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An initial review of residual symptoms after empirically supported trauma-focused cognitive behavioral psychological treatment

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ABSTRACT

Objective: Although residual symptoms remain following clinical treatment for posttraumatic stress disorder (PTSD), little is known about the characteristics of these residual symptoms. We aimed to determine the type, severity, and frequency of symptoms that remain after trauma-focused psychotherapy.
Methods: We conducted a systematic review of 51 randomized controlled trials of empirically supported psychosocial interventions for PTSD (68 total treatment arms). Outcomes included: 1) PTSD symptoms and 2) conditions commonly comorbid with PTSD: depression, anxiety, and quality of life impairment.
Results: In general, the results revealed that participants who completed PTSD treatment continued to report residual PTSD symptoms: 31% reported clinical symptom levels, and 59% reported subthreshold levels at posttreatment, particularly within the hyperarousal cluster. Residual symptoms also emerged for depression (19% clinical), anxiety (55% clinical), and quality of life (36% clinical). Few differences emerged across treatment types, but differential patterns were revealed for sample/trauma types.
Conclusions: Results suggest a need for focused research attention to and clinical assessment of individual residual symptoms following empirically supported treatment for PTSD to determine whether further treatment sessions are warranted.

1. Introduction

Posttraumatic Stress Disorder (PTSD) affects approximately 8% of the United States population (Kessler et al., 1995) and is associated with multiple mental and physical health comorbidities (Galatzer-Levy, Nickerson, Litz, & Marmar, 2013; Pacella, Hruska, & Delahanty, 2013) and reduced quality of life (QOL; Pagotto et al., 2015). The literature on empirically supported treatments for PTSD has become more sophisticated over time, and several treatments are effective across populations (Bisson & Andrew, 2007). However, even front-line treatments are not successful for every individual. Specifically, a review of randomized clinical trials (RCTs) of empirically supported PTSD treatments found that non-response rates range up to 50% (Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008), and a meta-analysis of psychotherapy for PTSD noted that, although the majority of patients continue to have substantial residual symptoms posttreatment, details regarding these symptoms and other posttreatment comorbidities are sparse (Bradley, Greene, Russ, Dutra, & Westen, 2005). Thus, it is important to gain a better understanding of the types of symptoms that are likely to persist following treatment, and whether these symptoms vary across populations.

1.1. Residual symptom literature review

Although many people with PTSD will respond to treatment, response does not necessarily equate to remission. Even among patients

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considered to be treatment responders, improvement is often defined as a specified point reduction on a specific PTSD assessment measure, rather than a complete absence of symptoms, suggesting that residual symptoms may remain. For instance, among those who completed cognitive behavioral therapy(CBT) treatment *and* lost their PTSD diagnosis, nearly 50% still demonstrated clinical levels of insomnia following treatment (Zayfert & DeViva, 2004). Additional studies have found that subthreshold PTSD or even "resolved" PTSD (i.e. no longer meeting criteria for PTSD 12 months post-injury) can still cause functional impairment (Bryant et al., 2016; Zlotnick, Franklin, & Zimmerman, 2002), and that specific symptom clusters at post-treatment (e.g., avoidance, numbing, and hyperarousal) are associated with lingering impairment (Lunney & Schnurr, 2007; Monson et al., 2012; Shnaider et al., 2014).

Given the problematic consequences associated with the presence of residual symptoms, trauma experts have highlighted the need for gaining a deeper understanding of the specific symptoms that remain following PTSD treatment (Schnyder et al., 2015). Two early treatment reviews indicated that certain types of psychosocial treatment for PTSD may address some symptoms better than others (Shalev, Bonne, & Eth, 1996; Sherman, 1998), and that patients may benefit from additional or sequential treatment based on the type of residual symptoms that remain. Yet, the extant literature is primarily focused on pharmacological- but not psychosocial-based PTSD treatments. Advancing our knowledge of the specific types and clinical severity of symptoms that remain after psychosocial treatment could inform practice recommendations, such as whether augmenting existing treatment (via additional sessions to target residual symptoms, a sequential therapy offered post-PTSD treatment, or a simultaneous pharmacological treatment) may be necessary to experience complete remission or better functional improvement. Further study of residual symptoms may also aide in development of modified PTSD theory and treatment: for instance, a growing body of evidence suggests that residual sleep problems may indicate that sleep disturbances are a core feature of PTSD rather than a secondary symptom (Spoormaker & Montgomery, 2008).

1.2. Goals for current review

Given the limited empirical focus on residual symptoms to date, the goal of this review was twofold: 1) to determine the types and levels of PTSD symptoms (global and individual cluster-level symptoms) that remain following RCTs of empirically supported trauma-focused psychosocial treatment, and 2) to determine the presence of residual symptoms in domains that are frequently comorbid with PTSD—depression, anxiety, and QOL. We focus on these secondary outcomes as well because, despite not being directly addressed in trauma-focused therapy, such comorbidities are affected by PTSD, may complicate recovery, and are typically associated with impairment on their own. For instance, comorbidity can lead to worse outcomes, such as greater PTSD symptom severity and lower self-efficacy (Adams et al., 2018), greater psycosocial impairment (see Flory & Yehuda, 2015 for review), and increased suicide attempts (Kimbrel, Meyer, DeBeer, Gulliver, & Morissette, 2016). Notably, treatment dropout is more common when comorbidities are pesent (see Flory & Yehuda, 2015), and there is some evidence that comorbidity may explain some gender differences in treatment response via predicting treatment dropout (Blain, Galovski, & Robinson, 2010). On the other hand, multiple mental health diagnoses can have shared etiological causes, meaning that treating one disorder may lead to improvements in others simultaneously (Spinhoven, Penninx, van Hemert, de Rooij, & Elzinga, 2014). To this end, a recent review reported that PE specifically leads to improvements in depression, general anxiety, trauma cognitions, and psychosocial functioning (van den Berg et al., 2015).

We aimed to examine PTSD symptoms at both the global *and* symptom cluster level for the following reasons: an early review (Sherman, 1998) reported that psychosocial treatments had differential effects at the symptom cluster level (i.e. largest effect sizes for avoidance, then intrusions, and moderate for arousal, depression and anxiety) highlighting the importance of assessing specific symptom clusters in addition to the total score. Our findings may indicate whether standard CBT interventions may be more effective at targeting intrusions and avoidance symptoms vs. hyperarousal symptoms; the latter symptom cluster may warrant specific intervention following CBT for PTSD (Zayfert & DeViva, 2004). Further, different symptom clusters may have differential effects on outcomes. For example, improvements in different domains of psychosocial functioning (e.g., work, family, and life satisfaction) are driven by improvement in different symptoms clusters: Specifically, one study found that improvement in the emotional numbing cluster of PTSD was associated with improved non-family relationships, whereas improvement in hyperarousal was associated with improvement in daily living and household tasks (Shnaider et al., 2014).

