	1.59 (2)[n.s.]	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Trauma load					
Estimate	3	386	0.37*** [0.13, 0.57]	5.37 (2)[n.s.]	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Service-related trauma					
Estimate	5	1,124	0.33**** [0.16, 0.48]	11.83 (3)*	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Pretreatment PTSD severity					
Estimate	33	4,970	0.29** [0.10, 0.47]	906.71 (25)****	
Moderator tests			Coef.		
Study quality			−0.04 [−0.20, 0.11]		0.01
ROB (ref high)					0.07
Low			0.77 [−0.15, 1.70]		
Medium			0.57 [−0.36, 1.49]		
No. of sessions (ref one—six sessions)					0.002
Seven–11 sessions			0.49 [−0.54, 1.53]		
12–20 sessions			0.60 [−0.51, 1.72]		
M_{age}			[c]		
% female			[c]		
Sample type			[c]		
Trauma type (ref combat/war)					0.04
IPV/acts of mass terror			−0.39 [−0.84, 0.05]		

(*table continues*)

Table 3 (*continued*)

Predictor variable	*k*	*N*	*r* [95% CI]	*Q* (*df*)	ΔT^2
Mixed traumas			−0.51 [−0.95, −0.06]*		
Trauma timing			[c]		
Psychiatric comorbidities					
Depression					
Estimate	18	1,680	0.42 [0.25, 0.57]****	252.95 (15)****	
Moderator tests			Coef.		
Study quality			0.001 [−0.14, 0.16]		0.003
ROB (ref high)					0.001
Medium			−0.16 [−0.56, 0.24]		
No. of sessions (ref seven–11sessions)					0.01
12–20 sessions			−0.47 [−1.34, 0.40]		
20+ sessions			−0.18 [−1.09, 0.74]		
Not reported/unknown			0.001 [−0.97, 0.99]		
M_{age}			0.03 [0.001, 0.07]*		0.04
% female			−0.002 [−0.001, 0.003]		
Sample type			[c]		
Trauma type			[c]		
Trauma timing			[c]		
Problematic alcohol use					
Estimate	7	24,620	0.19**** [0.12, 0.25]	18.07 (5)***	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		
Personality disorder screen					
Estimate	3	2,952	0.44* [0.03, 0.73]	65.85 (2)***	
Moderator tests			Coef.		
Study quality			[b]		
ROB			[b]		
No. of sessions			[b]		
M_{age}			[b]		
% female			[b]		
Sample type			[b]		
Trauma type			[b]		
Trauma timing			[b]		

Note. *r*, 95% CI, and *Q* statistics presented for the 13 predictor categories. Symbol (#) indicates collinearity among sample type, trauma timing moderator variables, and collinearity between sample type and trauma type moderators for the other social support and clinical-comorbidity categories, respectively; therefore, the moderator variable with the highest bivariate correlation with PTSD outcome was used in the respective multivariate metaregression tests. T-F = trauma-focused; CI = confidence interval; PTSD = posttraumatic stress disorder; n.s. = not significant; Coef. = coefficient; ROB = risk of bias; IPV = interpersonal violence; OR = odds ratio.

[a] Collinearity between characteristics prevented moderation tests. [b] Insufficient heterogeneity observed OR limited studies to test meaningful moderation tests. [c] Nil bivariate association precluding follow-up multivariate tests. [d] Nonsignificant random effects model test.

* $p < .05$. ** $p < .01$. *** $p < .001$. **** $p < .0001$.

Education. Years of education across four studies did not predict PTSD outcomes ($r = 0.07$; 95% CI [−0.30, 0.42], $Z = 0.37$, $p > .05$).

Race. Across six studies, individuals from a Caucasian or European background demonstrated better PTSD outcomes ($r = 0.16$; 95% CI [0.09, 0.23], $Z = 4.62$, $p < .0001$). Heterogeneity tests revealed 88% true between-study variance, $Q(5) = 42.59$, $p < .0001$, $I^2 = 88.26$, $\tau^2 = 0.01$. Egger's test of the intercept was not significant, $t(3) = 2.11$, $p > .05$.

