Comorbid Posttraumatic Stress Disorder and Major Depressive Disorder: The Usefulness of a Combined Treatment Approach

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Contents

Abstract

Objective: This thesis examined the utility of targeting depressive symptoms in those with comorbid posttraumatic stress disorder (PTSD) and major depressive disorder (MDD). Working from the perspective that MDD interferes with PTSD treatment efficacy by impeding optimal emotional engagement during therapy, this thesis tested a therapy approach that first addressed MDD, followed by cognitive processing therapy (CPT) for PTSD. The possible mechanisms through which MDD reduces optimal PTSD treatment outcomes were also examined. It was predicted that inhibited (i.e., underengagement) and elevated (i.e., overengagement) levels of emotional engagement would predict reduced PTSD and MDD outcomes.

Method: A randomised control, crossover design was used. Fifty individuals with comorbid PTSD and MDD were randomised to receive either CPT alone, CPT then behavioural activation (BA) for MDD, or BA then CPT. Participants were assessed at pre-, mid-, posttreatment, and at 6-month follow-up. PTSD and MDD symptom severity was further assessed every second session. PTSD and MDD symptoms were the main outcome variables of interest; emotional engagement, trauma cognitions, rumination, and emotional numbing were assessed as hypothesised mechanisms of change. Imputations were made for missing posttreatment, and follow-up data, and mixed, repeated-measures ANOVAs were run on each imputed dataset and results pooled. Emotional engagement was also assessed through therapy session coding based on the Client Expressed Emotional Arousal Scale-III. Specifically, all therapy sessions were coded for levels of under-, over-, and optimal emotional engagement. Mixed-effect models were used to analyse the relationship between under-, over- and optimal level of emotional engagement and PTSD and MDD outcome over the course of treatment.

Results: All conditions evidenced significant improvements on primary (PTSD and MDD) and secondary treatment outcomes (trauma cognition, rumination, emotional

numbing) from pre- to posttreatment, and pre- to 6-month follow-up. Effect sizes for the intent-to-treat sample were good with within group effect sizes ranging from 1.25 to 2.84 for PTSD symptoms, and 0.56 to 1.51 for depressive symptoms. At posttreatment, compared to CPT and BA/CPT, CPT/BA evidenced significantly greater improvements on all measures other than emotional numbing. At 6-month follow-up, compared to CPT and BA/CPT, CPT/BA evidenced significantly greater improvements on measures of rumination, and meaningfully larger improvements on measures of PTSD, MDD, and trauma cognitions. Further, CPT/BA demonstrated greater participant retention than CPT and BA/CPT.

Condition differences in the effects of under- and optimal emotional engagement emerged. For CPT and CPT/BA, elevated levels of underengagement predicted elevated PTSD (but not MDD) symptoms over the course of treatment, and elevated levels of optimal engagement predicted reduced PTSD and MDD symptoms over the course of treatment. However, this was not the case for BA/CPT, and BA/CPT participants appeared less sensitive to the effects of under- and optimal engagement. For all conditions, elevated levels of overengagement predicted elevated PTSD and MDD symptoms.

Conclusion: Findings support modifications to CPT and indicate that there is *added* benefit in targeting MDD in the treatment of comorbid PTSD/MDD. However, treatment order is imperative, with superior treatment outcomes only achieved when PTSD is targeted *prior* to MDD. That is, CPT/BA appeared to be the treatment of choice. Results also suggest that optimal levels of emotional engagement are critical to the therapeutic process in CPT, and that under- and overengagement are detrimental to achieving good treatment outcomes.

Declaration

'I certify that this thesis does not incorporate without acknowledgement any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.'

Samantha Angelakis

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research is what allows us to continue to learn more about posttraumatic stress disorder, and is what allows us to ensure that people in similar situations are provided with the best available treatments.

Chapter 1: Introduction

It is well established that posttraumatic stress disorder (PTSD) often co-occurs with depression. Over a range of sample and trauma types it has been observed that 30-50% of individuals with PTSD also meet the criteria for a diagnosis of depression (Creamer, Burgess, & McFarlane, 2001; Kessler et al., 2005a; Rytwinski, Scur, Feeny & Youngstorm, 2013). The high prevalence of PTSD and major depressive disorder (MDD) comorbidity is problematic as individuals with comorbid PTSD and MDD demonstrate a greater illness burden and lower levels of global functioning (Kessler, Chiu, Demler, & Walters, 2005b), a more chronic course of impairment (Post, Zoellner, Youngstorm, & Feeny, 2011), and a more delayed response to treatment (Green et al., 2006) than individuals with PTSD or MDD alone. Further, initial levels of depression have been associated with poorer treatment outcomes (Bryant, Moulds, Guthrie, Dang, & Nixon, 2003; Taylor et al., 2001) although this is not always a consistent finding (Gillespie, Duffy, Hackmann, & Clark, 2002). Despite such findings and irrespective of the high prevalence of PTSD/MDD comorbidity, few studies have tested treatments that address *both* PTSD and a comorbid condition, let alone PTSD and MDD specifically. That is, whilst research has begun to investigate this in relation to comorbid panic (Falsetti, Resnick, & Davis, 2005) and substance use (Cook, Walser, Kane, Ruzek, & Woody, 2006; Najavits, Weiss, Shaw, & Munez, 1998), research has not examined the efficacy of using combined treatments to target comorbid PTSD/MDD.

In line with the paucity of research surrounding the treatment of comorbid PTSD/MDD, in this thesis I tested the utility of a combined treatment approach that addressed PTSD *and* MDD symptoms. The possible mechanisms through which depression reduces optimal PTSD treatment outcomes were also examined. Working from the perspective that depression interferes with PTSD treatment efficacy by impeding optimal emotional engagement during therapy, I explicitly tested a therapy

approach that first addressed depression, followed by cognitive processing therapy (CPT) for PTSD. Participants were randomly allocated to three treatment groups: CPT alone, behavioural activation (BA) for depression then CPT, or CPT then BA. I also recorded and coded all therapy sessions for emotional engagement. The three groups were used to answer the following questions: (1) Does a combined treatment that targets both PTSD and MDD result in added benefits relative to PTSD treatment alone, (2) If a combined treatment is useful, does it matter in which order therapy is delivered and, (3) Does emotional engagement predict treatment outcome?

This first chapter serves as a literature review and examines explanations for the high PTSD/MDD comorbid relationship. Likely candidate variables that would explain the high level of comorbidity between PTSD and MDD are also reviewed with a focus on a shared vulnerability account of the PTSD/MDD relationship. The chapter then illustrates how depression may impede optimal recovery from PTSD through review of both theoretical and empirical findings. I argue that there is a need to examine treatments that target both PTSD and MDD symptoms. Chapter 2 outlines the methodology of the treatment study. Chapters 3 and 4 report results surrounding treatment outcomes and process measures. Finally, Chapter 5 discusses findings and provides suggestions for future research.

Pathways to Comorbid PTSD and MDD

While the high prevalence of PTSD/MDD comorbidity is well documented, the *nature* and *causes* of PTSD/MDD comorbidity are less understood. As a better understanding of the mechanisms underlying such comorbidity will ultimately improve treatment development, this chapter begins by exploring potential pathways to PTSD/MDD comorbidity. Research examining the temporal order of PTSD and MDD development has suggested several potential pathways to PTSD/MDD comorbidity.

Some researchers have proposed that pre-existing MDD may elevate one's susceptibility to traumatic events (Breslau, Davis, Peterson, & Schultz, 1997; Kessler, Sonnega, Bromet, & Nelson, 1995). It has also been suggested that MDD may be a reaction to PTSD, whereby PTSD is a risk factor for the development of MDD (Breslau, Davis, Peterson, & Schultz, 2000). Recently, Stander, Thomsen, and Highfill-McRoy (2014) examined the literature regarding the development of comorbid PTSD/MDD, in military samples. Although the reviewed literature generally supported the hypothesis that PTSD was a causal risk factor for the development of MDD, they acknowledged that the exact relationship between PTSD and MDD was likely to be complex, involving bidirectional causality, common risk factors, and common vulnerabilities.

Shared Vulnerability Pathways to Comorbid PTSD

The finding that MDD elevates the risk of developing PTSD after trauma exposure (Koenen et al., 2002), and the finding that PTSD increases the risk of developing first onset MDD following a trauma (Breslau et al., 2000; Kessler et al., 1995), does suggest a bidirectional relationship between the two disorders and that this may occur due to a shared vulnerability or a shared diathesis. Methodologically sound prospective studies have shed light on the shared vulnerability relationship between PTSD and comorbid MDD. Breslau et al. (2000) explored PTSD-MDD pathways using retrospective and prospective data from a large sample of trauma victims (n = 1,007) and found that those with pre-existing MDD were three times more likely to develop PTSD after trauma exposure, compared with those without pre-existing MDD. Further, relative to those who were not exposed to a trauma, those who were exposed to trauma and developed PTSD were 2.8 times more likely to develop MDD. However, those who were exposed to a trauma and did not develop PTSD were not significantly more likely to develop MDD. Using a similar prospective design with trauma victims recruited from

a hospital emergency room Shalev et al. (1998) found that the prevalence of MDD in patients with PTSD was 44% compared to 29% for those without PTSD. Cross sectional studies further support the shared vulnerabilities explanation (see Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Nixon, Resick, & Nishith, 2004).

PTSD/MDD Comorbidity Is Not an Artefact of Symptom Overlap

The frequent comorbidity of PTSD and MDD has also been explained in terms of symptom overlap with researchers positing that the disorders co-occur superficially due to the shared symptoms of sleep disturbances, diminished interest and involvement in everyday activities, and concentration difficulties. However, for the most part this explanation has been discounted (e.g., Blanchard, Buckley, Hickling, & Taylor, 1998; Ford, Elhai, Ruggiero, & Frueh, 2009). Elhai, Grubaugh, Kashdan, and Frueh (2008) found that the lifetime prevalence rate of depression amongst adults with PTSD (54.7%) remained essentially the same when overlapping symptoms were removed and a prorated PTSD diagnostic algorism was applied (54.41%). Further, using data taken from the National Survey of Adolescents, Ford et al. found that the rate of PTSD remained unchanged when overlapping symptoms were removed and an altered PTSD diagnostic algorithm was applied. In line with this, Stander et al. (2014) suggest that rather than PTSD and MDD comorbidity being the product of definitional confounds, common underlying dimensions for PTSD and MDD symptoms may actually be manifestations of common vulnerabilities. However, alternative findings exist. O'Donnell, Creamer, and Pattison (2004) explored the relationship between PTSD, MDD, and comorbid PTSD/MDD in a sample of 363 injury survivors. They found that PTSD and MDD presented as independent constructs three months post-trauma. However, at one year follow-up PTSD and MDD symptoms became less distinct and no

longer presented as unique constructs. While O'Donnell et al. found a merged traumatic stress response this is an exception to the majority of research.

The existence of a dysphoric factor within PTSD has also led researchers to posit that the elevated rate of PTSD/MDD comorbidity may occur superficially due to a shared, non-specific dysphoric factor. However, the factor structure of PTSD still remains unclear and contradictory findings exist. For instance, some researchers highlight a shared underlying latent structure whereby PTSD and MDD symptoms may be represented by a single, underlying structure (Elhai et al., 2011; Miller et al., 2010). Adding to this, others suggest that all PTSD symptoms are associated with general distress and that PTSD-specific symptoms are no less correlated with distress or depression than dysphoric symptoms (Elkit, Armour, & Shevlin, 2010; Marshall, Schell, & Miles, 2010). Alternatively, others posit that PTSD and MDD are distinct factors and highlight distinguishable features within PTSD (Forbes et al., 2010; Post et al., 2011). For instance, longitudinal research has identified PTSD symptoms that uniquely account for later PTSD adjustment and suggest that such symptoms are different from those that predict both PTSD and MDD severity. For example, hyperarousal has been found to be a defining feature of PTSD that is able to uniquely predict PTSD adjustment (Marshall, Schell, Glynn, & Shetty, 2006; Schell, Marshall, & Jaycox, 2004). There currently does not appear to be a clear pattern of methodological or sample differences that would account for the discrepancies in the literature.

Irrespective of the contradictory findings, and of most relevance to the current review, researchers have consistently demonstrated that PTSD and MDD remain distinct features when overlapping symptoms are removed, and, as will be highlighted later, findings suggest that MDD influences treatment outcomes in those with PTSD. Thus, although PTSD and MDD may share a common dysphoric factor that influences comorbidity, PTSD and MDD can be considered separate constructs. Accordingly, there

is still a critical need to further explore the relationship between PTSD and MDD and examine treatments for comorbid PTSD/MDD.

Possible Candidates to Explain Shared Vulnerability

In line with a shared vulnerability pathway numerous studies have indicated that risk factors for PTSD are also risk factors for MDD. For example, event severity, childhood trauma, female gender, and pre-existing anxiety and depressive disorders are all risk factors for PTSD *and* MDD development (Breslau et al., 1997; 2000). Although it is acknowledged that these risk factors are not confined to PTSD and MDD and have been found in other psychopathologies (e.g., Kessler et al., 2005b), there are a number of other potential mechanisms shared by PTSD and MDD that may explain the high comorbidity of the disorders. These are now briefly reviewed.

Cognitive and memory processes. PTSD and MDD share similarities across numerous cognitive and memory processes. For instance, individuals with MDD can experience intrusive memories at the same frequency and level of distress as those with PTSD (Brewin, Gregory, Lipton, & Burgess, 2010). Further, individuals with PTSD and MDD both demonstrate retrieval of overgeneralised autobiographical memories (Harvey, Bryant, & Dang, 1998; Kuyken & Dalgleish, 1995). Importantly, research demonstrates that overgeneralised memories are not simply a marker for psychopathology but can predict the onset and maintenance of PTSD and MDD (Kleim & Ehlers, 2008). As overgeneralised memories may influence the onset and development of psychopathology, and as this has been demonstrated so reliably in PTSD and MDD (Williams et al., 2007), it is reasonable to posit that such common memory processes may explain why individuals are vulnerable to this comorbidity.

Shared cognitive vulnerabilities such as rumination may further drive PTSD/MDD comorbidity. Rumination is well established in PTSD and MDD (Michael,

Halligan, Clark, & Ehlers, 2007; Nolen-Hoeksema, 2000) with rumination surrounding the causes, consequences, and implications of a trauma, along with repetitive thinking about the causes and consequences of one's distress, consistently found to predict PTSD and MDD symptom severity, onset, and maintenance (Ehlers, Mayou, & Bryant, 1998; Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema &Morrow, 1991; Steil & Ehlers, 2000). Further, longitudinal studies have repeatedly found that post-trauma rumination predicts later PTSD and MDD symptoms. For instance, in a longitudinal study of road traffic accident survivors, Ehring, Rank, and Ehlers (2008) found that trauma- and depressive-rumination not only predicted PTSD symptom severity, but that premorbid depressive rumination predicted post-trauma depressive symptoms. Michael, McLaughlin, Shepherd, and Nolen-Hoeskema (2013) explored the role of rumination in the development of depression and anxiety after stressful life events using a longitudinal design and found that self-reported exposure to stressful life events was related to increased levels of rumination. Further, rumination mediated the relationship between self-reported stressful life events and anxiety and depression symptoms. Such a body of literature suggests that a tendency to ruminate may influence the development of MDD and anxiety (i.e., PTSD) following traumatic events, and suggests that a shared predisposition towards rumination may in part promote PTSD/MDD comorbidity.

Maladaptive cognitions are also critical in the etiology of PTSD and MDD. Depressive cognitions often relate to the self, the world, and the future and are characterised by a sense of helplessness (Seligman, Abramson, Semmel, & von Baeyer, 1979). Further, individuals with PTSD tend to interpret information in a way that leads to a sense of fear or persistent state of threat (Ehlers & Clark, 2000). Unsurprisingly the maladaptive cognitions inherent in PTSD and MDD overlap. Individuals with PTSD and MDD both show a tendency to interpret events in a negative manner, to catastrophise, and to blame themselves for events (Beck, Riskind, Brown, & Steer,

1988; Dunmore, Clark, & Ehlers, 2001). Additionally, individuals with PTSD often exhibit negative beliefs in line with Beck's cognitive triad. As maladaptive cognitions influence the onset and development of PTSD and MDD, and as maladaptive cognitions overlap in the two disorders, one may again posit that common, maladaptive cognitions further potentiate the comorbid PTSD/MDD relationship. The reviewed literature suggests that PTSD and MDD share a series of maladaptive cognitive and memory processes that may contribute to the high rate of PTSD/MDD comorbidity. In other words, comorbid PTSD/MDD can be viewed as the product of shared cognitive vulnerabilities. Treatment issues are now reviewed.

Justification for Targeting Depression in the Treatment of Comorbid PTSD/MDD

A range of empirically supported treatments for depression exist which include, cognitive therapy, behavioural activation, interpersonal therapy, social skills training, short-term psychodynamic psychotherapy, and non-directive supportive therapy (National Institute for Clinical Excellence (NICE), 2009). Effective treatments for depression often work within a cognitive behavioural therapy (CBT) framework and incorporate components such as cognitive restructuring to challenge negative cognitions pertaining to Beck's cognitive triad (i.e., the world: "the world is unfair", the self: "I am worthless", and the future: "the future is helpless"), and behavioural activation to enhance pleasure and mastery in day-to-day life. Empirically supported PTSD treatments include cognitive processing therapy (CPT), prolonged exposure (PE), and eye movement desensitisation and reprocessing (EMDR). Again, most PTSD treatments take a trauma focused CBT approach and aim to facilitate emotional processing of the trauma memory and cognitive restructuring of maladaptive cognitions. In line with principles suggested by emotional processing theory and social-cognitive theories of PTSD, therapies such as CPT and PE aim to alleviate PTSD symptoms by facilitating:

1) emotional engagement with, and the emotional processing of the trauma memory and, 2) corrective learning and cognitive restructuring of maladaptive beliefs or stuck points (Gonzalez-Prendes & Resko, 2012; Hembree & Foa, 2004; Resick, Monson, & Chard, 2006). That is, both CPT and PE emphasise cognitive restructuring *and* emotional engagement with the trauma memory as a mechanism of change.

Cognitive behavioural therapy has been empirically supported as an effective treatment for both PTSD and MDD (e.g., Butler, Chapman, Forman, & Beck, 2006; Hollon, Shelton, & Davis, 1993) and is recommended as the first-line treatment for PTSD (Australian Centre for Posttraumatic Mental Health, 2007; NICE, 2005) and MDD (NICE, 2009). However, not all clients benefit from CBT. For prolonged and imaginal exposure, non-response rates range from 25 to 60%, and dropout rates between 0 to 50% (Bradley, Greene, Russ, Dutra, & Westen, 2005; Resick, Nishith, Waver, Astin, & Feuer, 2002; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008). Given such response rates there is a need to better understand factors associated with poorer treatment outcomes.

The Influence of Depression on PTSD Treatment Outcomes

Whilst the evidence is not universal, several studies have demonstrated that MDD influences PTSD treatment outcomes (Taylor et al., 2001). In a sample of civilian survivors Bryant et al. (2003) found that those who dropped out of CBT (including exposure) had higher levels of baseline depression than treatment completers. McDonagh et al. (2005) observed the same in a sample of childhood sexual abuse victims. Stein, Dickstein, Schuster, Litz, and Resick (2012) compared treatment response trajectories in participants allocated to CPT, CPT components, or prolonged exposure. Non-responders were more likely to have a diagnosis of MDD and report

more severe baseline hyperarousal symptoms than non-responders. They concluded that those with comorbid MDD may need additional treatment.

Although the above findings suggest that MDD inhibits treatment efficacy, this has not always been observed with some studies finding MDD to be unrelated to treatment response or dropout (e.g., Aderka et al., 2011; Aderka, Gillihan, McLean, Foa, 2013; Ehlers et al., 1998). For example, Gillespie et al. (2002) found that comorbidity (including depression, alcohol abuse, and panic) was not associated with reduced treatment outcomes in PTSD sufferers. However, individuals with comorbid conditions required a greater number of treatment sessions. Further, Liverant, Suvak, Pineles, and Resick (2012) found that changes in PTSD and MDD symptoms during CPT and CPT treatment components occurred concurrently and that changes in one disorder did not influence changes in the other.

While it is tempting to conclude that one does not need to target MDD in the treatment of comorbid PTSD/MDD, the literature is in its infancy and ignores that in certain contexts MDD appears to impact outcome. Further, research to date has primarily used mixed samples of individuals with PTSD only and individuals with comorbid PTSD/MDD, failing to analyse outcomes in terms of comorbidity. The use of such populations may partially account for contradictory findings and reduces one's ability to determine if trauma-based treatments are sufficient when comorbidity is present. Thus, there is a need for future research to clearly determine the best way of treating comorbid PTSD/MDD. It should also be noted that there is a paucity of literature examining the relationship between PTSD and MDD symptoms *over the course* of treatment and we consequently have little understanding of how PTSD and MDD symptoms interact during treatment.

As not all individuals recover from PTSD, and as MDD may interfere with PTSD treatment outcome, there is a critical need to identify factors that affect treatment

efficacy and explore the specific mechanisms through which MDD inhibits optimal recovery from PTSD. Examination of such factors would enhance PTSD treatment efficacy. The following sections consider these areas and examine the literature relevant to factors that inhibit PTSD treatment outcomes in the context of MDD. I argue that there is merit in targeting MDD explicitly in the treatment of comorbid PTSD/MDD.

Emotional Processing Theory: Emotional Engagement and Treatment Outcome

Cognitive-behavioural conceptualisations of PTSD are arguably the most relevant models to consider in light of the questions at hand, and a clear path is evident in the evolution of theoretical accounts from emotional processing theory (Foa, Huppert, & Cahill, 2006; Foa & Kozak, 1986), dual representation theory (Brewin, Dalgleish, & Joseph, 1996), social-cognitive theories (Horowitz, 1976, 1986; Janoff-Bulman, 1985, 1989; McCann & Pearlman, 1990), and Ehlers and Clark's (2000) cognitive theory of PTSD.

Social-cognitive theories of PTSD explain how traumatic events conflict with existing schemas or beliefs that people hold about themselves and the world (Horowitz, 1976, 1986; Janoff-Bulman, 1985; 1989; McCann & Pearlman, 1990). Beliefs proposed to be altered by a trauma include beliefs that the world is fair and predictable, the world is meaningful, and the self is worthy (e.g., Epstein, 2003; Janoff-Bulman, 1989). Social-cognitive theories suggest that in order to reconcile information about the traumatic event with prior beliefs, people tend to do one or more of three things: assimilate, accommodate, or over-accommodate. Assimilation occurs when an individual alters information to match a prior belief (e.g., "*Because a bad thing happened to me, I must have been punished for something I did"*). Accommodation relates to altering beliefs to allow the incorporation of new information (e.g., "*Although I didn't use good judgment in that situation, most of the time I make good decisions"*). Over-accommodation relates

to altering one's beliefs but in an extreme manner in order to feel safer and more in control (e.g., "*No one can be trusted"*). If an individual is unable to reconcile the traumatic event with prior beliefs, intrusions and avoidance ensue.

From a social-cognitive framework the goal of PTSD treatment is to facilitate the accommodation of the trauma memory and to create balanced beliefs that take into account the reality of the trauma without those beliefs becoming extreme (i.e., over-accommodated). This is achieved through explicit cognitive restructuring as well as exposure to and engagement with the trauma memory. While social-cognitive theories suggest that cognitive restructuring is necessary for treatment change, social-cognitive theories also suggests that affective expression and engagement with the trauma memory is required for symptom alleviation (Resick, 2001). Social-cognitive models suggest that affective expression of trauma-related emotions is needed for recovery and emphasise the importance of connecting with natural emotions (e.g., fear, anger, shame, disgust, sadness) in order to allow such emotions to take their course and reduce (Resick & Schnicke, 1993).

Social-cognitive theories share similarities with emotional processing theory. When exploring the context and development of such theories it becomes apparent that these theories have built upon one another and share numerous commonalities (Dalgleigh, 2004; Shipherd, Street, & Resick, 2006). For instance, both emotional processing theory and social-cognitive theories suggest that emotional engagement with the trauma memory, along with changes in maladaptive cognition are central to treatment change. Primarily, their major difference lies in the emphasis given to particular mechanisms of change. While emotional processing theory and social-cognitive theories both emphasise cognitive change and emotional processing as mechanisms of change, emotional processing theory places a larger emphasis on

emotional processing, while social-cognitive theories place a larger emphasis on cognitive change.

While all these theories have made significant contributions to our current understanding of PTSD, emotional processing theory will be used as the framework for examining comorbid PTSD/MDD. I made such a selection as emotional processing theory is still clearly embedded in more recent cognitive models of PTSD, it overlaps greatly with other theories, and because Foa and colleagues have been explicit in proposing certain mechanisms (MDD included) that are thought to influence not only natural recovery following trauma, but also treatment outcomes.

Emotional processing theory (Foa & Kozak, 1986; Foa, Steketee, & Rothbaum, 1989) suggests that PTSD is the product of pathological fear structures that promote avoidance and escape behaviours. The fear structure contains information about (a) feared stimuli (e.g., a man with a knife), (b) verbal, physiological, and behavioural responses (e.g., heart palpitations), and (c) meaning elements (e.g., a man with a knife is dangerous and palpitations mean that I am scared). The fear structure is activated when something in a person's environment matches information represented within their fear structure (Cahill & Foa, 2007). This in turn produces cognitive, behavioural, and physiological anxiety reactions as well as promoting avoidance behaviour and aiding in the escape of danger (Rauch & Foa, 2006; Resick, Monson, & Rizvi, 2008). When the fear structure accurately reflects reality it can be considered an adaptive structure that promotes escape and avoidance behaviour in the presence of danger. However, PTSD-related fear structures are considered maladaptive as the associations amongst stimulus elements do not accurately reflect reality. Specifically, PTSD-related fear structures contain erroneous relationships between trauma reminders (which are essentially safe) and meaning elements (such as a sense of incompetence), and contain excessive response elements that are resistant to modification.

Emotional processing theory also proposes that pathological cognitions underlie PTSD. Two specific kinds of unhelpful cognitions are believed to be evident. First, the world is dangerous and second, one's self is incompetent (Foa & Rothbaum, 1998). Individuals with pathological fear structures may also overestimate the probability that the feared stimuli will cause physical (e.g., heart attack) or psychological harm (e.g., going crazy), and individuals may assume that unless escape or avoidance of the feared stimuli is achieved, the anxiety will endure forever (Foa & Riggs, 1993).

Emotional Engagement with the Trauma Memory

Emotional processing theory posits that recovery from PTSD is a product of the degree to which one engages in emotional processing of the trauma memory. Emotional processing is defined as the modification of fear structures in which maladaptive associations amongst feared stimuli, responses, and meaning elements are replaced with more adaptive associations (Foa & Kozak, 1986). Two processes are thought necessary for emotional processing. First, the fear structure must be activated and emotionally engaged with (Foa, Riggs, Massie, & Yarczower, 1995; Jaycox, Foa, & Morall, 1998; Pitman et al., 1996). Activation occurs when one comes into contact with stimuli that are inherent in their fear structure and are associated with danger. Second, information that is incompatible with elements in the fear structure needs to be incorporated to allow maladaptive elements to be replaced (Foa & Cahill, 2001; Foa, Huppert, & Cahill, 2006). Emotional processing is said to have occurred when the fear structure is activated during exposure and when this is accompanied by a decrease in fear to trauma-related stimuli after repeated exposure. To put it simply, in order to recover from PTSD, or in order to achieve optimal treatment outcomes, emotional processing theory suggests that one must emotionally engage with trauma-related memories, thoughts, and emotions in a manner that allows corrective learning to take place (Foa et al., 1995).

However, as will be outlined, the mere presence of emotional engagement is not sufficient for positive treatment outcomes. Rather, emotional processing theory suggests that in order for treatment to be effective an *optimal* level of engagement must be maintained whereby both too little (underengagement) and too much activation (overengagement) may in fact reduce PTSD treatment efficacy (Rauch & Foa, 2006).

Underengagement refers to when the fear structure is not sufficiently activated and relates to lower levels of emotional arousal or detachment from one's emotions. Underengagement is said to occur when an individual experiences low levels of anxiety whilst recounting the trauma memory and appears to be unable or unwilling to recall details and emotional responses associated with the trauma. Overengagement occurs when an individual's fear structure becomes so activated during exposure that the client cannot focus on or incorporate new information into the fear structure. Overengagement is characterised by immense levels of distress or anxiety that overwhelm one's ability to process information whilst recalling the trauma. This distress impairs one's ability to remember that they are in the present moment, that they are safe, and that the trauma is in the past. Thus, within emotional processing theory treatment outcomes are viewed to be a product of the degree to which a client successfully (or optimally) emotionally engages with trauma-related feelings, perceptions, memories, and thoughts (Foa & Kozak, 1986). Importantly, in order to achieve good treatment outcomes, a central tenet of the theory is that one must emotionally engage at an *optimal* level.

Empirical Findings Related to Emotional Engagement

Emotional engagement has been conceptualised as a sign of working through unresolved issues (Greenberg, 2002a, 2002b), as an indicator of emotion transformation through exposure (Foa & Kozak, 1986), and as a sign of access to maladaptive cognitions (Teasdale & Barnard, 1993). Whilst research has begun to illustrate the role

emotional engagement plays in the acquisition of positive treatment outcome in a broad range of psychopathologies (e.g., Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002; Jaycox, Foa, & Morral, 1998), methodological shortcomings in the emotional engagement field reduce our ability to draw meaningful conclusions from such studies, and have left a number of important theoretical and clinical questions unanswered. Empirical findings related to emotional engagement are now reviewed along with methodological limitations and areas for future research. I will argue that there is a need to explore emotional engagement in the treatment of comorbid PTSD/MDD using more rigorous methodologies.

Methodological limitations in past examinations of emotional engagement. Researchers have attempted to explore the impact of emotional engagement on PTSD treatment outcomes for a number of years. For example, Jaycox et al. (1998) examined the influence of emotional engagement and habituation on outcome during cognitive behavioural exposure therapy for PTSD. Emotional engagement was assessed via subjective units of distress scores (SUDS) whereby distress was rated on a scale from 0 to 100 (0 = calm and free from distress, 100 = most distressed). Jaycox et al. found that those who demonstrated high engagement (high arousal/distress) and high habituation were eight times more likely than those who demonstrated low engagement and low habituation to meet criteria for good posttreatment outcome. Further, Foa et al. (1995) explored the relationship between fear activation and treatment outcomes in the treatment of PTSD. Facial fear expressions coded from videotapes and SUDS were used as assessments of fear activation. They found that SUDS and PTSD symptoms were correlated with one another and found fear activation to predict outcome whereby those who demonstrated more severe PTSD prior to treatment and displayed more intense facial fear expressions during exposure benefitted more from treatment than those who had less severe PTSD and displayed lower fear expression.

Although such studies suggest that emotional engagement influences PTSD treatment outcomes, limitations exist in regards to the use of SUDS as an index of emotional engagement. First, as individuals provide SUDS during exposure tasks the utility of such scores is limited to providing a snapshot of engagement during exposure. In other words, SUDS do not indicate engagement over an entire treatment session or program, and must therefore be considered a discrete assessment of emotional engagement. Second, SUDS may obscure fluctuations or patterns in engagement. For example, while an individual may report elevated SUDS during an exposure task they may still underengage for the remainder of the session. Related to this, SUDS do not allow one to explicitly assess under-, over-, and optimal engagement separately and only provide an overarching assessment of high distress or overengagement. Finally, as SUDS are a subjective measure, SUDS may not be in line with more objective assessments of engagement and may potentially be biased by a client's ability to accurately reflect upon and report his or her own level of distress. This is a salient point as psychometric data for SUDS is lacking. Therefore, although the noted studies provide some initial insight into emotional engagement, the sole reliance on SUDS is a limitation and does not allow one to explicitly determine how under-, over-, and optimal engagement influence treatment outcome. In order to gain a better understanding of the relationship between emotional engagement and treatment outcome future research must assess emotional engagement with more objective and in-depth measures, and should endeavour to explore the reliability of SUDS.

The importance of emotional engagement extends to other psychopathologies with research suggesting that experiential avoidance or an unwillingness to experience feelings and thoughts is a core component of many disorders and is not restricted to PTSD (Beutler, Clarkin, & Bongar, 2000; Greenberg, & Pascual-Leone, 2006; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996; Poss, Greenberg, Goldman, & Korman,

2003). Stringer, Levitt, Berman, and Matthews (2010) explored the relationship between emotional disengagement during therapy and treatment outcome in a sample of treatment seeking university students with a range of different presenting issues. Emotional disengagement was operationalised as the presence of disengaged silences during therapy sessions. Stringer et al. coded 52 sessions for silences and found that the presence of disengaged silences (i.e., emotional disengagement) predicted higher depressive symptoms at posttreatment. However, alternative explanations exist as disengaged silences may not solely reflect emotional disengagement but may also capture related concepts such as poor working alliance, treatment resistance, and underlying anger and depression. Therefore, it may be these factors or the interaction of these factors that influence treatment outcome rather than engagement alone. This is a significant issue given that in the context of emotional engagement in PTSD treatment, we are concerned with emotional engagement with the trauma memory itself rather than engagement in therapy per se (e.g., alliance, resistance). The presence of alternative explanations again underlines the need to explore emotional engagement using clearer definitions of engagement, and through the use of more objective measurements of the construct. I now review studies that have attempted to explore emotional engagement more directly through the use of coding. However, as will be reviewed, methodological limtiations still reduce the validity of findings.