For the purposes of this review, we defined a residual symptom as one that remains at either a "clinical" or "subthreshold" level, versus a "minimal/absent" level, following PTSD treatment (informed by established criteria; see "Coding and data abstraction" section below, and Supplemental Table 1).

An exploratory goal of the present review was to describe patterns of group differences in residual PTSD symptoms based on subgroups of treatment and sample types. In particular, we examined six potentially relevant groupings to see if different groups had noticeably different levels of residual symptoms. First, we examined treatment type (grouped into exposure versus cognitive versus all other treatments) to see whether different treatment types would produce different levels of residual PTSD symptoms. We next examined whether different sample types would lead to different levels of residual PTSD symptoms, given evidence of difference in rates of PTSD or response to treatment based on gender (Olff, Langeland, Draijer, & Gersons, 2007), interpersonal assault trauma vs. other types of trauma (Olff et al., 2007), and military/Veteran vs. civilian samples (Steenkamp, Litz, Hoge, & Marmar, 2015). For the secondary outcomes, we examined whether post-treatment symptom levels would be associated with pre-treatment symptom levels, and whether PTSD symptom levels were associated with secondary symptom levels.

1.3. Study inclusion

Studies included in this review were RCTs examining empirically supported psychosocial treatments for PTSD, regardless of whether they explicitly discussed or analyzed "residual" symptoms. This broad inclusion criteria allowed for the coding of potential residual symptoms based on a set of rules created for this study (Supplemental Table 1), and was vital for two primary reasons: 1) there is insufficient literature base ***directly*** examining and reporting upon residual symptoms after psychosocial treatment for PTSD (for exceptions, see Larsen, Fleming, & Resick, 2019; Schnurr & Lunney, 2019; Zayfert & DeViva, 2004), and 2) this method allowed for an examination of residual symptoms following a variety of empirically supported treatments in a broad cross-section of the treatment literature. We identified eligible studies based on empirical articles included in two recent systematic reviews of empirically supported treatment for PTSD: Cusack et al. (2016) and the American Psychological Association's (APA) Clinical Practice Guideline for the Treatment of PTSD in Adults (2017).

Specifically, Cusack et al. (2016) reviewed a comprehensive set of RCT's published between 1980 and 2014 that evaluated a psychosocial intervention compared with any of the following conditions: waitlist, usual care, no intervention, placebo, or a competing intervention. Results yielded a high level of support for *exposure-based therapies* (i.e., prolonged exposure (PE) and other exposure therapies), and moderate support for cognitive and cognitive-behavioral therapies (i.e., cognitive therapy [CT], cognitive processing therapy [CPT], and other cognitive behavioral therapies), with low-moderate support for narrative

exposure therapy [NET] and Eye Movement Desensitization and Reprocessing [EMDR]. The recent APA (2017) document regarding clinical practice guidelines paralleled and updated this literature search to include any RCT's published between 2012 and 2016. Similarly, this review determined that there was a strong body of evidence to support CBT, CPT, CT, and Exposure Therapy, including PE. Given our focus on residual symptoms following *the most efficacious* psychosocial treatments for PTSD, we used this set of reviews to compile and include the highest-quality PTSD psychosocial treatments (i.e., CBT/CT, CPT, and PE/exposure; see Cusack et al., 2016; APA 2017) deemed to have a medium to strong body of evidence for PTSD reduction. Thus, certain therapies with small to moderate bodies of promising evidence (e.g. EMDR, NET, Brief Eclectic Psychotherapy, relaxation) did not have sufficient support to be included in this review. Residual symptoms stemming from pharmacological treatments are beyond the scope of this review, and were not included in our analyses.

1.4. Outcome: categories of residual symptoms

The primary purpose of this review was to examine residual DSM-IV PTSD symptoms at both the global and the symptom-cluster level (i.e., re-experiencing, hyperarousal, avoidance and numbing)[2] . Beyond PTSD symptoms, we also examined symptoms of two frequently comorbid disorders (i.e., depression and anxiety) and QOL. Insufficient RCTs were available to examine other common comorbidities (e.g., dissocation, physiological indicators, substance use, sleep, cognitive symptoms).[3]

2. Methods

2.1. Procedure

Given that a literature search for "residual" or "refractory" symptoms and "PTSD" yielded very few studies usable for a review, we utilized RCTs of psychosocial PTSD treatment derived from the two systematic reviews described above (Cusack et al., 2016; APA, 2017). To expand upon these prior reviews, we created guidelines for the classification of symptom means following treatment, and re-analyzed the data in terms of the *prevalence of residual symptoms reported in each study, not the efficacy of the treatments per se*. That is, none of the studies directly reported on the presence or absence of residual PTSD symptoms, but they did provide sufficient data for re-coding and analysis according to guidelines that we created to determine whether residual symptoms were present.

2.2. Inclusion criteria

The studies included in this review are restricted to RCTs as opposed to non-randomized, observational, or other intervention designs, because RCTs provide the most stringent means of demonstrating a cause-effect relationship between the treatment intervention and target outcome. An RCT allows for the reduction and/elimination of confounding factors that are present in observation or single-group study designs. Further, RCTs were more likely to report outcomes in the detailed way we required to calculate the presence of residual symptoms. All studies included in this review were rated as low or medium risk of bias (vs. high risk of bias)[4] based on the systematic bias assessment included in the review by Cusack et al. (2016). However, the studies included in the new APA draft guidelines (2017; $n = 8$) did not undergo the same bias rating procedure. As such, two of five authors (SL, AB, RG, PH, & KL) individually recorded three typical indicators of quality (Jadad et al., 1996) for each study to assess bias: whether a study included randomization, blind assessors, and a detailed description of withdrawal and retention. Of these eight APA studies, all used randomization, all studies with clinical assessments used blind assessors ($n = 7$; one study included self-report assessments and did not qualify for this category), and all studies provided numbers of participants who withdrew or dropped out. Based on this information, we retained all eight of these studies in our final analysis.