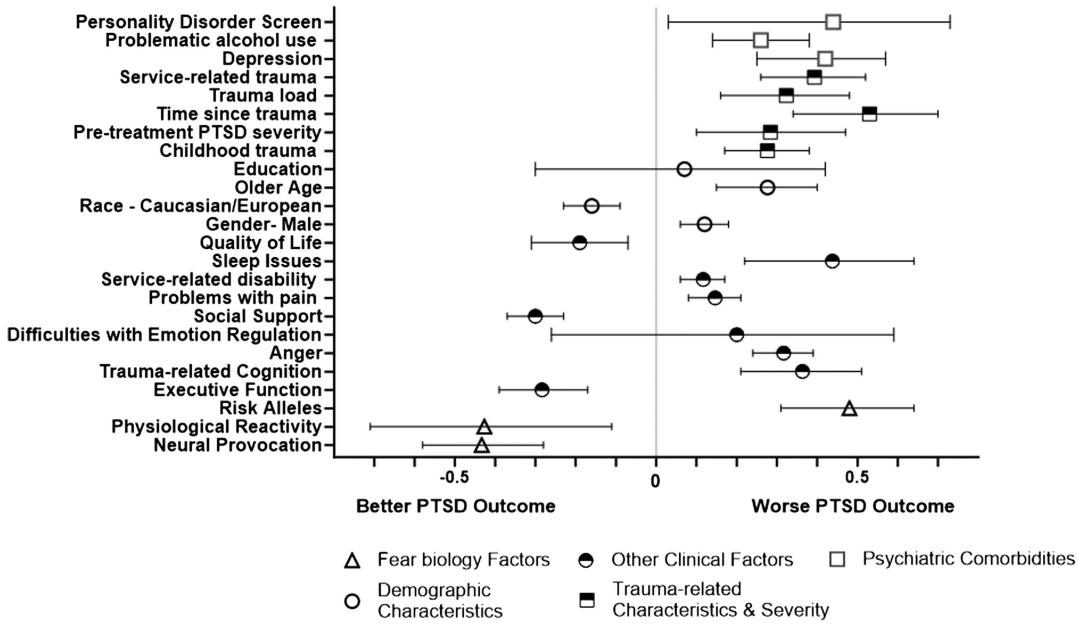
Age. Across five studies, older age was related to worse PTSD outcomes ($r = 0.28$; 95% CI [0.15, 0.40]; $Z = 4.13$, $p < .0001$). Between-study heterogeneity was not significant, $Q(3) = 5.23$, $p > .05$, $I^2 = 42.6$, $\tau^2 = 0.01$. Egger's test of the intercept was not significant, $t(2) = 2.56$, $p > .05$.

Trauma Characteristics and Severity

Trauma Onset. Experiencing trauma in childhood was associated with poorer PTSD outcomes across five studies ($r = 0.28$; 95% CI [0.17, 0.38], $Z = 4.86$, $p < .0001$). True between-study variance was not observed in this meta-analysis, $Q(4) = 1.57$, $p > .05$, $\tau^2 = 0.00$, and the Egger's test of the intercept was not significant, $t(3) = 3.96$, $p > .05$.

Figure 2

Pooled Mean Effect Sizes of 24 Predictor Categories



Note. Error bars reflect range. PTSD = posttraumatic stress disorder.

Trauma Load. A higher trauma load was associated with worse PTSD outcomes across three studies ($r = 0.37$; 95% CI [0.13, 0.57], $Z = 2.98$, $p < .001$). Between-study heterogeneity was not significant, $Q(2) = 5.37$, $p > .05$, $\tau^2 = 0.03$, and the Egger's test of the intercept was not significant, $t(1) = 5.54$, $p > .05$.

Time Since Trauma. A longer duration since traumatic event was associated with poorer outcomes across three studies ($r = 0.55$; 95% CI [0.34, 0.70]; $Z = 4.68$, $p < .0001$). Between-study heterogeneity was not significant, $Q(2) = 1.59$, $p > .05$, $\tau^2 = 0.00$, and the Egger's test of the intercept was not significant, $t(1) = 3.53$, $p > .05$.

Service-Related or Combat Trauma. Exposure to combat trauma was associated with worse PTSD severity across four studies ($r = 0.33$; 95% CI [0.16, 0.48]; $Z = 3.62$, $p < .0001$). One study was not quantified (Voelkel et al., 2015), and similarly evidenced that combat trauma was related to worse PTSD outcome. Heterogeneity tests revealed 75% between-study heterogeneity, $Q(3) = 11.83$, $p < .05$, $\tau^2 = 0.03$. Egger's test of the intercept was not significant, $t(2) = 2.99$, $p > .05$.