Missirlian, Toukmanian, Warwar, and Greenberg (2005) studied emotional arousal and client perceptual processing during early, middle, and late stages of experiential therapy for depression. Perceptual processing encompasses a variety of cognitive processes (i.e., cognitive reevaluation, differentiation, integration) that lead to the development of more complex understandings of experience (Toukmanian, 1992). Missirlian et al. found that mid-therapy emotional arousal predicted improvements in self-esteem. Further, mid-therapy arousal and perceptual processing predicted

reductions in depressive and psychopathological symptoms better than arousal or perceptual processing alone. They concluded that early and middle stage emotional arousal enhances the accessibility of emotions to cognitive processing such that arousal is reduced and reflective processing increased towards the end of therapy.

Similar to prior research that used SUDS as a key measure, there are several methodological issues associated with Missirlian et al.'s (2005) findings. Missirlian et al. used the Client Expressed Emotional Arousal Scale III—Revised[1] (CEAS; Warwar & Greenberg, 1999) to assess emotional arousal. Although the scale is frequently used and demonstrates sound psychometric properties, the manner in which scores were collected, summarised, and analysed is a potential criticism of the study. First, Missirlian et al. did not assess emotional arousal over a whole treatment program but rather assessed arousal during a selection of early (Session 2 and 3), middle (two middle sessions), and late (second to last and third to last sessions) sessions. Adding to this, sessions were not coded in their entirety but rather 'emotional episodes' were coded. An emotional episode is a segment of therapy in which a client specifically discusses an emotion in response to a situation (see Greenberg & Korman, 1993). By failing to code therapy sessions in their entirety, and by not coding the entire treatment program, patterns in engagement during treatment cannot be determined and valuable information is lost.

Second, whilst emotional episodes during the six selected sessions were coded minute-by-minute, modal emotional engagement scores were averaged across early, middle, and late sessions. That is, each time point was summarised with only one average, arousal score. While this simplifies data analysis (and leads to more simplistic interpretations of findings), mean arousal scores are an inexact measure that

[1] The CEAS provides a rating of emotional arousal based on coded videotapes of therapy sessions. Scores range from 1 to 7 whereby 7 indicates extreme arousal. When assessing emotional arousal during a therapy session raters rate each minute of a session, or each minute of a particular segment of a session. Each minute is provided with: (a) an emotional label, (b) a modal rating (i.e., overall level of arousal intensity within the minute) and/or (c) a peak rating (i.e., intensity of maximally aroused moment within the minute).

significantly obscure fluctuations in emotional engagement. Also, by using an average to summarise emotional engagement, engagement is treated as one all-encompassing construct, with specific states of under-, over-, and optimal engagement being overlooked. More precisely, by using averages to summarise engagement one is unable to determine if under- and overengagement reflect distinct or similar states, or if under- and overengagement have a different effect on outcome. Further, the use of averages does not allow one to determine how engagement operates during treatment sessions (i.e., do individuals either under- or overengagement during sessions, or do they fluctuate between both states). Therefore, while Missirlian et al. demonstrated a relationship between engagement and treatment outcome, the utilised methodology leaves one unable to determine how engagement, and more specifically, how *under*- and *over*engagement influence outcome.

The current literature is based on subjective and brief assessments of engagement (i.e., SUDS), has failed to code treatment sessions, or treatment programs in their entirety, and has used crude averages to summarise CEAS coding. Accordingly, we are unable to accurately understand the relationship between under-, over-, and optimal engagement and treatment outcome, nor able to stringently evaluate several key tenets of emotional processing theory. In addition to these limitations, it could also be argued that the cited research conceptualises the relationship between engagement and outcome too simplistically. While the above studies focus on a direct relationship between engagement and outcome (albeit with methodological flaws), more recent research has begun to illustrate the complexity of this relationship and suggests that the relationship between engagement and treatment outcomes is non-linear (Carryer & Greenberg, 2010; Coombs, Coleman, & Jones, 2002; Pascual-Leone & Greenberg, 2007). For instance, although some researchers have found engagement to relate to positive treatment outcomes in CBT, interpersonal therapy, and experiential therapy, others have found that

elevated levels of painful emotion may be associated with reduced treatment outcomes (Coombs et al., 2002) and emotion dysregulation (Greenberg, 2002b; Linehan, 1993). This is in line with studies that suggest that 'catharsis' alone is not an adequate explanation for therapeutic improvement and that emotional engagement has multiple facets that need to be assessed in order to best predict treatment outcome (Carryer & Greenberg, 2010).

Given the acceptance of a nonlinear relationship between general arousal and performance in other domains of functioning (i.e., Yerkes & Dodson, 1908), it is surprising that this principle has not been applied more readily to psychotherapies. Greenberg and colleagues (Goldman, & Greenberg, 2005; Pascual-Leone & Greenberg, 2007; Poss, et al., 2003) have begun to more intricately examine the impact of emotional engagement on experiential therapy where interpersonal problems were the target of treatment. Pascual-Leone and Greenberg found that distress reduction was associated with moving from states of high arousal and low meaning (i.e., no clear reference to internal experiences) to low arousal and high meaning (i.e., an orientation to, and awareness of internal experiences). They suggested that although high levels of arousal and engagement may be beneficial in the short-term, extended periods of high engagement may be detrimental as it produces mental fatigue that interferes with reflective processes that would usually lead to positive treatment outcomes. Consistent with such a proposition numerous theorists have posited that excessive emotional arousal (i.e., overengagement) can lead to dysregulation that can in turn interfere with an individual's ability to think coherently and reduce positive therapeutic outcomes (Greenberg, 2002a; 2002b; Linehan, 1993). However, while such research attempts to move away from a linear understanding of engagement, much of the referenced research still only assesses engagement during specific sessions rather than over a whole course of treatment, and does not separate the effects of under-, over- and optimal engagement.

Towards a more complete picture of emotional engagement. Carryer and Greenberg (2010) have addressed some of the aforementioned methodological limitations. The researchers examined how *varying levels* of emotional engagement, assessed by peak CEAS ratings, influenced treatment outcomes in a sample of participants with MDD who were treated with experiential therapy. Expressed emotional arousal (independently coded from session tapes) was assessed during participants' three highest arousal sessions (taken during mid- to late therapy). Unlike previous research Carryer and Greenberg did not rely on averaged arousal scores, rather they created a more sensitive way of summarising coding scores. First, each minute of a selected session was coded using the CEAS. Then, from such ratings a proportion score was created. Proportion scores reflected the proportion (or percentage) of the coded session spent in a highly aroused state (i.e., proportion of the session spent at a CEAS peak rating of 5 or greater). Carryer and Greenberg (2010) found that depressive symptom reduction was predicted by a non-linear pattern of expressed emotion in which optimal treatment gains were observed when clients experienced highly arousing emotions for 25% of their session. Deviations towards lower frequencies of high emotional arousal (i.e., underengagement) and deviations towards higher frequencies of high emotional arousal (i.e., overengagement) were associated with poorer treatment outcomes. The use of such a methodology allowed Carryer and Greenberg to expand on past research and predict an optimal level of engagement. Such findings highlight that the relationship between arousal and outcome is complex and that moderate levels of emotional arousal are required to produce optimal treatment outcomes.

Summary and future research. The reviewed literature provides some indication that both too much and too little emotional engagement is associated with reduced treatment outcomes. This is in line with emotional processing theory's proposition that effective treatment requires an optimal level of engagement, and that under- and

overengagement may predict reduced treatment efficacy. However, while findings suggest that emotional engagement influences treatment outcome, due to methodological issues this proposition remains tentative. There are significant gaps in the literature and several clinical and theoretical questions still remain. For instance (a) How do levels of under-, over-, and optimal engagement influence treatment outcome, and are the processes equally detrimental to PTSD and MDD outcome? (b) Are under- and overengagement similar or distinct states? (c) Do individuals show a tendency to either under- or overengage during treatment or do they show a tendency for both under- and overengagement? (d) Does the effect of under- and overengagement differ depending on treatment type? (e) How do peak and modal scores relate to one another, and what is the best means of summarising arousal? and (f) Do SUDS provide a valid estimate of emotional engagement?

The paucity of research on emotional engagement is especially evident when looking at anxiety disorders and comorbidity with most emotional engagement research confined to the experiential treatment of MDD. No study to date has explored the relationship between engagement and treatment outcome in the context of comorbid PTSD/MDD treatment or CPT. The scarcity of such research is especially poignant given emotion processing theory's propositions surrounding the importance of engagement during PTSD treatment. It must also be highlighted that the influence of comorbid MDD on the interplay between emotional engagement and outcome remains unknown. As MDD is associated with emotion dysregulation, flattening of affect, and cognitive difficulties it would be reasonable to suggest that MDD may inhibit emotional engagement during PTSD treatment. However, as stated, research is yet to test how MDD may influence emotional engagement and consequently treatment outcome.

Although coding treatment sessions and programs in their entirety is a laborious task, and although more discrete assessments of emotional arousal require more

complex statistical analysis, failure to do so may mask subtle effects and hinder one's ability to understand the mechanisms underlying treatment change. By using methodologically sound procedures that explore engagement in an in-depth manner previously unanswered questions can be explored. Answering such questions would advance our knowledge about the processes that occur during treatment and allow for better treatment conceptualisation and delivery. Further, by exploring engagement during the treatment of comorbid PTSD/MDD one may be better able to understand how MDD impedes recovery from PTSD and subsequently identify how to enhance treatment effectiveness. The influence of MDD on the treatment of comorbid PTSD/MDD will be explored in the following sections with evidence suggesting that MDD may impede engagement and optimal recovery from comorbid PTSD/MDD.

How May Depressive Symptoms Inhibit Optimal Emotional Engagement and Treatment Outcomes in Comorbid PTSD/MDD?

Emotional processing theory suggests that depressive symptoms may reduce treatment outcomes by promoting under- and overengagement with the trauma memory. In line with such a proposition, the following section outlines how various depressive features (i.e., numbing, rumination, maladaptive cognitions) may influence treatment efficacy and reduce treatment outcomes in those with PTSD. Initially this may seem contradictory (i.e., that MDD can lead to both under- and overengagement) however, it must be reiterated that MDD is a multifaceted disorder with numerous symptom types that can differ in severity. Accordingly, these numerous symptoms and varying severities may differentially affect how MDD influences engagement.

Depression and Emotional Numbing

Emotional numbing, a phenomenon inherent in the presentation of PTSD and MDD, reduces one's ability to experience and identify emotions and can be characterised as a mechanism that inhibits emotional engagement during exposure therapy (Jaycox & Foa, 1996; Zimering, Caddell, Fairbank, & Keane, 1993). Posttraumatic emotional numbing creates a barrier to the emotions experienced during the trauma and is maintained by its capacity to reduce distress. In line with this, individuals with PTSD often report feeling cut-off or numb from the world and their emotions (Feeny, Zoellner, Fitzgibbons, & Foa, 2000). Similarly, individuals with moderate MDD experience low mood and anhedonia while in severe cases of MDD there is a significant dulling of affect which is often accompanied by severe psychomotor retardation (Loas & Boyer, 1996; Loas, Salinas, Guelfi, & Samuel-Lajeunesse, 1992). For instance, Lemke, Puhl, and Winkler (1999) found that 75% of a depressed sample experienced anhedonia and 43% to 52% experienced psychomotor retardation. Consequently, an inability to fully experience affect, as seen in MDD, may lead to underengagement during trauma-based treatment and potentially inhibit treatment outcomes in those with comorbid PTSD/MDD.

Consistent with the notion that MDD-related emotional numbing may promote underengagement, research has found emotional numbing to influence PTSD and MDD treatment outcomes. McMakin et al. (2012) found that anhedonia predicted a longer and more severe course of MDD in a sample of youths receiving a new serotonin reuptake inhibitor or venlafazine, with or without CBT. They concluded that anhedonia was a robust predictor of time to remission and MDD-free days. Curry et al. (2006) found that adolescents with MDD who reported more melancholic symptoms (i.e., anhedonia, psychomotor retardation) benefited less from CBT interventions. In regards to PTSD, Taylor et al. (2001) demonstrated that partial responders to PTSD focused CBT tended

to have higher levels of pretreatment numbing and depressive symptoms relative to full responders. In line with such findings, McMillen, North, and Smith (2000) suggest that trauma victims with considerable numbing symptoms might require different interventions from those with considerable reexperiencing and hyperarousal symptoms. They suggest that interventions that explicitly address numbing symptoms, such as behavioural activation, might be of particular benefit to such individuals.

Related to emotional numbing, individuals with MDD often actively avoid negative emotional material (Kahn & Garrison, 2009). Salters-Pednealt, Tull, and Roemer (2004) posit that emotional concealment reduces ones' ability to engage with the environment, elevates distress, and inhibits the learning of non-threatening associations. Similarly, Baumeister, Bratslavsky, Muraven, and Tice (1998) suggest that any sort of self-regulation (i.e., concealment, suppression, avoidance) depletes mental resources as attempts to control emotions during one task reduces resources needed to perform subsequent tasks. Thus, emotional concealment or emotional numbing, as seen in MDD, may inhibit optimal treatment outcomes by interfering with cognitive processes that would otherwise be available for therapy.

Several lines of research support such a proposal. Richards and Gross (1999) found that university students asked to suppress their feelings whilst being shown a series of neutral or negative slides and information demonstrated impaired cued-recall and cued-recognition memory compared to those in the no-suppression condition. They concluded that successful emotion suppression may require an internal dialogue where one must constantly remind oneself to suppress, self-monitor their outward signs of emotion, and conduct evaluations on how well one is doing. This dialogue may then consume finite attentional resources that would otherwise be used to process one's surroundings and events. In a similar vein, using an MDD sample, Muraven, Tice, and Baumesiter (1998) found that individuals asked to restrict the outward experience of

emotion whilst watching an emotional film persevered for a shorter time on a handgrip task relative to no-regulation controls. They concluded that emotional concealment may be associated with quicker disengagement from distressing tasks in those with MDD. Thus, emotion concealment, as seen in those individuals with MDD, may inhibit treatment outcome by reducing cognitive resources available during therapy and by hastening disengagement during distressing tasks. However, this is yet to be tested.

Again related to emotional numbing, a reduced ability to recognise emotions has also been found to predict heightened levels of psychopathology and poorer treatment outcomes (Rufer et al., 2004). For example, in a study of 86 outpatients with MDD, alexithymia at baseline was associated with reduced remission and higher depression rates at 6-month follow-up (Viinamäki et al., 2002). Further, Sloan and Kring (2007) propose that lower emotional awareness is associated with psychological disorders such as anxiety, MDD, and personality disorders. As individuals with MDD demonstrate reduced emotional awareness and difficulties expressing emotion (Suveg, Southam-Gerow, Goodman, & Kendall, 2007; Zeman, Klimes-Dougan, Cassano, & Adrian, 2007) it is possible that such difficulties may again inhibit emotional engagement and consequently reduce treatment outcomes in those with comorbid PTSD/MDD.

To review, emotional numbing and the concealment of emotion may potentially reduce treatment outcomes by: 1) reducing cognitive resources, 2) by hastening disengagement from anxiety provoking tasks (i.e., exposure tasks), 3) inhibiting one's ability to recall and utilise the information presented during therapy, 4) reducing one's ability to engage with their memories during therapy, and 5) reducing emotional awareness. Thus, emotion concealment and a general difficulty experiencing and identifying emotions, as seen in MDD where numbing is a component of the depressive presentation, is likely to contribute to underengagement during PTSD treatment and

reduce outcomes. However, as current research is limited, the effects of emotional numbing on comorbid PTSD/MDD treatment outcome must be examined further.

Depression and Emotion Regulation

Emotion regulation is critical in the initiation and organisation of adaptive behaviours and reduces the likelihood of experiencing stressful levels of unpleasant emotions (Garnefski et al., 2002). Emotion dysregulation is associated with ruminative responses to distress, poor emotional awareness, and dysregulated anger and sadness expression across disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Research indicates that individuals with MDD exhibit difficulties regulating their emotions and utilise more maladaptive emotion regulation strategies than non-depressed individuals (Garnefski, Kraaij, & Spinhoven, 2001; Gotlib & Joormann, 2010). Specifically, the dysfunctional attention, memory, and interpretation processes associated with MDD have been found to promote the use of maladaptive emotion regulation strategies and reduce the use of more adaptive strategies (Joormann & D'Avanzato, 2010). As adaptive emotion regulation reduces stressful levels of emotions (i.e., under-, and overengagement), and as individuals with MDD experience a reduced ability to regulate emotion it is possible that MDD interferes with engagement during PTSD treatment. Specifically, reduced emotional regulation may make individuals with MDD more prone to under- or overengagement during PTSD treatment and therefore reduce outcomes for those with comorbid PTSD/MDD. However, this proposal is speculative as no study to date has examined how emotion dysregulation impacts engagement during PTSD treatment. Despite this, as discussed next, the specific types of emotion regulation strategies people with MDD use may shed light on the likely mechanisms through which this could influence the emotional processing of traumatic material.

Individuals with MDD use maladaptive emotion regulation strategies such as rumination, avoidance, and suppression (Garnefski et al., 2002; Nolen-Hoeksema, 2000). For example, although suppression alleviates the expression, and possibly the experience of negative emotions in the short-term, it contributes to the maintenance of emotional and physiological distress in the long-term. Research demonstrates that suppression can lead to hypersensitivity and increased accessibility to unwanted material, and concomitant emotional and physiological arousal (Cioffi & Holloway, 1993; Gross, 1998; Gross & Levenson, 1997; Wegner & Gold, 1995; Wenzlaff & Wegner, 2000). Therefore, in PTSD sufferers with MDD, such suppression may promote overengagement with the trauma memory and prevent the incorporation of new information into the fear structure. However, it may also be the case that individuals with MDD suppress so well that they exhibit underengagement by failing to emotionally engage with the trauma memory. Research is yet to test these hypotheses.

An effective way to regulate emotion is to redirect attention away from emotion-eliciting stimuli (Gotlib & Joormann, 2010). However, attention and eye gaze studies demonstrate that individuals with MDD have difficulties disengaging from negative material (Caseras, Garner, Bradley, & Mogg, 2007) and show biases towards depression relevant stimuli such as socially threatening or depression-related words (Bradley, Mogg, & Lee, 1997; Broomfield Davies, MacMahon, Ali, & Cross, 2007; Matthews, Ridgeway, & Williamson, 1996; Nunn, Mathews, & Trower, 1997). This is problematic as maintained attention on such negative material may maintain dysphoric mood in individuals with MDD as it enhances their tendency to mull over negative information, increases reactivity to that information, and elevates distress (Joormann & Gotlib, 2010). Along similar lines Brewin and colleagues (Brewin, Hunter, Carroll, & Tata, 1996; Brewin et al., 2010), drawing on work from the depression field (e.g., Dalgleish & Watts, 1990), proposed that MDD in the context of PTSD enhances access to

negative memories and reduces access to positive memories. This greater accessibility of negative memories further potentiates MDD, leading to a vicious cycle. Brewin et al. (1996) suggest that MDD may prolong unhelpful emotional processing (i.e., increased focus on negative material) and result in more chronic PTSD. Therefore, the tendency individuals with MDD have to dwell on negative information, or more precisely, their difficulty disengaging from negative material, may cause them to become preoccupied with negative content during therapy. This may consequently reduce attention to positive material and contribute to overengagement during PTSD treatment. Of course it may not necessarily be the accessibility of negative memories that further potentiates MDD, but rather the distress associated with such negative memories. In summary, problems disengaging from negative material and elevated levels of suppression and avoidance may frustrate emotional engagement in people with MDD and inhibit the optimal treatment of comorbid PTSD/MDD.

Depression and emotion reactivity. Associated with emotional regulation difficulties, MDD is also related to altered emotional reactivity. The emotion context-insensitivity hypothesis (ECI) posits that individuals with depression demonstrate a generalised flattening of emotional responding to both positive and negative-valence stimuli (Rottenberg, Kasch, Gross, & Gotlib, 2002). It is proposed that whilst reduced reactivity may allow one to avoid emotional disturbances in the short-term, in the long-term it contributes to greater depression severity, longer episodes of depression, and reduced psychosocial functioning (Rottenberg et al. 2002). The ECI hypothesis is well supported (see Bylsma, Morris, & Rottenberg, 2008). For instance, Rottenberg, Gross, and Gotlib (2005) exposed individuals who were currently depressed, formerly depressed, or never depressed to stimuli that elicited happy, sad, and neutral states and found that currently depressed individuals reported less sadness reactivity and less happiness experiences than the other participants. This is of relevance to the present

chapter as the reduced or flattened reactivity associated with MDD is likely to contribute to underengagement during the treatment of comorbid PTSD/MDD.

Depression and Maladaptive Cognitive Processes

Depression is associated with a range of maladaptive cognitive processes including persuasive negative appraisals and ruminative response styles (Haaga, Dyck, & Ernst, 1991). Such processes not only influence the onset and maintenance of MDD (Nolen-Hoeksema, 2000) but also influence treatment outcomes (Beevers, Wells, & Miller, 2007). Although the literature is scarce, research is also beginning to indicate that individuals with comorbid PTSD/MDD possess stronger negative cognitions than those with PTSD or MDD alone (Menne, 2005). Nixon et al. (2004) observed that individuals with comorbid PTSD/MDD demonstrated more maladaptive beliefs than those with PTSD or MDD alone. Using a cross sectional design, Gonzalo, Kleim, Donaldson, Moorey, and Ehlers (2012) examined attributional styles and post-trauma cognitions. Depressive attributions and post-trauma negative beliefs were found to independently predict MDD and PTSD severity. Further, those with PTSD alone endorsed fewer depressive attributions than those with comorbid PTSD/MDD or MDD. As MDD is associated with maladaptive cognitions and as research implicates negative cognitions as inhibiting good treatment outcomes in MDD and PTSD (Hamilton & Dobson, 2002), one would expect that the negative cognitions associated with MDD could impede recovery from comorbid PTSD/MDD. For instance, depressive cognitions may have an additive effect that enhances the potency of negative, trauma cognitions and consequently increases the difficulty of altering these unhelpful beliefs.

Foa and Kozak (1986) outline how negative cognitions may reduce emotional engagement and treatment outcomes. They suggest that for individuals with MDD, maladaptive beliefs such as self-perceptions of ineffectiveness or helplessness may

facilitate learning deficits (i.e., a reduced ability to incorporate corrective information within one's fear structure). As emotional processing depends on the integration of fear-relevant information, such deficits may reduce emotional engagement and the likelihood that corrective information is incorporated into the fear structure. Further, the tendency in MDD to attribute success to external sources and failure to internal causes may reduce self-efficacy regarding one's ability to cope with stress and strong emotion. This may consequently promote underengagement in trauma-focused therapy as an individual with MDD may believe that they cannot handle their emotions and must therefore inhibit their emotional expression.

Depressive rumination[2] may also influence treatment outcomes in those with comorbid PTSD/MDD. Posited by the response style theory of depression and supported by research, individuals who ruminate about the causes, consequences, and implications of their depressive symptoms are likely to become and remain depressed for longer (Just & Alloy, 1997; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991). Further, rumination is related to numerous cognitive correlates of depression such as reduced problem solving (Lyubomirsky & Nolen-Hoeksema, 1995; Watkins & Moulds, 2005), overgeneralised autobiographical memories (Watkins & Teasdale, 2004), and negative cognitions (Lavender & Watkins, 2004). Some models of depression conceptualise rumination as a problematic escape behaviour that is not dissimilar to overt forms of avoidance (Martell, Addis, & Jacobson, 2001). The behavioural activation model explicitly acknowledges the role of rumination and whilst it suggests that rumination is distinct from other more obvious forms of avoidance (e.g., social withdrawal), it posits that the function is the same. That is, that rumination

[2] Watkins (2008) differentiates between abstract-analytic and concrete-experiential rumination. Abstract-analytic rumination relates to the evaluation of the cause, meaning, and implications of self-experiences and is associated with increased negative global self-evaluations (Rimes & Watkins, 2005) and reduced emotional recovery from prior failure (Moberly & Watkins, 2006). Concrete-experiential rumination focuses on contextual and concrete details of self-experience and is associated with reduced emotional reactivity to stressors (Watkins, Moberly, & Moulds, 2008) and reduced depressive mood (Watkins, Baeyens, & Read, 2009). Abstract-analytic rumination can be considered maladaptive while concrete-experiential rumination is considered adaptive. This review focuses on the negative impact of abstract-analytic rumination.

operates to actively avoid engagement with the environment. In line with such a conceptualisation Moulds, Kandris, Starr, and Wong (2007) found rumination, avoidance, and depression to be significantly correlated in a non-clinical sample. Further, rumination and behavioural avoidance remained related when anxiety was controlled. By conceptualising rumination as an active form of avoidance, rumination is likely to inhibit the processing and elicitation of trauma memories and emotions, and depressed individuals with a ruminative response style are likely to underengage during PTSD treatment and show poorer outcomes.

Others however conceptualise rumination differently, suggesting that its effects extend beyond being a form of avoidance. It is well established that individuals with MDD exhibit cognitive deficits such as impaired memory and problems with attention set shifting (Levens, Muhtadie, & Gotlib, 2009; Paelecke-Habermann, Pohl, & Leplow, 2005). Recent findings also indicate that rumination further captures cognitive resources thereby preventing these resources from being used in other effortful tasks (e.g., Levens et al.). That is, when faced with complex tasks, individuals with MDD find it hard to stop ruminating and organise cognitive resources to maximise their performance. If MDD is associated with a reduced ability to manage cognitive resources, and if rumination indeed *drains* cognitive resources, depressive rumination may inhibit emotional engagement during therapy as the individual has fewer cognitive resources available to engage with the trauma memory.

Finally, Smith and Alloy (2009) provide yet another explanation as to how rumination impedes treatment outcome. Smith and Alloy suggest that depressive rumination inhibits one's ability to adaptively and fully experience negative emotions. From this perspective depressive rumination may again inhibit one's ability to fully experience trauma-related emotions and memories, consequently facilitating underengagement and reducing treatment outcomes during the treatment of comorbid

PTSD/MDD. The literature suggests that depressive rumination may inhibit engagement with the trauma memory as rumination reduces one's ability to adaptively experience negative emotions, reduces the availability and allocation of cognitive resources, and is akin to a form of avoidance. This would therefore impede outcomes in those with comorbid PTSD/MDD. However, as depressive rumination has not been explicitly examined as a barrier to engagement such predictions require exploration.

Depression and Overgeneralised Memories

As noted previously, people with MDD exhibit problems in the retrieval of specific memories and have a tendency to retrieve overgeneralised memories (Williams et al., 2007; Williams & Scott, 1988). Although not yet studied in samples of individuals with comorbid PTSD/MDD, studies of separate samples of PTSD and MDD sufferers have shown overgeneralised memory to be associated with the onset and maintenance of PTSD (Kleim, & Ehlers, 2008) and MDD (Brittlebank, Scott, Williams, & Ferrier, 1993; Summer, Griffith, & Mineka, 2010). Further, with specific relevance to the present chapter, the overgeneral recall of autobiographical memories has been found to influence treatment outcome (Harvey et al., 1998). For example, Brittlebank et al. (1993) found that the overgeneral recall of autobiographical memories predicted less complete recovery from MDD at seven-month follow-up assessment. Dalgleish, Spinks, Yiend, and Kuyken (2001) additionally found that the extent to which individuals retrieved overgeneral memories predicted delayed recovery from affective disorders.

The retrieval of overgeneralised memories may in part reflect a form of functional avoidance whereby the retrieval of general memories reduces the distress that the retrieval of *specific* memories would usually elicit (Williams et al., 2007). Dalgleish, Rolfe, Golden, Dunn, and Barnard (2008) explored the mechanisms underpinning overgeneralised memories where participants completed a standard autobiographical

memory test (AMT) and a reversed AMT (i.e., provide general memories rather than specific). In line with an affect regulation hypothesis, distress was associated with reduced recall specificity. Boritz et al. (2011) explored the relationship between autobiographical memory specificity and emotional arousal in brief emotion-focused and client-centred therapy for MDD. They found that the retrieval of more specific autobiographical memories was positively associated with peak emotional arousal. This suggests that retrieval of specific memories requires the evocation of visual and experiential imagery that consequently evokes deeper emotional arousal. They concluded that individuals with MDD may retrieve memories in a more general manner in an attempt to avoid deeper emotional experiences and that this may consequently inhibit levels of engagement during therapy. While this avoidance may be another maladaptive emotion regulation strategy discussed earlier, it is also possible that the underlying processing that leads to this retrieval style has implications for how trauma memories are retrieved and the level of engagement exhibited by a suffer with comorbid PTSD/MDD during this processing. More explicitly, this retrieval style may hinder processing and reduce engagement (i.e., underengagement) during PTSD treatment.

The contribution of impaired executive ability to autobiographical memory deficits has also been highlighted (see Dalgleish et al., 2007). From this perspective reduced executive control (i.e., reduced planning and monitoring abilities) and the subsequent creation of overgeneralised autobiographical memory may reduce the ability individuals with MDD have to incorporate corrective information into their fear structures, subsequently influencing treatment success. Overall, depression-related overgeneralised memories and reduced executive control may promote underengagement and reduce one's ability to incorporate corrective information during PTSD treatment, consequently hindering outcomes.

Summary

I have proposed that MDD symptoms may hinder optimal engagement during trauma-focused therapy and consequently reduce treatment outcomes in those with comorbid PTSD/MDD. As such, the practice of treating PTSD alone may not be the most effective means of treating comorbid PTSD/MDD as it may not always address the mutual relationship between the two disorders nor the detrimental effects depressive symptoms may have on treatment outcomes. Thus, I propose that the combined treatment of both disorders may be more effective for this patient group. The following section will now review potential treatment pathways for comorbid PTSD/MDD.

Potential Treatment Pathways for Comorbid PTSD/MDD

The reviewed literature suggests that a closer examination of the role of MDD symptoms on PTSD therapy outcomes is warranted. Given the high prevalence of PTSD and MDD comorbidity it is somewhat surprising that intervention research to target the two is lacking, especially considering adjunctive therapies for other PTSD comorbidities such as panic, and substance abuse have been examined (Falsetti, Resnick, & Davis, 2005; Najavits et al., 1998). The one study to date that has explicitly examined treating comorbid PTSD and MDD is described and potential ways of treating this comorbidity highlighted.

Nixon and Nearmy (2011) tested a combined treatment program for individuals with comorbid PTSD/MDD (n = 20). Individuals underwent six sessions of behavioural activation (BA) therapy for MDD followed by up to ten sessions of trauma-focused therapy. They found a decrease in PTSD (d = 0.47) and MDD (d = 0.36) severity between pre- and midtreatment, and a decrease in PTSD symptoms (d = 0.81) from mid- to posttreatment. Symptom reduction was maintained at 3-month follow-up (d =

1.49 and 0.75, respectively). Such findings point to the utility of using BA in the treatment of comorbid PTSD/MDD.

Other researchers have explored the value of using BA as a stand-alone treatment of PTSD (Jakupcak, Wagner, Paulson, Varra, & McFall, 2010; Wagner, Zatzick, Ghesquiere, & Jurkovich, 2007). As PTSD and MDD are both in part maintained by withdrawal and avoidance (Ehlers & Clark, 2000; Martell et al., 2001) researchers have justified using BA for the treatment of PTSD as BA aims to reverse such patterns of avoidance. Researchers have argued that by reducing avoidance, BA is similar to invivo exposure typically used for PTSD. However, small scale pilot and treatment studies have found BA to only have small to moderate effects on PTSD symptoms and often PTSD symptoms still remain within the moderate to severe range at posttreatment (Jackupcak et al., 2006; Mulick & Naugle, 2004). Interestingly, BA for PTSD has also been found to only have limited effects on MDD symptoms with studies failing to find significant reductions on self-reported depression scores at posttreatment and follow-up (Wagner et al., 2007) and some studies even reporting increases, albeit nonsignificant, in depression severity (Jakupcak et al., 2006).

Whilst comorbidity in PTSD is the rule rather than the exception, there is no clear clinical consensus based on empirical grounds as to whether one should treat the other disorder when it co-occurs with PTSD. The treatment of comorbid PTSD/MDD can be approached in a variety of ways. For instance, one could simply treat PTSD for as a long as needed to also resolve MDD. Or, as MDD is proposed to inhibit optimal treatment outcomes, one could develop a combined treatment that targets *both* PTSD and MDD symptoms. Such treatment avenues are now discussed.

Trauma-focused Therapy for Comorbid PTSD/MDD

The effects of trauma-focused CBT are not confined to PTSD symptoms, with trauma-focused therapy often leading to significant reductions in depressive symptoms (Foa et al., 1999a; Resick & Schnicke, 1992). For instance, Resick et al. (2008) conducted a dismantling study of CPT in which CPT, the cognitive component of CPT, and the written component of CPT were compared. The researchers found that at pretreatment 51% of treatment completers had an MDD diagnosis, whereas 16% continued to meet the diagnosis at posttreatment and only 13% at the 6-month follow-up. Similarly, Resick et al. (2002) reported that at pretreatment 53% of CPT participants met criteria for MDD compared to only 18% at follow-up. However, the question still remains as to whether a single focus on PTSD is the most effective means of treating comorbid PTSD/MDD. Although no study to date has explored whether a single focus on PTSD is superior to a treatment program that also focuses on MDD, this has been examined in non-PTSD comorbidities (Craske, et al., 2007; Schulte, Künzel, Pepping, & Schulte-Bahrenberg, 1992) and findings suggest that treatments focused on principal diagnoses may reduce comorbidity severity.