Ultimately, we included 51 studies (43 from Cusack et al., 2016, eight from APA, 2017) with a total of 68 active treatment arms consisting of either PE or exposure-based treatments, CPT or cognitive therapy, or CBT[5] . For studies that included more than one active treatment arm, we included only those arms utilizing one of the above-named treatments. We do not report on the control/placebo or treatment arms including other treatments (e.g., NET).

2.3. Coding and data abstraction

Each study was coded by two different authors for population and trauma type, treatment type, methodology (i.e., assessment type, bias), and statistical characteristics (i.e., symptom means; see below). Disagreements, most of which were non-substantive, were discussed to come to consensus. Within each study, we recorded the mean symptom levels for each outcome at the pre-treatment and immediate post-treatment assessment. If a follow-up was conducted[6], we recorded the mean symptom level at the last reported follow-up assessment (typically 3, 6 or 12 months post-treatment). Thus, we were not solely interested in recording symptom *changes* or *effect sizes*, but rather in examining *absolute levels* of remaining symptoms. Further, we used means from the treatment completer sample (vs. the intent to treat sample) when available to evaluate participants who had engaged in a full course of treatment.

To facilitate comparison across measures and symptom types, we used two primary methods to create a coding system to define residual post-treatment symptom levels for each outcome: 1) established guidelines in the literature were applied as cutoff values in this review, and 2) when these established guidelines were not available, we applied the Jacobson and Truax (1991) method (detailed description below). See Supplemental Table 1 for a full list of measures and our criteria for determining clinical cutoffs for this study resulting from both of these methods. Coding involved categorizing the residual symptoms into three main categories based on cutoffs established in the literature: 1) *clinical* symptoms i.e. significant symptoms still present at a level indicating a clinical disorder or significant level of distress or impairment; 2) *subthreshold* symptoms i.e. mild-moderate symptoms that remain at a level that may cause distress or impairment; 3) *good end-state functioning*

[2] We did not find studies that utilized DSM-5 that met our other inclusion criteria; all included studies used DSM-IV.

[3] There were several studies reporting on dissociation and physiological symptoms, but unfortunately, not enough to produce reliable findings. Thus, although we initially conducted the same analyses with these two outcomes, we will not fully report them here (contact authors for details). Briefly, five studies reported dissociative symptoms; dissociative symptoms decreased from pre to post treatment, but were all at a less than residual level at all time periods. Seven studies reported physiological symptoms, but each one of a different type (many without agreed-upon cutoffs in the literature). Non-trauma-cued symptoms stayed consistent over time, whereas trauma cued measures decreased with treatment.

[4] Cusack et al. (2016) excluded 30 studies judged to be at high risk of bias from main analyses; we did the same.

[5] We excluded two other studies with empirically supported treatments because they were not representative of results overall: Gamito et al., 2010(compared imaginal exposure and virtual reality exposure but only included 10 total participants and had unclear reporting of outcomes); and Rothbaum et al., 2006 (compared sertraline augmented with PE vs. placebo; in order to isolate the effects of PE we examined pre-PE symptom levels which were misleadingly low due to previous treatment with sertraline).

[6] In a small number of cases there was either no follow-up, or a follow-up occurred more than 1 year after treatment.

or *minimal/absent* symptoms, i.e. symptoms are no longer present and/or there is little evidence of impairment or distress.

When established cutoff guidelines were not available, we applied the Jacobson and Truax (1991) methods for using population norms to create a cutoff to define whether study outcomes fall closer to a healthy or dysfunctional population norm. The most notable difference with this method, compared to using established guidelines, is that it resulted in the creation of two categories (versus the aforementioned three categories) to describe the outcomes: 1) *clinical symptoms* (significant residual symptoms), and 2) *good end-state functioning* (minimal/absent symptoms). We did not define a subthreshold category because the level of detail required to compute that information was not available. To create these categories, we calculated the value that represented either two standard deviations below the clinical sample mean or two standard deviations above the healthy population mean (or, if both clinical and healthy population means were available, we used a mid-point as a cutoff; see Jacobson & Truax, 1991, p. 13 for formula).

Below, we detail the coding process for each outcome.

2.3.1. PTSD

For the global PTSD outcome, established cutoff guidelines were available for many of the PTSD measures (e.g. Clinician-Administered PTSD Scale [CAPS; Blake et al., 1990]; Posttraumatic Diagnostic Scale [PDS; Foa et al., 1999]). For instance, for the CAPS total score, we identified 0–19.99 as *minimal/absent* symptoms (Weathers, Keane, & Davidson, 2001), 45 or greater as *clinical* symptom levels (Blake et al., 1990), and any score in between as *subthreshold* symptom levels.

Given that these measures do not offer cutoffs at the subscale level, we extrapolated symptom subscale cutoffs from the guidelines for the full scales.[7] Specifically, we divided the total scale cutoffs by the total number of items on the CAPS (i.e., 17), then multiplied by the number of items in each subscale to determine the cutoffs for that subscale. For example, to calculate the cutoff for clinical levels of re-experiencing symptoms, we divided the total PTSD clinical symptom level of 45 by 17 (the number of items on the CAPS), then multiplied that number by the number of symptoms that characterize the re-experiencing subscale (i.e., 5 symptoms) $(45 \div 17 = 2.65) \times 5 = 13.25)$. The resulting number represents the cutoff for clinical level symptoms for the re-experiencing subscale, and so on.

Many of the studies included in this review utilized more than one measure of PTSD. For brevity, we elected to focus on the results of the interview-based measure when one was available, and on the self-report measure when one was not. When more than one self-report measure was utilized ($n = 3$), we reported on the measure used most frequently (i.e., the PDS). For the primary outcome of PTSD, we also provide a total summary of results across all measures, then separate the results by assessment type (interview vs. self-report).

2.3.2. Depression and anxiety

For the outcomes of depression and anxiety, categories were primarily based on established cutoffs: moderate-severe symptoms were categorized as *clinical*, mild-moderate as *subthreshold*, and low-minimal as *minimal/absent* (e.g., Beck Depression Inventory-II [BDI-II], Beck, Steer, & Brown, 1996; Beck Anxiety Inventory [BAI], Beck, Esptein, Brown, & Steer, 1988).