Pretreatment PTSD Severity. More severe pretreatment PTSD across 27 samples predicted poorer outcome ($r = 0.29$; 95% CI [0.10, 0.47]; $Z = 2.89$, $p < .01$). Sensitivity analyses with three studies removed (Blanchard et al., 2003; Bragdon & Lombardo, 2012; Owens et al., 2008) who utilized subthreshold (vs. full) PTSD diagnosis revealed a similar effect size ($r = 0.29$; 95% CI [0.10, 0.47]), suggesting that the presence of these studies minimally influenced the overall effect size. One study could not be quantified and reported a similar positive relationship between overall PTSD severity at baseline and PTSD symptoms at outcome (Chard et al., 2010). Heterogeneity tests revealed 97% true between-study variance, $Q(25) = 906.71$, $p < .0001$, $I^2 = 97.24$, $\tau^2 = 0.24$, suggesting follow-up moderation tests with sample characteristics were appropriate. A significant Egger's test of the intercept, $t(24) = 2.66$, $p < .05$, was followed up with trim-and-fill analyses. This revealed that 11 studies were missing to the left of the mean, with an adjusted correlation that was not considerably different from the computed estimate ($r = 0.24$; 95% CI [0.05, 0.41]).

Psychiatric Comorbidities

Depression. Depression symptoms prior to treatment was associated with poorer PTSD outcome across 16 studies ($r = 0.42$; 95% CI [0.25, 0.57]; $Z = 4.49$, $p = .0001$). Sensitivity analyses with one study removed (Owens et al., 2008), who utilized subthreshold (vs. full) PTSD diagnosis, was conducted and this revealed comparable effects with a similar overall correlation ($r = 0.42$; 95% CI [0.23, 0.57]), suggesting that this study did not influence the overall effect size. One study could not be quantified and detailed that depression and guilt symptoms were associated with poorer PTSD outcome (Phelps et al., 2018). Heterogeneity levels indicated 94.73% true between-study variances, $Q(15) = 252.95$, $p < .0001$, $I^2 = 94.07$, $\tau^2 = 0.14$. Egger's test of the intercept was not significant, $t(14) = 0.06$, $p > .05$, suggesting likely minimal publication bias.

Alcohol Use. Alcohol misuse and related problems at baseline was associated with poorer outcomes across seven studies ($r = 0.19$; 95% CI [0.12, 0.25]; $Z = 5.45$, $p < .001$). There was significant heterogeneity, $Q[5] = 18.07$, $p < .05$, $I^2 = 72.33$, $\tau^2 = 0.00$. Egger's test of the intercept was not significant, $t(4) = 0.15$, $p > .05$.

Personality Screen. The presence of problematic personality traits was associated with worse outcomes across three studies ($r = 0.44$; 95% CI [0.03, 0.73]; $Z = 2.09$, $p < .05$). Between-study heterogeneity was high in this category, $Q(2) = 65.85$, $p < .001$, $I^2 =$

96.96, $\tau^2 = 0.15$. Egger's test of the intercept was not significant, $t(1) = 1.51$, $p > .05$.

Moderating Effects of Study Characteristics

As detailed above, heterogeneity tests involving Q and T^2 statistics were computed for all 24 effect sizes to determine whether there was sufficient heterogeneity within each meta-analysis to warrant exploration of whether the different types of baseline factors differed by study design and sample characteristics.

Study Design Characteristics. Multivariate metaregression analyses involving bias variables related to the quality of the observed predictor analysis, ROB in the overall study design, and number of therapy sessions together did not influence effect size estimates of meta-analyses relating to fear-related factors, other clinical factors, and pretreatment severity (see Table 3; range $\Delta T^2 =$ 0.00–0.06). Specifically, observed bias at a study design and predictor analysis level explained negligible variance in the effect sizes (see Supplemental Figure S1).

Study Sample Characteristics. Table 3 presents the details of relevant study sample moderation analyses. Study sample characteristics including age, proportion of females, sample type, or trauma timing did not influence most predictor categories tested for moderation effects. However, age was found to moderate baseline depression insofar as depressive symptoms in older individuals was associated with worse T-F psychotherapy outcome. Further, trauma type moderated pretreatment PTSD severity, whereby high PTSD severity in those reporting combat trauma was associated with poorer T-F psychotherapy outcomes.