For example, Craske et al. (2007) allocated participants with panic disorder and another comorbid anxiety or mood disorders (n = 65) to either CBT that focused solely on the panic disorder, or CBT that focused on the panic disorder and the most severe comorbid condition. They found that both treatments led to reductions in panic symptoms (ES = 0.72) and a decline in the severity (ES = 0.63) and number of comorbid disorders (ES = 0.42). Further, panic disorder focused CBT produced greater improvements in the most severe baseline comorbid condition (ES = 0.11). The idea that one may not need to include extra treatment components for individuals with comorbid PTSD/MDD would further seem supported by dismantling studies that suggest that additional therapeutic components may not translate to better treatment outcomes (Foa

et al., 1999a; 2005). However, it should be noted that studies exploring the utility of a single treatment focus on comorbidity often report small effect sizes with some studies failing to demonstrate maintained treatment superiority at follow-up. Additionally, such studies have included mixed samples of PTSD and comorbid PTSD/MDD sufferers and have not separately examined outcomes.

Trauma-focused therapy may have positive effects on comorbidity for numerous reasons. For instance, individuals may apply core CBT strategies to comorbid symptoms, or such treatments may target and improve emotion processes that are shared across emotional disorders (i.e., negative affectivity, perceived lack of emotional control). Further, trauma-focused CBT may be effective in reducing MDD symptoms as the treatment shares much in common with typical MDD treatments. For instance, CPT and cognitive therapy (the latter used to treat MDD) both target maladaptive cognitions.

Evidence supporting the efficacy of trauma-focused therapy in reducing comorbid symptoms at face value suggests that PTSD treatment alone may be sufficient to treat comorbid PTSD/MDD. However, such a proposition is tentative as research to date has not specifically explored if a single focused PTSD treatment is *superior* to a treatment program that focuses both on PTSD and MDD. Further, as the above findings primarily relate to mixed samples of individuals with PTSD alone and those with comorbid PTSD/MDD, such results may not extend to samples that consist entirely of individuals with comorbid PTSD/MDD. It may be the case that when depression is significantly elevated (as is the case in comorbidity), trauma-focused therapy alone is not sufficient to reduce such symptoms.

Combined Treatment for Comorbid PTSD/MDD

Research has also explored the efficacy of using a combined treatment approach whereby a focus is placed on treating *both* PTSD and the comorbid condition in either an integrated or sequential manner. As stated, while research has rarely targeted MDD

in the treatment of comorbid PTSD/MDD, research has examined the comorbid relationship between PTSD and disorders other than MDD such as panic and substance abuse (Cloitre, Koenen, Cohen, & Han, 2002; Falsetti et al., 2005; Najavits et al., 1998). Current literature surrounding the combined treatment of PTSD and another comorbid condition suggests that combined treatments lead to significant reductions in PTSD and comorbid symptoms and suggest that there may be utility in targeting both PTSD and the comorbid condition (Cloitre et al., 2002; Falsetti et al., 2005; Najavits et al., 1998; Wald, Taylor, Chiri, & Sica, 2010). Further, some studies suggest that by treating the comorbid condition initially, trauma-focused therapy may be made more tolerable for clients (Cloitre et al.; Cook et al., 2006; Falsetti et al., 2005).

As an example, combined treatments have been developed for comorbid PTSD and panic. As panic attacks frequently co-occur with PTSD, and as panic symptomatology can reduce one's ability to tolerate exposure techniques, Falsetti, Resnick, Davis, and Gallagher (2001) developed multiple channel exposure therapy (M-CET). The treatment combines CPT, a well-established PTSD treatment, with panic control treatment, a panic disorder treatment. Falsetti et al. (2005) compared M-CET (n = 12) to a minimal attention control (n = 15) and found that participants in the M-CET condition reported a greater decrease in PTSD symptoms and the number of panic attacks than those in the control.

Further, seeking safety is a cognitive-behavioural treatment designed to teach and facilitate coping strategies to reduce PTSD *and* substance abuse symptoms. It has been studied within numerous contexts including a multisite controlled trial with homeless women veterans (Desai, Harpaz-Rotem, Najavits, & Rosenheck, 2008), two randomised control trials with low-income women and adolescent girls (Hien, Cohen, Miele, Litt, & Capstick, 2004; Najavits, Gallop, & Weiss, 2006), a controlled trial (Gatz et al., 2007), and eight uncontrolled pilot studies (e.g., Cook, Walser, Kane, Ruzek, & Woody, 2006;

Najavits, Weiss, Shaw, & Muenz, 1998; Zlotnick, Najavits, & Rohsenow, 2003). Prior studies have suggested that seeking safety is an acceptable treatment that produces superior effects on PTSD and substance use compared to treatment-as-usual control (Desai et al., 2008; Gatz et al., 2007; Najavits et al., 2006). However, contradictory findings exist. For instance, Hein et al. (2009) randomised 353 women to either seeking safety or an active comparison health education group. They reported clinically significant reductions in PTSD symptoms for both conditions but no reliable difference between conditions. Substance use outcomes did not differ between seeking safety and the control at posttreatment, and at follow-up substance use outcomes showed no significant change from baseline. Further, in their randomised controlled trial, Hein et al. (2004) compared seeking safety to an active treatment control (cognitive–behavioral relapse prevention), and treatment as usual (community control). While both seeking safety and the active control produced larger changes than treatment as usual, improvements were not significantly different between seeking safety and the active control. Contradictory findings highlight the difficulty associated with treating such a population, and the need to further explore the utility of combined treatments for PTSD when comorbidity exists.

Along with contradictory findings, methodological issues are also apparent. Current examinations of combined treatments for PTSD and comorbid conditions tend to compare combined treatments to no-treatment controls. There appears to be a near absence of studies that compare combined treatments to single focused treatments that target PTSD alone. Further, sample sizes remain relatively small consequently reducing statistical power and generalisability. As such, one is unable to determine if combined treatments that target PTSD and comorbidity produce superior outcomes compared to single treatments that focus on PTSD alone.

It should also be noted that while combined treatments may be used to target comorbid conditions, there has been a surge of research examining unified treatment protocols that target a variety of unipolar mood and anxiety disorders simultaneously (Erickson, Janeck, & Tallman, 2007; Moses & Barlow, 2006; Norton & Philipp, 2008; Wilamowska et al., 2010). Research suggests that anxiety and mood disorders may be more similar than previously acknowledged, therefore reducing the need for diagnostic-specific treatments (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). For instance, anxiety and mood disorders have common cognitive, behavioural, and emotional regulation processes that may serve as targets for therapeutic change (e.g., avoidance, emotional regulation, processing biases) (Brown & Barlow, 2009; Brown, Chorpita, & Barlow, 1998; Dalgleish & Watts, 1990; Liverant, Brown, Barlow, & Roemer, 2008). Further, single-diagnosis treatments for anxiety and mood disorders share common features such as cognitive restructuring, prevention of avoidance, and exposure procedures. In line with such similarities, some have argued that in an attempt to be parsimonious and pragmatic, unified or transdiagnostic approaches should be applied. Thus, it may not necessarily be that a combined or sequential treatment approach is required in the treatment of comorbid PTSD/MDD but that a unified treatment that targets common underlying factors and deficits inherent within this presentation is sufficient. However, there is currently contention surrounding the usefulness of transdiagnostic treatments (Mansell, Carey, & Tai, 2013), and although transdiagnostic treatments have been found to be effective for eating disorders and emotional disorders (e.g., Mansell, Harvey, Watkins, & Shafran, 2008; 2009), no study to date has explored the effectiveness of a transdiagnostic approach for PTSD when comorbidity is present. Adding to this, studies of transdiagonstic treatments seldom compare transdiagonstic treatments approaches to single-diagnosis treatments.

In sum, although some studies suggest that a single focus on PTSD is sufficient to treat PTSD when comorbidity is present, other studies highlight the merit of using combined or sequential treatment approaches that targets both PTSD and the comorbid disorder. As single focused PTSD treatments have not been consistently compared to sequential or combined treatments, one is unable to determine the best means of treating PTSD when comorbidity exists, and one is unable to determine the *superiority* of a single versus combined treatment approach. As MDD symptoms may interfere with treatment outcomes, conceptually a combined treatment approach appears beneficial. In line with the reviewed research, it would not be misguided to propose that if MDD was addressed prior to PTSD treatment the negative effect depressive symptoms have on engagement could be minimised and PTSD treatment outcomes enhanced. However, given the paucity of research surrounding combined and sequential treatment approaches this remains speculative.

Summary

This chapter has highlighted several critical areas in relation to comorbid PTSD/MDD. First, it was argued that PTSD/MDD comorbidity may be the product of shared vulnerabilities. Second, within an emotional processing framework the barriers to treatment outcomes were identified with specific discussion of the processes through which MDD may inhibit optimal levels of emotional engagement. Finally, avenues of treating comorbid PTSD/MDD were noted and the merit of a combined treatment approach highlighted.

A number of important research avenues have been suggested. Although theory and clinical observations support the proposition that MDD impedes emotional engagement and subsequently treatment outcomes, systematic and methodologically sound investigation is lacking. Consequently, there is a growing need for experimental

studies and treatment trials to examine the interplay between emotional engagement and treatment outcome in more depth. Researchers must extend beyond summarising coded scores with averages, must code engagement over an entire treatment program, and test the impact of under-, over-, and optimal engagement on outcomes separately.

This chapter further highlighted the paucity of research comparing single, focused treatments to combined or sequential treatments for PTSD when comorbidity is present. Research must test varying treatment approaches for comorbid PTSD/MDD and should examine if those with comorbid PTSD/MDD require combined or sequential treatments, or if they simply need *more* of the same therapy. Additionally, when examining sequential treatments, crossover designs should be used to determine the influence of treatment presentation order on treatment outcome. A better understanding of these processes would enhance the treatment of comorbid PTSD/MDD and contribute to our understanding of the relationship between PTSD and MDD.

Aims of Thesis

In this thesis I tested the utility of a combined treatment approach that targeted *both* PTSD and MDD symptoms in individuals with comorbid PTSD/MDD. A crossover design was used whereby three active treatment groups were compared. Individuals were randomly allocated to either CPT for PTSD, BA for MDD followed by CPT, or CPT followed by BA. I videorecorded all therapy sessions and coded each minute of each therapy sessions for emotional engagement using the Client Expressed Emotional Arousal Scale (CEAS) which is a standardised coding system. PTSD, MDD, and secondary outcome measures were assessed at pretreatment, mid-phase, posttreatment, and 6-month follow-up. PTSD and MDD measures were also taken every second session. The aims of the thesis were two-fold. First, I determined whether a combined treatment was necessary for individuals with comorbid PTSD/MDD or if a

single focus on PTSD was sufficient (i.e., within the framework of a randomised treatment trial). Second, I explored the mechanisms underlying treatment change and more specifically examined the relationship between emotional engagement and treatment outcomes (i.e., process research).

As noted, CPT and PE are both congruent with the tenets of emotional processing theory. Additionally, both treatments emphasise the importance of emotional engagement with the trauma memory and cognitive restructuring in the reduction of PTSD symptoms. In this thesis I targeted PTSD with CPT as CPT is considered a 'first line' treatment for PTSD, as CPT has been empirically tested in shorter forms (e.g., 12 vs. 6 sessions), and as CPT is amenable to being condensed into 10 sessions. I further selected CPT as it is distinct to BA and does not provide considerable overlap in terms of behavioural components. Further, I selected CPT as the use of imaginal exposure within specific treatment sessions allowed emotional engagement with the trauma memory to be assessed clearly and discretely. In regards to the treatment of comorbid depression, while numerous treatments for depression exist (e.g., cognitive therapy, interpersonal therapy, behavioural activation), I selected behavioural activation as its combination with CPT ensured that clients were not overloaded with cognitive therapy, and as BA lends itself well to a reduced treatment length (e.g., Lejuex, Hopko, & Hopko, 2001).

Aims Related to Treatment Trial

The treatment component of this thesis addressed two overarching research questions: 1) Does a combined treatment that targets both MDD and PTSD result in added benefits relative to PTSD treatment alone, and 2) If a combined treatment is useful, does it matter in which order therapy is delivered? The thesis therefore

represents the first study to explicitly compare the utility of a combined versus single treatment approach for individuals with comorbid PTSD/MDD.

Aims Related to Process Research

I also explored the influence of emotional engagement on treatment outcomes in those with comorbid PTSD/MDD. Specifically, I tested emotional processing theory's propositions surrounding emotional engagement, and examined if under-, over-, and optimal engagement predicted PTSD and MDD treatment outcomes. Therefore, this thesis addressed gaps in the literature and moved beyond answering *if* treatment worked to exploring the *processes* underlying treatment change. I also aimed to address methodological flaws inherent in past research by coding all undertaken therapy sessions, by summarising coded scores using proportions rather than averages, by exploring the relationship between peak and modal scores, and by examining the influence of under-, over-, and optimal engagement separately.

Hypotheses

Treatment outcome. Working from the perspective that MDD impedes treatment outcomes in those with comorbid PTSD/MDD the following predictions were made.

1. Participants in all conditions would demonstrate a significant reduction in PTSD and MDD symptoms.
2. If targeting depression is required for optimal treatment outcomes, then in comparison to CPT participants, BA/CPT and CPT/BA participants would show a greater reduction in PTSD and MDD symptoms.
3. If depressive symptoms reduce emotional engagement during PTSD treatment (and if initial BA sessions successfully target depressive symptoms such as low mood, rumination, and emotional numbing), BA/CPT participants would show a

greater reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms than CPT/BA participants.

Emotional engagement. Given that research explicitly exploring the effect of emotional engagement on comorbid PTSD/MDD treatment outcomes is limited, and as such research suffers a range of methodological issues, the broad goals of this component of the thesis are summarised (with specific predictions made were justifiable):

1. To determine whether under- and overengagement reflected similar, overlapping processes or distinct, separate processes.
2. To determine how under- and overengagement operate during treatment (e.g., do individual have a tendency to either under- or overengage during sessions, or do individuals demonstrate both under- and overengagement during sessions).
3. To determine the effects of under-, over-, and optimal engagement on PTSD and MDD treatment outcomes, and explore whether under- and overengagement had a different impact on outcome. In line with the cited research it was hypothesised that elevated levels of under- and overengagement would predict elevated PTSD and MDD symptoms, and that elevated levels of optimal engagement would predict reduced PTSD and MDD symptoms.
4. To examine whether the relationship between under-, over-, and optimal engagement and PTSD and MDD treatment outcome differed between CPT, BA/CPT, and CPT/BA conditions.
5. Determine the convergent and discriminant validity of self-reported subjective units of distress scores (SUDS) and objectively coded Client Expressed Emotional Arousal Scale (CEAS) scores. It was hypothesised that if convergent validity was adequate, SUDS and CEAS ratings of overengagement would be significantly correlated with one another, as these scales should be tapping into

the same construct of elevated emotional engagement. Additionally, it was hypothesised that if discriminant validity was adequate that SUDS and CEAS ratings of underengagement would only demonstrate a small or weak (i.e., $r <$ .10) correlation with one another, as these measures should tap into different constructs.

Chapter 2: Method

Participants

Participants were recruited throughout the Adelaide area through referrals from local hospitals, victim support centres, police departments, mental health centres, universities, and community therapists. Participants were included if they were over 16 years of age and had been directly or indirectly exposed to a trauma and met criteria for PTSD and MDD at the time of initial assessment. Participants were required to be a least one-month post trauma (no upper limits) and if on medication, were on a stable dose (typically for a 4-week period prior to assessment). Participants with current substance abuse were included if they agreed to minimise (or desist) usage during the period of treatment. Participants were allowed to continue in other psychotherapeutic intervention as long as it was not specifically related to the treatment of PTSD (this consisted of self-help groups or occasional attendance with another professional). Exclusion criteria included illiteracy, current uncontrolled psychosis, severe traumatic brain injury, active suicidality, and current substance or alcohol dependence[3] that clinically required primary attention. Individuals were further excluded if they were at significant risk of harm (i.e., in an abusive relationship).

Following telephone screening, potential participants were invited to take part in a pretreatment assessment at which point they discussed and signed informed consent. This study was conducted in compliance with the Southern Adelaide Clinical Human Research Ethics Committee and the Women's & Children's Health Network Human Research Ethics Committee. The study was registered with the Australian New Zealand Clinical Trials Registry (Trial ID: 12611000541909). Treatment occurred in three locations in the community: Flinders University, Yarrow Place Rape Crisis Service, and Victim Support Service.

[3] It should be emphasised that substance and alcohol dependence was only deemed an exclusion criteria if the dependence clearly needed to be treated first (e.g., if the client was at significant risk of withdrawal).

A total of 294 individuals made contact to enter the trial (see Figure 2.1). Following contact, 39 individuals were initially excluded due to not wanting ongoing treatment or not wishing to participate in the trial, 34 were excluded due to requesting phone counselling or being unable to attend weekly therapy sessions, 5 were allocated to a non-trial therapist, and 3 could not be re-contacted. Thus a total of 213 individuals were assessed for eligibility by an assessor who was blind to group assignment. The most common reason for exclusion (n = 163) included not meeting criteria for MDD diagnosis (n = 25), already being engaged in trauma-focused therapy (n = 25), current and significant substance or alcohol dependence (n = 17), and still being at risk of revictimisation (n = 16). One participant failed to complete the initial assessment. Of the 50 participants randomised into the study, one participant from BA/CPT was withdrawn from the study, by design, due to changes in medication. Therefore, the intent-to-treat (ITT) sample[4] included 49 participants.

Demographic and clinical sample and trauma characteristics are shown in Table 2.1. For the assessment of PTSD and initial treatment focus, participants identified their worse trauma (i.e., index event). There were no significant differences between treatment conditions in the ITT sample on type of index trauma, time since trauma, or other aspects of trauma history. Average length of time since index trauma was 4.68 years (SD = 6.95), with a range of 2 months to 27 years. As noted in Table 2.1, in terms of the index trauma focussed on during treatment, 8.2% identified child sexual abuse, 2% identified child physical abuse, 53.1% identified adult sexual assault, 18.4% identified adult physical assault, 10.2% identified armed hold-up or home invasion, and 8.2% identified traumatic loss.

[4] The ITT sample included all randomised participants irrespective of the amount of treatment received.

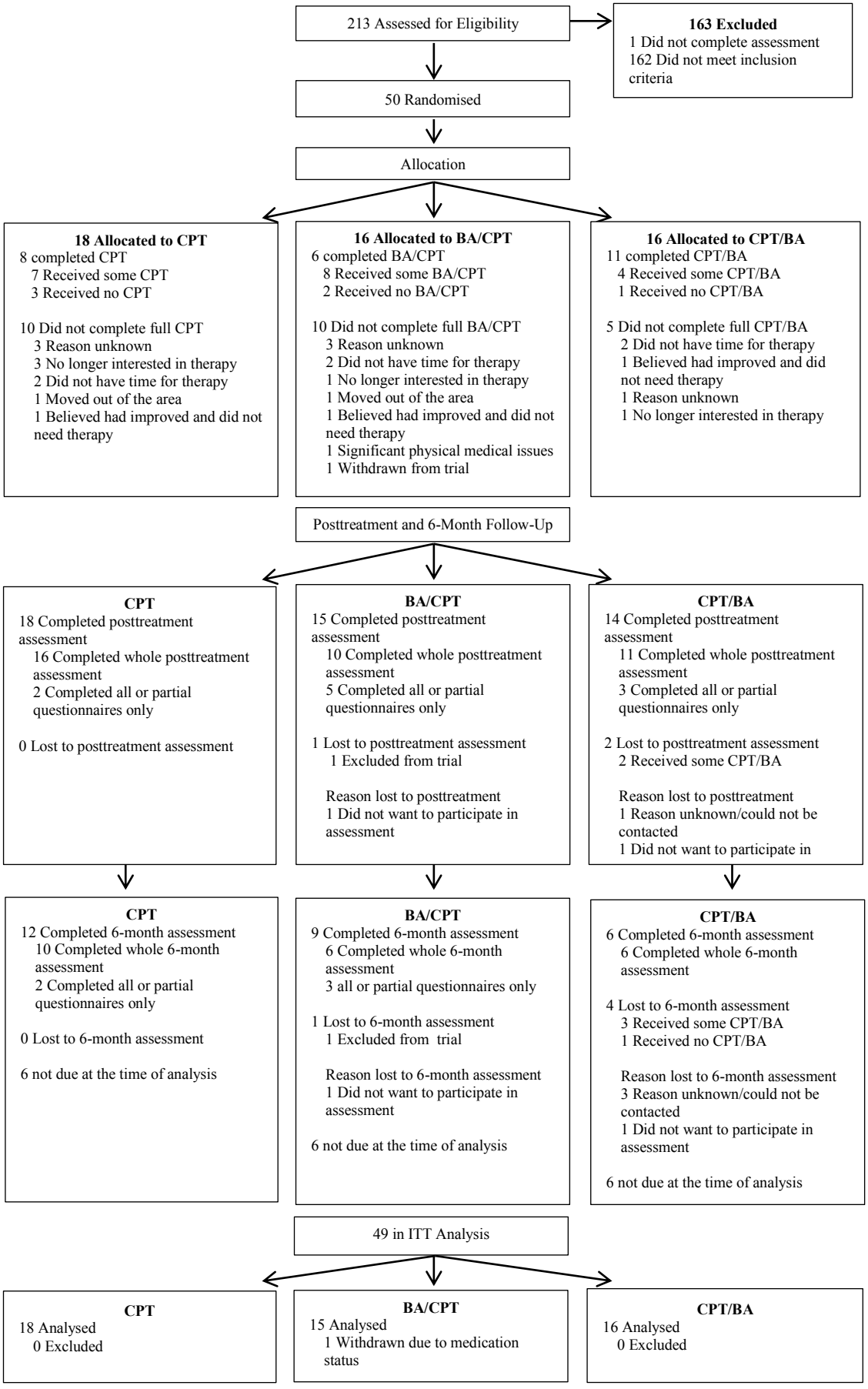

Figure 2.1. Flow chart of progression through study protocol. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; ITT = Intent-to-treat.

Table 2.1

Participant Demographic and Baseline Characteristics for Intent-to-Treat Sample

Characteristics Mean (SD) or number (%)	CPT (*n* = 18)	BA/CPT (*n* = 15)	CPT/BA (*n* = 16)	Test	*p*	*ES for* φ
Age years, mean (SD)	31.44 (12.51)	34.20 (13.97)	32.44 (11.84)	$F = 0.19$	.83	0.09[a]
Female	17 (94.4%)	13 (86.7%)	16 (100%)	$\chi^2 = 2.41$	.30	0.22[b]
Caucasian ethnicity	16 (88.9%)	13 (86.7%)	15 (93.8%)	$\chi^2 = 10.09$	.26	0.45
Total years of education, mean (SD)	13.39 (3.39)	12.70 (1.49)	13.93 (2.70)	$F = 0.82$	.78	0.45
Currently employed	10 (55.6%)	8 (53.3%)	10 (62.5%)	$\chi^2 = 0.30$	.86	0.08
Income						
Less than $10,000	2 (11.1%)	4 (26.7%)	1 (6.3%)			
$10,001 – 30,000	4 (22.2%)	4 (26.7%)	4 (25.0%)			
$30,001 – 50,000	6 (33.3%)	3 (20%)	7 (43.8%)	$\chi^2 = 5.74$	.68	0.34
$50,001 – 70,000	4 (22.2%)	4 (26.7%)	3 (18.8%)			
$70,001 – 90,000	0 (0%)	0 (0%)	0 (0%)			
More than $90,000	2 (11.1%)	0 (0%)	1 (6.3%)			
Marital status						
Single	11 (61.1%)	7 (46.7%)	7 (43.8%)			
Married/cohabiting	4 (22.2%)	5 (33.3%)	3 (18.8%)	$\chi^2 = 3.00$	.81	0.25
Divorced/separated/widower	1 (5.6%)	1 (6.7%)	2 (12.5%)			
Relationship not living together	2 (11.1%)	2 (13.3%)	4 (25%)			

Characteristics Mean (SD) or number (%)	CPT (*n* = 18)	BA/CPT (*n* = 15)	CPT/BA (*n* = 16)	Test	*p*	*ES for* φ
Index Trauma						
Adult physical assault	6 (33.3%)	2 (13.3%)	1 (6.3%)			
Adult sexual assault	9 (50%)	7 (46.7%)	10 (62.5%)			
Child physical abuse	1 (5.6%)	0 (0%)	0 (0%)	χ^2 =11.10	.35	0.48
Child sexual abuse	1 (5.6%)	1 (6.7%)	2 (12.5%)			
Armed hold up or home invasion	1 (5.6%)	2 (13.3%)	3 (12.5%)			
Traumatic loss	0 (0%)	3 (20%)	1 (6.3%)			
Years since index trauma, mean, (SD)	4.06 (4.87)	4.47 (7.78)	5.57 (8.38)	F = 0.20	.82	0.09
Current comorbid diagnoses						
Additional mood disorder	3 (16.7%)	3 (20%)	7 (43.8%)	χ^2 = 3.66	.16	0.27
Additional anxiety disorder	11 (61.1%)	6 (40%)	11 (68.8%)	χ^2 = 2.80	.25	0.24
Substance abuse or dependence	5 (27.8%)	2 (13.3%)	5 (31.3%)	χ^2 = 1.51	.47	0.18
Total number of disorders, mean, (SD)	2.89 (1.50)	2.33 (1.50)	2.88 (0.96)	F = 0.87	.43	0.19
Currently on psychotropic medication	4 (22.2%)	6 (40%)	9 (56.3%)	χ^2 = 4.15	.13	0.29
Order of PTSD/MDD Onset						
PTSD onset prior to MDD	5 (27.8%)	2 (13.3%)	2 (12.5%)			
MDD onset prior to PTSD	11 (61.1%)	7 (46.7%)	7 (43.8%)	χ^2 = 5.53	.24	0.34
PTSD and MDD onset concurrently	2 (11.1%)	6 (40%)	7 (43.8%)			

Characteristics Mean (SD) or number (%)	CPT (*n* = 18)	BA/CPT (*n* = 15)	CPT/BA (*n* = 16)	Test	*p*	*ES for* φ
Baseline Assessment Measures (SD)						
CAPS	72.50 (23.09)	81.60 (14.79)	84.94 (14.39)	*F* = 2.14	.13	0.31
PCL	56.06 (11.28)	60.20 (7.49)	61.12 (10.35)	*F* = 1.26	.29	0.23
DASS-D	21.11 (10.68)	21.20 (9.91)	26.12 (9.76)	*F* = 1.29	.28	0.24
PTCI	130.67 (36.62)	139.27 (39.87)	152.94 (26.36)	*F* = 1.76	.18	0.28
RRS	51.94 (13.97)	57.07 (13.71)	61.88 (11.28)	*F* = 2.45	.10	0.33
SRRS	1159.17 (462.47)	1310.33 (439.30)	1546.25 (313.98)	*F* = 3.76	.03	0.40
EN	25.67 (7.93)	28.47 (4.67)	28.62 (5.15)	*F* = 1.23	.30	0.23
TAS	55.28 (12.41)	62.47 (9.80)	60.94 (10.67)	*F* = 1.97	.15	0.29

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; ES = Effect Size; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

[a.] Effect size conventions for Cohen's *f*: 0.1 = small, 0.25 = medium, and 0.4 = large.

[b.] Effect size conventions for φ: 0.1 = small, 0.3 = medium, and 0.5 = large.

With regards to trauma history more generally, participants were exposed to multiple traumas with the following rates of lifetime endorsement of sexual abuse in childhood (34.7%), physical abuse in childhood (28.6%), adult sexual assault (73.5%), adult physical assault (59.2%), domestic violence (26.5%), armed hold-up (10.2%), traumatic loss (10.2%), and motor vehicle accident (MVA) (20.4%). 30.6% of participants had experienced a single trauma (of these, 20.4% had experienced a single adult sexual assault, 8.2% a single adult physical assault, and 2% a single sexual assault in childhood). One participant from CPT, BA/CPT, and CPT/BA each had occasional sessions with another professional that did not meet study exclusion criteria for such contact (i.e., contact was not trauma related).

As per inclusion criteria, all participants met the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association, 1994) criteria for PTSD and MDD at initial interview. At pretreatment, using the MINI International Neuropsychiatric Interview (MINI) modules for MDD, dysthymia, manic episodes, panic disorder, agoraphobia, social phobia, obsessive compulsive disorder (OCD), alcohol dependence, substance dependence, and generalised anxiety, there were no significant differences between treatment conditions on these disorders or total number of disorders. Additional comorbidity was common with 82.7% of participants having an additional diagnosis aside from PTSD and MDD. The most common comorbid conditions were as follows: major depression (100%), panic disorder (31.4%), social phobia (25.5%), alcohol dependence (25.5%), and dythymia (25.5%).

Of participants in the ITT sample, six participants did not return for the first therapy session. Dropout was defined as completing anything less than the 12 sessions. Across groups, 55.6% (n = 10) of CPT, 60% (n = 9) of BA/CPT, and 31.2% (n = 5) of CPT/BA dropped out after pretreatment assessment or received some therapy. All

dropout occurred prior to Session 6 with no participant dropping out after Session 6. The overall dropout rate was 49% (n = 24) with 25 participants completing treatment.

In regards to demographic and trauma history, completers endorsed a greater number of years of education than non-completers. There were no other significant differences in treatment status based on age, ethnicity, employment status, income, marital status, index trauma, trauma history, or order of PTSD/MDD onset (see Appendix A for completer and non-completer descriptive statistics, inferential statistics, and effect sizes).

Instruments

Interviews

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). The CAPS is an interviewer-administered diagnostic assessment of PTSD that demonstrates excellent psychometric properties (Weathers, Ruscio, & Keane, 1999). For each symptom, a clinician rates symptom frequency and intensity on a scale ranging from 0 to 4. Items rated with a frequency of one or higher and an intensity of two or higher are deemed diagnosable symptoms (Blake et al.). Symptom severity is determined by adding frequency and intensity ratings. Scores range from 0 to 136. PTSD diagnosis on the CAPS was based on meeting the symptom criteria as defined by the DSM-IV as well as having a minimum severity score of 45. The CAPS was administered at pretreatment, posttreatment, and 6-month follow-up.

MINI International Neuropsychiatric Interview (MINI; Sheehan et al., 1997). The MINI is a diagnostic interview that investigates the presence of 17 DSM-IV Axis 1 disorders. The MINI assesses the existence, severity, and disability associated with current disorders. Psychometric properties are excellent (Sheehan et al.). Inter-rater reliability of the interview is good with kappa coefficients ranging from .88 to 1.0

(Sheehan et al.). Test-retest reliability is between .79 and .93. The entire MINI was administered at pretreatment and 6-month follow-up and the MDD module administered at posttreatment.

Trauma interview. A brief, semi-structured interview was conducted at pretreatment to gain relevant information about participants. The interview covered the following areas: demographic information, information about the trauma, social support, treatment history, and involvement in the criminal justice system.

Interrater reliability of structure interview. Individual and group meetings were held throughout the project to discuss diagnostic conceptualisations and to reconcile conflicting diagnostic decisions. Assessments were discussed in detail with a senior project staff member (Reg Nixon). A random sample of 24 tapes was selected for evaluation of interrater reliability for the CAPS. Categorical diagnostic analyses revealed that the kappa coefficient for overall PTSD diagnosis was .92 with 92% agreement. The correlation between interviewer and rater total CAPS, PTSD severity scores was .98 ($p < .001$). Kappa values and percentages of agreement for the three PTSD symptoms clusters were: re-experiencing ($\kappa = .92$, 96% agreement), avoidance ($\kappa = .75$, 92% agreement), and hyperarousal ($\kappa = .83$, 93% agreement). A random sample of 24 tapes was selected for evaluation of diagnostic reliability of the MINI. Kappa values for diagnoses ranged from .86 to 1.00, except for generalised anxiety ($\kappa = .68$, 93.3%). Kappa value for MDD diagnosis was .88 with 95.8% agreement.

Self Report Scales

All self-report scales were completed at pre-, post-, and 6-month follow-up assessments. Additional assessments were also collected during treatment. First, a condensed battery of self-report scales was administered at mid-phase (i.e., treatment phase cross-over point). Mid-phase assessment included the following scales:

Posttraumatic Stress Disorder Check List (PCL), Depression Anxiety and Stress Scale (DASS), Posttraumatic Cognitions Inventory (PTCI), Emotional Numbing Questionnaire (EN), Twenty-Item Toronto Alexithymia Scale (TAS), Ruminative Response Scale of the Response Style Questionnaire (RRS), and Stress-Reactive Rumination Scale (SRRS). The timing of the mid-phase assessment differed between conditions. While the mid-phase assessment occurred at the end of Session 5 for the CPT condition, the mid-phase assessment was provided at treatment cross-over points for the BA/CPT and CPT/BA conditions. That is, BA/CPT participants completed the mid-phase assessment at the end of Session 5 just prior to the initiation of CPT, and CPT/BA participants completed the assessment at the end of Session 10 just prior to the initiation of BA. Mid-phase assessments were provided at such points to allow investigation of treatment sequencing and to provide conceptual information regarding the process of treatment change. Second, PCL and DASS scales were completed every second session.

Posttraumatic Stress Disorder Check List (PCL; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). The 17-item PTSD self-report rating scale is an assessment of PTSD severity that produces three subscale scores that correspond to DSM-IV PTSD symptoms clusters B, C, and D as well as a total symptomatology score. Each item is rated on a 5-point scale ranging from 1 (*Not at all*) to 5 (*Extremely*). The measure has sound psychometric properties. Weathers, Litz, Herman, Huska, and Keane (1993) found PCL scores to have a coefficient alpha of .97 and test-retest reliability to be .96. Further, convergent validity with the CAPS is .92 (Blanchard et al.).