2.3.3. Quality of life (QOL)

The outcome of QOL is generally heterogeneous and is composed of varying measures and constructs reflecting life satisfaction, functioning, disability, and impairment (we will herein collectively refer to these as "quality of life"; Schnurr, Lunney, Bovin, & Marx, 2009). Although QOL is an important indicator of treatment-related improvement, the available data is hampered by the lack of clarity regarding the category of "remission" for disability outcomes (Sheehan & Sheehan, 2008), and the lack of standardized guidelines across assessments to determine clinical, subthreshold, and good end-state functioning. As such, we primarily utilized the Jacobson and Truax (1991) guidelines for creating clinical cutoffs for the varied measures reflecting QOL (e.g., SF-36, Ware & Sherbourne, 1992; Social Adjustment Scale [SAS], Weissman & Bothwell, 1976). When guidelines were available, indicators of *good end-state functioning* included categories labeled as "subclinical impairment" (Work and Social Adjustment Scale [WSAS], Mundt, Marks, Shear, & Greist, 2002), "average" or "good" QOL (e.g., Quality of Life Inventory, Frisch, 1994), and signs of "remission" (Sheehan Disability Scale [SDS], Sheehan & Sheehan, 2008). For the category of *subthreshold* symptoms, we coded for a "response" but not "remission" (e.g., SDS), or a level of remaining impairment without severe symptoms (e.g., WSAS).

2.4. Group differences

For the primary outcome of PTSD symptoms, we examined the percent of study arms with residual clinical, subthreshold, or minimal/absent symptoms at the post-treatment assessment (the timepoint most closely tied to treatment and most consistently available across all measures) to determine whether patterns emerged based on theoretically relevant subgroups. In particular, we examined treatment type (exposure vs. cognitive therapy vs. other CBT), population type (military/Veterans vs. civilians; female vs. male samples), and trauma type (interpersonal assault trauma vs. non-interpersonal assault trauma). For the secondary outcomes, samples sizes were generally too small to support examining such group differences. However, we did examine two groupings that included information from each study: whether clinical symptoms at pre-treatment were associated with greater residual symptoms (since pre-treatment severity for the secondary symptoms varied across the clinical, subthreshold, and minimal categories), and whether post-treatment PTSD symptom levels were associated with each secondary symptom level at post-treatment. We report the frequencies within each subgroup to allow for a comparison. It is important to note that statistical analysis of group differences was outside the scope of this review; any patterns that emerge here will serve to highlight potential factors that warrant further investigation.

3. Results

For an overall summary of results, see Table 1; for detailed results separated by each study and each outcome, please see Supplemental Table 2. The articles that provided data for the review are cited in the reference list for Supplemental Table 2.

3.1. Outcome: PTSD

A total of 51 studies were included, with 68 active treatment arms. Thirty-two of the study arms included more than one measure to capture PTSD symptoms, resulting in 104 assessments of PTSD across the 68 treatment arms. In all 104 cases, these measures indicated pre-treatment mean scores in the clinical range for PTSD. We focus here on the results of only one PTSD measure per treatment arm: an interview-

[7] For PTSD subscales, we considered using a cutoff based on DSM-IV criteria. For instance, for the CAPS reexperiencing (Criterion B) subscale for DSM-IV, one reexperiencing symptom is needed to meet this criterion. Therefore, a score of 3 (indicating a "1–2" score on a single reexperiencing item) could be counted as a residual symptom. However, this presents three problems: first, scoring was not available at this granular level, meaning that a "3" could indicate a "3" on one item, or a "1" on 3 items. Second, some studies did not subdivide symptoms according to DSM-IV categories (e.g. subdivided symptoms into numbing and dysphoria clusters). Third, the IES, a frequently used measure, does not follow the DSM-IV symptoms precisely. Therefore, to keep interpretation as consistent as possible across studies, we opted to utilize a cutoff reflecting a "3" for *each CAPS item* in a given symptom cluster.

Table 1
Percent of Study Arms with Residual Symptoms at Threshold Levels.

	PRE	POST	FOLLOW-UP
Total Sample, PTSD	(*n* = 68)	(*n* = 68)	(*n* = 60)
%CLINICAL	100	30.9	25.0
%SUBTHRESHOLD	0	58.8	50.0
%MINIMAL/ABSENT	0	10.3	25.0
Interview-Based PTSD Measures	(*n* = 53)	(*n* = 54)	(*n* = 47)
%CLINICAL	100	29.6	25.5
%SUBTHRESHOLD	0	61.1	44.7
%MINIMAL/ABSENT	0	9.3	29.8
Self-Report PTSD Measures	(*n* = 15)	(*n* = 14)	(*n* = 12)
%CLINICAL	100	35.7	25.0
%SUBTHRESHOLD	0	50.0	66.7
%MINIMAL/ABSENT	0	14.3	8.3
Intrusion/Re-experiencing	(*n* = 20)	(*n* = 20)	(*n* = 16)
%CLINICAL	100	55.0	68.8
%SUBTHRESHOLD	0	35.0	12.5
%MINIMAL/ABSENT	0	10.0	18.8
Avoidance (with Numbing)[a]	(*n* = 17)	(*n* = 17)	(*n* = 13)
%CLINICAL	100	47.1	69.2
%SUBTHRESHOLD	0	52.9	23.1
%MINIMAL/ABSENT	0	0	7.7
Hyperarousal	(*n* = 11)	(*n* = 11)	(*n* = 7)
%CLINICAL	100	54.5	42.9
%SUBTHRESHOLD	0	45.5	42.9
%MINIMAL/ABSENT	0	0	14.3
Depression	(*n* = 54)	(*n* = 54)	(*n* = 49)
%CLINICAL	88.9	18.5	24.2
%SUBTHRESHOLD	11.1	53.0	36.7
%MINIMAL/ABSENT	0	31.6	38.8
Anxiety	(*n* = 38)	(*n* = 38)	(*n* = 35)
%CLINICAL	100.0	55.3	54.3
%SUBTHRESHOLD	0	23.7	22.9
%MINIMAL/ABSENT	0	21.1	22.9
Quality of Life	(*n* = 27)	(*n* = 28)	(*n* = 22)
%CLINICAL	81.5 [b]	35.7 [b]	31.8 [b]
%SUBTHRESHOLD	3.7	42.9 [c]	31.8 [c]
%MINIMAL/ABSENT	14.8	21.4	36.4

[a] Some studied including avoidance or numbing separately. To facilitate comparison, this table reports those that reported combined avoidance and numbing (the most common breakdown of symptoms).

[b] Three studies had scores that were in the clinical range for the mental components subscale of the SF-12 and SF-36, while the physical components score was in the non-clinical range; these were counted as clinical (Mueser et al., 2008, pre-treatment and follow-up; Schnurr et al., 2003, each timepoint; Schnurr et al., 2007, pre-treatment, which also had a QOLI score just in the non-clinical range).