Discussion

In this systematic and meta-analytic review, we examined the relationship between a range of baseline factors and PTSD severity following T-F psychotherapy. The analysis included 237 effect sizes across 114 studies, representing for the first time, an aggregate approach to quantifying the relationships between baseline factors and PTSD treatment outcome. We utilized a data-driven framework in the categorization of these predictors, which led to 24 predictor categories. Better response to T-F psychotherapy was predicted by greater activation of neural regions implicated in fear responses and psychophysiological reactivity in response to fear provocation stimuli, as well as higher levels of pretreatment executive functioning, social support, being female, and being Caucasian. Poorer response to T-F psychotherapy was predicted by being a carrier of risk alleles of genes modulating fear expression, as well as higher levels of depression, cognitive distortions, anger, pretreatment PTSD severity, childhood trauma, trauma exposure, combat exposure, service-related disability, pain, sleep difficulties, poor quality of life, alcohol use, and problematic personality traits. In short, while fear biology factors did play an important role in predicting treatment response, there were comparable contributions of other factors that may not be directly related to fear. These findings align with recent narrative reviews of the extant literature relating to predictors of treatment response (Fonzo et al., 2020; Kline et al., 2023), and they importantly highlight a role for nonfear-related factors in driving response to first line interventions (Alpert et al., 2023) for PTSD.

The Role of Fear Circuitry

Consistent with the current focus on augmenting T-F psychotherapy by promoting extinction or inhibitory learning processes (Lebois et al., 2019), each of the factors that were biologically related to fear (i.e., neural and psychophysiological responses to provocation, as well as genetic risk alleles) conferred medium effects in predicting treatment outcomes. It is worth comparing the predictive strength of this category relative to pretreatment PTSD severity, which was measured by the same clinical interviews or self-report measures that were used to measure treatment response. One can expect a stronger association between measures when the same measurement is employed, and so the finding that fear-related factors (that were assessed using neural imaging, psychophysiological indicators, or genetic alleles) performed as strongly as studies that employed the same measures for predictors and outcomes underscores the predictive strength of the fear-related factors. It is also worth noting the assessment of fear-related predictors were largely derived in response to an experimental or provocation cue (e.g., reminders of the trauma, affective stimuli). Specifically, greater biological arousal related to trauma processing involving physiological indices of arousal and activation in the fear neurocircuitry accords with the theory that exposure involves accessing affective components of trauma memories (Foa, 2006). Specifically, the proposal that extinction mechanisms involve learning the mismatch between initial distress and postexposure reductions in distress is regarded as a central tenet of successful response to T-F psychotherapy (Foa, 2006). Supporting this conclusion is the finding that reductions in the Research Domain Criteria of arousal/regulatory systems arousal are associated with improved PTSD severity following exposure-based treatments (Zambrano-Vazquez et al., 2017). We note that a number of predictive factors that are not directly related to fear may actually be indirectly influenced by extinction-related processes. For example, the finding that females displayed better outcomes may be attributed to females having stronger consolidation of fear extinction learning (Velasco et al., 2019). It is also possible that negative beliefs about expressing emotions that tend to be held more frequently in males (McLean & Anderson, 2009) may prevent engagement with exposure-based interventions, and this may reduce opportunities for fear extinction learning. The analysis also indicated that older people displayed worse outcomes, which may be related to difficulties with emotion disclosure (Litz et al., 2019) and diminished executive function (Olff et al., 2014), both of which can reduce effective extinction. In short, we recognize that many predictors can involve multiple mechanisms, which may to different degrees implicate some level of fear processing.

The Role of Other Predictive Factors

In terms of socioeconomic factors, the finding that Caucasian/European ethnicity was associated with better outcomes accords with previous reports that African Americans have been shown to have more negative appraisals about the world, especially in regards to mistrust and perceptions that the world is malevolent (Zoellner et al., 1999). This pattern may be attributed to the frequency of negative events African Americans experience, which is related to the extent they hold less benevolent beliefs about the world (Poulin

& Cohen Silver, 2008). It is also possible that non-Caucasians often respond more poorly to treatment because this group tends to come from lower socioeconomic standing, which can limit their capacity to seek and attend treatment (Davis et al., 2008). This group can also experience considerable stigma about attending therapy, fear of being discriminated by health providers, and can also attend therapy with more race-based trauma that can compound their PTSD (Williams et al., 2014). In short, it is possible that treatment may have less benefit for more disadvantaged people, including non-Caucasians, because they have fewer resources, more personal and social demands, and are vulnerable to ongoing stressors (Reeves et al., 2016). It is also worth noting evidence that whereas non-Caucasians, such as African Americans, may have difficulty seeking and persisting with treatment, they can achieve comparable outcomes as Caucasians if they complete treatment (Lester et al., 2010). The reason for the current observation that non-Caucasians tend to fare worse with T-F psychotherapy appears to be a complex issue that requires closer investigation to understand the factors underpinning this trend. Further, the observation that years of education was not associated with treatment outcome is somewhat surprising in light of evidence that lower education levels have been associated with risk for PTSD (Brewin et al., 2000). Relatedly, pretrauma levels of intelligence are associated with PTSD risk (Macklin et al., 1998). These findings have been interpreted as possibly reflecting the pattern of people with higher education or intelligence having better coping resources or socioeconomic advantages to manage stress, and one could also suggest these factors may contribute to better response to T-F psychotherapy. It is noteworthy that three of the four studies reporting the predictive role of education involved military populations (Sripada et al., 2019; van Rooij et al., 2015; Walter et al., 2014), and it is possible that entry criteria and training requirements may have led to limited range of education levels in these military organizations. Alternatively, it is conceivable that education levels are not influential in T-F psychotherapy response because extinction-based strategies have been shown to be effective across species, and so exposure therapy may not require optimal education levels for it to be effective.