Depression Anxiety and Stress Scale (DASS-21; Lovidbond & Lovibond, 1995). The DASS-21 is a short-form version of the 42-item DASS and assesses depression, anxiety, and stress. Each construct is assessed with seven statements with ratings ranging from 0 (Did not apply to me at all) to 3 (Applied to me very much, or

most of the time). The scale has good construct validity (Henry & Crawford, 2005) and high internal consistency (.87 - .94) (Lovibond & Lovibond). The depression subscale of the DASS (i.e., DASS-D) is reported throughout as a primary treatment outcome.

Posttraumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tolin, & Orsillo, 1999b). The PTCI is a 36-item, self-report measure that assesses trauma-related thoughts and beliefs. The PTCI has three main factors: negative cognitions about one's self, negative cognitions about the world, and self blame. Participants are asked to rate how much they agree with each item on a scale from 1 (Totally disagree) to 7 (Totally agree). Total scores range from 36 to 252. Internal consistency across the total scale (.97) and the three subscales (self: .97; world: .88; blame: .86) is acceptable and convergent validity is good (Foa et al.).

Cognitive Emotion Regulation Questionnaire – Short (CERQ; Garnefski & Kraaij, 2006; Garnefski, Kraaij, & Spinhoven, 2001). The CERQ contains nine distinct scales that identify cognitive emotion regulation strategies (i.e., self blame, other blame, acceptance, refocus on planning, refocus positive, rumination, positive reappraisal, putting into perspective, and catastrophising). Each scale consists of two items that are rated from 1 (*Almost never*) to 5 (*Almost always*), with each subscale score ranging between 2 and 10. The scale has previously demonstrated reasonable internal reliability with Cronbach's alphas between .68 (other-blame) and .83 (rumination) (Garnefski et al., 2006). However, discriminant and convergent validity is still to be determined.

Although the CERQ was collected at pre-, post-, and 6-month follow-up, CERQ scores were not included in final analyses as the clinical utility and reliability of the scale became questionable (non-imputed, descriptive statistics are presented in Appendix B). As the study progressed I began to question the clinical utility of the scale and explicitly questioned the ability of two items to properly assess each subscale,

especially given that subscales reflected relatively large and encompassing constructs. Adding to this, participants often had difficulty determining if CERQ items related to their traumatic experiences or experiences more generally. For instance, while some participants completed the questionnaire in reference to general stress, others completed the questionnaire in reference to their index trauma.

To further assess the validity of the CERQ I correlated pretreatment CERQ subscales with other conceptually, related questionnaires used in this thesis (i.e., pretreatment PTCI, RRS, and SRRS questionnaires). See Appendix C for correlations. If the CERQ was a valid measure it was predicted that:

1. Positive CERQ subscales (i.e., acceptance, positive reappraisal, positive refocus, and refocus on planning subscales) would be negatively correlated with PTCI total, negative world, negative self, and self blame scores, and negatively correlated with scores on the Ruminative Response Scale of the Response Style Questionnaire (RRS), and the Stress-Reactive Rumination Scale (SRRS).
2. Scores on the CERQ self blame subscale would be positively correlated with scores on the PTCI self blame subscale, and scores on the RRR, and SRRS.
3. Scores on the CERQ catastrophising subscale would be positively correlated with scores on the PTCI negative self, negative world, and self blame subscales, and positively correlated with scores on the RRS and SRRS.

Prediction 1 was refuted as positive CERQ subscales were not significantly correlated with the PTCI, RRS, and SRRS. Prediction 2 was also refuted. Although the CERQ self blame and PTCI self blame subscales were significantly correlated, this was not the case for the RRS and SRRS. Furthermore, Prediction 3 was refuted. While a significant correlation was found for the CERQ catastrophising subscale and the PTCI negative world subscale, this was not the case for other PTCI subscales, and the RRS

and SRRS. Surprisingly, the CERQ catastrophising subscale was more strongly related to measures of emotional numbing. In sum, as CERQ subscales did not correlate well with other expected measures, and due to the questionable clinical utility of the scale, the CERQ was not used in final analyses nor reported any further.

Emotional Numbing. Emotional numbing was assessed in two ways. First, the Acute Stress Disorder Scale (Bryant, Moulds, & Guthrie, 2000) and the CAPS contain items that assess emotional numbing. These items were extracted and used as an assessment of emotional numbing (labelled Emotional Numbing Questionnaire (EN), see Appendix D). The seven extracted items included: *"Have you felt emotionally numb or had trouble experiencing feelings like love or happiness?", "Did you feel in a daze?", "Did things around you feel unreal or dreamlike?", "Have you been less interested in activities you used to enjoy?"* Each item was rated on a scale from 1 (*Not at all*) to 5 (*Very much*) with total scores ranging between 5 and 35. Questions were applied using a one week time-frame.

Emotional numbing was also assessed with the Twenty-Item Toronto Alexithymia Scale (TAS; Bagby, Parker, & Taylor, 1994). This scale assesses a person's difficulty identifying feelings (e.g., "*I have feelings that I can't quite identify*"), difficulty describing feelings (e.g., "*I find it hard to describe how I feel about people*"), and externally oriented thinking (e.g., "*Being in touch with emotions is essential*"). The TAS is the most widely used and validated self-report measure of alexithymia. The scale has demonstrated good internal consistency and test-retest reliability (Bagby, Taylor, Parker, & Loiselle, 1990). Further, the scale has good factorial (Parker, Taylor, & Bagby, 2003) and concurrent validity (Bagby, Parker, Taylor, 1994; Bagby et al., 1990).

Ruminative Response Scale of the Response Style Questionnaire (RRS; Treynor, Gonzalez, Nolen-Hoeksema, 2003). As posited in Chapter 1, rumination is suggested to reduce emotional engagement and consequently inhibit treatment outcomes

in individuals with comorbid PTSD/MDD. Thus, rumination was considered a variable of interest and measured within the current study. The RRS assesses one's tendency to ruminate in response to depressed mood. The scale contains 22 items that examine responses to dysphoric mood that are focused on the self (e.g., "*Think why do I always react this way?*"), focused on symptoms (e.g., "*Think about how hard it is to concentration*"), or focused on possible consequences and causes of low mood (e.g., "*Think I won't be able to do my job/work because I feel so badly*"). Items are answered on a scale ranging from 1 (*Almost never*) to 4 (*Almost always*). A total score is calculated by summing all 22 items. The scale further contains two subscales labelled brooding and reflection. Each subscale consists of 5 items. The RRS has good internal consistency (.90) (Treynor et al., 2003). Coefficient alphas for the brooding and reflection subscales are .77 and .72, respectively (Treynor et al.) and previous studies report acceptable convergent and predictive validity (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema & Morrow, 1991).

Stress-Reactive Rumination Scale (SRRS; Robinson & Alloy, 2003). The SRSS contains subscales that assess three cognitive tendencies: tendency to use a negative inferential style, tendency to focus on hopeless cognitions, and tendency to focus on active coping strategies. Participants are presented with 25 statements (e.g., "*Think about how the stressful event is all your fault*") and are asked to indicate how frequently they do or think about each item in response to their trauma. Each statement is rated on a scale ranging from 0 (*Not focus on this at all)* to 100 (*Focus on this to a great extent*). The SRRS has good internal consistency (α = .89), good test-retest reliability 1-month later (r = .71), and good convergent validity with the RRS (r = .69) (Robinson & Alloy).

Timeline for PTSD and MDD development. At pretreatment participants were presented with a timeline to determine the order of PTSD and MDD onset. Participants

were presented with a timeline representing their life and were asked to indicate when they first noticed the onset of depression. They were then asked when they viewed PTSD as occurring. Depression onset was determined prior to determining PTSD onset to reduce the likelihood that participants would be biased to index their depression onset to the time of their trauma.

Trauma History Questionnaire. Adapted from Resick et al. (2002), the trauma history questionnaire is a 24-item self-report questionnaire that assesses the frequency and severity of past traumatic experiences. Participants are asked to rate each traumatic experience (e.g., military combat) in terms of how often they occurred (e.g., 0 – *Never* to 6 – *More than 20 different times*) and how distressing they found the worst incident (e.g., 1 – *Minimally distressing* to 10 – *Extremely distressing*). This questionnaire was administered at pretreatment assessment.

Working Alliance Inventory (WAI; Tracey & Kokotovic, 1989). The WAI is a 12-item self-report scale that assesses therapist relationship variables. The client version asks participants to indicate how often they feel a certain way about their therapist (e.g., "*My therapist and I agree about the things I will need to do in therapy to help improve my situation*"). The therapist version asks the therapist to indicate how often they feel a certain way about their client (e.g., "*The client and I agree about the steps to be taken to improve his/her situation*"). Items are scored on a scale ranging from 1 (*Never*) to 7 (*Always*). The WAI was administered at Session 2 and posttreatment. The WAI demonstrates good internal consistency ranging from .87 to .93, and good predictive validity (Horvath & Greenberg, 1989).

Credibility/Expectancy Questionnaire (CEQ; Devilly & Borkovec, 2000). The CEQ is a 6-item questionnaire that assesses perceived treatment expectancy and credibility. The credibility factor is based on responses to three items measuring how logical the therapy seems, how successful one thinks the therapy will be in reducing

symptoms, and how confident one would be in recommending the therapy to a friend with similar symptoms. The expectancy factor is based on three responses reflecting how much one thinks they will improve by the end of treatment, the percentage they believe their symptoms will reduce by the end of treatment, and how much they feel they will improve by the end of treatment. The questionnaire was administered at Session 2 and participants' last treatment session (or posttreatment if the participant did not complete treatment). The scale demonstrates high internal consistency with a standardised α of between .84 and .85 for the whole scale (Devilly & Borkovec). Test-retest reliability over one week is also good at .82 for expectancy and .75 for credibility (Devilly & Borkovec).

Process Measures

The following measures were taken to address my aim of testing emotional processing theory's proposition that emotional engagement influences treatment outcome. Specifically, such measures were assessed to explore the prediction that elevated levels of under- and overengagement would predict elevated PTSD and MDD symptoms, and that elevated levels of optimal engagement would predict reduced PTSD and MDD symptoms. Emotional engagement was assessed using two measures: Subjective units of distress scores (SUDS) and the Client Expressed Emotional Arousal Scale-III (CEAS-III; Warwar & Greenberg, 1999).

Subjective Units of Distress Scores (SUDS)

SUDS were used to assess subjective distress during exposure tasks conducted in session (i.e., participants' reading of their trauma accounts). SUDS range between 0 to 100 whereby 0 indicates feeling calm and free from distress and 100 indicates experiencing maximal distress. A SUDS score of 100 was anchored as the most distress

imaginable (i.e., overengagement). At the start, middle, and end of trauma accounts participants were asked to indicate their SUDS levels. For each participant a mean SUDS score was calculated for each in-session trauma account task. Scores served as a measure of emotional engagement whereby higher scores indicate higher engagement.[5] It should be noted that the use of SUDS is a modification to the typical CPT protocol.

As stated in Chapter 1, SUDS provide a snapshot of engagement and do not provide a discrete indication of emotional engagement over a whole treatment session. Consequently, SUDS were used in the present study not to provide an overall measure of emotional engagement, but to provide an assessment of engagement during trauma-account exposure tasks. SUDS were also collected to allow the accuracy and validity of such scores to be explored (i.e., How do SUDS correlated with more in-depth objective CEAS measures of engagement?).

Client Expressed Emotional Arousal Scale-III (CEAS; Warwar & Greenberg, 1999)

The CEAS provides ratings of emotional expression that are based on coded videotape recordings of therapy sessions (see Appendix E for manual). The CEAS is a 7-point scale that assesses the quality and intensity of a participant's emotions. Ratings are based on the degree of arousal in a participant's voice and body, and the degree to which they restrict the expression of emotion. An "emotional voice" is defined as an "an overflow of emotion into a speech pattern" and is identified by attending to the following features: accentuation patterns, regularity of pace, terminal contours, and whether there has been a disruption in speech patterns (Warwar & Greenberg, 1999).

[5] Craske et al. (2008) posit that habituation is not predictive of therapeutic outcome or new learning but rather that *inhibitory* learning is central to the extinction of negative trauma associations. As such, it should be emphasised that in this thesis I conceptualised SUDS as a measure of emotional engagement and SUDS habituation was not the primary outcome of interest.

Accentuation patterns refer to the emphasis patterns in a sentence. Regularity of pace refers to the extent to which pace varies within a particular utterance. Terminal contours refer to increases or descents in pitch. Finally, the disruption of speech patterns relates to the extent to which speech is disrupted by emotional overflow. More explicitly, an emotional voice is considered present when accentuation patterns are irregular, when pace is uneven, and when terminal contours are unexpected.

Coding based on the CEAS is separated into two parts. First, the participant's primary expressed emotion is identified and labelled. Ratings on expressed arousal can fit into one of 15 categories identified to be relevant to psychotherapy sessions (Warwar & Greenberg, 1999): pain/hurt, sadness, hopelessness/helplessness, loneliness, anger/resentment, contempt/disgust, fear/anxiety, love, joy/excitement, content/calm/relief, shame/guilt, pride/self-confidence, surprise/shock, anger/sadness and pride/anger. A 16th category labelled 'unspecified bad' feeling was added to the emotional categories to avoid losing information about emotionally aroused moments that did not specifically fit into existing categories or were deemed ambiguous.

Second, the overall level of intensity (i.e., modal), as well as the peak level of intensity of the primary emotion is rated such that every coded segment is given a modal and peak arousal score. Ratings are based on a 7-point scale whereby higher scores indicate higher arousal intensities (i.e., 1 - *Client does not express emotions. Voice or gestures do not disclose any emotional arousal*; 4 - *Arousal is moderate in voice and body. Emotional voice is present; ordinary speech patterns are moderately disrupted by emotional overflow as represented by changes in accentuation patterns, unevenness of pace, changes in pitch. Although there is some freedom from control and restraints, arousal may still be somewhat restricted*; 7 - *Arousal is extremely intense and full in voice and body. Usual speech patterns are completely disrupted by emotional overflow. Arousal appears uncontrollable and enduring. There is a falling apart*

quality). A modal score reflects a participant's overall, average level of expressed arousal for a coded segment. A peak score reflects the highest level of arousal present during a segment. That is, whilst modal scores provide an overview of emotional engagement during an entire coded segment, peak scores reflect momentary elevations in arousal. A person's baseline emotional arousal is also considered during coding.

In this thesis all therapy sessions were coded in their entirety using the CEAS (Warwar & Greenberg, 1999). Four raters were used: I rated all sessions while the additional three raters (undergraduate research assistants) rated a randomly selected 90 sessions to provide an assessment of inter-rater reliability (results of which are reported below). My ratings were used for the final analyses. Raters viewed videotapes of each session one minute at a time. Each minute was given a peak and modal rating of arousal and a corresponding emotion category label.

In line with past research (Carryer and Greenberg, 2010), and given that a rating of seven on the CEAS (i.e., the highest level of intensity) occurred too rarely to allow for any meaningful analysis, in this thesis a score of five and above was conceptualised as reflecting overengagement. Underengagement was defined as a rating of two or below as the scale's definition of a rating of two (i.e., "*Very little arousal in voice, body or words, any arousal is almost completely restricted and there is no disruption of normal speech patterns*") is in line with the theoretical conceptualisation of underengagement. Finally, a score of three and four was conceptualised as optimal engagement. The use of such ratings is supported by Carryer and Greenberg (2010) and Missirlian et al. (2005) who proposed a rating of four to be an ideal level of arousal and scores of five and above to indicate high arousal.

Once every minute of a session was coded, proportion scores (i.e., percentage frequency) were then used to summarise raw, *modal* emotional arousal scores for each session. For each session the proportion of time a participant spent in an overengaged,

underengaged, and optimally engaged state was calculated separately (see Appendix F for an example). Proportion scores were calculated by dividing the number of minutes of a session spent underengaged, overengaged, and optimally engaged by the total number of minutes for that session, and multiplying this by 100. For example, if a participant underengaged for 20 minutes, overengaged for 10 minutes, and optimally engaged for 30 minutes of a 60-minute session, this translated to a participant who would have been considered to have underengaged for 33.33% of the session, overengaged for 16.67% of the session, and been at an optimal level of engagement for 50% of the session.

To recap, each session was summarised with three proportion scores: one score reflected the proportion of time spent underengaged, one score reflected the proportion of time spent overengaged, and one score reflected the proportion of time spent optimally engaged. An underengagement proportion score reflected the proportion of a treatment session spent at a CEAS modal rating of two or below, an overengagement proportion score reflected the proportion of a treatment session spent at a CEAS modal rating of five and above, and an optimal engagement proportion score reflected the proportion of a treatment session spent at a CEAS modal rating of three and four. It should be noted that emotional labels, or the type of emotion displayed, was not analysed as the aims of this thesis related to the *intensity* of emotional engagement, not the type of emotion per se.

Justification for CEAS coding and analysis. As noted in Chapter 1, using averages to summarise modal or peak scores may smooth out or conceal arousal fluctuations during a single treatment session or over a treatment program. Further, the use of average scores does not allow under-, over-, and optimal engagement to be analysed separately. In order to overcome the shortcomings of using averages I summarised modal arousal ratings using proportions scores (i.e., the proportion of a

session spent over-, under-, or optimally engaged). The use of proportion scores allows for a more discrete or sensitive measure of arousal to be gained, and allows one to determine the effects of under-, over-, and optimal emotional engagement on PTSD and MDD treatment outcomes separately. Further, the use of proportion scores allows one to determine whether under- and overengagement have a different impact on outcome.

Proportion scores were calculated based on modal ratings rather than peak ratings. Conceptually, modal ratings illustrate a participants' overall level of arousal rather than momentary elevations or fluctuations in arousal. For instance, whilst a peak score may reflect a few seconds of elevated arousal during a coded segment, a modal score provides an overview of arousal for a whole coded segment. Modal scores were conceptually selected as they were believed to be more representative of a participant's general level of emotional arousal during a coded segment, and consequently more representative of engagement during a treatment session.

To extend beyond a conceptual justification and to be confident in the validity of modal scores, I explored the relationship between modal and peak CEAS ratings. Descriptive statistics are presented in Table 2.2. Raw modal and peak ratings were significantly correlated with one another, $r = .79$, $p < .001$. Further, absolute agreement measured by interclass correlation coefficient was .85. This suggests that raw modal and peak scores were relatively consistent with one another and captured similar levels of arousal. Additionally, the large intraclass correlation coefficient suggests that the peak level of engagement observed during a rated segment was often sustained for the majority of that rated segment.

I also correlated proportions of over-, under-, and optimal engagement calculated based on raw peak ratings, with proportions of over-, under-, and optimal engagement calculated based on modal ratings. Proportion scores of underengagement based on peak ratings were significantly correlated with proportion scores of underengagement based

on modal ratings, $r = .84$ $p < .001$. Proportions scores of overengagement assessed by peak ratings were also significantly correlated with proportions scores of overengagement assessed by modal ratings, $r = .88$, $p < .001$. Finally, proportion scores of optimal engagement based on peak and modal ratings were also significantly correlated, $r = .75$, $p < .001$. Whilst proportions of over-, under-, and optimal engagement assessed by peak and modal scores were related to one another, examination of descriptive statistics unsurprisingly demonstrated that proportions of underengagement were smaller when based on peak ratings as compared to modal ratings. Further, proportions of overengagement were larger when based on peak ratings as compared to modal ratings. This suggests that the use of peak scores may underestimate proportions of underengagement and overestimate proportions of overengagement.

Table 2.2

Descriptive Statistics for the Client Expressed Emotional Arousal Scale: Peak and Modal Ratings

Measure	*M*	*SD*
Raw peak scores	3.97	0.74
Raw modal scores	3.69	0.76
Proportion of underengagement assessed by peak scores	2.68%	8.53%
Proportion of overengagement assessed by peak scores	18.48%	19.52%
Proportion of optimal engagement assessed by peak scores	78.84%	19.86%
Proportion of underengagement assessed by modal scores	6.27%	14.27%
Proportion of overengagement assessed by modal scores	10.75%	15.53%
Proportion of optimal engagement assessed by modal scores	82.97%	18.90%

As peak and modal scores were strongly correlated with one another and provided a similar assessment of engagement, my results suggest that the selection of modal or

peak scores may relate more strongly to the aims of the undertaken study, or how one conceptualises engagement. As emotional processing theory emphasises the detrimental impact of underengagement it was imperative that the measure selected for this thesis did not mask proportions of underengagement. Thus, as peak scores were likely to conceal or minimise proportions of underengagement, modal scores were selected as they were more sensitive to levels underengagement and less likely to mask proportions of underengagement. Additionally, as I was more interested in participants' general level of engagement during treatment sessions, rather than momentary elevations in engagement, modal scores were deemed an appropriate unit of measurement.

Interrater reliability was assessed based on a randomly selected 90 sessions (5327 data points). Interrater reliability, measured by intraclass coefficient, was .80 [range: .79, .81]. This is in line with previous studies that have reported interrater reliability scores ranging from .75 to .81 (Auszra, Greenberg, & Hermann, 2013; Missirlian et al., 2005).

Therapists and Training

Therapists were six women and three males. Four therapists (including myself) were currently undertaking postdoctoral clinical psychology training. One therapist was Associate Professor Reg Nixon who had advanced CPT training. The remaining four therapists had degrees in social work and were regular ongoing staff at Yarrow Place and Victim Support Service. As the lead researcher I conducted the majority of therapy (n = 43) and all pretreatment assessments.

Yarrow Place and Victim Support Service staff were provided with training in the following manner. After therapists read treatment manuals, they undertook a one-day training workshop for behavioural activation (BA) and a two-day workshop for CPT conducted by Reg Nixon (RN). Therapists additionally watched clinical training tapes.

Training was followed by weekly 1-hr supervision with the expert trainer (RN) or myself (i.e., the primary investigator) to ensure treatment adherence and competence.

Postdoctoral clinical students (including myself) completed training in the following way. Once manuals were read an online CPT training course was undertaken.[6] Following this, BA and CPT training and clinical tapes were viewed and discussed in detail. While undertaking training students attended supervision with RN to gain further clinical experience and continued to attend ongoing weekly supervision sessions following training. All therapy sessions were videotaped and were closely supervised by RN and myself to maintain treatment adherence and ensure therapist competence. Due to funding and time constraints, the videos were not formally coded for adherence or competence.

Design and Treatment Overview

Procedure

In line with the crossover design of the study participants were randomly allocated to CPT, BA/CPT, or CPT/BA. Participants were allocated through block randomisation whereby a researcher independent of the study possessed the randomisation sheet. Participants were allocated to a treatment condition following pretreatment assessment to insure that the pretreatment assessor was unaware of group allocation. Treatment was conducted in three sites throughout Adelaide, South Australia: Flinders University, Yarrow Place, a sexual assault counselling service, and Victim Support Service, a not-for-profit support service for individuals who have experienced a crime. Research was conducted at Yarrow Place and Victim Support Service as pre-existing research links existed with such services and to enhance recruitment and potential sample size.

[6] The CPT training course can be accessed at https://cpt.musc.edu/. The web-based CPT course is a reputable and good quality form of training that teaches all components of CPT.

Treatment was scheduled to be completed after 12 to 15 therapy sessions depending on condition allocation. Treatment in the BA/CPT and CPT/BA conditions consisted of 15 weekly sessions either 60 or 90 minutes in length. Irrespective of sequence, participants completed five BA sessions and 10 CPT sessions. All BA sessions were 60 minutes. As CPT is usually conducted in 12 hour-long sessions and as participants in the current study received 10 sessions of CPT, content delivery of CPT was slightly altered. The initial six CPT sessions were 60 minutes long while Sessions 7 through 10 were 90 minutes. This therefore equated to the traditional 12 hours of CPT over 10 sessions. Participants in the CPT only condition received a minimum of 12 sessions. All sessions were 60 minutes in length and followed the traditional 12-session CPT protocol. Following completion of the 12-session protocol participants in the CPT condition were offered up to three additional sessions based on clinical need, and with a view of matching the 15 session protocols available to CPT/BA and BA/CPT participants.

Following a brief telephone screening, potential participants who met eligibility criteria were scheduled to complete a pretreatment assessment. Following informed consent, the pretreatment assessment, comprised of diagnostic interviews and self-report measures, was undertaken. Random allocation to CPT, BA/CPT, or CPT/BA occurred after participants were assessed. This allowed the investigator, assessor, and therapist to remain blind to condition at pretreatment assessment. Therapy was then initiated. Participants completed PCL and DASS questionnaires every second session. Participants further completed a mid-phase questionnaire package. Posttreatment assessments were completed two weeks after treatment had ceased (or if a participant stopped treatment two weeks after treatment would have ended) and again 6-months after treatment ended. Posttreatment and 6-month follow-up assessments were completed by assessors unaware of treatment condition. All attempts were made to

collect as much posttreatment and 6-month follow-up data as possible. Therefore, some participants did not complete the whole posttreatment or follow-up assessment battery but only completed questionnaires, or in some cases, only the PCL and DASS. The proportion of data collected is reported throughout results. Data were collected between October 2010 and January 2014.

Treatment

Cognitive Processing Therapy (CPT). CPT followed the manual written by Resick, Monson, and Chard (2007). Therapy was conducted over 12 to 15 weekly sessions. CPT is a structured and manualised treatment that allows clients to develop skills necessary to identify and challenge dysfunctional trauma related beliefs. Session 1 is a psychoeducation session that introduces the concepts of PTSD and CPT. Clients are provided with an overview of PTSD and the treatment rationale. During Session 1 clients are given the homework task ('practice assignment') to write an impact statement about the personal meaning of the index trauma. During Session 2 the impact statement is read and stuck points (i.e., problem areas in thinking, also described as negative automatic thoughts) identified. In this session clients are taught to identify the connection between events, thoughts, and feelings. During Session 3 clients are further taught to identify and label their thoughts and feelings and taught to identify the relationship between events, thoughts, and emotions. Clients are further assigned the task of writing a detailed account of their index trauma. During Sessions 4 and 5 clients generate and read their trauma account to the therapist. The primary aim of the trauma account is to enhance emotional processing and to allow therapist and client to identify stuck points or cognitive distortions. Through Socratic questioning the therapist begins to question self-blame and other distorted thoughts. This is continued during Session 6. Thus, Sessions 1 through 6 allow clients to process the trauma and aim to teach clients

to identify problematic cognitions and challenge trauma-related thoughts. While writing about a second trauma may occur following Session 5, subsequent sessions focus on teaching clients to challenge and change unhelpful beliefs surrounding the meaning and implications of the trauma.

The remaining sessions teach clients cognitive therapy skills and focus on a range of beliefs (described as 'themes') that have been disrupted by the trauma. During Sessions 6 and 7 clients use worksheets to develop and practice using more balanced self-statements. They are taught to challenge single unhelpful beliefs and then identify problematic patterns of thinking. Sessions 8 through 12 ask clients to focus on one theme each week (i.e., safety, trust, power/control, esteem, intimacy) and correct overgeneralised beliefs related to each theme. At the end of Session 11 clients are assigned to write another impact statement that reflects their current beliefs and this revised statement is used in Session 12 to evaluate treatment gains. During Session 12 clients also receive information on relapse prevention.

Although the core therapy protocol consisted of 12 sessions, up to three additional sessions were offered. These additional sessions were offered to increase the clinical validity of the protocol and to target any residual PTSD or MDD symptoms that were present. This is routine practice in a community setting and is supported by studies that suggest that clients require a variable or extended number of sessions in order to achieve optimal treatment gains (Chard. 2005; Galovski, Blain, Mott, Elwood, Houle, 2012). Glavoski et al. state that additional CPT sessions are warranted if PTSD and MDD symptoms have not resolved by the end of the standard 12-session protocol.

Behavioural Activation then Cognitive Processing Therapy (BA/CPT). A manualised BA program was adapted from Addis and Martell's (2004), and Martell, Addis, and Jacobson's (2001) behavioural activation intervention for depression. CPT for PTSD was based on Resick et al.'s (2007) protocol (described above). Therapy

consisted of 15 sessions lasting between 60 to 90 minutes each. The first five sessions were depression focussed and utilised behavioural activation techniques. Sessions focused on the link between behaviour and mood and involved psychoeducation, activity monitoring, scheduling of mastery and pleasure tasks, and specific consideration of the outcome of certain helpful and unhelpful behaviours (Martell et al.). All five BA sessions were 60 minutes in length.

During Session 1 clients receive an overview of PTSD and MDD and the rational for treating MDD *and* PTSD is provided. In Session 2 the cycle of depression is introduced and the effects of inactivity discussed. Clients develop an understanding of the link between inactivity and depression and begin to see the benefits of activity and taking an "outside-in" approach to target depression. Client are assigned a weekly activity monitoring task in which they are asked to monitor what they do during the week and how these activities make them feel. Activity monitoring aims to allow the client to become aware of their mood, anxiety levels, triggers, and consequences. During Session 3 the activity monitoring task is reviewed and the TRAP (trigger, response, avoidance pattern) model discussed. Therapists exemplify the importance of scheduling pleasant events and the significance of elevating activity. The client is assigned a pleasant event scheduling task to complete during the following week. Mastery and pleasurable activities are the focus of the scheduling task. The pleasant event scheduling task is reviewed in Session 4 and incomplete activities are problem solved and reset. The continued importance of being active is discussed in session and the ACTION model (assess, choose, try, integrate, observe, never give up) is introduced. Clients are assigned to continue with the pleasant event scheduling of pleasurable and mastery based activities. In Session 5 clients use worksheets and role plays to demonstrate the importance of elevating pleasant activities in everyday life. Time is available within Session 5 to discuss further depression symptoms the client views as

impacting them (e.g., sleep, appetite, and concentration) and how to address these. Pleasant event scheduling is further assigned for homework.

Treatment then turns to a trauma focus with Sessions 6 through 15 taking a condensed CPT format. That is, Sessions 6 through 15 mimicked the exact treatment provided in the CPT condition, the only difference being the reduced treatment length. Such sessions incorporate exposure and cognitive therapy with a focus on the themes of safety, trust, intimacy, power/control, and esteem (Resick, et al., 2007). In Session 6 clients are presented with an overview of PTSD and a rationale for CPT. Clients are assigned the task of writing the impact statement. During Session 7 clients read the impact statement and develop an understanding of the connection between events, thoughts, and feelings. In Session 8 clients are further taught to identify thoughts and feelings and observe the connection between thoughts and feelings. The trauma account is assigned for homework in which clients are asked to write about the index trauma in as much detail as possible. During Sessions 9 and 10 clients work through their trauma accounts and stuck points are identified and challenged using Socratic questioning. During Session 11 clients are further taught to identify and challenge distorted beliefs and develop more balanced beliefs. Clients are taught to identify and challenge single unhelpful belief and acknowledge and change problematic patterns of thinking. Consequently Sessions 6 through to 11 allow the client to process the trauma and alter unhelpful trauma-beliefs. Sessions 6 through to 11 are all 60 minutes in length.

Sessions 12 to 15 ask clients to work through themes of safety, trust, power/control, esteem, and intimacy and correct overgeneralised beliefs related to each theme. In order to condense the typical 12-session CPT treatment into a 10-session format these remaining sessions were 90 minutes and two themes were discussed per session. This allowed 12 hours of therapy to be condensed into 10 sessions. In Session 12 the themes of safety and trust are introduced and overgeneralised beliefs challenged.

Within Session 13 the themes of power/control and esteem are introduced and overgeneralised beliefs are challenged. Within Session 14 the theme of intimacy is introduced and clients are assigned the task of writing a new impact statement that reflects their current beliefs. In Session 15 the new impact statement is reviewed and relapse prevention information delivered.

Cognitive Processing Therapy then Behavioural Activation (CPT/BA). The CPT/BA protocol was identical to the BA/CPT protocol except the CPT and BA components were presented in the reverse order. That is, the first 10 sessions were trauma-focussed (CPT) whilst the latter five sessions delivered BA.

Analysis Plan

Analysis of Treatment Outcomes

This thesis attempted to determine whether a combined treatment that targets *both* PTSD and MDD resulted in added benefits relative to PTSD treatment alone, and whether the order of therapy delivery was important. I analysed CAPS, PCL, and DASS-D scores, along with supplementary measures of emotions and cognitions to determine changes from pre- to posttreatment, and pre- to 6-month follow-up. Specifically, imputations were made for missing posttreatment, and 6-month follow-up data.[7] Once missing data was imputed I ran mixed, repeated-measures ANOVAs on each imputed dataset and pooled results according to the formula specified by Raghunathan and Dong (2013). Effect sizes and confidence intervals are reported throughout. Primary analyses were conducted with the ITT sample in which every effort was made to retain dropouts for assessments. I also repeated analyses on participants who completed all treatment sessions (‘completers’) and examined condition differences present at mid-phase assessments.

[7] I present a review of imputation strategies in Appendix G, and a summary of predictor and imputed variables is presented in Appendix H.