[c] Ehlers et al. (2014, Arms 1 and 2) had one measure that indicated subthreshold functioning (SDS) and one that indicated clinical life dissatisfaction (QLESQ), at both post-treatment and follow-up; these were counted as subthreshold.

based measure (*n* = 54) when available, or a self-report measure (*n* = 14)[8] . We first report on the total sample, then separate results based on those studies utilizing 1) an interview-based measure when available or 2) only a self-report measure.

3.1.1. Total sample (N = 68; Table 1)

Although PTSD symptoms decreased from pre- to post-treatment in all 68 study arms, only 10.3% of the active treatment arms yielded outcomes in the minimal/absent symptom range at post-treatment. This number increased to 25.0% at follow-up.

3.1.2. Interview-based assessments (n = 54; Table 1)

The CAPS is considered the "gold standard" for diagnosing PTSD (Weathers et al., 2001), and was the most frequently used measure among the studies using interview-based assessments (*n* = 45; 83.3%); the remaining nine studies (16.7%) used the PSS-I. As noted previously, participants' scores consistently decreased from pre-treatment to post-treatment across all studies; however, post-treatment scores fell into the minimal/absent symptom range in only 9.3% of the studies using an interview-based PTSD measure. This percentage increased to 29.8% at follow-up.

3.1.3. Self-report assessments (n = 14; Table 1)

The PDS (*n* = 7) was the most frequently used self-report measure. PTSD symptoms were again reduced at post-treatment, and a slightly larger percentage of studies (14.3%) (compared to the interview measures) yielded outcomes in the minimal/absent symptom range at post-treatment. Slightly fewer study arms had minimal/absent symptoms at follow up (8.3%).

3.1.4. Outcome: PTSD symptom clusters

Cluster-level data was available for 20 (29.4%) of the 68 active treatment arms, but the number of symptom clusters examined was not consistent: nine arms included a sub-analysis of only two clusters (intrusion and avoidance using the IES), eight arms evaluated the three standard DSM-IV clusters (reexperiencing, avoidance and numbing, hyperarousal), and three arms evaluated a four-cluster grouping that separated avoidance and numbing into individual clusters. All of the pre-treatment scores for the PTSD symptom cluster analysis were categorized as clinical.

For intrusions, at post-treatment, 35% of the samples decreased to subthreshold, and 10% decreased to the minimal/absent symptom range (18.8% at follow-up). Given the small number of study arms in which avoidance and numbing were reported separately (*n* = 3), we focused our analyses on those study arms in which avoidance (with numbing) is reported (*n* = 17). Although 52.9% had decreased to the subthreshold range at post-treatment, none had decreased to minimal/absent. At follow-up, 7.7% had decreased to the minimal/absent range, however there was also an increase in the percentage reporting clinical level symptoms post-treatment (47.1%) to follow-up (69.2%). At post-treatment, 45.5% of studies were categorized as subthreshold hyperarousal symptoms, while none decreased to the minimal/absent symptom range. By follow-up, 14.3% had decreased to the minimal/absent symptom range.

As with the overall PTSD measure, we examined symptom clusters by interview vs. self-report measure. However, here we report only on the interview measure (i.e. CAPS, *n* = 9) because 1) the IES was the only self-report symptom measure used to report individual symptom clusters and 2) of studies reporting the IES symptom clusters, all but two report on the version of the IES using only the intrusion and avoidance symptom clusters, thus not allowing for a comparison across all symptom clusters. Post-treatment symptom clusters for five of the nine studies remained at the same level as each other. However, in four studies, there was a differential pattern, with hyperarousal remaining at a more severe level than the other symptoms clusters (i.e. in three of the five, hyperourousal was clinical, whereas other clusters were subthreshold; in one of the five, hyperarousal was subthreshold whereas all other symptom clusters were minimal).

3.2. Outcome: depression (n = 54; Table 1)

The BDI was the most commonly used measure of depression (*n* = 46; 85%).Nearly all study populations were within the clinical range for depression at pre-treatment (88.9%). At post-treatment, studies most commonly were in the subthreshold range (53.0%), and depression continued to drop further at follow-up, with roughly equal levels of subthreshold and minimal symptoms (each 37.3%).

Further inspection of the data revealed that pre-treatment depression levels had some relationship to post-treatment levels: study arms

[8] Among those studies that did not use interview-based measures, the PDS was the most frequently used self-report measure (*n* = 7). Given its more frequent use and the fact that it is designed as a diagnostic scale, in cases where the PDS was used along with another self-report measure, we report here on the PDS results.

with post-treatment clinical or subthreshold symptom levels all had pre-treatment depression at clinical levels, whereas those with post-treatment minimal/absent depression levels were either at clinical (n = 11) or subthreshold (n = 6) levels at pre-treatment. Likewise, PTSD levels had some association with depression levels (e.g., 70% of studies with post-treatment clinical depression levels also had clinical post-treatment PTSD levels, 70% of those with subthreshold levels of depression also had subthreshold PTSD, and 30% of those with minimal/absent depression also had minimal/absent PTSD).

3.3. Outcome: anxiety (n = 38; Table 1)

Six different measures were used to assess anxiety; the State-Trait Anxiety Inventory (STAI) and the BAI were the most commonly used measures (47% and 30.6% respectively) (Beck et al., 1988; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). At pre-treatment, all study arms were within the clinical range for impairment and most remained in the clinical range at post-treatment (55.3%) and at follow-up (54.3%). At post-treatment, roughly equal numbers of studies were categorized as subthreshold (23.7%) and minimal symptoms (21.1%). At follow-up, anxiety symptoms were roughly the same (21.2% subthreshold and 24.2% minimal).

For anxiety, we could not examine the relationship of pre- to post-treatment symptom levels, given that all studies were in the clinical range at pre-treatment. Examining post-treatment PTSD levels revealed that they had some association (of studies with post-treatment clinical anxiety, most [62%] had post-treatment clinical PTSD, of studies with subthreshold anxiety, most [78%] had subthreshold PTSD, and all with minimal anxiety had subthreshold PTSD [none had minimal/absent PTSD]).

3.4. Outcome: quality of life (n = 28[9]; Table 1)

QOL outcomes included the varied constructs of disability (SDS [most commonly used n = 7], CAPS disability item), functioning (SF-36, SF-12, WSAS, SAS, variations of the World Health Organization Well-Being Index [Bech, Olsen, Kjoller, & Rasmussen, 2003]), and life satisfaction measures (QOLI, Quality of Life Enjoyment and Satisfaction Questionnaire [QLESQ; Endicott, Nee, Harrison, & Blumenthal, 1993].