A longer duration since the event, a higher trauma load, experiencing multiple types of traumas during childhood, and service-related trauma predicted worse posttreatment PTSD severity. This is not surprising within the context that cumulative and sustained trauma load beginning in childhood and/or sustaining across the lifetime is often associated with a more complex PTSD presentation that includes enduring emotional and psychosocial difficulties, as well as other comorbidities (Cloitre et al., 2013). These trauma-related demographics may moderate treatment response insofar as fear extinction-based psychotherapies may not address these factors. Indeed, sexual assault and combat trauma, relative to other trauma types, have been associated with worse treatment outcomes following prolonged exposure-based therapies (Markowitz et al., 2017; Zandberg et al., 2016).

Additionally, general functioning and psychiatric comorbidities had significant small to medium effects in modulating worse PTSD outcomes. This finding is significant because most people with PTSD have a comorbid condition (Kessler et al., 1995). The more traumatic events a person experiences, including the range of different types of traumas, the greater the likelihood the person will have comorbidity and complex clinical presentations (Karam et al., 2014). In this sense, the risk for poor outcome posed by comorbidity may be related to the association of different trauma types and earlier exposure to trauma on poor treatment response. It is worth noting that some applications of T-F psychotherapy have specifically attempted to address comorbid issues, including physical health comorbidity, pain, anxiety, sleep, and concurrent alcohol use (Cloitre et al., 2010; Hien et al., 2017; Kline et al., 2023). In this context, it is worth mentioning that increasing attention is being given to complex PTSD, which is characterized by a form of PTSD that is compounded by significant problems in emotion regulation, relationships, and self-identity (Brewin et al., 2017). Controlled trials have often been (sometimes correctly) criticized for excluding more complex cases that clinicians regularly treat in daily practice. The finding that comorbidity and impaired functioning predicts poor T-F psychotherapy response may suggest that special attention needs to be given to improve treatment response in these cases.

The Role of Other Clinical Factors and Comorbidities

In terms of factors that are more amenable to change in psychotherapy, cognitive and affective factors exerted a moderate association with PTSD severity following T-F psychotherapy. The role of distorted trauma-related beliefs about oneself and the world attest to the role of unhelpful cognitive styles in influencing poorer treatment response (Ehlers & Clark, 2000). Some T-F psychotherapy protocols emphasize cognitive reframing techniques (Ehlers et al., 2005; Resick et al., 2017), and this is informed by evidence that changes in cognitive style can precede PTSD symptom change (Kleim et al., 2013). This empirical literature fits within broader evidence for the role of changes in appraisals in preceding symptom change in other anxiety disorders, including social phobia and panic disorder (Hoffart et al., 2009; Hofmann et al., 2007). This cumulative evidence suggests that more attention on how to modify maladaptive thinking could potentially enhance treatment response for PTSD.

The observation that anger is predictive of worse PTSD treatment outcome is consistent with proposals that anger is distinct from fear-related emotions and can involve reward motivation as distinct from the avoidance that characterizes fear (Harmon-Jones et al., 2017). Network analyses of PTSD symptoms indicate that anger is not strongly connected to fear-based symptoms, such as re-experiencing or avoidance (Bryant et al., 2017), which supports the notion that anger may function differently from fear-based processes. It has been shown that people whose primary response to recalling their trauma is anger, rather than fear, do not respond optimally to exposure to the trauma memory because extinction processes are not operating in this nonfear response to the memory (Foa et al., 1995). This finding accords with reports that anger is commonly a residual symptom after T-F psychotherapy (Schnurr & Lunney, 2019). Overall, it appears that T-F psychotherapy, or at least those variants that place emphasis on exposure therapy, may not target posttraumatic anger. This interpretation suggests that evidence-based approaches that target anger may be more appropriate in these cases (Del Vecchio & O'Leary, 2004). It is interesting that emotion regulation was not a predictor of treatment response because it is commonly thought to be an important component of T-F psychotherapy, and hence one may expect people who have better emotion regulation skills to respond more positively to treatment (Gross & Jazaieri, 2014). One possible explanation for the observed finding is that the studies that measured emotion regulation as a potential predictor each targeted

emotion regulation skills as a focus of the treatment (e.g., the Skills Training in Affective and Interpersonal Regulation program, Cloitre et al., 2002; Hoeboer et al., 2021). The content of the treatments may have minimized the extent to which baseline emotion regulation levels may have influenced treatment response as each treatment taught emotion regulation skills as part of the treatment prior to engaging in trauma-focused aspects of treatment.