I also examined if the proportion of participants who met criteria for PTSD and MDD diagnosis at posttreatment and follow-up differed between CPT, BA/CPT, and CPT/BA. Further, as loss of diagnosis does not equate to good end-state functioning or imply reduced functional disability, achievement of good end-state functioning was also examined. Good end-state functioning was conservatively defined as achieving a CAPS scores of 19 or below (as per Schnurr et al., 2007) *and* a DASS-depression subscale score (DASS-D) of 6 or below. Achievement of good end-state functioning reflected that a participant was relatively asymptomatic at posttreatment or 6-month follow-up. To determine if the proportion of participants who met criteria for PTSD, MDD, and good end-state functioning at posttreatment and follow-up differed between CPT, BA/CPT, and CPT/BA I conducted a logistic regression with PTSD and MDD diagnostic status, as well as good end-state functioning at posttreatment and follow-up as outcomes, and condition as predictor. I conducted analyses on imputed data and used CPT as the reference group. I first conducted analyses on the ITT sample and then completer sample.

Analysis of Process Outcomes

With regards to process outcomes, this thesis aimed to: 1) determine whether under- and overengagement were similar or distinct processes, 2) determine how under- and overengagement operated during treatment, 3) determine the effects of under-, over-, and optimal levels of emotional engagement on PTSD and MDD treatment outcomes, and explore if under- and overengagement had a different impact on outcome, 4) determine if the relationship between under-, over-, and optimal engagement and treatment outcome differed between CPT, BA/CPT, and CPT/BA conditions and, 5) determine the convergent and discriminant validity of self-reported SUDS and objectively coded CEAS scores.

I used mixed-effects models to analyse the relationship between treatment outcome and each of under-, over-, and optimal emotional engagement. All mixed-effects models used a normal distribution with PCL and DASS-D scores collected every second session as outcome. I used likelihood ratio tests to assess the extent to which the addition of predictors (i.e., under-, over-, and optimal engagement) improved the fit of the model. I used correlations to determine whether under- and overengagement were similar or distinct processes, and to determine the convergent and discriminant validity of SUDS and CEAS ratings.

Chapter 3: Treatment Outcome Results

An alpha level of .05 was used for all inferential analyses and Cohen's *d* was reported as a measure of effect size. Although recognised as subjective, the cut-offs for small, medium, and large effects are 0.2, 0.5, and 0.8, respectively (Cohen, 1988). All confidence intervals were reported at a 95% accuracy level. I do not report effect sizes for omnibus tests of main effects but rather report effect sizes for specific pairwise comparisons. Doing so provides a more explicit understanding of differences in effects. Imputed descriptive statistics for the intent-to-treat (ITT) sample at pretreatment, posttreatment, and 6-month follow-up[8] assessment are presented in Table 3.1 (pg. 86), pooled inferential statistics are presented in Table 3.2 (pg. 88), and pooled effect sizes are presented in Table 3.3 (pg. 89). I discuss imputation and pooling procedures in detail in Appendix G. Unless otherwise specified all analyses were conducted in R, an open-source language and environment for statistical computing (R Development Core Team, 2011).

Due to the small sample size (n = 49) this thesis was underpowered. It is well established that low statistical power increases the probability of Type II error, or in other words, reduces the probability of detecting a difference between groups where a difference exists. Further and paradoxically, low statistical power can also increase the likelihood that a statistically significant finding is actually falsely positive. In order to minimise the consequences of being underpowered, and in order to achieve a clearer picture of results, I report effect sizes and their confidence intervals throughout all analyses. By reporting effect sizes and confidence intervals a better estimate of effects can be gained and the uncertainty or precision of results is made explicit. The reporting of effect sizes and confidence intervals is increasingly supported by prominent

[8] At the time of writing six CPT, six BA/CPT, and six CPT/BA 6-month follow-up assessments had not yet been collected from the ITT sample. Thus, a proportion of 6-month follow-up data was not included in reported analyses. I report the proportion of imputed and missing data throughout analyses.

psychological statisticians (see Cumming, 2014; Cumming et al., 2007; Cumming & Finch, 2001) and is part of good scientific practice (Wilkinson & Task Force on Statistical Inference [TFSAI], 1999). Indeed, Psychological Science, the flagship journal of the Association for Psychological Science (APS), is moving away from the reporting of *p* values and requires all authors to report confidence intervals and effect sizes. Further, although the sample size was small, and a larger proportion of data was missing (i.e., while only up to 4% of data was missing at posttreatment, up to 55% was missing at follow-up), multiple imputation should still be considered an appropriate means of working with missing data. For instance, Schafer and Graham (2002) posit that a large sample is not required for multiple imputation to provide precise estimates. Further, Rubin (1987) specifically calculated that even when sample size was small, and 50% of data was missing, multiple imputation provided a rate of 95% efficiency (i.e., accuracy).

Treatment Outcomes

Restatement of Aims and Analysis Plan

In this thesis I determined if a combined treatment approach was necessary in the treatment of comorbid PTSD/MDD, or if a single focus on PTSD was sufficient. I also determined if treatment presentation order affected outcome. I hypothesised that:

1. Participants in all conditions would demonstrate a significant reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms.
2. If targeting depression is required for optimal treatment outcomes, then in comparison to CPT participants, BA/CPT and CPT/BA participants would show a greater reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms.
3. If depressive symptoms reduce emotional engagement during PTSD treatment (and if initial BA sessions successfully target depressive symptoms such as low

mood, rumination, and emotional numbing), BA/CPT participants would show a greater reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms than CPT/BA participants?

Multiple Imputation and Data Analysis in This Thesis

To answer hypotheses I imputed missing posttreatment and 6-month follow-up data using multiple imputation. Pretreatment values did not require imputation as all pretreatment values were collected. A detailed description of multiple imputation is presented in Appendix G, and predictors and imputed variables are summarised in Appendix H. Once imputed datasets were created analyses were then carried out on PTSD and MDD symptom measures (i.e., primary outcomes) as well as other supplementary measures of cognitions, rumination, and emotional numbing. Specifically, I analysed pre- to posttreatment, and pre- to 6-month follow-up changes on all symptom measures using mixed, repeated-measures ANOVAs. Results were then pooled according to the specifications of Raghunathan and Dong (2013) to allow for single and meaningful interpretations of imputed datasets. Analyses were undertaken on the ITT sample first and then the completer sample. Specifics of undertaken analyses are reported throughout.

As multiple imputation is considered an appropriate strategy for handling missing data when data is considered missing at random, I first examined whether missingness was non-random for any collected variable other than time. More specifically, I examined if pretreatment CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS scores differed between those who provided data at posttreatment and follow-up and those that did not. Independent sample *t*-tests demonstrated that pretreatment CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN and TAS scores did not differ significantly between participants who did and did not provide posttreatment data, with effect sizes

all being small to medium (i.e., most effects between $d = 0.01$ and 0.23, with effects on the SRRS and RRS being $d = 0.30$ and $d = 0.38$ respectively). The same was observed at follow-up. Pretreatment CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN and TAS scores did not differ significantly between participants who provided follow-up data and those that did not. Effects sizes were again small to medium (i.e., most effects between $d = 0.01$ and $d = 0.29$, and the effect on the RRS being $d = 0.44$). Pretreatment demographic variable also did not differ significantly between those who did and did not provide data at posttreatment and those who did and did not provide data at follow-up. As differences were generally not large nor systematically related to the same variables, there was not sufficient evidence to suggest that missingness was associated with collected variables (i.e., non-random).

It should also be noted that I repeated analyses using available-case analysis in the ITT sample. In available-case analysis missing data were not imputed but rather, only available data-points were analysed using 3 (Condition: CPT, BA/CPT, CPT/BA) × 3 (Time: pretreatment, posttreatment, 6-month follow-up) mixed, repeated-measures ANOVAs on CAPS, PCL, DASS-D, PTCI, RRS, SRRS, TAS, and EN scores. Although available-case analysis can reduce sample size and does not recognise that non-responders may differ from responders, I used available-case analysis to identify the extent to which the pattern of results observed in the imputed data was the product of missing data-points. If available-case analysis and imputation results were found to be similar this would suggest that results were not driven by missing data. Alternatively, if available-case analysis and imputation results were found to be different this would suggest that available and missing data followed different patterns. In this thesis, analyses using available-case analysis, and analyses based on imputed data provided the same pattern of results. This therefore suggests that results in the ITT sample were not driven by missing data. It also suggests that if missing data points had been collected

and observed, results are likely to have looked similar to those achieved in the imputed data. Descriptive statistics for available-case analysis are presented in Appendix I.

Treatment Outcomes: Preliminary Analyses

Before examining posttreatment and 6-month follow-up outcomes I examined pretreatment symptom severity and retention rates for CPT, BA/CPT, and CPT/BA. I first examine pretreatment symptom severity in the ITT sample and then the completer sample. I then turn attention to retention rates. It should be reiterated that imputations were not made for pretreatment variables as all data was available.

Pretreatment Symptom Severity in the Intent-to-Treat Sample

In order to examine pretreatment symptom severity for CPT, BA/CPT, and CPT/BA participants, and in order to gain a better sense of what conditions looked like prior to the commencement of treatment, I conducted one-way ANOVAs on pretreatment symptom scores (i.e., CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN and TAS) as well as the number of endorsed comorbid conditions for the ITT sample. Descriptive statistics and effect sizes for pretreatment variables in the ITT sample are presented in Tables 2.1 (pg. 51) and 3.1 (pg. 86).

A significant main effect was found for pretreatment SRRS scores, $F(2, 46) = 3.76$, $p = .03$. Pairwise comparisons demonstrated that CPT/BA participants reported significantly higher pretreatment SRRS scores ($p = .03$, $d = 0.97$, 95% CI $d = [0.25, 1.68]$) than CPT participants. Pretreatment SRRS scores did not differ significantly between CPT and BA/CPT ($p = .99$, $d = 0.33$, 95% CI $d = [-0.36, 1.02]$), and BA/CPT and CPT/BA ($p = .35$, $d = 0.62$, 95% CI $d = [-0.11, 1.34]$). No other significant main effects or pairwise comparisons emerged.

As sample size was small I examined descriptive statistics and pairwise comparison effect sizes to determine if nonsignificant results were the product of reduced statistical power. Effect sizes for the number of endorsed comorbid conditions was small suggesting that CPT, BA/CPT, and CPT/BA did not meaningfully differ on the number of endorsed conditions. Effect sizes from pairwise comparison on the CAPS, PCL, DASS-D, PTCI, RRS, EN, and TAS were small to medium. Descriptive statistics and effect sizes suggested that at pretreatment, CPT/BA participants reported slightly higher (although nonsignificant) CAPS (p = .16, d = 0.64, 95% CI d = [-0.06, 1.33]), DASS-D (p = .47, d = 0.49, 95% CI d = [-0.20, 1.17]), PTCI (p = .21, d = 0.70, 95% CI d = [0.001, 1.39]), RRS (p = .10, d = 0.78, 95% CI d = [0.07, 1.47]), and TAS (p = .43, d = 0.49, 95% CI d = [-0.20, 1.17]) scores than CPT participants. Further, CPT/BA participants reported higher DASS-D (p = .55, d = 0.50, 95% CI d = [-0.23, 1.20]) scores than BA/CPT participants. All other pairwise comparisons produced small effects sizes. Findings suggest that at pretreatment CPT/BA participants tended to report slightly higher symptom severity scores than CPT participants.

Pretreatment Symptom Severity in Completers and Non-Completers

I then conducted a series of independent samples t-tests to compare pretreatment symptom severity in completers and non-completers. Specifically, I conducted independent samples t-tests on CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS pretreatment scores, as well as the number of endorsed comorbid conditions for completer and non-completers (descriptive statistics and effect sizes presented in Appendix A). Non-completers (M = 3.13, SD = 1.42) endorsed a greater number of pretreatment comorbid conditions than completers (M = 2.32, SD = 1.15), $t(47)$ = -2.19, p = .03, d = 0.63, 95% CI d = [0.05, 1.20]. No other significant pretreatment differences were found between completers and non-completers. Examination of descriptive

statistics and effect sizes revealed that completers and non-completers reported similar pretreatment CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS scores and that effect sizes were mostly negligible. Effect sizes did not exceed .27 and the majority of effects were between .01 and .14. Findings therefore suggest that aside for the number of endorsed comorbid conditions, there was little evidence of meaningful difference between non-completers and completers. Perhaps most interestingly, completers did not report significantly lower pretreatment symptom severity scores than non-completers. Participants reported no adverse effects over the course of the study.

Retention and Number of Attended Sessions

A dropout was defined as any participant who completed anything less than 12 sessions. The overall dropout rate was 49% (n = 24) with 25 participants completing treatment. All dropout occurred before Session 6. In light of the high dropout rate I examined dropout more explicitly to determine if dropout rates and the number of attended sessions differed between treatment conditions in the ITT sample.

I first examined condition differences in the proportion of dropout using chi square analyses. Chi square analyses did not reveal a significant difference in dropout rates between CPT/BA and CPT $\chi^2(1) = 2.03$, $p = .15$, $\varphi = .24$, or CPT/BA and BA/CPT $\chi^2(1) = 2.58$, $p = .11$, $\varphi = .29$ in the ITT sample. However, effect sizes suggest that this may be a power issue with 31.2% (n = 5) of CPT/BA participants compared to 55.6% (n = 10) of CPT, and 60% (n = 9) of BA/CPT participants dropping out. In fact, compared to CPT/BA, almost double the number of CPT and BA/CPT participants dropped out. Effect sizes therefore suggest an effect of condition on retention in which CPT/BA demonstrated better participant retention than CPT and BA/CPT.

To further explore differences in retention I examined the number of attended sessions using independent samples t-tests. CPT/BA participants tended to attend a

greater number of sessions (M = 11.25, SD = 6.16) than CPT (M = 7.50, SD = 6.33), $t(32) = -1.75$, $p = .09$, $d = 0.59$, 95% CI d = [-0.10, 1.27] and BA/CPT participants (M = 7.13, SD = 6.51), $t(29) = -1.81$, $p = .08$, $d = 0.65$, 95% CI d = [-0.08, 1.37]. Although differences were nonsignificant, as effect sizes were medium this suggests that this may again be a power issue. CPT/BA participants were likely to attend 3.77 and 4.14 more sessions than CPT and BA/CPT participants respectively. This is a clinically significant difference as four treatment sessions represents over a quarter of the treatment program. In sum, CPT/BA retained participants more effectively than CPT and BA/CPT.

As treatment structure differed between conditions with the CPT protocol being 12 to 15-sessions, and the BA/CPT and CPT/BA protocol being 15-sessions, I also examined the number of sessions completers attended to determine if treatment structure led to attendance differences. The number of sessions completers attended did not differ significantly between conditions, $F(2, 22) = 2.04$, $p = .16$. On average, CPT completers (n = 8) attended 14.00 (SD = 2.07) sessions, BA/CPT completers (n = 6) attended 14.67 (SD = 0.82) sessions, and CPT/BA completers (n = 11) attended 15.18 (SD = 0.40) sessions. Thus, completers attended a similar number of sessions and CPT completers attended two extra sessions beyond the typical 12-session CPT protocol.

Treatment Outcomes: Intent-to-Treat Sample

Summary of Hypothesis Testing Approach

In line with imputation procedures discussed in Appendix G, to test hypotheses in the ITT sample missing posttreatment and 6-month follow-up values were imputed using multiple imputation. Once data was imputed I analysed pre- to posttreatment changes on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS using 3 (Condition: CPT, BA/CPT, CPT/BA) × 2 (Time: pre-treatment, posttreatment) mixed, repeated-measures ANOVAs. I then analysed pre- to 6-month follow-up changes on all

outcome measures using 3 (Condition: CPT, BA/CPT, and CPT/BA) × 2 (Time: pre-treatment and 6-month follow-up) mixed, repeated-measures ANOVAs. Pooled results are reported throughout. Imputed descriptive statistics for the ITT sample, pooled inferential statistics, and pooled effect sizes are presented in Tables 3.1 to 3.3 (pp.86-89).

I used two, 3 (Condition: CPT, BA/CPT, and CPT/BA) × 2 (Time: pre-treatment, posttreatment, *or* 6-month follow-up) mixed, repeated-measures ANOVAs instead of a single 3 (Condition: CPT, BA/CPT, and CPT/BA) × 3 (Time: pre-treatment, posttreatment, and 6-month follow-up) ANOVA as main effects produced by 3 × 3 ANOVAs do not allow one to explicitly determine where condition and time differences occur and consequently would not have allowed my research questions to be addressed. More explicitly, when conducting 3 × 3 ANOVAs, follow-up analyses such as planned comparisons are required to determine where differences exist. However, conducting planned comparisons is much more difficult when data are imputed. Further, conducting every pairwise comparison (i.e., six, 2 × 2 ANOVAs) would have been impractical. Thus, to allow condition and time differences to be examined more explicitly, while still using imputed data, I conducted two, 3 × 2 ANOVAs. The use of two, 3 × 2 ANOVAs allowed me to gain a clearer examination of Condition × Time interactions and allowed me to determine if effects were the product of pre- to posttreatment, or pre- to 6-month follow-up changes. Although one may argue that this approach still does not explicitly determine where condition differences exist, examination of effect sizes and confidence intervals allow this to be done reasonably well. In sum, in order to test all hypotheses in the ITT sample I conducted two, 3 (Condition: CPT, BA/CPT, and CPT/BA) × 2 (Time: pre-treatment, posttreatment, *or* 6-month follow-up) mixed, repeated-measures ANOVAs on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS. I now address each hypothesis and report ANOVA results relevant to each hypothesis.

Table 3.1

CPT, BA/CPT, and CPT/BA Imputed Means, Standard Deviations, Sample Sizes, and Proportion of Imputed Data Over Time on All Measures: Intent-to-Treat Sample

			Pretreatment			Posttreatment			6-Month Follow-up		
Measure	Condition	*n*	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
CAPS	CPT	18	72.50	23.09	0	36.38	29.05	12 (2)	41.03	27.10	44.45 (8)
	BA/CPT	15	81.60	14.79	0	48.83	21.55	26.67 (4)	36.33	28.56	60 (9)
	CPT/BA	16	84.94	14.39	0	25.36	26.38	25 (4)	34.55	26.68	62.50 (10)
PCL	CPT	18	56.06	11.28	0	36.33	16.57	0	38.14	17.98	33.33 (6)
	BA/CPT	15	60.20	7.49	0	44.20	14.70	0	43.14	17.63	40 (6)
	CPT/BA	16	61.12	10.35	0	28.64	14.09	12.50 (2)	32.74	15.31	62.50 (10)
DASS - D	CPT	18	21.11	10.68	0	11.00	12.62	0	14.01	11.83	33.33 (6)
	BA/CPT	15	21.20	9.91	0	15.87	8.99	0	14.51	12.81	40 (6)
	CPT/BA	16	26.12	9.76	0	9.45	12.26	12.50 (2)	9.96	13.15	62.50 (10)
PTCI	CPT	18	130.67	36.62	0	103.89	48.34	0	97.51	35.24	38.89 (7)
	BA/CPT	15	139.27	39.87	0	107.81	41.47	20 (3)	100.83	36.59	60 (9)
	CPT/BA	16	152.94	26.36	0	79.13	43.53	25 (4)	80.67	31.47	62.50 (10)
RRS	CPT	18	51.94	13.97	0	46.56	16.57	0	43.76	11.77	38.89 (7)
	BA/CPT	15	57.07	13.71	0	47.63	14.32	20 (3)	40.73	11.79	60 (9)
	CPT/BA	16	61.88	11.28	0	37.34	13.46	25 (4)	36.35	11.71	62.50 (10)

Measure	Condition	*n*	Pretreatment			Posttreatment			6-Month Follow-up		
			M	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
SRRS	CPT	18	1159.17	462.47	0	865.00	542.67	0	732.64	407.97	38.89 (7)
	BA/CPT	15	1310.33	439.30	0	820.27	439.16	20 (3)	656.17	451.74	60 (9)
	CPT/BA	16	1546.25	313.98	0	695.94	462.79	25(4)	584.75	423.18	62.50 (10)
EN	CPT	18	25.67	7.93	0	15.72	8.96	0	16.69	8.73	38.89 (7)
	BA/CPT	15	28.47	4.67	0	20.27	9.54	20 (3)	18.03	9.30	60 (9)
	CPT/BA	16	28.62	5.15	0	14.14	9.45	25 (4)	16.62	10.09	62.50 (10)
TAS	CPT	18	55.28	12.41	0	49.44	14.54	0	43.88	19.71	38.89 (7)
	BA/CPT	15	62.47	9.80	0	54.31	14.88	20 (3)	45.05	19.39	60 (9)
	CPT/BA	16	60.94	10.67	0	41.11	14.31	25 (4)	42.85	18.33	62.50 (10)

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS–D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

[a] Six CPT, six BA/CPT, and six CPT/BA, 6-month follow-up assessments were not due at the time of writing and were therefore not included in reported analyses.

Table 3.2

Pooled Inferential Statistics from Imputed Datasets for Intent-to-Treat Sample on All Measures: Pre- to Posttreatment, and Pre- to 6-Month Follow-Up

	Posttreatment				6-Month Follow-up			
Measure	*df1*	*df2*	*F*	*p*	*df1*	*df2*	*F*	*p*
CAPS								
Time (T)	0.99	33.29	123.72	< .001	0.95	25.15	102.30	< .001
Condition (C)	1.74	42.88	1.73	.19	0.10	32.72	0.01	.33
C × T	1.69	33.29	4.49	.02	1.30	25.15	2.06	.16
PCL								
T	1.00	44.37	113.73	< .001	0.92	27.17	74.07	< .001
C	1.97	44.12	2.14	.13	0.34	28.67	0.46	.31
C × T	1.93	44.37	5.24	.01	0.69	27.17	1.85	.18
DASS – D								
T	0.99	42.61	39.34	< .001	0.78	28.61	20.13	< .001
C	1.93	44.39	0.35	.70	0.63	22.53	0.11	.62
C × T	1.79	42.61	3.38	.05	0.72	28.61	1.53	.22
PTCI								
T	0.98	33.17	51.03	< .001	0.86	19.79	55.97	< .001
C	1.14	42.86	0.20	.69	0.37	23.74	0.16	.44
C × T	1.95	33.17	6.03	.01	1.26	19.79	3.67	.06
RRS								
T	0.98	33.01	29.22	< .001	0.96	26.61	39.00	< .001
C	1.12	42.58	0.29	.62	1.12	29.24	0.18	.70
C × T	1.88	33.01	6.02	.01	1.52	26.61	3.56	.05
SRRS								
T	0.98	33.80	59.92	< .001	0.93	27.34	81.00	< .001
C	0.38	44.57	0.21	.42	0.86	38.03	0.51	.45
C × T	1.86	33.80	5.62	.01	1.45	27.34	4.23	.04
EN								
T	0.98	38.61	58.29	< .001	0.93	29.54	50.20	< .001
C	1.71	43.83	1.55	.22	0.65	26.65	0.48	.42
C × T	1.69	38.61	1.60	.22	0.21	29.54	0.21	.30
TAS								
T	0.96	30.23	26.55	< .001	0.71	26.34	24.06	< .001
C	1.80	42.91	2.11	.14	1.26	23.74	0.81	.40
C × T	1.68	30.23	3.92	.04	0.92	26.34	0.48	.48

Note. CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table 3.3

Pooled Effect Sizes (Cohen's d) [and 95% Confidence Intervals for Effect Sizes] from Pre- to Posttreatment, and Pre- to 6-month Follow-Up on All Symptom Measures: Intent-to-Treat Sample

	CPT		BA/CPT		CPT/BA	
Measure	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up
CAPS	1.38 [0.64, 2.10]	1.25 [0.53, 1.96]	1.77 [0.91, 2.62]	2.01 [1.11, 2.89]	2.84 [1.84, 3.83]	2.37 [1.44, 3.27]
PCL	1.39 [0.65, 2.12]	1.20 [0.48, 1.91]	1.37 [0.56, 2.16]	1.28 [0.48, 2.06]	2.64 [1.67, 3.59]	2.23 [1.33, 3.11]
DASS – D	0.87 [0.18, 1.54]	0.63 [-0.04, 1.30]	0.56 [-0.17, 1.29]	0.59 [-0.15, 1.32]	1.51 [0.71, 2.30]	1.44 [0.65, 2.21]
PTCI	0.62 [-0.05, 1.29]	0.93 [0.24, 1.62]	0.78 [0.03, 1.51]	1.01 [0.24, 1.77]	2.08 [1.20, 2.94]	2.65 [1.68, 3.60]
RRS	0.35 [-0.31, 1.01]	0.63 [-0.04, 1.30]	0.67 [-0.07, 1.41]	1.28 [0.48, 2.06]	2.01 [1.14, 2.86]	2.24 [1.34, 3.13]
SRRS	0.58 [-0.09, 1.25]	0.98 [0.28, 1.67]	1.12 [0.34, 1.89]	1.48 [0.66, 2.29]	2.18 [1.28, 3.05]	2.62 [1.65, 3.57]
EN	1.18 [0.46, 1.88]	1.09 [0.38, 1.78]	1.09 [0.32, 1.86]	1.44 [0.62, 2.24]	1.92 [1.07, 2.76]	1.53 [0.73, 2.31]
TAS	0.43 [-0.23, 1.10]	0.70 [0.02, 1.37]	0.65 [-0.09, 1.38]	1.15 [0.37, 1.92]	1.91 [0.79, 2.40]	1.23 [0.46, 1.98]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Hypothesis 1: Intent-to-Treat Sample

Hypothesis 1 predicted that all participants would demonstrate a significant reduction in PTSD and MDD symptoms. In other words, it was predicted that a main effect of time would emerge on PTSD (CAPS and PCL) and MDD (DASS-D) measures from pre- to posttreatment, and pre- to 6-month follow-up on each of the ANOVAs. Supporting Hypothesis 1, significant main effects of time emerged on all measures from pre- to posttreatment. Specifically, CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS scores decreased significantly from pre- to posttreatment. Similarly, significant main effects of time emerged on all measures from pre- to 6-month follow-up with all symptom measures decreasing. Effect sizes and confidence intervals indicated that all conditions consistently produced medium to large effects. Thus, results support Hypothesis 1 and the efficacy of CPT, BA/CPT, and CPT/BA in reducing symptoms from pre- to posttreatment, and pre- to 6-month follow-up. Treatment conditions not only reduced PTSD and MDD symptoms, but also led to significant reductions in trauma cognitions, rumination, and emotional numbing. Findings support CPT, and modifications to CPT as effective treatments for comorbid PTSD/MDD.

Hypothesis 2 and 3: Intent-to-Treat Sample

Hypothesis 2 predicted that compared to CPT alone, the combined BA/CPT and CPT/BA conditions would show a greater reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms. Hypothesis 3 predicted that BA/CPT participants would demonstrate greater reductions in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms than CPT/BA participants. Therefore, Hypothesis 2 and 3 related to the interaction between condition and time. Results largely refuted Hypothesis 2 and 3 and suggested that compared to CPT and BA/CPT, CPT/BA produced larger effects on outcome measures. Findings related to Hypothesis 2 and 3 in the ITT sample are now

discussed in detail (see Tables 3.1 to 3.3 for imputed descriptive statistics, pooled inferential statistics, and pooled effect sizes).

Pre- to posttreatment changes. I first examined pre- to posttreatment changes on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS as assessed by the undertaken 3 (Condition: CPT, BA/CPT, and CPT/BA) × 2 (Time: pretreatment, posttreatment) ANOVA. Significant Condition × Time interactions emerged on all measures other than the EN. Refuting Hypothesis 2 and 3, descriptive statistics and effect sizes demonstrated that compared to CPT and BA/CPT, *CPT/BA* produced larger symptom reductions. I first review significant Condition × Time interactions on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, and TAS, and then turn attention to the EN.

Effect sizes demonstrated that while all conditions consistently produced medium to large effects on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, and TAS, CPT/BA produced larger effects than CPT and BA/CPT. CPT/BA produced effect sizes on these measures 1.74 to 5.71 times larger than that produced by CPT, and 1.60 to 3.00 times larger than that produced by BA/CPT. Estimated effect sizes for CPT/BA scores were not within the bounds of CPT and BA/CPT effect size confidence intervals, and overlap of effect size confidence intervals was not considerable. Thus, results suggest that compared to CPT and BA/CPT, CPT/BA produced substantially larger reductions on all measures other than the EN from pre- to posttreatment.

Although a significant interaction did not emerge for EN scores I examined effect sizes and confidence intervals to determine if this was the product of reduced statistical power. Effect sizes for the EN demonstrated the same pattern of results as that reported above. That is, compared to CPT and BA/CPT, CPT/BA appeared to produce a meaningfully larger reduction on the EN. CPT/BA produced an effect on the EN 1.63 times larger than that achieved by CPT, and 1.76 times larger than that achieved by BA/CPT. Further, CPT and BA/CPT effect size confidence interval for the EN did not

encompass the effect size estimated for CPT/BA. This suggests that even if the effect CPT and BA/CPT had on the EN reached the upper-bounds of reported confidence intervals, the effect would still be smaller than that estimated for CPT/BA. Thus, there appeared to be evidence of a difference in effect in which CPT/BA produced meaningfully larger reductions on the EN than CPT and BA/CPT, but this study was not sufficiently powerful to detect it as significant.

To review, compared to CPT and BA/CPT, CPT/BA produced significantly larger effects on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, and TAS from pre- to posttreatment. Further, meaningful differences in effect sizes emerged for the EN favouring CPT/BA. Thus, Hypothesis 2 was refuted as CPT/BA, but not BA/CPT, was found to be more effective than CPT alone. Hypothesis 3 was also refuted as CPT/BA demonstrated better treatment outcomes than BA/CPT. These findings suggest that compared to CPT alone, there is added benefit in targeting PTSD *and* MDD symptoms in those with comorbid PTSD/MDD. However, this benefit is only achieved when PTSD is targeted *before* MDD.

Pre- to 6-month follow-up changes. I then examined pre- to 6-month follow-up changes on the CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS as assessed by the undertaken 3 (Condition: CPT, BA/CPT, and CPT/BA) × 2 (Time: pre-treatment, 6-month follow-up) ANOVA. For many outcome variables Condition × Time interactions were nonsignificant and for those that were, CPT/BA produced larger effects than CPT and BA/CPT, refuting Hypothesis 2 and 3. I first discuss significant interactions and then review nonsignificant interactions.

A significant Condition × Time interaction emerged on the RRS and SRRS. Descriptive statistics and effects sizes suggested that compared to CPT and BA/CPT, CPT/BA had a larger effect on the RRS and SRRS. CPT/BA produced an effect on the RRS 3.56 times larger than CPT, and 1.75 times larger than BA/CPT. Further, CPT/BA

produced an effect on the SRRS 2.67 times larger than CPT, and 1.77 times larger than BA/CPT. CPT and BA/CPT effect size confidence intervals for the RRS and SRRS did not encompass estimated effect sizes for CPT/BA, and overlap of confidence intervals between CPT/BA and CPT and BA/CPT was not considerable. Thus, refuting Hypothesis 2 and 3, compared to CPT and BA/CPT, CPT/BA produced substantially larger reductions from pre- to 6-month follow-up on the RRS and SRRS.

Although no significant Condition × Time interaction emerged on the CAPS, PCL, DASS-D, PTCI, EN, and TAS, I examined effect sizes and confidence intervals for these measures to determine the magnitude of change, and to determine if nonsignificant results were potentially the product of reduced statistical power. I first compare effects on the CAPS, PCL, DASS-D, PTCI, EN, and TAS for CPT/BA and CPT, and then compare effects between CPT/BA and BA/CPT. Descriptive statistics and effect sizes demonstrated that compared to CPT, CPT/BA produced meaningfully larger effects on all measures other than the EN at follow-up. CPT/BA produced effects on the CAPS, PCL, DASS-D, PTCI, and TAS 1.76 to 2.85 times larger than that produced by CPT. Effect size confidence intervals for the CAPS, PCL, DASS-D, PTCI, and TAS for CPT did not encompass estimated effect sizes for CPT/BA. This suggests that even at the upper-bounds of CPT's effect, CPT did not produce an effect on these measures as large as that estimated for CPT/BA. Alternatively, CPT and CPT/BA demonstrated a similar effect on the EN with effect size confidence intervals overlapping greatly. These findings refute Hypothesis 2 and 3 and suggest that compared to CPT, CPT/BA produced meaningfully larger effects on the CAPS, PCL, DASS-D, PTCI, and TAS at follow-up. It is possible that a more powerful study would have found such differences to be significant.

In comparison to BA/CPT, CPT/BA produced meaningfully larger changes from pre- to 6-month follow-up on the PCL, DASS-D, and PTCI. CPT/BA produced effects

on the PCL, DASS-D, and PTCI 1.74 to 2.62 times larger than BA/CPT. BA/CPT confidence intervals did not encompass the effect size estimated for CPT/BA and overlap was not considerable. Thus, again refuting Hypothesis 2 and 3, there appeared to be a difference in effect whereby CPT/BA produced meaningfully larger effects on the PCL, DASS-D, and PTCI than BA/CPT. Again, with greater statistical power such differences may have reached significance. Effect sizes for the CAPS, EN, and TAS did not meaningfully differ between BA/CPT and CPT/BA, with CPT/BA producing effects only 1.06 to 1.19 times larger than that achieved by BA/CPT.

To review, compared to CPT and BA/CPT, CPT/BA demonstrated substantially larger effects on the RRS and SRRS at follow-up. Further, CPT/BA produced meaningfully (although nonsignificant) larger effects on the PCL, DASS-D, and PTCI than CPT and BA/CPT, and meaningfully larger effects on the CAPS and TAS than CPT. Taken together with significant Condition × Time interactions found from pre- to posttreatment, results suggest that while CPT/BA produced substantially larger effects from pre- to posttreatment, symptom change was more similar across conditions from pre- to 6-month follow-up, as illustrated by nonsignificant interactions. That is, although CPT/BA appeared to lead to larger changes from pre- to posttreatment, by follow-up the size of change was more similar and CPT and BA/CPT participants appeared to 'catch-up.' However, one must remember that compared to CPT and BA/CPT, at 6-month follow-up CPT/BA still produced significantly larger effects on the RRS and SRRS, and meaningfully larger effects on many other measures that a more power study may have found to be significant. Thus, at 6-month follow-up Hypothesis 2 and 3 were refuted and CPT/BA still appeared to be the treatment of choice.