Most study populations were within the clinical range for impairment at pre-treatment (81%), whereas at post-treatment most were in the subthreshold range (43%). Studies were roughly evenly split between clinical, subthreshold, and minimal/absent symptom ranges at follow-up, with most in the minimal/absent range (36%).

Further inspection of the data revealed that pre-treatment QOL levels had some relationship to post-treatment levels: study arms with post-treatment clinical levels were all clinical at pre-treatment, those with post-treatment subthreshold levels were mostly (92%) clinical at pre-treatment, and those with post-treatment minimal/absent levels of impairment mostly (67%) at minimal/absent levels of impairment at pre-treatment. Unlike depression and anxiety, post-treatment QOL levels were not consistently associated with post-treatment PTSD levels (e.g., those with post-treatment clinical QOL levels were most likely to have subthreshold post-treatment PTSD symptoms [50%], those with post-treatment subthreshold QOL symptoms were also most likely to have subthreshold post-treatment PTSD levels [92%], but those with post-treatment minimal impairment were most likely to have post-treatment clinical QOL levels [50%]).

[9] Forbes et al. (2012) also reported on quality of life. However, reported baseline symptom levels were different in Tables 1 and 2. We were unable to clarify this difference and therefore excluded this study from this review.

3.5. Exploratory Descriptive Group Differences (Table 2): PTSD

A secondary aim of our review was to determine whether residual PTSD symptoms were more likely to emerge among certain subgroups. To examine this aim, we calculated and compared the percentage of study arms with residual symptoms across different sub-groupings (see Table 2) at the first post-treatment assessment. Given the small sample sizes across the multiple symptom levels after the groupings were established, this aim is exploratory to highlight patterns and prompt further exploration. For the grouping of treatment type across clinical, subthreshold, and mimimal/absent symptoms levels, there are few interpretable differences. On the other hand, differences among population subtypes reveal some notable findings. That is, military/Veteran samples comprised a higher percentage of clinical outcomes (28.6% of clinical outcomes, but 0% of minimal/absent symptom outcomes) compared to civilian samples (71.4% of clinical outcomes but 100% of minimal/absent outcomes). On the contrary, samples who experienced interpersonal trauma (assault, abuse) comprised only 4.8% of the clinical outcomes, but 71.4% of the minimal/absent outcomes. Relatedly, all-female samples comprised only 4.8% of clinical outcomes but 71.4% of minimal/absent symptom outcomes.

4. Discussion

To date, residual symptoms have not been a primary focus of the broad PTSD psychotherapy literature and have not been addressed in a unified manner; therefore, little is known regarding their presence following psychosocial treatment. This review provides preliminary work toward quantifying residual symptoms following empirically supported RCT's of psychotherapy for PTSD. Our findings indicate that residual PTSD symptoms continue to be common after treatment, with minimal/absent symptoms achieved for roughly 10% of study arms at post-treatment, and 25% at a later follow-up. Regarding the secondary outcomes of depression, anxiety, and QOL, roughly one-quarter to one-third of the study arms had samples with minimal/absent residual symptoms/impairment at post-treatment.

Across all outcomes, no substantial group differences emerged across treatment types. This is not surprising, as direct comparisons of active trauma-focused therapies tend to find little difference in clinical outcome (e.g., Resick, Nishith, Weaver, Astin, & Feuer, 2002). However, pre-treatment symptom severity affected post-treatment secondary outcomes, with clinical samples being more likely to display residual symptoms than samples starting in a subthreshold or minimal/absent range. Most comorbid symptoms (except for QOL) tended to parallel decreases in PTSD symptoms. Some patterns emerged based on sample type: samples with the best outcomes were more likely to include all-female samples, non-military/Veteran samples, and those who had experienced abuse/assault as the index trauma. These results are consistent with a prior review suggesting that military samples may not respond as well to empirically supported treatments as non-military samples (Steenkamp et al., 2015). The literature suggests that interpersonal assault traumas lead to higher levels of PTSD symptoms (Olff et al., 2007), but our study would indicate that they respond well to treatment, ending with lower levels of residual symptoms (this is confounded by the overlap with more female samples, who also appear to have fewer residual symptoms following treatment, as has been found more generally, Kirshner, Genack, & Hauser, 1978). This may also speak to the fact that at least two treatments (PE and CPT) were initially designed specifically for female interpersonal assault survivors, though they have now been used much more broadly.

4.1. Global posttraumatic stress disorder

Our findings indicate that following empirically-supported PTSD psychosocial treatment, participants continue to experience residual symptoms (90% total: 31% at the clinical level and 59% at the

Table 2
Number and Percent of Study Arms with Residual PTSD Symptoms at Threshold Levels at Post-Treatment Divided by Treatment Type, Trauma Type, and Sample Type (n = 68).

CATEGORY	CLINICAL n (%)	SUBTHRESHOLD n (%)	MINIMAL/ABSENT n (%)
Treatment Type	Exposure 5 (23.8%)	Exposure 16 (40.0%)	Exposure 1 (14.3%)
	Cognitive 5 (23.8%)	Cognitive 5 (12.5%)	Cognitive 2 (14.3%)
	Other CBT 11 (52.4%)	Other CBT 17 (42.5%)	Other CBT 4 (57.1%)
Military/Veteran Sample[a]	Yes 6 (28.6%)	Yes 4 (10%)[a]	Yes 0 (0%)
	No 15 (71.4%)	No 36 (90%)	No 6 (100%)
Interpersonal Trauma	Yes 1 (4.8%)	Yes 9 (22.5%)	Yes 5 (71.4%)
	No 20 (95.2%)	No 31 (77.5%)	No 2 (28.6%)
Gender	Both 19 (90.5%)	Both 30 (75%)	Both 2 (28.6%)
	Male 1 (4.8%)	Male –	Male –
	Female 1 (4.8%)	Female 10 (25%)	Female 5 (71.4%)

Note: Percentages are calculated as a percent of those study arms in the same post-treatment category. "Exposure" includes PE, imaginal exposure, and in vivo exposure. "Cognitive" includes CPT, cognitive therapy, cognitive restructuring, and meta-cognitive therapy. "Other CBT" includes CBT, treatments that address both PTSD and substance use disorders, and treatments that combine exposure and cognitive elements. "Interpersonal trauma" includes adult or childhood assault (sexual or physical).