Depression symptoms were associated with poorer outcomes and this points to the complexity of PTSD as a heterogenous condition in which depressive features represent both a subset of PTSD symptoms that can be categorized as a dysphoric phenotype of the disorder but also a comorbid condition (Flory & Yehuda, 2015; Forbes et al., 2011, 2015). In this context, it is noteworthy that deficits in executive function and attentional control had a medium effect in predicting poorer PTSD treatment outcomes. Dysphoric and numbing symptoms of PTSD are associated with executive function impairments, and through this process depressive symptoms may mediate the relationship between PTSD and executive function processes (Dretsch et al., 2012; Olff et al., 2014), thereby contributing to treatment response.

Implications for Augmenting Treatment

On the premise that predictors of treatment outcome may inform potential strategies to augment current treatments of PTSD, this meta-analysis points to a number of opportunities for potential strategies. The finding that treatment response was associated with fear and extinction processes supports the focus of recent attempts to augment T-F psychotherapy by promoting extinction processes (Lebois et al., 2019). In addition, however, this review highlights that a range of other processes should also be targeted to improve treatment response. For example, the association between poor treatment response with comorbidity and negative emotional responses suggests that strategies to target maladaptive emotional coping styles may augment T-F psychotherapy responses. One example is the Unified Protocol that focuses on emotion management strategies and has been shown to effectively reduce a range of anxiety disorders (Barlow et al., 2017). Of relevance is one pilot study demonstrating that use of the Unified Protocol ameliorated PTSD and other comorbid symptoms (O'Donnell et al., 2021) in a PTSD sample. Specifically, the effect size of Unified Protocol in conferring improved PTSD symptoms (Hedges' g = 1.20–1.47; O'Donnell et al., 2021) was comparable in magnitude to purely exposure-based therapies (Hedges' g = 1.25; Mclean et al., 2022) for the treatment of PTSD. The extent to which this approach can be used to augment T-F psychotherapy has yet to be tested.

Maladaptive appraisals had a strong impact on poorer response to treatment. There are a range of evidence-based strategies targeting appraisals that contribute to psychopathology, with considerable evidence supporting mindfulness-based cognitive therapy techniques that can alleviate rumination on repetitive negative thoughts that contribute to negative mood states (Perestelo-Perez et al., 2017; Schumer et al., 2018). Recognizing the role of diverse emotional responses related to appraisals in treatment outcome, it is worth noting the recent attention on moral injury after trauma. This construct, which remains somewhat loosely operationalized in the literature, has been defined in terms of the emotional difficulties of trauma survivors who experience moral transgressions by themselves or others (Litz & Kerig, 2019). Increasing numbers of studies have reported the role of moral injury in nonfear-related psychopathology after trauma (Griffin et al., 2019). For example, one study of deployed military personnel found that those who were exposed to moral injury-related events (not involving threat to one's life) by oneself were more likely to report guilt and self-blame, whereas those exposed to morally injurious events by others were more likely to report sadness and humiliation (Litz et al., 2018). Although the evidence for treating moral injury is sparse, there have been calls for focused research on treating moral injury in PTSD; one such approach that has been proposed is adaptive disclosure therapy (Litz et al., 2016), however, this has yet to be subjected to evaluation.