Summary of Treatment Outcomes in the Intent-to-Treat Sample

Results supported Hypothesis 1 and suggested that all treatment conditions were effective in reducing, PTSD, MDD, and secondary outcomes in the ITT sample. Hypothesis 2 and 3 were refuted as results failed to demonstrate the superior efficacy of BA/CPT. Refuting Hypothesis 2, CPT/BA, but not BA/CPT, demonstrated better treatment outcomes than CPT alone. Also, refuting Hypothesis 3, CPT/BA demonstrated larger effects on PTSD, MDD, and secondary outcomes than BA/CPT. While differences between conditions were more pronounced at posttreatment than 6-month follow-up, significant and meaningful differences still existed at follow-up supporting the superior efficacy of CPT/BA compared to CPT and BA/CPT. Findings suggest that a combined treatment that targets PTSD *and* MDD results in added benefits relative to PTSD treatment alone. However, the added utility of targeting MDD is only achieved when PTSD treatment is presented *prior* to MDD treatment.

Supplementary Analysis: Controlling for Number of Sessions Attended

As CPT/BA demonstrated enhanced retention compared to CPT and BA/CPT, one may argue that CPT/BA participants only evidenced superior treatment outcomes because they attended a greater number of sessions than CPT and BA/CPT participants. In other words, the superiority of CPT/BA over CPT and BA/CPT may merely be a product of treatment dose. To explore such an explanation I repeated the above ANOVAs controlling for number of sessions. More explicitly, rather than carrying out mixed, repeated measures ANOVAs, I conducted two, 3 (Condition: CPT, BA/CPT, CPT/BA) × 2 (Time: pretreatment, posttreatment *or* 6-month follow-up) mixed, repeated-measures ANCOVAs on all outcome measures that controlled for number of sessions. The main difference between analyses controlling for number of sessions and previous analyses was that effect sizes used pooled ANCOVA models to estimate

adjusted means. To calculate Cohen's *d* I used variance taken from pretreatment scores for each of the conditions to determine the relevant standard deviations. In essence, I calculated confidence intervals as though values were estimated directly from the data. Confidence intervals therefore do not take into account the error inherent in either: 1) the imputation process, or 2) estimating the mean with the effect of the covariate removed. As CPT, BA/CPT, and CPT/BA completers all attended a similar number of sessions, analyses were carried out on the ITT sample only using imputed data. Inferential statistics and effects sizes are reported in Appendix J.

ANCOVA results were largely consistent with initial results and refuted the notion that CPT/BA superiority was a product of treatment dose. First, when number of sessions was controlled, main effects of time were significant for all measures from pre- to posttreatment, and pre- to 6-month follow-up. Importantly, significant Condition × Time interactions emerged on the PTCI, RRS, and SRRS at posttreatment, and RRS and SRRS at 6-month follow-up, whereby CPT/BA reported larger effects on the PTCI, RRS, and SRRS than CPT and BA/CPT. While some interactions were nonsignificant, meaningful differences in effect sizes still emerged in which CPT/BA demonstrated meaningfully larger effects on the CAPS and DASS-D at posttreatment and 6-month follow-up compared to CPT and BA/CPT. Thus, while some interactions were nonsignificant when number of sessions was controlled, results remained largely the same as initial results and initial patterns in the data were maintained. To recap, as some interactions were nonsignificant when number of sessions was controlled, treatment dose should be considered a potential predictor of treatment outcome. However, as initial results and results obtained when controlling for number of sessions were very similar, and as a number of interactions supporting the efficacy of CPT/BA were also significant when number of sessions was controlled, the superiority of CPT/BA is not completely explained by treatment dose.

Treatment Outcomes: Completer Sample

Preliminary Analyses for Completer Sample

I next examined hypotheses in the completer sample (i.e., only those who completed the whole 12 to 15-session treatment program). However, before examining post- and 6-month follow-up treatment outcomes I examined pretreatment symptom severity. To examine pretreatment symptom severity for CPT, BA/CPT, and CPT/BA completers I conducted a series of one-way ANOVAs on pretreatment CAPS, PCL, DASS-D, RRS, SRRS, EN, and TAS scores (see Appendix K for inferential statistics and Table 3.4 for descriptive statistics). Results suggested that compared to BA/CPT and CPT/BA completers, CPT completers reported lower pretreatment symptom scores.

A significant main effect emerged on the CAPS, RRS, and SRRS in which CPT completers reported lower pretreatment scores than BA/CPT and CPT/BA completers (see Appendix K). Pairwise comparisons demonstrated that CPT completers reported significantly lower pretreatment CAPS scores than BA/CPT (p = .02, d = 1.54, 95% CI d = [0.29, 2.74]) and CPT/BA completers (p = .01, d = 1.45, 95% CI d = [0.40, 2.47]). Pretreatment CAPS scores did not significantly differ between BA/CPT and CPT/BA completers (p = 1.00, d = 0.18, 95% CI d = [-0.82, 1.17]). In regards to RRS scores, CPT completers reported significantly lower pretreatment RRS scores than BA/CPT (p = .05, d = 1.45, 95% CI d = [0.22, 2.63]) and CPT/BA completers (p = .04, d = 1.25, 95% CI d = [0.23, 2.24]). RRS scores did not differ significantly between BA/CPT and CPT/BA completers (p = 1.00, d = 0.18, 95% CI d = [0.82, 1.17]). Further, CPT completers reported significantly lower SRRS scores than CPT/BA completers (p = .04, d = 1.48, 95% CI d = [0.43, 2.50]). SRRS scores did not differ significantly between CPT and BA/CPT (p = .06, d = 1.24, 95% CI d = [0.05, 2.39]) and BA/CPT and CPT/BA (p = 1.00, d = 0.11, 95% CI d = [-0.89, 1.10]).

Table 3.4

CPT, BA/CPT, and CPT/BA Imputed Means, Standard Deviations, Sample Sizes, and Proportion of Imputed Data Over Time on All Measures: Completer Sample

			Pretreatment			Posttreatment			6-Month Follow-up		
Measure	Condition	*n*	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
CAPS	CPT	8	59.25	19.14	0 (0)	19.00	17.49	0	22.05	14.96	37.5 (3)
	BA/CPT	6	87.00	16.22	0 (0)	27.68	13.78	16.67 (1)	22.53	25.24	50.00 (3)
	CPT/BA	11	84.18	15.63	0 (0)	13.12	14.64	9.09 (1)	23.72	23.73	54.55 (6)
PCL	CPT	8	51.75	8.45	0 (0)	24.75	8.36	0 (0)	25.00	7.32	37.5 (3)
	BA/CPT	6	61.83	8.61	0 (0)	31.83	10.09	0 (0)	26.92	12.38	50.00 (3)
	CPT/BA	11	59.64	11.78	0 (0)	21.00	4.20	0 (0)	26.69	8.74	54.55 (6)
DASS - D	CPT	8	17.75	8.45	0 (0)	4.00	5.76	0 (0)	6.17	3.92	37.5 (3)
	BA/CPT	6	29.00	8.65	0 (0)	11.00	9.70	0 (0)	5.23	6.33	50.00 (3)
	CPT/BA	11	24.36	10.11	0 (0)	4.73	6.08	0 (0)	2.56	4.24	54.55 (6)
PTCI	CPT	8	118.75	36.64	0 (0)	79.75	47.84	0 (0)	99.13	46.20	37.5 (3)
	BA/CPT	6	154.17	36.14	0 (0)	94.17	29.82	0 (0)	85.01	39.16	50.00 (3)
	CPT/BA	11	148.91	24.26	0 (0)	64.36	37.73	0 (0)	79.23	31.74	54.55 (6)
RRS	CPT	8	46.12	10.55	0 (0)	42.62	16.23	0 (0)	46.20	10.00	37.5 (3)
	BA/CPT	6	61.83	11.23	0 (0)	44.33	10.05	0 (0)	39.72	13.05	50.00 (3)
	CPT/BA	11	59.82	11.25	0 (0)	32.82	8.24	0 (0)	40.37	12.02	54.55 (6)

			Pretreatment			Posttreatment			6-Month Follow-up		
Measure	Condition	*n*	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
SRRS	CPT	8	994.38	303.37	0 (0)	507.50	400.16	0 (0)	551.81	397.92	37.5 (3)
	BA/CPT	6	1518.33	547.92	0 (0)	714.17	419.60	0 (0)	634.67	556.16	50.00 (3)
	CPT/BA	11	1470.91	333.39	0 (0)	608.36	334.34	0 (0)	589.82	349.29	54.55 (6)
EN	CPT	8	25.88	8.59	0 (0)	22.50	6.74	0 (0)	16.76	14.00	37.5 (3)
	BA/CPT	6	30.33	4.13	0 (0)	15.00	9.44	0 (0)	14.35	9.38	50.00 (3)
	CPT/BA	11	27.27	5.57	0 (0)	11.64	6.99	0 (0)	14.67	9.12	54.55 (6)
TAS	CPT	8	55.75	10.91	0 (0)	42.75	14.33	0 (0)	43.02	24.32	37.5 (3)
	BA/CPT	6	63.67	8.59	0 (0)	49.17	12.42	0 (0)	47.30	17.57	50.00 (3)
	CPT/BA	11	61.36	9.10	0 (0)	36.55	11.08	0 (0)	40.92	17.60	54.55 (6)

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Checklist; DASS-D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognition Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaires; TAS = Twenty- Item Toronto Alexithymia Scale.

[a] Three CPT, three BA/CPT, and six CPT/BA, 6-month follow-up assessments were not due at the time of writing and were therefore not included in reported analyses.

Table 3.5

Pooled Inferential Statistics from Imputed Datasets for Completer Sample on All Measures: Pre- to Posttreatment, and Pre- to 6-Month Follow-Up

	Posttreatment				6-Month Follow-up			
Measure	*df1*	*df2*	*F*	*p*	*df1*	*df2*	*F*	*p*
CAPS								
Time (T)	1.00	19.43	195.44	< .001	0.99	13.07	178.69	< .001
Condition (C)	1.92	20.17	3.78	.04	1.67	15.38	2.40	.13
C × T	1.96	29.43	5.05	.02	1.60	13.07	3.94	.05
PCL								
T	1	22	293.85	< .001	0.96	9.04	172.55	< .001
C	2	22	2.44	.11	1.27	12.22	1.25	.30
C × T	2	22	3.77	.04	1.44	9.04	1.07	.36
DASS – D								
T	1	22	117.55	< .001	0.99	11.43	102.06	< .001
C	2	22	2.78	.08	1.36	14.87	1.50	.25
C × T	2	22	1.28	.30	1.74	11.43	3.66	.06
PTCI								
T	1	22	62.36	< .001	0.95	10.36	55.42	< .001
C	2	22	1.26	.31	1.00	19.15	0.22	.65
C × T	2	22	2.96	.07	1.59	10.36	5.11	.03
RRS								
T	1	22	45.89	< .001	0.94	11.50	19.57	.001
C	2	22	1.57	.23	1.45	13.54	0.91	.39
C × T	2	22	7.94	.003	1.40	11.50	4.67	.04
SRRS								
T	1	22	82.55	< .001	0.98	8.76	67.91	< .001
C	2	22	2.70	.09	1.78	17.15	1.98	.17
C × T	2	22	2.18	.14	1.58	8.76	2.65	.13
EN								
T	1	22	66.75	< .001	0.67	16.99	37.84	< .001
C	2	22	1.08	.36	1.47	14.30	0.20	.75
C × T	2	22	0.04	.96	1.27	17.00	0.99	.36
TAS								
T	1	22	46,86	< .001	0.76	18.09	13.24	.003
C	2	22	1.57	.23	0.94	18.44	0.64	.43
C × T	2	22	2.11	.15	0.54	18.09	0.28	.47

Note. CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table 3.6

Pooled Effect Sizes (Cohen's d) [and 95% Confidence Intervals for Effect Sizes] from Pre- to Posttreatment and Pre- to 6-month Follow-Up on All Symptom Measures: Completer Sample

	CPT		BA/CPT		CPT/BA	
Measure	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up
CAPS	2.20	2.22	3.95	3.08	4.70	3.84
	[0.90, 3.44]	[0.92, 3.47]	[1.87, 5.96]	[1.30, 4.79]	[3.01, 6.35]	[2.38, 5.27]
PCL	3.21	3.48	3.20	3.29	4.36	3.28
	[1.66, 4.72]	[1.85, 5.07]	[1.38, 4.95]	[1.45, 5.08]	[2.77, 5.94]	[1.95, 4.57]
DASS – D	1.90	1.76	1.96	3.14	2.35	2.83
	[0.68, 3.08]	[0.56, 2.91]	[0.52, 3.34]	[1.34, 4.88]	[1.23, 3.44]	[1.61, 4.02]
PTCI	0.92	0.47	1.81	1.86	2.67	2.58
	[-0.14, 1.94]	[-0.53, 1.46]	[0.41, 3.16]	[0.45, 3.25]	[1.48, 3.82]	[1.41, 3.72]
RRS	0.26	-0.02	1.64	1.85	2.74	1.70
	[-0.73, 1.24]	[-1.00, 0.96]	[0.28, 2.95]	[0.43, 3.20]	[1.53, 3.91]	[0.70, 2.67]
SRRS	1.37	1.26	1.65	1.61	2.58	2.63
	[0.25, 2.45]	[0.16, 2.33]	[0.28, 2.96]	[0.26, 2.91]	[1.41, 3.72]	[1.45, 3.78]
EN	1.86	1.05	2.10	2.24	2.47	1.75
	[0.65, 3.03]	[-0.02, 2.09]	[0.62, 3.52]	[0.72, 3.70]	[1.33, 3.59]	[0.74, 2.73]
TAS	1.02	0.68	1.36	1.22	2.45	1.49
	[-0.04, 2.05]	[-0.35, 1.67]	[0.06, 2.61]	[-0.05, 2.45]	[1.31. 3.56]	[0.52, 2.43]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Significant main effects and significant pairwise comparisons did not emerge on the PCL, DASS-D, RRS, SRRS, EN, and TAS. However, as this thesis was underpowered I examined effect sizes comparing CPT to BA/CPT, and CPT to CPT/BA completers to determine if nonsignificant results were the product of reduced statistical power. Effect sizes suggested that CPT completers reported meaningfully lower pretreatment PCL, DASS-D, RRS, SRRS, EN, and TAS scores than BA/CPT and CPT/BA completers. More explicitly, effect sizes from pairwise comparisons suggested that compared to BA/CPT completers, CPT completers tended to report lower pretreatment PCL (p = .24, d = 1.18, 95% CI d = [0.01, 2.32]), DASS-D (p = .11, d = 1.31, 95% CI d = [0.11, 2.47]), PTCI (p = .15, d = 0.97, 95% CI d = [-0.17, 2.08]), EN (p = .64, d = 0.63, 95% CI d = [-0.47, 1.71]), and TAS (p = .42, d = 0.79, 95% CI d = [-0.33, 1.88]) scores. Further, compared to CPT/BA completers, effect sizes suggested that CPT completers tended to report lower pretreatment PCL (p = .32, d = 0.75. 95% CI d = [-0.36, 1.84]), DASS-D (p = .42, d = 0.70, 95% CI d = [-0.25, 1.63]), and PTCI (p = .15, d = 1.00, 95% CI d = [0.02, 1.96]) scores. Results suggest that compared to BA/CPT and CPT/BA completers, CPT completers reported lower pretreatment symptoms.

Summary of Hypothesis Testing Approach for Completer Sample

Hypotheses in the completer sample were tested using the same procedures reported for the ITT sample. That is, to test hypotheses in the completer sample I imputed missing posttreatment and 6-month follow-up data and pooled results. To test hypotheses I conducted two, 3 (Condition: CPT, BA/CPT, CPT/BA) × 2 (Time: pretreatment, posttreatment *or* 6-month follow-up) mixed, repeated-measure ANOVAs on CAPS, PCL, DASS-D, PTCI, RRS, SRRS, EN, and TAS scores for the completer

sample.[9] I now address each hypothesis separately and report ANOVA results relevant to each hypothesis. Imputed descriptive statistics, pooled inferential statistics, and pooled effect sizes are presented in Tables 3.4 to 3.6 (pp. 98-101).

Hypothesis 1: Completer Sample

Hypothesis 1 predicted that all conditions would demonstrate a significant reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms. As such, main effects of time were the analysis of interest. Results supported Hypothesis 1 and suggested that if participants completed treatment they were likely to show good treatment outcomes. Specifically, significant main effects of time emerged on all measures from pre- to posttreatment. Other than RRS scores for CPT completers, all effect sizes were large with effect sizes ranging from 0.92 to 4.70. Interestingly, the effect size for RRS scores reported by CPT completers was only 0.26. With regards to pre- to 6-month follow-up changes, significant main effects of time emerged on all measures, again supporting Hypothesis 1. With the exception of RRS and PTCI scores for CPT completers, effect sizes were medium to large with the majority of effect sizes being greater than one. In contrast, CPT completers evidenced smaller effects on the RRS (d = -0.02) and PTCI (d = 0.47) at follow-up.

Results support Hypothesis 1 and suggest that if participants completed treatment they were likely to demonstrate very large changes on PTSD, MDD, and secondary outcomes. It should be emphasised that completers evidenced very large effect sizes from pre- to posttreatment, and pre- to 6-month follow-up. For example, 6-month

[9] Although CPT completers reported lower pretreatment scores than BA/CPT and CPT/BA completers, I did not control for pretreatment scores in analyses. Pretreatment scores were not controlled as I was not interested in determining if posttreatment and follow-up scores differed between conditions, but rather I was interested in determining if the magnitude of change from pre- to posttreatment, and pre- to 6-month follow-up differed between conditions. By examining the latter such analyses already take into account pretreatment differences, as analyses look at differences in the magnitude of change irrespective of starting point.

follow-up effect sizes on the CAPS were 2.22, 3.08, and 3.84 for CPT, BA/CPT, and CPT/BA respectively.

Hypothesis 2 and 3: Completer Sample

Hypothesis 2 predicted that compared to CPT completers, BA/CPT and CPT/BA completers would show a greater reduction in PTSD (CAPS and PCL) and MDD (DASS-D) symptoms. Hypothesis 3 predicted that BA/CPT completers would show a greater reduction in PTSD and MDD symptoms than CPT/BA completers. To test Hypothesis 2 and 3 I examined Condition × Time interactions as analysed by the undertaken ANOVAs (pooled inferential statistics and effect sizes are presented in Tables 3.5 and 3.6). Results largely refuted Hypothesis 2 and 3 and suggested that all treatment completers were able to achieve good outcomes irrespective of condition.

Pre- to posttreatment changes. When examining pre- to posttreatment changes most Condition × Time interactions for the completer sample were nonsignificant and significant interactions only emerged on the CAPS, PCL, and RRS. Descriptive statistics and effect sizes demonstrated that compared to BA/CPT and CPT/BA completers, CPT completers reported smaller effects on the CAPS and RRS. Alternatively, for PCL scores CPT/BA completers showed larger decreases on the PCL compared to CPT and BA/CPT completers. I now discussed significant interactions in detail.

CPT completers demonstrated an effect on the CAPS 1.80 times smaller than that demonstrated by BA/CPT, and 2.14 times smaller than that demonstrated by CPT/BA. However, it should be emphasised that although CPT completers evidenced smaller reductions on the CAPS, the effect CPT had on the CAPS was still very large ($d = 2.20$). Interestingly, CPT completers only evidenced small reductions on the RRS ($d = 0.26$) and produced an effect on the RRS 6.30 times smaller than BA/CPT, and 10.53 times

smaller than CPT/BA. PCL scores evidenced a different interaction in which CPT/BA completers demonstrated a larger reduction on PCL scores compared to CPT and BA/CPT completers. CPT/BA produced an effect 1.36 times larger than that achieved by CPT and BA/CPT.

As most interactions were nonsignificant Hypothesis 2 and 3 were refuted. Results suggest that if individuals completed treatment they were likely to demonstrate large changes on PTSD, MDD, and secondary outcomes from pre- to posttreatment irrespective of condition. However, there was a small caveat to this whereby CPT participants evidenced only small changes on the RRS.

Pre- to 6-month follow-up changes. Similar to posttreatment results, and refuting Hypothesis 2 and 3, most Condition × Time interactions were nonsignificant for completers at follow-up (see Tables 3.5 and 3.6 for pooled inferential statistics and effect sizes). The only significant interactions to emerge were on the CAPS, PTCI, and RRS. Compared to BA/CPT and CPT/BA completers, CPT completers demonstrated smaller effects on the PTCI and RRS. Alternatively, CAPS scores showed a different pattern in which CPT/BA completers produced a larger effect on the CAPS than CPT completers. I first discuss interactions for the PTCI and RRS and then discuss results for the CAPS.

While BA/CPT and CPT/BA produced very large effects on the PTCI at follow-up, CPT only produced a small effect. CPT produced an effect on the PTCI 3.96 times smaller than BA/CPT, and 5.49 times smaller than CPT/BA. The CPT effect size confidence interval for the PTCI did not encompass the effect sizes estimated for BA/CPT and CPT/BA. Further, CPT did not appear to produce a significant effect on the RRS (d = -0.02) and CPT produced an effect on the RRS 92.5 times smaller than BA/CPT, and 85 times smaller than CPT/BA. Interestingly, although RRS scores did not appear to change meaningfully from pre- to 6-month follow-up for CPT completers,

follow-up RRS scores were quite similar across all conditions (see descriptive statistics in Table 3.4). In line with this, as CPT completers reported lower pretreatment RRS scores than BA/CPT and CPT/BA completers, smaller changes on the RRS may in part reflect a floor effect whereby CPT completers did not have as much room for improvement as the other groups. However, as BA/CPT and CPT/BA were able to reduce RRS scores by approximately 20 points whilst CPT only reduced RRS scores by 3.5 points, and as CPT/BA demonstrated an ability to reduce RRS scores to a lower point than CPT, lower pretreatment scores and floor effects do not fully explain why CPT completers did not show a meaningful change on the RRS.

Turning attention to the CAPS, compared to CPT completers, CPT/BA completers produced a larger effect on the CAPS at follow-up. CPT/BA produced an effect on the CAPS 1.73 time larger than that demonstrated by CPT. However, this again does not suggest that CPT did not have a significant effect on the CAPS as CPT produced a very large effect (d = 2.22). Rather, results suggest that CPT/BA simply had a larger effect than CPT. Follow-up results for completers suggest that if individuals completed treatment they showed large changes on PTSD, MDD, and secondary outcomes. However, there was an exception to this as effects on the RRS and PTCI seemed to be reduced for CPT completers.

Summary of Treatment Outcomes in the Completer Sample

In sum, results from the completer sample refute Hypothesis 2 and 3 and suggest that if participants completed treatment they were likely to achieve good treatment outcomes with most effect sizes being very large (especially large for CPT/BA). However, there was a small exception to this in which CPT completers only showed small changes on the RRS and PTCI. While lower pretreatment scores and floor effects may partially account for this, as CPT/BA and BA/CPT demonstrated quite large

changes on these measures this does not fully account for findings. As such, results suggest that CPT may not be sufficient to challenge and reduce rumination (RRS) and negative cognitions (PTCI) in a wholly comorbid sample, and that a combined treatment approach may be needed to produce meaningful changes in these symptoms.

Treatment Outcomes: PTSD and MDD Diagnostic Status and Good End-State Functioning

Results so far have examined symptom severity and have suggested that compared to CPT and BA/CPT, CPT/BA produced better PTSD, MDD, and secondary treatment outcomes (ITT sample). I now turn attention to PTSD and MDD diagnostic status, and good end-state functioning (i.e., binary outcomes). To determine if the proportion of participants who met criteria for PTSD, MDD, and good end-state functioning at posttreatment and 6-month follow-up differed between CPT, BA/CPT, and CPT/BA I conducted a logistic regression with PTSD and MDD diagnostic status, as well as good end-state functioning at posttreatment and follow-up as outcomes, and condition as predictor. Analyses were conducted on imputed data and CPT was used as the reference group.[10] By predicting a binary outcome for CPT (i.e., reference group), we can tell if the likelihood of achieving that outcome differed significantly between CPT and BA/CPT, and CPT and CPT/BA. Good end-state was conservatively defined as at or below a cut-off of 19 on the CAPS, and at or below a cut-off of 6 on the DASS-D. Those who achieved good end-state functioning were considered asymptomatic. I first discuss findings in the ITT sample and then turn attention to completers.

[10] By using CPT as a reference group inferential statistics allow one to determine if the proportion of BA/CPT or CPT/BA participants who met criteria for PTSD, MDD, and good end-state functioning differed significantly from the proportion observed in CPT.

PTSD and MDD Diagnostic Status and Good End-State Functioning: Intent-to-Treat Sample

Descriptive statistics for the ITT sample are presented in Table 3.7, and pooled inferential statistics are presented in Table 3.8. When interpreting Table 3.8 the 'Intercept' relates to the CPT reference group and as such, statistics reported in Table 3.8 demonstrate to which extent proportions of participants who met criteria for PTSD, MDD, or good end-state functioning in BA/CPT and CPT/BA differed significantly to proportions reported in CPT. As the limitations of reduced statistical power are emphasised when analysing binary outcomes the importance of descriptive statistics and confidence intervals is accentuated.

Table 3.7
Imputed Proportion of Participants Who Met Criteria for PTSD, MDD, and Good End-State Functioning at Posttreatment and 6-Month Follow-Up: Intent-To-Treat Sample

		Posttreatment		6-Month Follow-up	
	n	Proportion (%)	95% Confidence Interval	Proportion (%)	95% Confidence Interval
PTSD diagnosis					
CPT	18	36.1	13.3 - 58.9	56.7	33 - 80.3
BA/CPT	15	70	46.0 - 94	54.7	28.6 - 80.7
CPT/BA	16	27.5	4.9 - 50	31.2	7.7 - 54.8
MDD diagnosis					
CPT	18	42.2	18.8 - 65.7	40	16.7 - 63.3
BA/CPT	15	70.7	46.9 - 94.5	53.3	27.2 - 79.5
CPT/BA	16	20	0 - 40.2	26.2	3.8 - 48.7
Good end-state					
CPT	18	38.9	15.7 - 62.1	17.8	0 - 36
BA/CPT	15	6.7	0 - 19.7	22.7	0.7 - 44.6
CPT/BA	16	53.1	27.9 - 78.3	38.1	13.6 - 62.7

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder.

Table 3.8

Pooled Inferential Statistics for Proportion of Participants Who Met Criteria for PTSD, MDD, and Good End-State Functioning at Posttreatment and 6-Month Follow-Up: Intent-To-Treat Sample

	Posttreatment			6-Month Follow-up		
	b	SEb	*p*	b	SEb	*p*
PTSD diagnosis						
Intercept	-0.57	0.51	-	0.27	0.60	-
CPT compared to BA/CPT	1.42	0.78	.07	-0.08	0.80	.92
CPT compared to CPT/BA	-0.41	0.79	.61	-1.01	0.88	.23
MDD diagnosis						
Intercept	-0.31	0.49	-	-0.42	0.56	-
CPT compared to BA/CPT	1.20	0.78	.13	0.54	0.87	.53
CPT compared to CPT/BA	-1.08	0.82	.20	-0.69	1.04	.52
Good end-state						
Intercept	-0.45	0.48	-	-1.57	0.73	-
CPT compared to BA/CPT	-2.19	1.14	.06	0.29	1.04	.79
CPT compared to CPT/BA	0.58	0.73	.43	1.08	0.92	.25

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder.

PTSD diagnosis. On the CAPS interview for the assessment of PTSD, all participants were positive for PTSD at pretreatment. For the ITT sample at posttreatment, the proportion of participants who maintained a PTSD diagnosis did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA. However, this may be a power issue. Descriptive statistics and confidence intervals demonstrated that compared to CPT and CPT/BA, BA/CPT participants were more likely to maintain a PTSD diagnosis at posttreatment. In fact, quite a large proportion (i.e., 70%) of BA/CPT participants maintained a PTSD diagnosis at posttreatment. The proportion of BA/CPT participants who met criteria for PTSD was 34% larger than that reported by CPT, and 42.5% larger than that reported by CPT/BA. Additionally, confidence intervals between BA/CPT and CPT did not overlap considerably, and confidence intervals between BA/CPT and CPT/BA only overlapped by 4%. This suggests that if BA/CPT was used as a reference group, the difference between BA/CPT and CPT/BA may have been

significant. The large difference between BA/CPT and CPT and CPT/BA proportions, and the small overlap between BA/CPT and CPT and CPT/BA confidence intervals suggests that meaningful differences existed in which BA/CPT participants were more likely to maintain a PTSD diagnosis at posttreatment compared to CPT and CPT/BA participants.

For the ITT sample at 6-month follow-up, PTSD diagnostic status did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA with the proportion of participants who met criteria for PTSD being more similar across conditions. In sum, results at posttreatment provide some evidence to suggest that compared to CPT and CPT/BA participants, BA/CPT participants were more likely to still meet criteria for PTSD. However, at follow-up the proportion of participants who maintained a PTSD diagnosis was more similar across conditions.

MDD diagnosis. On the MINI interview at pretreatment, all participants were positive for MDD. For the ITT sample at posttreatment, MDD diagnostic status did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA (see Tables 3.7 and 3.8). Although results were nonsignificant I further examined descriptive statistics to determine if this was again a power issue. Descriptive statistics did indeed demonstrate a meaningful difference in MDD diagnostic status at posttreatment. Specifically, the proportion of participants who met MDD criteria at posttreatment appeared to be meaningfully smaller for CPT/BA compared to CPT and BA/CPT. The proportion of CPT/BA participants who met MDD criteria at posttreatment was 22.2% smaller than that reported by CPT, and 50.7% smaller than that reported by BA/CPT. Supporting the notion that a meaningful difference existed, confidence interval for CPT/BA did not overlap greatly with CPT, and did not overlap at all with BA/CPT. Thus, at posttreatment there was evidence that CPT/BA reported a meaningfully smaller proportion of participants who met MDD criteria compared to CPT and BA/CPT.

The proportion of participants who maintained an MDD diagnosis at 6-month follow-up did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA. However it should be noted that the proportion of CPT/BA participants who maintained an MDD diagnosis at 6-month follow-up was still 27.1% smaller than that reported by BA/CPT.

Good end-state functioning. At posttreatment the proportion of participants in the ITT sample who met criteria for good end-state functioning did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA (see Tables 3.7 and 3.8). However, descriptive statistics and confidence intervals suggest that this may again be a power issues. Descriptive statistics demonstrated that a very small proportion of BA/CPT participants met criteria for good end-state functioning (i.e., 6.7%). The proportion of BA/CPT participants who met criteria for good end-state functioning was 32.2% smaller than that reported by CPT, and 46.4% than that reported by CPT/BA. BA/CPT and CPT confidence intervals only overlapped by 4%. Further, BA/CPT and CPT/BA confidence intervals did not overlap at all which suggests that if BA/CPT was used as a reference group, the difference between BA/CPT and CPT/BA may have been significant. Thus, results suggest that at posttreatment there were meaningful differences in proportions in which BA/CPT participants were less likely to meet criteria for good end-state functioning than CPT and CPT/BA participants. For the ITT sample at 6-month follow-up, the proportion of participants who met criteria for good end-state functioning did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA, and proportions were more similar across conditions.

Summary. Results suggested that compared to CPT and CPT/BA participants, BA/CPT participants were more likely to still meet criteria for PTSD and were less likely to meet criteria for good-end state functioning at posttreatment. Further, at posttreatment, compared to CPT and BA/CPT, CPT/BA reported a meaningfully smaller

proportion of participants who met MDD criteria. Proportions of participants who met criteria for PTSD, MDD, and good-end state functioning were more similar at follow-up. I now turn attention to the completer sample.

PTSD and MDD Diagnosis, and Good End-State Functioning: Completer Sample

Analyses were repeated on the completer sample. In line with results previously reported for completers, examination of PTSD and MDD diagnosis, and good end-state functioning generally suggested that if individuals completed treatment they were likely to lose their PTSD and MDD diagnosis, and achieve good end-state functioning (see Table 3.9 for descriptive statistics, and Table 3.10 for pooled inferential statistics).

Table 3.9

Imputed Proportion of Participants Who Met Criteria for PTSD, MDD, and Good End-State Functioning at Posttreatment and 6-Month Follow-Up: Completer Sample

		Posttreatment		6-Month Follow-up	
	n	Proportion (%)	95% Confidence Interval	Proportion (%)	95% Confidence Interval
PTSD Diagnosis					
CPT	8	0	- [a]	3.8	0 - 17.9
BA/CPT	6	31.7	0 - 72.2	23.3	0 - 59.9
CPT/BA	11	17.3	0 - 40.7	6.4	0 - 21.4
MDD Diagnosis					
CPT	8	12.5	0 - 37.0	0	- [a]
BA/CPT	6	30.0	0 - 69.8	0	- [a]
CPT/BA	11	8.2	0 - 25.1	0	- [a]
Good end-state					
CPT	8	62.5	26.6 - 98.4	35.0	0 - 70
BA/CPT	6	16.7	0 - 49.3	46.7	3.8 - 89.5
CPT/BA	11	72.7	45.1 -100	46.4	15.6 - 77.1

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder.
[a] As variance was small, and as all collected data demonstrated a proportion of zero, standard deviations and confidence intervals could not be calculated.