[a] Two study arms in the subthreshold category had mixed civilian/veteran samples. Thus, one was counted in the military sample category and one in the non-military sample.

subthreshold level). Our findings also suggest that improvements in PTSD symptoms continue after treatment has ended, such that at a later follow-up assessment, residual symptoms remained in 75% vs. 90% of study arms (25% clinical; 50% subthreshold). These results suggest that it may be beneficial to assess residual symptoms at end of treatment or later follow-up appointment to determine the longer-term effects, and to describe which symptoms remain. Although the current study cannot determine whether these residual symptoms are problematic, there is some evidence from other studies that remaining symptoms may contribute to residual impairment (Lunney & Schnurr, 2007; Monson et al., 2012; Shnaider et al., 2014), to the risk for increased PTSD following any future stressors (Boe, Holgerson, & Holen, 2010), and even to the risk of re-victimization (Risser, Hetzel-Riggin, Thomsen, & McCanne, 2006). However, it is possible that following successful therapy, clients are equipped with the coping skills to manage the residual symptoms. Further research is warranted to directly address these questions.

4.2. PTSD symptom clusters

None of the studies examined in this review directly reported on post-treatment PTSD symptoms at the individual symptom level, and only about one-third reported on symptoms at the symptom-cluster level, making a thorough and detailed analysis of the specific nature of residual PTSD symptom-cluster data difficult. Using the coding system created for this study, our findings indicate that although symptoms generally improved from pre-to post-treatment and follow-up, no more than 20% of studies displayed minimal/absent symptom levels at either timepoint.

When we examined changes in symptom clusters for only those studies using the CAPS, our findings suggested that hyperarousal was most likely to persist immediately following treatment. This finding, though it should be considered quite tentative given the small sample size, is consistent with prior literature that suggests that standard CBT interventions may be more effective at targeting intrusions and avoidance; hyperarousal symptoms may be more dependent on a broad range of maintaining factors which may warrant specific intervention following CBT for PTSD (Zayfert & DeViva, 2004). Schnurr and Lunney (2015) also reported that PE had larger effect sizes for the reduction of avoidance and numbing symptoms vs. hyperarousal symptoms. Thus, the current front-line cognitive-behavioral treatments may not target arousal symptoms to the degree necessary for full symptom abatement, potentially calling for a specific arousal-targeted intervention or intervention component. Further, hyperarousal may be the symptom cluster linked most closely to emotion dysregulation and the evolutionarily older fear system that is more automatic and less cognitively mediated. It is possible that pre-trauma emotion dysregulation (or other risk factors) both put people at risk for PTSD, and are more likely to remain following treatment. If that is the case, there may be targeted methods to both treat and prevent PTSD based on emotion dysregulation prior to trauma or immediately post-trauma. Notably, patterns were different when examining multiple measures (i.e. in Table 1, our overall findings show roughly equal levels of all symptom clusters at post-treatment, with more clinical levels of intrusions and avoidance than hyperarousal at follow-up), highlighting the importance of using best-practice measures. Given our small sample size, we would suggest future studies use the CAPS subscales at all follow-up timepoints to allow for larger comparisons.

4.3. Depression and anxiety

Although PTSD treatment has proven effective in reducing symptoms of depression and anxiety (Foa et al., 1999; Resick et al., 2002; Tarrier, Pilgrim et al., 1999; van den Berg et al., 2015), residual symptoms may be expected given that therapies for PTSD do not specifically target these comorbid conditions. To this end, depression remained at a clinical level in one fifth of all study arms, and anxiety in one half of all study arms at post-treatment, generally paralleling the results for PTSD improvement. Neither treatment type nor sample type appeared to be associated with residual symptoms of depression or anxiety. However, both baseline symptom severity and changes in PTSD symptoms were associated with post-treatment outcomes, such that improvements in PTSD generally translated into improvements in anxiety and depression. Many treatments examined here were explicitly cognitive and it is possible that cognitive change reducing dysfunctional attitudes or modifying the processing of depression-related material may contribute to decreases in both PTSD and depression; however, depression- or anxiety-specific processes and symptoms may not be as fully addressed as trauma-specific cognitions. Relatedly, Flory and Yehuda (2015) also suggest that if a comorbidity reflects a phenotype or subtype of PTSD (vs. pure symptom overlap), then treatment approaches may not be as effective for depressive symptomology, and may require more targeted treatment. Simiarly, van den Berg et al. (2015) highlight that there are different pathways and development of comorbidity, and these differences may impact PTSD symptoms and treatment differently.

4.4. Quality of life

Ideally, treatment effects will go beyond symptom remission to improve QOL. Our results are consistent with some authors' suggestion that changes in functioning may be slower than changes in symptoms, and may only emerge with longer follow-up (Sheehan & Sheehan, 2008). That is, in this study, QOL continued to improve from pre-treatment to post-treatment and further at follow-up. But interestingly, there was a less clear relationship between PTSD symptoms and QOL over time than with depression or anxiety. This is contrary to a recent study that found a dose-response relationship between PTSD symptom remission and QOL improvement (Schnurr & Lunney, 2016). Given the small number of studies included for QOL in this review, caution is warranted in interpreting these results; further research is needed to better understand the relationship between PTSD symptoms and QOL.

4.5. Limitations

Results from the current study should be considered in light of certain limitations. We focused primarily on residual symptoms within responder/completer samples to include participants who had completed a full course of treatment; as such, it is possible that the levels of residual symptoms are present to a greater extent within intent to treat samples. Further, our methodology of grouping together different forms of cognitive behavioral treatments may have masked any true treatment differences across samples; however, a statistical moderation analyses was beyond the scope of this review, and there was an insufficient number of studies to focus on only one type of therapy. Likewise, larger samples would have allowed us to more thoroughly analyze subgroups (for instance, there was only one all-male sample included in this review). Further, the fact that interpersonal trauma samples and all-female samples substantially overlapped makes it difficult to tease apart the effects of those sub-groupings; future research including males in interpersonal trauma studies would help in this regard.

Additional limitations are based on the limitations present in the larger literature, e.g., the lack of studies routinely using DSM-5 assessments and capturing newly introduced symptoms of PTSD (e.g., recklessness); the limited reporting on individual symptoms or symptom clusters (especially numbing); the discrepant ways of measuring QOL; and the lack of reporting on—and established cutoffs for—physiological indicators and dissociation.