In terms of depressive symptoms, there are a range of interventions that could potentially be used to target the dysphoric features of PTSD, including problem solving, cognitive therapy, and behavioral activation (Cuijpers et al., 2007a, 2007b; Cuijpers et al., 2009). Other potential strategies have emerged that can address dysphoria by enhancing capacity for hedonic pleasure through positive affect training (Craske et al., 2019). Other approaches have targeted the memory deficits often seen in people with dysphoric presentations, including training people to retrieve more specific autobiographical memories, including those involving positive experiences (Moradi et al., 2014). Providing mnemonic strategies has also been shown to augment the gains made by cognitive behavioral therapy in other conditions, and may also be beneficial for PTSD (Harvey et al., 2016). The utility of addressing depression with targeted cognitive behavioral therapy is indicated by evidence that this approach can augment clinical outcomes in people with treatment-resistant depression (Hauksson et al., 2017; Strawbridge et al., 2019). These are typically not included in T-F psychotherapy interventions, and they may be useful in addressing specific executive control processes that in part could underlie the dysphoria and anhedonic features of the disorder. In terms of improving attentional control, attentional control training has been used successfully in military populations as an early intervention to ameliorate PTSD symptoms (Badura-Brack et al., 2015). This form of training is thought to target hyper fluctuations in attentional flexibility between threat avoidance and hypervigilance in a manner that balances attentional control between opposingly valanced stimuli (Alon et al., 2019). This strategy has yet to be used as an adjunct in enhancing T-F psychotherapy and is an avenue worth pursuing with the goal of inducing cognitive flexibility in PTSD.

The evidence that positive social support had a medium effect on T-F psychotherapy outcomes suggests that socially oriented interventions may have potential in augmenting PTSD outcomes. In this regard, interpersonal psychotherapy is the most validated treatment that purports to address relational issues and has been shown to be an effective treatment for depression (Cuijpers et al., 2016), as well emotional reactivity in borderline personality disorder (Sinnaeve et al., 2015). There is initial evidence that it is also effective for PTSD and is not significantly less effective than exposure therapy (Markowitz et al., 2015). Interpersonal psychotherapy builds on attachment theory and presumes that depression is maintained by adverse interpersonal events, such as bereavement, role loss, or conflict with others. Accordingly, it teaches strategies in managing these events and the emotional reactions one has to these events. Although interpersonal psychotherapy is effective in treating depression, it has not been evaluated as an adjunct for T-F psychotherapy; nonetheless, it may offer potential to promote better

social support and thereby augment the gains of -F psychotherapy. Insofar as the presence of interpersonal trauma-related baseline factors (i.e., moral injury, combat trauma) is in part associated with problems with interpersonal and social functioning in a person presenting with PTSD, there may be a particular need for these to be targeted in common T-F interventions in the context that these factors conferred worse outcomes in the current review. To this end, counteracting problematic social relations (e.g., high expressed emotion in one's partner, negative partner reactions) with positive social support experiences may facilitate engagement with trauma-focused strategies, which in turn may facilitate positive coping responses during treatment (Cohen & Wills, 1985). Therefore, there is evidence that people respond better to treatment for PTSD when they are able to choose the treatment modality (Le et al., 2018; Zoellner et al., 2019). Thus, adapting the type of treatment, and adjusting how treatment is delivered, may augment how patients respond to T-F psychotherapy.

Potential for a Systems Approach to Improving PTSD Outcomes

The limited success of attempts to date to augment T-F psychotherapy by focusing predominantly on promoting extinction processes may be explained, in part, by the observation in this meta-analysis that there is a broad array of patient and clinical factors that are associated with T-F psychotherapy outcomes. We acknowledge that these predictor categories likely interact in many complex ways in driving response to T-F psychotherapy. Emerging attempts to explore multifactorial patient risk factor profiles that drive treatment outcome (Cloitre et al., 2016; Herzog et al., 2021; Norr et al., 2018) speak to the shift toward understanding the interactions among various static and malleable patient characteristics. Such frameworks inch closer toward the utility of adopting a more personalized approach to targeting specific symptoms in PTSD rather than a standard "package" of one of the variants of T-F psychotherapy. In this context, recent commentary has focused on process-based therapy approaches that promote adoption of evidence-based strategies to target the specific symptoms as a function of individual patient problems rather than a diagnostic entity (Hofmann & Hayes, 2018). This approach allows flexibility to address problems and processes most affecting patients, which in the case of PTSD could involve applying evidence-based strategies to target trauma memories, anhedonia, cognitive style, substance abuse, rumination, anger, or relational difficulties. For example, problems related to shame, guilt, or anger may be optimally addressed by strategies that do not utilize extinction paradigms, but focus more on cognitive and emotion-focused approaches that specifically address these problems. Alternately, problems involving social relations may be addressed with interpersonal psychotherapy strategies because interpersonal and social functioning have been repeatedly documented in people with PTSD (Jellestad et al., 2021). These therapeutic approaches are not typically embodied in T-F psychotherapy protocols; however, the current review highlights that this flexible approach may provide an important avenue to improve treatment response for PTSD. This tailored framework has not been evaluated to date, and so it is worthy of careful evaluation to determine its utility in overcoming the limitations of current standardized T-F psychotherapies.