Table 3.10

Pooled Inferential Statistics for Proportion of Participants Who Met Criteria for PTSD, MDD, and Good End-State Functioning at Posttreatment and 6-Month Follow-Up: Completer Sample

	b	SEb	*p*
Good end-state – posttreatment			
Intercept	0.51	0.73	-
CPT compared to BA/CPT	-2.12	1.32	.12
CPT compared to CPT/BA	0.47	1.00	.64
Good end-state – 6-month follow-up			
Intercept	-0.64	0.89	-
CPT compared to BA/CPT	0.51	1.30	.70
CPT compared to CPT/BA	0.49	1.08	.65

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder.

PTSD and MDD diagnosis. Examination of descriptive statistics for PTSD and MDD diagnostic status illustrated that there was very little variability between treatment conditions in the proportion of completers who continued to meet PTSD and MDD diagnostic criteria at posttreatment and 6-month follow-up (see Table 3.9 for descriptive statistics). Specifically, proportion scores for PTSD and MDD diagnoses only ranged from 0 to 31.7% across conditions. Reduced variability in scores, and the small sample size (n = 25) therefore precluded meaningful examination with inferential statistics. As such, interpretation of PTSD and MDD diagnostic status in the completer sample relied on descriptive statistics and confidence intervals alone.

The proportion of completers who maintained a PTSD diagnosis at posttreatment and 6-month follow-up was similar and did not appear to differ meaningfully between CPT and BA/CPT and CPT/BA. Similarly, MDD diagnostic status did not appear to differ meaningfully between CPT and BA/CPT and CPT/BA completers at posttreatment and 6-month follow-up. In fact, at follow-up all completers no longer met

criteria for MDD. These results suggest that irrespective of condition, a large proportion of completers no longer met criteria for PTSD and MDD at posttreatment and follow-up. This again supports the proportion that if individuals completed treatment they were likely to show good outcomes.

Good end-state functioning. As proportions of good end-state functioning showed more variability between treatment conditions inferential statistics were calculated. Analyses conducted on the ITT sample were repeated for completers. The limitations of a small sample and reduced power should be kept in mind and attention should be paid to descriptive statistics and confidence intervals. Proportions are reported in Table 3.9, and inferential statistics are presented in Table 3.10.

For completers at posttreatment, no significant difference emerged between CPT and BA/CPT, and CPT and CPT/BA on good end-state functioning. However, examination of descriptive statistics and confidence intervals suggests that this may relate to power. Similar to results seen in the ITT sample, only a small proportion of BA/CPT completers met criteria for good end-state functioning at posttreatment (16.7%). The proportion of BA/CPT participants who met criteria for good end-state functioning at posttreatment was 45.8% smaller than that reported by CPT, and 56% smaller than that report by CPT/BA. Additionally, confidence intervals between BA/CPT and CPT did not overlap considerably suggesting a meaningful difference. Furthermore, BA/CPT and CPT/BA confidence intervals only overlapped by 4.2% that suggests that if BA/CPT was used as a reference group the difference between BA/CPT and CPT/BA may have been significant. Taken together, posttreatment results suggest a meaningful difference in effect whereby BA/CPT completers were less likely to achieve good end-state functioning at posttreatment compared to CPT and CPT/BA completers. The proportion of completers who met criteria for good end-state functioning at 6-

month follow-up did not differ significantly between CPT and BA/CPT, and CPT and CPT/BA.

To summarise, completer results suggest that if participants completed treatment they were likely to no longer meet criteria for PTSD and MDD at posttreatment and follow-up irrespective of condition. Additionally, CPT and CPT/BA participants were particularly likely to achieve good end-state functioning at posttreatment.

Why Did BA/CPT Fail to Produce Superior Outcomes?

Results so far refute Hypothesis 2 and 3. With respect to Hypothesis 2, while CPT/BA was found to be more effective than CPT, this was not the case for BA/CPT. Also, refuting Hypothesis 3, CPT/BA produced effects larger than that demonstrated by BA/CPT. Put simply, results failed to find support for the superiority of BA/CPT and have rather suggested that CPT/BA is actually the treatment of choice. As results refuted predictions there is a need to explore the course of symptom change during BA/CPT.

Hypothesis 3 predicted that BA/CPT participant would show superior treatment outcomes compared to CPT/BA participants as initial BA sessions would target and reduce depressive symptoms (i.e., low mood, rumination, and emotional numbing), and consequently place participants in a better position to engage in CPT. Thus, to gain a better understanding of results I explored the initial efficacy of BA sessions, and determined if initial BA sessions did indeed reduce depressive symptoms and place individuals in a better position to undertake CPT. Specifically, I was interested in determining: 1) the efficacy of initial BA sessions, 2) if initial BA sessions did prepare participants to benefit from CPT, and 3) if initial BA sessions led to better treatment outcomes than initial CPT sessions.

Analysis of mid-phase assessments addresses these questions as mid-phase assessments were taken after the initial BA component for BA/CPT participants (after

Session 5), after the initial CPT component for CPT/BA participants (after Session 10), and after Session 5 for CPT participants.[11] As Hypothesis 2 and 3 predicted the superiority of BA/CPT over CPT and CPT/BA, one would expect that initial BA sessions would lead to larger symptoms reductions than initial CPT sessions. That is, BA/CPT participants would report greater symptom reductions on mid-phase measures than CPT and CPT/BA participants. Additionally, in line with Hypothesis 3 and the notion that initial BA sessions should reduce depressive symptoms, one would also expect initial BA sessions to produce meaningful changes on depressive symptoms such as low mood, rumination, and emotional numbing.

Analyses were carried out only on those who completed the mid-phase assessment. Imputations were not made for missing mid-phase scores as such scores were only missing if participants were no longer engaged in treatment. That is, all participants present at the mid-phase assessment point completed mid-phase questionnaires. It should be noted that as all dropout occurred prior to Session 6 the analysed sample was comprised solely of completers. As imputations were not required, I examined pre- to mid-phase changes on the PCL, DASS-D, PTCI, RRS, SRRS, EN and TAS with 3 (Condition: CPT, BA/CPT, CPT/BA) × 2 (Time: pretreatment, mid-phase) mixed, repeated-measures ANOVAs conducted in SPSS. Descriptive statistics, inferential statistics, and effect sizes are presented in Tables 3.11 to 3.13. Given the small sample size (n = 25) particular emphasis is given to effect sizes and confidence intervals.

[11] As mid-phase assessments occurred at different points for the different conditions (i.e., after Session 5 for CPT and BA/CPT, and after Session 10 for CPT/BA), and as CPT and BA/CPT participants completed mid-phase assessments after a similar number of sessions, the comparison of CPT and BA/CPT is of particular importance.

Table 3.11

CPT, BA/CPT, and CPT/BA Means, Standard Deviations, and Sample Sizes at Mid-phase Assessment

		Pretreatment			Mid-phase Assessment		
Measure	Condition	*n*	*M*	*SD*	*n*	*M*	*SD*
PCL	CPT	18	56.06	11.28	8	39.50	12.13
	BA/CPT	15	60.20	7.49	6	58.00	15.23
	CPT/BA	16	61.12	10.35	11	30.64	9.01
DASS – D	CPT	18	21.11	10.68	8	16.00	7.86
	BA/CPT	15	21.20	9.91	6	21.33	9.09
	CPT/BA	16	26.12	9.76	11	10.09	10.85
PTCI	CPT	18	130.67	36.62	8	111.75	39.80
	BA/CPT	15	139.27	39.87	6	143.67	25.85
	CPT/BA	16	152.94	26.36	11	90.00	14.55
RRS	CPT	18	51.94	13.97	8	48.12	15.05
	BA/CPT	15	57.07	13.71	6	56.17	10.19
	CPT/BA	16	61.88	11.28	11	47.82	14.60
SRRS	CPT	18	1159.17	462.47	8	968.13	466.95
	BA/CPT	15	1310.33	439.30	6	1395.00	539.21
	CPT/BA	16	1546.25	313.98	11	849.55	579.64

Measure	Condition	Pretreatment			Mid-phase Assessment		
		n	*M*	*SD*	*n*	*M*	*SD*
EN	CPT	18	25.67	7.93	8	21.88	8.24
	BA/CPT	15	28.47	4.67	6	26.67	5.88
	CPT/BA	16	28.62	5.15	11	15.60	9.62
TAS	CPT	18	55.28	12.41	8	56.13	18.13
	BA/CPT	15	62.47	9.80	6	67.67	10.53
	CPT/BA	16	60.94	10.67	11	43.54	12.04

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table 3.12

Inferential Statistics for Pre- to Mid-phase Outcomes

Measure	*df*	*F*	*p*
PCL			
Time (T)	1	53.41	< .001
Condition (C)	2	5.12	.02
C × T	2	14.05	< .001
DASS – D			
T	1	23.75	< .001
C	2	2.03	.16
C × T	2	5.95	.01
PTCI			
T	1	10.56	.004
C	2	2.72	.09
C × T	2	5.43	.01
RRS			
T	1	5.85	.02
C	2	1.48	.25
C × T	2	1.50	.25
SRRS			
T	1	9.55	.01
C	2	2.19	.14
C × T	2	5.90	.01
EN			
T	1	14.25	.001
C	2	2.32	.12
C × T	2	1.91	.17
TAS			
T	1	2.89	.10
C	2	3.32	.06
C x T	2	7.47	.003

Note. PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table 3.13

Effect Sizes (Cohen's d) [and 95% Confidence Intervals for Effect Sizes] from Pre- to Mid-phase: Intent-to-Treat Sample

Measure	CPT (*n* = 8)	BA/CPT (*n* = 6)	CPT/BA (*n* = 11)
PCL	1.44 [0.50, 2.34]	0.22 [-0.73, 1.17]	3.10 [1.94, 4.23]
DASS - D	0.51 [-0.34, 1.35]	-0.01 [-0.96, 0.84]	1.57 [0.68, 2.44]
PTCI	0.50 [-0.35, 1.34]	-0.12 [-0.96, 0.94]	2.81 [1.71, 3.89]
RRS	0.27 [-0.57, 1.10]	0.07 [-0.87, 1.00]	1.11 [0.27, 1.93]
SRRS	0.41 [-0.44, 1.25]	-0.18 [-0.96, 0.93]	1.58 [0.69, 2.45]
EN	0.47 [-0.38, 1.31]	0.36 [-0.60, 1.29]	1.79 [0.86, 2.68]
TAS	-0.06 [-0.89, 0.77]	-0.52 [1.47, 0.44]	1.55 [0.66, 2.42]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

In order to determine the efficacy of initial BA sessions, and in order to determine if BA/CPT participants reported greater symptom reductions on mid-phase measures compared to CPT and CPT/BA participants, Condition × Time interactions were the analyses of interest. Significant Condition × Time interactions emerged on all measures other than the RRS and EN. Descriptive statistics and effect sizes suggested that compared to CPT and BA/CPT, CPT/BA demonstrated larger effects on such measures. CPT/BA demonstrated effects on the PCL, DASS-D, PTCI, and SRRS 2.15 to 5.14 times larger than that reported by CPT, and the effect size for the TAS was 25.83 times larger for CPT/BA. Further, CPT/BA demonstrated effects on the PCL, PTCI, SRRS,

and TAS that were 2.98 to 21.42 time larger than those reported by BA/CPT and differences on the DASS-D were even more pronounced (CPT/BA *d* = 1.57 vs. BA/CPT *d* = -0.01). Thus, results suggest that at mid-phase, compared to CPT and BA/CPT, CPT/BA produced significantly larger effects on the PCL, DASS-D, PTCI, SRRS, and TAS. Further, and most important to the current discussion, results suggest that initial BA session did not reduce depressive symptoms (i.e., DASS-D) to the same extent as initial CPT sessions for CPT/BA participants. These findings refute the prediction that initial BA sessions would lead to larger symptom reductions than initial CPT sessions.

However, as CPT/BA received a greater number of sessions at mid-phase assessment it is not surprising to see such results, and one cannot rule out the possibility that the superior outcomes achieved by CPT/BA were merely the product of treatment dose. Consequently, I undertook planned comparisons and directly compared mid-phase assessments for CPT and BA/CPT. This allowed me to determine if five initial BA sessions led to larger symptom reductions than five initial CPT sessions.

Planned comparisons suggested that initial CPT sessions led to meaningfully larger reductions on PTSD, MDD, and secondary symptoms than initial BA sessions. More explicitly, planned comparisons demonstrated a significant difference between CPT and BA/CPT on the PCL (p = .01, d = 0.68, CI d = [-0.43, 1.76]), PTCI (p = .04, d = 0.50, CI d = [-0.59, 1.57]), and SRRS (p = .048, d = 0.45, CI d = [-0.63, 1.51]) at mid-phase. CPT demonstrated an effect on the PCL that was 6.55 times larger than that achieved by BA/CPT. While BA/CPT achieved only a small change on the PCL by mid-phase, CPT achieved a very large change. Further, BA/CPT did not produce a meaningful change on the PTCI with the effect size being small and negative. CPT demonstrated an effect on the PTCI that was 4.17 times larger than that achieved by BA/CPT. The same pattern emerged for the SRSS. While, BA/CPT produced a small, negative effect on the SRRS, CPT produced a moderate effect that was 2.28 times larger

than that produced by BA/CPT. Thus, at mid-phase, and after the completion of BA, there was evidence that initial BA sessions did not produce large or meaningful reductions on the PCL, PTCI, and SRRS, and that CPT reported significantly larger mid-phase effects on these measures than BA/CPT. However, while differences were significant, as pairwise comparision effect size confidence intervals contain negative values one must keep in mind that it is possible that true effects were negligible.

No significant difference was found between CPT and BA/CPT on the DASS-D (p = .09, d = 0.43, CI d = [-0.65, 1.49]), RRS (p = .10, d = 0.12, CI d = [-0.94, 1.18]), EN (p = .23, d = 0. 18, CI d = [-0.89, 1.24]), and TAS (p = .09, d = 0.54, CI d = [-0.55, 1.61]) at mid-phase. Given the nonsignificant Condition × Time interactions on the RRS and EN, nonsignificant differences on these scores are not surprising. However, it should be noted that although interactions and pairwise comparisons were nonsignificant for the RRS, EN, and TAS, initial BA sessions only had a small effect on the RRS and EN, and appeared to increase TAS scores. This is surprising given that rumination and emotional numbing are specifically associated with MDD, and BA is designed to target MDD.

Further examination of effect sizes and confidence intervals suggested that for the DASS-D nonsignificant findings might have been a power issue. While BA/CPT did not appear to produce a meaningful change on the DASS-D, with the effect size being only -0.01, CPT produced a moderate effect of 0.51. In other words, CPT produced an effect on the DASS-D 51 times larger than that achieved by BA/CPT. This should be considered a meaningful difference. As BA is designed to target depressive symptoms it is interesting to see such a small effect on the DASS-D. Such a finding explicitly refutes the prediction that initial BA sessions would reduce MDD severity, and subsequently place individuals in a better position to undertake CPT. However, as noted previously,

one must keep in mind that as confidence intervals contain negative values it is possible the true effects were small.

To recap, mid-phase findings suggest that initial BA sessions did not produce meaningful change on a number of outcomes, including depressive symptoms (i.e., DASS-D, RRS, EN). Further, compared to BA/CPT, CPT and CPT/BA consistently produced larger effects on PTSD, MDD, and secondary outcomes by mid-phase. Thus, findings indicate that initial BA sessions did not meaningfully reduce depressive symptoms, and consequently did not better prepare participants for CPT. In line with this conclusion, it is also worth highlighting that within group effect sizes at posttreatment and follow-up were similar for CPT and BA/CPT (see Table 3.3 pg. 89). That is, the efficacy of CPT, or reductions in PTSD, MDD, and secondary symptoms, did not differ meaningfully between those who had initial BA sessions (i.e., BA/CPT) and those that did not (i.e., CPT alone). This further supports the notion that initial BA sessions did not prepare clients to better engage in CPT, nor boost the efficacy of CPT.

Contrary to Hypothesis 3, these findings suggest that BA/CPT may have failed to produce superior outcomes as initial BA sessions did not prepare BA/CPT participants for subsequent CPT sessions, and because initial BA sessions did not meaningfully reduce depressive symptoms. In fact, results suggest that rather than initial BA sessions increasing the efficacy of CPT, initial CPT sessions may have enhanced the efficacy of BA. In line with this, it may also be the case that by reducing PTSD symptoms first (as was seen in CPT/BA) subsequent change in MDD and secondary symptoms was facilitated. Such an explanation is in line with the superior outcomes demonstrated by CPT/BA.

Supplementary Analyses

I also carried out supplementary analyses to explore condition differences in credibility/expectancy and working alliance, and to examine the effect of order of PTSD and MDD onset on treatment outcome. I now provide a brief summary of findings and results are reported in detail in Appendix L.

Credibility/Expectancy of Treatment and Working Alliance

I explored credibility/expectancy and working alliance as a potential explanation for the superiority of CPT/BA. At pretreatment, although nonsignificant, effect sizes from pairwise comparisons suggested that compared to CPT and BA/CPT participants, CPT/BA participants reported meaningfully higher credibility/expectancy scores. Pretreatment credibility/expectancy did not meaningfully differ between CPT and BA/CPT participants. Working alliance did not appear to significantly or meaningfully differ between CPT, BA/CPT and CPT/BA at pretreatment.

At posttreatment, CPT/BA participants reported significantly higher credibility/expectancy scores than CPT participants, and meaningfully (although nonsignificant) higher credibility/expectancy scores than BA/CPT participants. Further, BA/CPT reported meaningfully (although nonsignificant) higher credibility/expectancy scores than CPT participants. No significant or meaningful condition differences emerged on working alliance at posttreatment.

These findings suggest that differences in credibility/expectancy may in part explain CPT/BA superiority. However, as effects were not large (and in most cases nonsignificant), and as the sample size was small, findings require replication. Working alliance did not appear to meaningfully differ between conditions and as such working alliance does not account for treatment differences.

Order of PTSD and MDD Onset

I then examined if order of PTSD and MDD onset influenced treatment outcomes in participants (see Appendix L for full details). To enhance sample size and power, treatment conditions were collapsed. PTSD and MDD treatment outcome at posttreatment and follow-up did not differ significantly between participants who reported PTSD onset prior to MDD onset (PTSD/MDD onset), participants who reported MDD onset prior to PTSD onset (MDD/PTSD onset) or, participants who reported simultaneous PTSD and MDD onset (simultaneous PTSD/MDD onset). Thus, results suggested that order of onset did not meaningfully influence PTSD and MDD outcome. The following chapter explores the processes underlying treatment change and specifically examines the effects of under-, over- and optimal levels of emotional engagement on PTSD and MDD treatment outcome.

Chapter 4: Emotional Engagement Results

Restatement of Aims Related to Emotional Engagement and Coding Procedures

In addition to evaluating the efficacy of CPT, BA/CPT, and CPT/BA, in this this thesis I also explored the *processes* underlying treatment change and explicitly examined the relationship between emotional engagement and PTSD and MDD treatment outcome. To recap, I aimed to:

1. Determine whether under- and overengagement reflected similar, overlapping processes or distinct, separate processes.
2. Determine how under- and overengagement operated during treatment (e.g., do individual have a tendency to either under- or overengage during sessions, or do individuals demonstrate both under- and overengagement during sessions).
3. Determine the effects of under-, over-, and optimal engagement on PTSD and MDD treatment outcomes, and explore whether under- and overengagement had a different impact on outcome.
4. Examine whether the relationship between under-, over-, and optimal engagement and PTSD and MDD treatment outcome differed between CPT, BA/CPT, and CPT/BA.
5. Determine the convergent and discriminant validity of self-reported subjective units of distress scores (SUDS) and objectively coded Client Expressed Emotional Arousal Scale (CEAS) scores.

As explained in Chapter 2, and as illustrated in Appendix F, all therapy sessions were video recorded and every minute of every session was coded for emotional engagement using the Client Expressed Emotional Arousal Scale-III (CEAS; Warwar & Greenberg, 1999). The CEAS allowed each recorded minute to be coded on a scale

ranging from 1 to 7. A score of 1 indicated a total lack of emotional engagement while a score of 7 indicated extreme emotional overengagement. Each minute was given an emotional label, a peak rating, and a modal rating. Peak scores reflected the highest level of arousal present during the coded minute. Modal scores reflected a participant's overall, average level of arousal during the coded minute. In Chapter 2 correlations of peak and modal scores demonstrated that peak scores underestimated proportions of underengagement, and overestimated proportions of overengagement. Accordingly, I used modal scores for analyses as I wanted to ensure that the measure used was sensitive to levels of underengagement and did not conceal or minimise proportions of underengagement. I also selected modal scores as I was interested in participants' overall level of engagement rather than momentary elevations or changes in engagement. CEAS scores of 1 and 2 reflected underengagement, scores of 3 and 4 reflected optimal engagement, and scores of 5 to 7 reflected overengagement. For each session a proportion score was calculated that represented the proportion of the session a participant spent under-, over-, or optimally engaged. Proportion scores were calculated by dividing the raw number of minutes spent under-, over-, and optimally engagement, by the total number of minutes in the session, and multiplying this by 100. For example, if a participant underengaged (i.e., gained a CEAS rating of 1 or 2) for 30 minutes of a 60-minute session this translated to an underengagement proportion scores of 50. Proportion scores were used in the undertaken analyses.

Preliminary Analyses: Connection Between Under- and Overengagement

Past research has examined emotional engagement as a unified construct and has failed to determine if under- and overengagement are in fact separate or distinct states. Further, research has not examined if individuals are likely to either underengage or

overengage during treatment, or if individuals are likely to move between the two states during treatment. It is surprising that past research has not addressed these issues as such questions directly influence data analysis strategies. For instance, if correlations show under- and overengagement to be separate states that are not strongly, or reliably related to one another, coding and analysis techniques currently being used must be adapted to explore under- and overengagement separately.

When examining the relationship between under- and overengagement three possibilities exist:

1. Individuals show a common underlying problem with engagement whereby those who are more or less likely to overengage will also be more or less likely to underengage. This would be reflected by a positive correlation between proportion scores of under- and overengagement.
2. Under- and overengagement are separate states and an individual's propensity to underengage is separate from their propensity to overengage, and vice versa. This would be reflected by proportion scores of under- and overengagement not being meaningfully correlated with one another.
3. Under- and overengagement reflect the same construct such that when underengagement is high overengagement will be low, when underengagement is moderate overengagement will also be moderate, and when underengagement is low overengagement will be high. This would imply that individuals have a tendency towards either under- or overengagement. This would be reflected by a negative correlation between proportion scores of under- and overengagement.

In order to address Aims 1 and 2, I correlated proportions of under- and overengagement. Correlation coefficients are presented in Table 4.1. The correlations of under- and overengagement during the same treatment session are of primary interest. Such correlations are presented in bold in Table 4.1. With regards to the relationship between under- and overengagement during the same treatment session (bolded correlations) correlations ranged from -.33 to .14, with no correlation reaching significance. As this thesis was underpowered, and as meaningful results may have therefore not reached significance, explicit attention should be paid to the magnitude and direction of correlations. Although nonsignificant, correlations of under- and overengagement during the same treatment session spanned from small and negative to very small and positive. Thus, for some sessions participants demonstrated a tendency towards both under- and overengagement, while in other sessions participants demonstrated higher proportions of underengagement and lower proportions of overengagement, or vice versa. That is, in some sessions under- and overengagement acted as separate constructs and in other sessions they were more similar. Putting this together, as correlations were small and nonsignificant results suggest that under- and overengagement are indeed separate constructs. However, the results also suggest that the relationship between under- and overengagement is complex and that this relationship is not always consistent across sessions. These results highlight the importance of examining under- and overengagement as separate constructs.

I next examined the relationship between under- and overengagement over the course of treatment. Correlations during the same treatment session should again be examined (see bolded values in Table 4.1). As treatment progressed correlations, although nonsignificant, moved from small and negative to very small and positive. More explicitly, early on in therapy participants demonstrated a small tendency towards

Table 4.1

Correlation of Proportion of Under- and Overengagement During Each Treatment Session

	Session 1 UE	Session 2 UE	Session 3 UE	Session 4 UE	Session 5 UE	Session 6 UE	Session 7 UE	Session 8 UE	Session 9 UE	Session 10 UE	Session 11 UE	Session 12 UE	Session 13 UE	Session 14 UE	Session 15 UE
Session 1 OE	**-.22**														
Session 2 OE	-.26	**-.27**													
Session 3 OE	-.27	-.27	**-.33**												
Session 4 OE	-.23	-.33	-.36	**-.27**											
Session 5 OE	-.35	-.46*	-.35	-.23	**-.30**										
Session 6 OE	.01	-.36	-.27	-.07	-.17	**-.29**									
Session 7 OE	-.26	-.28	-.26	-.25	-.20	-.26	**-.25**								
Session 8 OE	.02	-.27	-.24	-.15	-.14	-.21	-.13	**-.06**							
Session 9 OE	-.33	-.12	-.26	-.28	-.26	-.30	-.30	-.33	**-.31**						
Session 10 OE	-.24	.004	-.12	-.11	-.16	-.18	-.17	-.17	-.21	**-.16**					
Session 11 OE	-.25	-.18	-.15	-.21	-.21	-.17	-.27	-.37	-.22	-.23	**-.25**				
Session 12 OE	-.18	.03	-.09	-.22	-.07	-.14	-.15	-.19	-.08	-.15	-.15	**-.12**			
Session 13 OE	-.27	-.13	-.05	-.01	-.07	-.09	-.14	-.21	-.20	-.08	.04	.01	**.07**		
Session 14 OE	.13	.34	.81**	.81**	.63*	.66*	.39	.40	.44	.73**	.67*	.84**	.94**	**.14**	
Session 15 OE	-.32	-.14	-.09	-.03	-.12	-.16	-.17	-.27	-.24	-.13	.01	-.03	.03	.10	**.11**

Note. UE = Underengagement; OE = Overengagement. Bolded correlations are the main outcome of interest.
n = 42 - 19
* $p < 0.05$; ** $p < 0.01$.

either under- or overengagement as demonstrated by the small and negative correlation during early therapy sessions. However, as therapy progressed the negative correlation, and thus the tendency towards either under- or overengagement disappeared and under- and overengagement became more distinct constructs. This suggests that as participants progressed through therapy their tendency to either under- or overengage reduced, and that treatment appeared to disrupt the relationship between under- and overengagement. Although correlations were nonsignificant, as results were relatively consistent over a series of observations, the mean correlation is likely to be significantly different from zero suggesting a meaningful pattern of results.

In light of such findings I then examined correlations of under- and overengagement for CPT, BA/CPT, and CPT/BA separately to determine if the relationship between under- and overengagement differed between conditions (see Table 4.2). Completers were used for such analyses to ensure that correlations were not influenced by dropout. Again, correlations were largely nonsignificant. However, in turning attention to the magnitude and direction of correlations results suggested that for CPT and BA/CPT, correlations of under- and overengagement were largely negative for the first 10 sessions. This suggests that during the early stages of therapy CPT and BA/CPT participants demonstrated a tendency towards either under- or overengagement. However, as correlations were weak and nonsignificant results also suggest that this tendency did not completely determine or predict an individual's level of engagement and that individuals who demonstrated a tendency towards underengagement were still likely to overengage, or vice versa. CPT/BA participants did not demonstrate this tendency.

Correlations also indicated that as therapy progressed CPT participants appeared to lose their tendency towards either under- or overengagement while this was not the case for BA/CPT and CPT/BA. Or in other words, although the tendency towards

under- and overengagement reduced over the course of therapy for CPT, change was more flat for BA/CPT and CPT/BA. As this pattern of change only occurred for the CPT condition, and as correlations were generally weak it is hard to determine the impact of therapy on this tendency.

Table 4.2

Correlations of Under- and Overengagement During the Same Treatment Session by Condition: Completer Sample

Session	CPT	BA/CPT	CPT/BA
Session 1	-.31	-.21	-.32
Session 2	-.37	-.67	.03
Session 3	-.61	-.47	-.02
Session 4	-.77*	-[b]	.07
Session 5	-.45	-.16	-[b]
Session 6	-.37	-.42	-.09
Session 7	-.47	-.49	-.11
Session 8	-.45	.10	.14
Session 9	-.37	-.53	-.33
Session 10	.31	-.72	.05
Session 11	-.16	-.61	-.24
Session 12	-.10	-.30	-[b]
Session 13	.85	-.06	.24
Session 14	1**[a]	.54	-.19
Session 15	1**[a]	-[b]	-[c]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation.
[a] Under- and overengagement did not occur in this session
[b] As underengagement did not occur in this session correlations of under- and overengagement could not be conducted
[c] As overengagement did not occur in this session correlations of under- and overengagement could not be conducted
n = 11 - 4
* $p < 0.05$; ** $p < 0.01$.

In sum, results suggest that the relationship between under- and overengagement is complex. As results were generally weak and nonsignificant under- and overengagement should be considered separate constructs that appear to fluctuate largely independently. Further, results provide some indication that CPT and BA/CPT participants demonstrated a tendency towards under- or overengagement, and that CPT reduced this tendency throughout therapy. Future research with larger samples is needed to explore this in more detail.

Effect of Under-, Over-, and Optimal Emotional Engagement on PTSD and MDD Outcome Over the Course of Treatment

I used mixed-effects models[12] to analyse the relationship between treatment outcome and each of under-, over-, and optimal emotional engagement in the ITT sample (Aims 3 and 4). Mixed-effects models were used as this technique extends on regressions and allows each recorded outcome assessment to be used as a data point rather than relying on the use of aggregated scores. Specifically, a mixed-effects modelling approach allowed for PCL and DASS-D scores collected every second session to be used and entered into the model. All mixed-effects models used a normal distribution with PCL and DASS-D scores collected every second session as outcome. I used likelihood ratio tests to assess the extent to which the addition of predictors improved the fit of the model, and as a basis for determining the best-fitting model, and profile likelihood confidence intervals for inference regarding fixed effect parameters within the model of best fit. I concluded that a coefficient was significantly different from zero when that coefficient's confidence interval did not include zero.

The interpretation of the models themselves is essentially equivalent to that of regressions. I assessed whether a predictor should be included in the model of best fit by

[12] All mixed-effects models were created using the `lme4` package (Bates, Maechler, Bolker, & Walker, 2013) in `R`, an open-source language and environment for statistical computing (R Development Core Team, 2011).

examining the improvement in the fit when the variable was added as a predictor. The most complex model that was a significant improvement in fit was the model of best fit. As in standard regression, the relationship between a predictor and the outcome is described by the regression coefficient (*b*) that represents the amount of change in the outcome with a 1-unit change in the predictor (in this case changes in PCL and DASS-D scores with every 1-unit change in engagement).

The reader is reminded that PCL and DASS-D scores were collected every second session, rather than every session. As such, proportions of under-, over-, and optimal engagement during the two proceeding treatment sessions were collated and I then examined how well these proportions predicted the next collected PCL and DASS-D outcome. A simple way of thinking of this is that proportions of under-, over-, and optimal engagement for the two preceding sessions were essentially calculated as though the two preceding sessions were one long session. For example, Session 1 and Session 2 were collapsed and proportions of under-, over-, and optimal emotional engagement during these two sessions were collated. I then examined how well these proportions of under-, over- and optimal engagement predicted PCL and DASS-D scores collected at Session 2. As scores were organised in such a way proportions of engagement during Session 15 were not included in analyses.

In sum, I created a mixed-effects model with PCL and DASS-D as the outcome, under-, over-, or optimal engagement as a fixed-effect, and session as a random effect. This allowed me to assess the relationship between engagement and the outcome simultaneously across all sessions. This creates two important advantages. First, including data from all sessions produces a more reliable estimate of the relationship in question. Second, we can directly examine the extent to which the relationship differs from session to session. CPT was used as the comparison condition (i.e., reference group). Analyses were conducted on the ITT sample.

As treatment structure differed between conditions with behavioural activation and trauma account exposure sessions occurring at different sessions for the different conditions, one may suggest that the relationship between emotional engagement and treatment outcome would of course vary across sessions for the different conditions. This is a valid point and if this were indeed the case it would not be appropriate to examine the relationship between engagement and outcome simultaneously across all sessions. Consequently, to test such a proposition I conducted initial analyses in which the relationship between emotional engagement and PCL and DASS-D outcome was allowed to vary between sessions. No evidence was found to suggest that the model obtained when the slope for each session was permitted to vary provided a significantly better fit than the model obtained when the slope was the same across sessions. I therefore report analyses that include random intercept, but not slope, models. This allowed the overall relationship between emotional engagement and treatment outcome to be examined. Coefficients are displayed in Tables 4.3, 4.6, and 4.7, and interactions displayed in Figures 4.1 through 4.3. I created plots such that x-axis scores (i.e., proportions of under-, over-, or optimal engagement) covered two standard deviations below through to two standard deviations above the mean for each treatment condition. A total of 392 therapy sessions (sessions 1 to 14) were coded and analysed.

Underengagement and Treatment Outcome

The relationship between underengagement and PCL and DASS-D scores was examined. I first created a mixed-effects model with PCL as the outcome (see Table 4.3 for coefficients). Adding proportion of underengagement did not improve the fit of the model, $\chi^2(1) = 2.01$, $p = .16$. The addition of condition $\chi^2(2) = 14.57$, $p < .001$ significantly improved the fit of the model. Differences in condition are not surprising given that results in Chapter 3 revealed outcome differences between conditions.