Our study was not a true systematic review in that we utilized studies identified in two published systematic reviews; at the time of data collection for this study (2017), those reviews represented recent and relevant RCT data to address our primary research question. Of note, the most recent RCTs (published in 2018) are not represented here. Further, as the literature advances, evidence for therapies not included in this review continue to grow; for instance, an initial online publication of the International Society for Traumatic Stress Studies (2018) found strong support for EMDR in adults, indicating that future studies should carefully examine residual symptoms after EMDR (and any others that continue to gain support).

In general, the coding system that was created for this study was designed to maximize the available data from published manuscripts; however, this data was not always ideal for creating cutoffs and interpreting the outcomes. Specifically, our use of group means to determine whether symptoms were still present at post-treatment may wash out differences within a given treatment arm. Regarding residual PTSD symptoms, we created total symptom scale (and subscale) cut-off scores that reflected the number of items in that (sub)scale rather than using diagnostic criteria to determine the presence or absence of residual PTSD symptoms. This method leads to some lack of clarity regarding whether a particular symptom severity would reach a diagnostic threshold or not, particularly on the CAPS. Thus, it is still unknown whether these residual symptoms are sufficient for a diagnosis.

Finally, given that psychological distress and health may differ between individuals with single- versus multiple-incident traumas, we note that many studies reviewed here did not assess lifetime/previous trauma exposure which may contribute to residual symptoms following treatment of a single trauma.

4.6. Research implications

First, in order to bring higher resolution to particular symptoms/processes that are not adequately targeted by current "first-line" interventions, we recommend increased attention to and reporting on individual PTSD symptoms or clusters (ideally via the CAPS; see Zayfert & DeViva, 2004; Schnurr & Lunney, 2015) to allow detection of patterns of residual PTSD symptoms. Specifically, reporting on the *frequency* of patients with remaining residual individual symptoms, rather than *average* residual symptoms in the sample may allow for the detection of specific symptoms within any given cluster that are particularly likely to remain as residual.

Second, we recommend consistent use of agreed-upon measures when conducting RCTs for PTSD (e.g., see Phen-X toolkit: a catalog of recommended and standard measures in biomedical research; Hamilton et al., 2011). Using consistent measures (along with the gold standard CAPS) across studies would allow for heightened comparability and usefulness of meta-analysis results. Based on this review, the BDI-II and STAI-S are the most widely used measures for depression and anxiety and may be most appropriate given their quick administration. In general, we recommend further reporting in the area of QOL, given that many studies did not include any such measures: the SDS and WSAS are relatively widely used and provide more clarity for interpretation than other measures. For dissociation, we recommend the DES-II (Carlson & Putnam, 1993) due to its widespread use and the availability of population subtype means, norms, and cutoff values. At a minimum, the three CAPS dissociation items could be used as an alternative.

Future studies could also consider including and examining several other types of outcomes that were not included here due to small sample sizes, e.g., dissociation, physiological symptoms, substance use, anger, cognitive symptoms/functioning (Kent, Davis, Stark, & Stewart, 2011; Walter, Palmieri, & Gunstad, 2010), trauma-related cognitions, sleep, and startle response. Further, given the overlapping but distinct features of complex PTSD as defined by ICD (e.g., disturbances in self-concept, emotion regulation, and relationships), it would be useful to examine residual symptoms among patients with complex PTSD to determine whether similar patterns emerge. Likewise, future reviews can include other treatments that had not gathered sufficient support at the time of this review to be included (e.g., NET). In addition, it would be useful to examine how residual symptoms are related to and may maintain the presence of one another (e.g., network analysis).

Third, we would recommend continued conversation to establish consensus on scale cutoffs to indicate clinical, subthreshold, and good end-state functioning for the relevant outcomes (particularly for PTSD symptom clusters).

Finally, a movement toward the Research Domain Criteria (RDoC) framework would involve the continued use of self-report and clinical interview measures, in combination with the inclusion of: 1) additional units of analysis (e.g., physiological measures), 2) fear, anxiety, and sustained threat constructs within the *negative valence systems* domain, and 3) social communication and affiliation/attachment constructs within the *social processes* domain. Specifically, physiological symptoms are integral to the fear response, and serve as the theoretical basis of PTSD. Physiological outcome measures are rarely examined, but seem to persist despite successful treatment. Given the significant individual variability in many baseline physiological measures, such as resting heart rate, we anticipate it would be more beneficial to examine malleable physiological measures *in response to stressors* (e.g., trauma-script imagery protocol) or heart rate variability, as these factors may be responsive to intervention.

Taken together, future studies should have a goal of identifying the effects of gold standard PTSD treatments on specific symptoms at cluster and sub-cluster levels, as well as upon relevant RDoC constructs, to elucidate understanding of the mechanisms underlying both PTSD symptomatology and treatment response.

4.7. Clinical implications

Our findings highlight the need for a more detailed assessment of residual symptoms at both short- and long-term follow-up periods after treatment, and for more empirical attention focused on the clinical management of residual symptoms. For example, it is unknown whether treatment should continue until PTSD can no longer be diagnosed, or until a response of a certain magnitude is indicated. This is particularly important considering research suggesting that the former approach has been associated with further improvements in QOL (Schnurr & Lunney, 2016), and that there are associations between subthreshold PTSD and impaired social and work functioning, anger, hostility, and suicide attempts (Zlotnick et al., 2002).

Specifically, one study examined variable-length CPT, in which treatment could be completed with more or less than 12 sessions depending on when "good end-state functioning" was achieved. This individualized approach resulted in the loss of diagnosis for nearly the full sample, but at different time-points for each person (Galovski, Blain, Mott, Elwood, & Houle, 2012). Further, comorbid conditions may become mutually maintaining, and transdiagnostic treatments may ultimately be more suitable to address comorbid conditions as opposed to single disorder treatment protocols. Our findings also support an argument for modular treatments that can specifically address symptoms still remaining (e.g., pivoting to a treatment focused specifically on insomnia if that is still residual).

4.8. Conclusions

There has been a long-standing call within psychotherapy research to look beyond effect sizes of treatments toward *clinical significance* (e.g., Jacobson & Truax, 1991). The move toward examining residual symptoms gives the field another means to evaluate empirically supported treatments. Although the PTSD treatment literature has made advances in solidifying evidence for PTSD treatment, most active study arms in this review demonstrated evidence of subthreshold or clinical levels of residual PTSD and associated comorbidities. These results highlight the need for focused attention to residual symptoms within the PTSD literature. By directly examining which individual PTSD and comorbid symptoms are residual following empirically supported treatment, we could gain a better understanding of both the strengths and limitations of empirically support treatments, and methods to improve their efficacy.

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Appendix A. Supplementary data

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