Limitations

We note several study limitations. First, we recognize that our grouping of predictors into categories that would permit meta-analytic quantification was somewhat arbitrary and the predictors in different categories may not be mutually exclusive. For example, appraisals of the traumatic event can overlap strongly with fear interpretations and in this sense are not fully independent of the fear-related construct. In noting this limitation, we also recognize that quantifying predictors into groupings requires some level of arbitrary categorization otherwise meta-analysis is not possible. Second, we did not include mediation or dropout-prediction studies given that this was beyond the scope of the current review; these matters are relevant to T-F psychotherapy and so are an important future research question within the context of identifying individual processes that modulate treatment response. Third, it is worth noting that the term "predictor" was broadly used in the studies cited insofar as a range of statistical analyses (e.g., correlations, cross-lagged regression methods) were used across studies to identify predictors of treatment outcome; here causation cannot be inferred. Fourth, the ROB for studies included in this review was mixed given our expansive inclusion of controlled and uncontrolled clinical trials; having said this, trials consisting of a high level of ROB were not included in the analyses to ensure sufficient confidence in the results of our analyses. Fifth, we note there is no consensus on how to define treatment response to T-F psychotherapy, and so to incorporate the findings of as many studies as possible, we followed the various methods of defining treatment response used by each study. This decision was taken to mirror the current state of the available research on efficacy of evidence-based PTSD interventions that at this point of time do not converge on a definition of PTSD response or nonresponse (Varker et al., 2020). To this end, a more comprehensive definition of treatment response is encouraged in future reviews (e.g., specific functional impairment outcomes) such that the true burden of this condition is more appropriately captured. This need is underscored by recent evidence which suggests that T-F psychotherapy may confer moderate gains in a range of functioning factors (Bonfils et al., 2022; Swerdlow et al., 2023), highlighting a need for more focused attention to functional outcomes of treatment rather than focusing only on symptom reduction. Sixth, trials that combined T-F and non-T-F conditions in observed predictor analyses were excluded as our research question focused on the role of predictors of T-F psychotherapy. However, in these trials, active and control conditions were often combined when a lack of a difference in PTSD outcome was observed. Seventh, we note that our searches were limited to articles published in English, in peer-reviewed journals, and comprised predominantly studies from Western and high-income settings (see Supplemental 1 and Table 1); these factors may limit the generalizability of the findings to more diverse cultures. Future research would benefit from conducting multilingual searches and consider non-English sources in their database search strategies. Eighth, our meta-analytic findings may represent inflated effects given our decision to only include published articles. We adopted this approach as we attempted to synthesize the more rigorously evaluated and publicly available information on pretreatment predictors of T-F psychotherapy outcome. We note however that excluding unpublished studies tends to inflate meta-analytic finings as these reports tend to have higher proportions null- and smaller effects than published

studies. We conducted publication bias analyses to adjust for this, which indicated minimal differences between observed and imputed effect sizes accounting for bias (see Supplemental Table S5). As such, true effects are likely to fall between those reported and these imputed effect estimations. Notwithstanding these limitations, our conclusions from the present article remain the same: A range of fear- and nonfear-related factors drive response T-F psychotherapy for PTSD. Finally, our research is beyond the scope of answering questions relating to treatment nonresponse and resistance, where factors such that the number of times a person with PTSD has had prior T-F psychotherapy and displayed limited response, cannot be learned from the current review.

Conclusions

In the context of many patients not responding to T-F psychotherapy, which is the treatment of choice for PTSD, this review points to several key conclusions. First, the current finding that factors related to extinction are an important predictor of treatment response underscores the importance of exposure therapy and is consistent with T-F psychotherapy being a frontline treatment for PTSD. This suggests that pharmacological and psychological strategies focused on extinction processes are worthy of ongoing investigation. Second, the review indicated that there are multiple other factors that can influence a person's response to T-F interventions that do not seemingly involve fear or extinction mechanisms. Many people with PTSD may require a broader approach to optimizing treatment response by targeting these other risk factors for poor treatment outcomes. The success rate of T-F psychotherapy has not improved over four decades, and this barrier may require a more comprehensive approach to treatments by targeting the range of factors that are associated with limited successful outcomes. A likely interpretation of the current findings is that the optimal approach to treating PTSD does not involve a "one-size fits all" solution but may require a more personalized approach that addresses specific issues that a patient may have that are likely to interfere with treatment success. While recognizing that identifying risk factors for poor treatment response does not necessarily indicate causative change mechanisms that occur in treatment, both clinicians and researchers may achieve their goals more effectively by assessing factors that are predictive of treatment response, which may lead to more informed targeting of potential candidates that may influence treatment response.

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