Further, as the underengagement fixed effects confidence interval did not include zero, the results indicated that there was a significant relationship for CPT in which higher proportions of underengagement predicted higher PCL scores. The addition of the Underengagement × Condition interaction $\chi^2(2) = 8.10$, $p = .02$ also significantly improved the fit of the model. Examination of Figure 4.1 revealed that for CPT and CPT/BA, elevated proportions of underengagement predicted elevated PCL scores. However, for BA/CPT underengagement did not predict PCL scores. In other words, BA/CPT participants were less sensitive to the effects of underengagement.

Table 4.3

Fixed Effect Coefficients for Best Fitting Mixed-Effects Models Predicting PCL and DASS-D Scores for Underengagement

Fixed effect	*b*	SE_b	95% CI[a]
PCL Outcome			
Intercept	33.62	4.02	[25.39, 41.77]
Underengagement	0.26	0.11	[0.06, 0.47]
Condition (C): BA/CPT	12.38	2.74	[7.04, 17.69]
C: CPT/BA	2.58	2.37	[-2.05, 7.18]
Underengagement × BA/CPT	-0.38	0.14	[-0.66, -0.10]
Underengagement × CPT/BA	0.13	0.38	[-0.60, 0.87]
DASS-D Outcome			
Intercept	11.75	2.17	[7.40, 15.99]
Underengagement	0.17	0.08	[0.002, 0.33]
C: BA/CPT	3.40	2.18	[-0.86, 7.61]
C: CPT/BA	0.19	1.88	[-3.51, 3.83]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL: Posttraumatic Stress Disorder Check List; DASS-D = Depression Anxiety and Stress Scale - Depression Subscale.

[a] Confidence intervals are 95% profile likelihood confidence intervals.

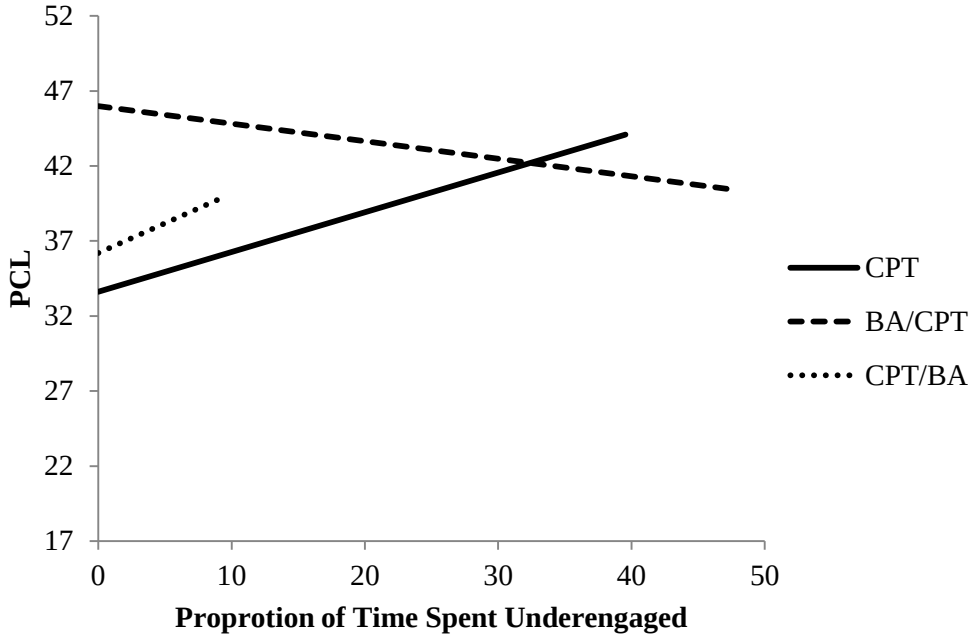

Figure 4.1. Plots of predicted PCL scores by proportion of time spent underengaged per session, for each treatment condition. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL = Posttraumatic Stress Disorder Checklist.

To explore the influence of underengagement on depression outcome a mixed-effects model with DASS-D as the outcome was created (see Table 4.3 for coefficients). Underengagement $\chi^2(1) = 1.24$, $p = .26$, condition $\chi^2(2) = 2.45$, $p = .29$, as well as their interaction $\chi^2(2) = 4.73$, $p = .09$, were added to the model. This did not significantly improve the fit of the model.

Supplementary analyses. I examined descriptive statistics to aid in interpretation of the interaction (see Table 4.4). First, descriptive statistics indicated that the proportion of underengagement experienced by participants remained low, with the overall, average proportion of underengagement being only 6.84 (SD = 14.40). This suggests that higher, or more extreme levels of underengagement were not consistently exhibited. Further, descriptive statistics and Figure 4.1 suggested that compared to CPT and BA/CPT, CPT/BA demonstrated a lower mean proportion of underengagement. To examine this further I created a mixed-effects model with underengagement as the

outcome and condition as the fixed-effect. Adding condition significantly improved the fit of the model, $\chi^2(2) = 10.19$, $p = .01$ (see Table 4.5 for coefficients). Coefficients and confidence intervals indicated that CPT/BA participants were less likely to underengage than CPT and BA/CPT participants. These findings suggest that while underengagement predicted elevated PCL scores for CPT/BA participants, CPT/BA participants were also less likely to underengage. This is in line with treatment outcomes presented in Chapter 3. More explicitly, although underengagement was detrimental for CPT/BA participants when it did occur, as CPT/BA was associated with lower levels of underengagement these participants were still able to achieve superior PTSD outcomes.

Table 4.4

Range, Mean, and Standard Deviations for Proportion of Underengagement, Overengagement and Optimal Engagement by Condition

Condition	Mean Proportion	Standard Deviation	Proportion Range
		Underengagement	
CPT	9.50	15.02	0 - 59.13
BA/CPT	9.80	18.73	0 - 79.35
CPT/BA	1.52	3.79	0 - 17.79
		Overengagement	
CPT	8.64	11.83	0 - 52.54
BA/CPT	8.12	12.60	0 - 66.67
CPT/BA	11.35	16.32	0 - 79.51
		Optimal Engagement	
CPT	81.86	15.67	37.78 – 100
BA/CPT	82.07	19.63	20.65 – 100
CPT/BA	83.63	16.27	20.49 – 100

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation.

Table 4.5

Fixed Effect Coefficients for Best Fitting Mixed-Effects Predicting Underengagement for Condition

Fixed effect	*b*	SE_b	95% CI[a]
Intercept	9.33	1.73	[5.89, 12.71]
Condition (C): BA/CPT	0.28	2.48	[-4.57, 5.14]
C: CPT/BA	-7.67	2.16	[-12.16, -3.67]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation.
[a] Confidence intervals are 95% profile likelihood confidence intervals.

In sum, underengagement was found to predict reduced PCL, but not DASS-D outcome over the course of treatment. Interestingly, the relationship between underengagement and PCL outcome differed by condition. Although elevated proportions of underengagement predicted reduced PCL outcomes for CPT and CPT/BA participants, this was not the case for BA/CPT participants. Or, put differently, BA/CPT participants were not as sensitive to the effects of underengagement. Supplementary analyses helped explain treatment outcomes suggesting that although underengagement predicted reduced PCL outcomes for CPT/BA participants, these participants were also less likely to underengage.

Overengagement and Treatment Outcome

I next examined the influence of overengagement on PCL and DASS-D outcome through mixed-effects modelling (see Table 4.6 for coefficients). A model was first created with PCL as the outcome. The addition of overengagement $\chi^2(1) = 19.42$, $p < .001$, and condition $\chi^2(2) = 23.38$, $p < .001$, significantly improved that fit of the model. The positive overengagement and condition fixed effect coefficients indicate a positive relationship whereby elevated proportions of overengagement predicted elevated PCL scores. The addition of the Overengagement × Condition interaction $\chi^2(2) = 3.64$,

p = .16, did not improve the fit of the model. Therefore, there was insufficient evidence to conclude that the relationship between overengagement and PCL scores differed by condition.

Table 4.6

Fixed Effect Coefficients for Best Fitting Mixed-Effects Models Predicting PCL and DASS-D Scores for Overengagement

Fixed effect	b	SE_b	95% CI[a]
	PCL Outcome		
Intercept	33.43	3.68	[25.86, 40.94]
Overengagement	0.33	0.06	[0.21, 0.45]
C: BA/CPT	8.72	2.25	[4.32, 13.11]
C: CPT/BA	-1.52	2.01	[-5.47, 2.39]
	DASS-D Outcome		
Intercept	11.23	1.87	[7.46, 14.91]
Overengagement	0.26	0.05	[0.17, 0.36]
C: BA/CPT	1.31	1.77	[-2.15, 4.76]
C: CPT/BA	-3.52	1.58	[-6.65, -0.46]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL: Posttraumatic Stress Disorder Check List; DASS-D = Depression Anxiety and Stress Scale - Depression Subscale.
[a] Confidence intervals are 95% profile likelihood confidence intervals.

Analyses were repeated with DASS-D scores and the same pattern of results was observed (See Table 4.6). The addition of overengagement $\chi^2(1) = 21.35$, $p < .001$, and condition $\chi^2(2) = 9.47$, $p = .01$, significantly improved that fit of the model while the addition of the Overengagement × Condition interaction did not, $\chi^2(2) = 2.89$, $p = .34$. In line with PCL scores, a positive relationship existed between overengagement and DASS-D outcome whereby high overengagement was associated with high DASS-D scores. As the Overengagement × Condition interaction was nonsignificant there was

not sufficient evidence to suggest that the relationship between overengagement and DASS-D outcome differed between conditions. Thus, elevated levels of overengagement predicted reduced PTSD and MDD treatment outcome irrespective of treatment condition. Findings suggest that in general, overengagement is a detrimental therapeutic process that inhibits PTSD and MDD treatment outcome.

Optimal Engagement and Treatment Outcome

Results so far have examined the influence of under- and overengagement on treatment outcome. However, these analyses alone do not allow one to determine if it is the presence of under- and overengagement that influences treatment outcome, or if it the mere absence of optimal engagement that influences outcome. As such I also examined the relationship between optimal engagement and PTSD and MDD outcome. I again used mixed-effects modelling to determine the relationship between PTSD and MDD treatment outcome and optimal emotional engagement.

I first created a mixed-effects model with PCL as the outcome (see Table 4.7 for coefficients). The addition of optimal engagement $\chi^2(1) = 25.04$, $p < .001$, and condition $\chi^2(2) = 17.02$, $p < .001$, significantly improved the fit of the model. While the significant effect of condition is not surprising given treatment outcomes reported in Chapter 3, the effect of optimal engagement is of particular interest. The negative coefficient for optimal engagement indicates a negative relationship between optimal engagement and PCL outcome whereby an elevated level of optimal engagement was associated with lower PCL scores (i.e., good treatment outcomes), while a reduced level of optimal engagement was associated with elevated PCL scores (i.e., poorer treatment outcomes). The addition of the Optimal Engagement × Condition interaction $\chi^2(2) = 24.13$, $p < .001$ improved the fit of the model. Examination of Figure 4.2 revealed that for CPT and CPT/BA elevated optimal engagement predicted lower PCL scores, and

low optimal engagement predicted elevated PCL scores. However, for BA/CPT optimal engagement did not predict PCL scores. That is, BA/CPT participants did not appear to be sensitive to the effects of optimal engagement.

I then repeated analyses with DASS-D scores (see Table 4.7 for coefficients). Results were similar to that achieved with PCL scores. The addition of optimal engagement improved the fit of the model $\chi^2(1) = 24.24$, $p < .001$. The negative coefficient indicates that elevated proportions of optimal engagement predicted reduced DASS-D scores, while reduced proportions of optimal engagement predicted elevated DASS-D scores. The addition of condition did not improve the fit of the model $\chi^2(2) = 3.15$, $p = .21$. The Optimal Engagement × Condition interaction was then added and this significantly improved the fit of the model $\chi^2(2) = 13.73$, $p = .001$. Examination of Figure 4.3 demonstrated that for CPT and CPT/BA elevated proportions of optimal engagement predicted lower DASS-D scores, and lower proportions of optimal engagement predicted higher DASS-D scores. However, optimal engagement did not significantly predict DASS-D scores for BA/CPT.

To gain a better understanding of the interaction between optimal engagement and condition I first examined descriptive statistics (see Table 4.4, pg. 138) and Figures 4.2 and 4.3. Average proportions of optimal engagement did not appear to differ between conditions. To examine this in more detail I created a mixed-effects model with optimal engagement as the outcome and condition as the fixed-effect. As suggested by descriptive statistics and Figures 4.2 and 4.3, adding condition did not significantly improved the fit of the model, $\chi^2(2) = 0.28$, $p = .87$ (see Table 4.8 for coefficients). Thus, proportions of optimal engagement did not appear to meaningfully differ between CPT, BA/CPT, and CPT/BA. As proportions of optimal engagement did not significantly differ between CPT, BA/CPT, and CPT/BA, differing proportions of optimal engagement do not account for the interaction.

Table 4.7

Fixed Effect Coefficients for Best Fitting Mixed-Effects Models Predicting PCL and DASS-D Scores for Optimal Engagement

Fixed effect	*b*	SE_b	95% CI[a]
	PCL Outcome		
Intercept	73.80	8.33	[57.77, 89.92]
Optimal Engagement	-0.45	0.09	[-0.64, -0.27]
C: BA/CPT	-34.00	10.61	[-54.67, -13.43]
C: CPT/BA	1.82	9.94	[-17.42, 21.24]
Optimal Engagement x BA/CPT	0.52	0.13	[0.27, 0.77]
Optimal Engagement x CPT/BA	-0.01	0.12	[-0.24, 0.22]
	DASS-D Outcome		
Intercept	38.66	6.43	[26.22, 51.15]
Optimal Engagement	-0.31	0.08	[-0.45, -0.16]
C: BA/CPT	-23.73	8.60	[-40.36, -6.94]
C: CPT/BA	0.02	8.10	[-15.59, 15.95]
Optimal Engagement × BA/CPT	0.30	0.10	[0.10, 0.50]
Optimal Engagement × CPT/BA	-0.02	0.10	[-0.21, 0.17]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL: Posttraumatic Stress Disorder Check List; DASS-D = Depression Anxiety and Stress Scale, Depression Subscale.
[a] Confidence intervals are 95% profile likelihood confidence intervals.

Table 4.8

Fixed Effect Coefficients for Best Fitting Mixed-Effects Predicting Underengagement for Condition

Fixed effect	*b*	SE_b	95% CI[a]
Intercept	82.68	2.75	[77.32, 88.21]
Condition (C): BA/CPT	-0.12	3.21	[-6.39, 6.62]
C: CPT/BA	1.17	2.80	[-4.28, 6.74]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation.
[a] Confidence intervals are 95% profile likelihood confidence intervals.

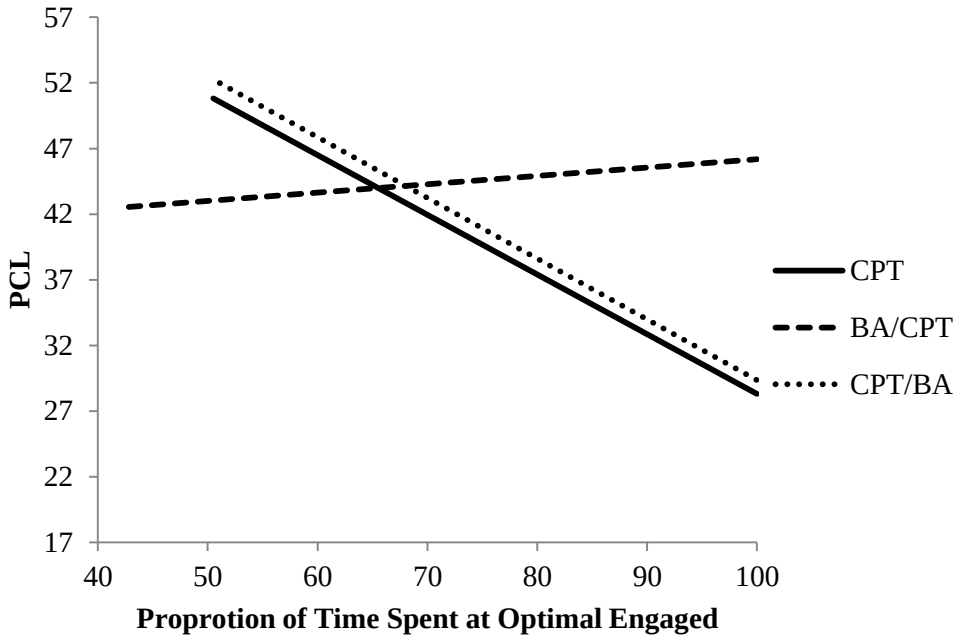

Figure 4.2. Plots of predicted PCL scores by proportion of time spent optimally engaged per session, for each treatment condition. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; PCL = Posttraumatic Stress Disorder Checklist.

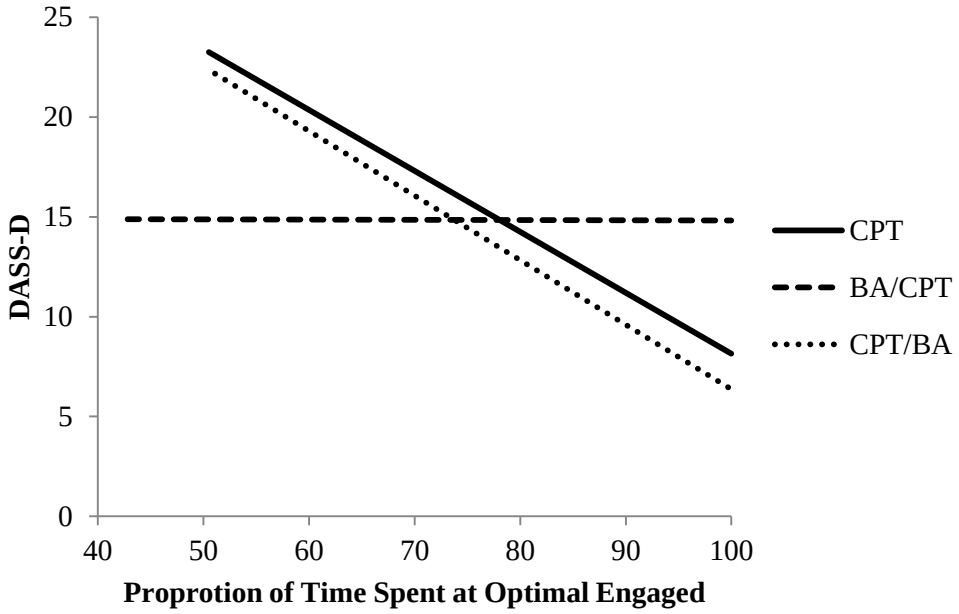

Figure 4.3. Plots of predicted DASS-D scores by proportion of time spent optimally engaged per session, for each treatment condition. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; DASS-D = Depression, Anxiety and Stress Scale – Depression Subscale.

To review, the relationship between optimal engagement and outcome was found to differ by condition. For CPT and CPT/BA participants, elevated levels of optimal engagement predicted better PTSD and MDD treatment outcomes, and reduced levels of optimal engagement predicted reduced PTSD and MDD outcome. However, for BA/CPT optimal engagement did not predict outcome. These findings highlight the importance of optimal engagement in achieving good PTSD and MDD outcomes for CPT and CPT/BA participants.

Summary of the Effects of Emotional Engagement on PTSD and MDD Outcome

Emotional engagement findings suggested that elevated levels of overengagement predicted elevated PTSD and MDD symptoms for all conditions. However, condition differences emerged in the effects of under- and optimal emotional engagement. For CPT and CPT/BA, elevated levels of underengagement predicted elevated PTSD (but not MDD) symptoms over the course of treatment. Further, for CPT and CPT/BA elevated levels of optimal engagement predicted reduced PTSD and MDD symptoms, while lower levels of optimal engagement predicted elevated PTSD and MDD symptoms. This was not the case for BA/CPT. Put simply, BA/CPT participants were less sensitive to the effects of under- and optimal engagement.

Convergent and Discriminant Validity of SUDS and CEAS Scores

I further tested the validity of using self-reported SUDS as a measure of emotional engagement. Specifically, I examined the convergent and discriminant validity of self-reported SUDS and objectively coded CEAS ratings. As SUDS and CEAS ratings of overengagement both represent a measure of distress and elevated arousal, I predicted that if convergent validity was adequate that SUDS and CEAS ratings of overengagement would be significantly correlated with one another, as these measures

should be tapping into the same construct. Alternatively, as underengagement relates to reduced affect and the reduced experience of distress, whereas SUDS relate to increased distress and arousal, one would not predict a strong or meaningful correlation between underengagement and SUDS. In fact, if SUDS are a valid assessment of emotional engagement, it should demonstrate discriminant validity by way of a minimal correlation with CEAS ratings of underengagement. Thus, I also predicted that if discriminant validity was adequate that SUDS and CEAS ratings of underengagement would only demonstrate a small or weak (i.e., $r < .10$) correlation with one another, as these measures should be tapping into different constructs.

To recap, SUDS were collected during participants' two trauma account sessions. SUDS were gained at the start, middle, and end of trauma account readings and an average SUDS was then calculated for each trauma account session. Therefore, for both trauma account sessions two measures of engagement were gained: first, an average SUDS, and second, proportions of over-, under-, and optimal engagement based on CEAS scores.

I aggregated average SUDS and CEAS ratings of over-, under-, and optimal engagement for the two trauma account sessions. Scores were then correlated (Appendix M provides correlations for the trauma account sessions separately). As expected, SUDS were significantly correlated with CEAS ratings of overengagement, $r = .51$, $p < .05$, and with CEAS ratings of optimal engagement, $r = -.55$, $p < .05$. Also, as predicted, the correlation between SUDS and CEAS ratings of underengagement was nonsignificant, $r = -.05$, $p = .82$. Therefore, SUDS demonstrated good convergent validity with CEAS ratings of overengagement, and good discriminant validity with CEAS ratings of underengagement. The significant negative correlation between SUDS and CEAS ratings of optimal engagement is not surprising given that optimal engagement reflects a state in which distress and affect is reduced.

Chapter 5: Discussion

This thesis determined the utility of targeting PTSD and MDD symptoms in those with comorbid PTSD/MDD. Additionally, to better understand the mechanisms underlying symptom change, and in order to move beyond simply examining treatment outcomes, the influence of emotional engagement was also explored. The following overarching research questions were addressed: (1) Does a combined treatment that targets both PTSD and MDD result in added benefits relative to PTSD treatment alone? (2) If a combined treatment is useful, does it matter in which order therapy is delivered? and, (3) Does emotional engagement during therapy predict treatment outcome?

This chapter begins by discussing the effects of treatment condition on PTSD, MDD, and secondary outcomes. Treatment outcomes are briefly summarised and explanations for the superiority of CPT/BA are offered. I then focus on emotional engagement. The effects of under-, over-, and optimal engagement are reviewed and methodological issues related to the assessment of engagement discussed. Finally, limitations are addressed, theoretical and clinical implications of findings highlighted, and areas for future research proposed. I argue that a combined treatment that targets PTSD *and* MDD results in added benefits relative to PTSD treatment alone. However, the added utility of targeting MDD is only achieved when PTSD treatment is presented *prior* to MDD treatment. I also argue that under- and overengagement are detrimental processes that inhibit treatment outcomes in those with comorbid PTSD/MDD.

Summary of Treatment Outcomes

This is the first study to explicitly test the usefulness of a combined treatment approach for individuals with comorbid PTSD/MDD using appropriate control and comparison conditions. As hypothesised, all conditions demonstrated significant reductions in PTSD and MDD symptoms, as well as secondary outcomes. However,

refuting hypotheses, *CPT/BA* produced larger effects than CPT and BA/CPT. The benefits of CPT/BA were more pronounced in the ITT sample than the completer sample, with all completers evidencing very good treatment outcomes. The following sections revisit hypotheses related to treatment outcomes in the ITT sample.

Hypothesis 1: All Treatments are Effective in Reducing PTSD and MDD

Supporting my first hypothesis, all conditions reduced PTSD and MDD symptoms in a wholly comorbid sample. Moreover, the positive effects of CPT, BA/CPT, and CPT/BA extended beyond PTSD and MDD symptoms to include improvements in trauma cognitions, rumination, and emotional numbing. In the ITT sample, effect sizes were mostly medium to large and effect sizes were similar to those reported in previous CPT trials (Monson et al., 2006; Forbes et al., 2012; Resick et al., 2002; 2008; Resick & Schnicke, 1992). The proportion of CPT and CPT/BA participants who maintained a PTSD and MDD diagnosis was consistent with the literature, with approximately 20 to 55% of participants still meeting PTSD and MDD diagnostic criteria at posttreatment and follow-up (Monson et al., 2006; Resick et al., 2002; 2008). As the cited trials use mixed samples of individuals with PTSD alone and comorbid PTSD/MDD, and as my results are consistent with these trials, results suggest that even when comorbidity exists CPT-based treatments are effective in reducing PTSD and MDD. Rates of good end-state functioning in CPT and CPT/BA were slightly smaller than that typically seen in other CPT trials (Chard, 2005; Resick et al., 2002). This may in part be the product of using a wholly comorbid sample and using a strict definition of good end-state functioning whereby participants needed to essentially be asymptomatic (i.e., CAPS $\leq$ 19 *and* DASS-D $\leq$ 6). One should also keep in mind that the proportion of BA/CPT participants that lost diagnoses or achieved good end-state functioning was smaller than

typically seen in the literature (the reduced efficacy of BA/CPT is discussed in a subsequent section).

My results suggest that treatment modifications did not hinder the effectiveness of CPT. While it is positive to see that all treatments were effective, this is not necessarily a new or surprising finding and it is examination of condition differences that is likely to yield a more interesting clinical picture. Accordingly, condition differences are now reviewed and the added utility of CPT/BA explained.

Hypothesis 2: A Combined CPT/BA Treatment is More Effective than CPT Alone

Hypothesis 2 predicted that the combined BA/CPT and CPT/BA treatments would be more efficacious than CPT alone. This was refuted in the ITT sample with CPT/BA, but not BA/CPT, demonstrating better outcomes than CPT alone. At posttreatment, CPT/BA produced significantly larger effects on measures of PTSD, MDD, trauma cognitions, rumination, and emotional numbing (as assessed by the TAS) than CPT, and BA/CPT. Thus, at posttreatment CPT/BA presented as a more effective treatment.

At 6-month follow-up, CPT/BA produced significantly larger effects on measures of rumination than CPT and BA/CPT, with no other significant interaction emerging. While this may suggest that by 6-month follow-up CPT and BA/CPT were able to catch-up, one must keep in mind that compared to CPT and BA/CPT, CPT/BA still produced meaningfully (albeit nonsignificantly) larger effects at 6-month follow-up on measures of PTSD, MDD, trauma cognitions, and emotional numbing (as assessed by the TAS), and that potentially with a larger sample or greater power, significant findings may have emerged. Thus, although CPT and BA/CPT demonstrated some catch-up, at follow-up CPT/BA still appeared to produce significantly larger changes in rumination, and meaningfully larger effects on many other measures than CPT and BA/CPT.

For the ITT sample, data for CPT/BA was compelling with effect sizes for this condition all being large and consistently larger than that achieved in other CPT trials (e.g., Alvarez et al., 2011; Forbes et al., 2012; Resick & Schnike, 1992). CPT/BA effect sizes ranged from 1.23 to 2.84. Comparing CPT with CPT/BA explicitly, CPT/BA produced effects on PTSD, MDD, and secondary symptoms 1.74 to 5.71 times larger than that produced by CPT. At posttreatment, CPT/BA participants were also less likely to maintain an MDD diagnosis than CPT participants. Further, CPT/BA demonstrated better retention. Thus, the advantage of CPT/BA over CPT was pronounced. By explicitly comparing CPT to two combined treatments I have shown that a combined CPT/BA treatment may lead to better outcomes compared to CPT alone. However, such a conclusion is made tentatively and requires replication given the small sample size and high rate of missing data.

The finding that CPT/BA led to better treatment outcomes than CPT alone in the ITT sample suggests that there is room for improvement in the traditional CPT protocol and that by having an additional BA focus after CPT, outcomes can be maximised. As noted in Chapter 1, although CPT shares components with traditional cognitive therapy for MDD, CPT does not explicitly target MDD and does not have a significant emphasis on the promotion of activity, and feelings of enjoyment and mastery in everyday life. Thus, although CPT may facilitate some reduction in MDD due to the overlap it shares with cognitive therapy for MDD, outcomes may not be maximised as MDD is not directly targeted. By incorporating BA at the end of CPT individuals are able to explicitly address their depression, learn different skills to work through depressive symptoms, and consequently show enhanced treatment outcomes. This is supported by CPT/BA producing an effect on the DASS-D 1.75 to 3.62 times larger than CPT.

The utility of combined treatments approaches has been questioned with some researchers suggesting that comorbid conditions do not need to be purposely targeted

using combined treatments, and that treatments focused on the primary disorder are sufficient (Craske et al., 2007). In particular, some suggest that combined treatments inhibit outcomes as presenting interventions simultaneously promotes overload such that none of the intervention is learnt well (Craske et al.; Foa et al., 1999a). However, my findings contradict such propositions. Indeed, my findings are more in line with previous PTSD studies that have found combined or sequential approaches to be useful when comorbidity is present (Cloitre et al., 2002; 2010; Falsetti et al., 2005; Najavits et al., 1998) and are congruent with van Minnen et al's (2012) suggestion to use combined treatments when individuals present to treatment with PTSD and complex comorbidity.

Although this section has noted the utility of a combined treatment over CPT, one must keep in mind that the usefulness of a combined treatment was dependent on the sequencing of treatment components and that the added utility of a combined approach was only achieved when PTSD was targeted *prior* to MDD. I now discuss the influence of treatment presentation order and the superiority of CPT/BA over BA/CPT.

Hypothesis 3: CPT/BA Produces Superior Outcomes Compared to BA/CPT

Hypothesis 3 predicted that if depressive symptoms reduced emotional engagement, that BA/CPT would demonstrate better outcomes compared to CPT/BA. As already noted, Hypothesis 3 was refuted and CPT/BA produced effects on PTSD, MDD, and secondary symptoms 1.60 to 3.00 times larger than BA/CPT. Further, compared to BA/CPT participants, CPT/BA participants were more likely to lose their PTSD and MDD diagnosis and achieve good end-state functioning at posttreatment. CPT/BA also showed better retention of clients in therapy. However, such findings should be interpreted in light of the small sample size and high rate of missing data.

In order to gain a better understanding of why BA/CPT failed to produce superior treatment outcomes I examined mid-phase outcomes in Chapter 3. In line with

Hypothesis 3 and the notion that initial BA sessions should reduce depressive symptoms and place participants in a better position to engage in CPT, one would expect initial BA sessions to produce meaningful changes on depressive symptoms (i.e., low mood, rumination, and emotional numbing). However, contrary to such predictions (and refuting Hypothesis 3), initial BA sessions did not reliably reduce MDD and secondary depression-related symptoms by mid-phase. Additionally, CPT and CPT/BA evidenced significantly larger effects on most measures at mid-phase compared to BA/CPT. Specifically, initial BA sessions almost had no effect on MDD (within group effects: DASS-D: d = -0.01), trauma cognitions (PTCI: d = -0.12), and rumination (SRRS: d = -0.18), and appeared to increase emotional numbing (TAS: d = -0.52). Furthermore, in line with the notion that initial BA sessions did not prepare participants for CPT, within group effect sizes suggested that CPT and BA/CPT produced similar effects on PTSD, MDD, and secondary symptoms at posttreatment and follow-up. That is, the efficacy of CPT did not differ between those who had initial BA sessions (i.e., BA/CPT), and those that did not (i.e., CPT).

Taken together findings suggest that an initial BA focus was not effective in reducing symptoms, and suggest that an initial BA focus did not place individuals in a better position to engage in CPT, nor enhance the efficacy of subsequent CPT sessions. Indeed, findings demonstrate a pattern opposite to that which was predicted, that is, an initial CPT focus was associated with a more effective reduction of symptoms and may have actually placed individuals in a better position to undertake BA.

Results are consistent with previous studies that have demonstrated that for individuals with PTSD, BA only has modest effects on PTSD and MDD symptoms (Mulick & Naugle, 2004; Jakupcak et al., 2006). For instance, using a sample of injury survivors with PTSD, Wagner et al. (2007) conducted a small-scale (n = 8) randomised effectiveness trial that compared BA for PTSD to treatment as usual. Consistent with

my findings, BA did not lead to significant improvements on depression scores. However, this should not be interpreted to mean that targeting MDD is unnecessary in the treatment of comorbid PTSD/MDD. Rather, it seems that a focus on MDD symptoms is beneficial and leads to superior outcomes when it is presented *after* CPT. In line with this, BA sessions that followed CPT (i.e., CPT/BA) reduced DASS-D scores from 10.09 to 4.73[13] which represents a clinically meaningful change. Thus, findings suggest that past studies may have failed to find significant effects of BA on PTSD and MDD symptoms not because BA is ineffective, but because the timing of BA was not optimal. I now provide specific explanations for the superiority of CPT/BA.

Why Did CPT/BA Demonstrate Superior Outcomes Compared to BA/CPT?

Various explanations exist for the superiority of CPT/BA. For instance, CPT/BA may have produced superior outcomes compared to BA/CPT because PTSD symptom change drives MDD symptom change, rather than vice versa. For example, PTSD symptoms such as hypervigilence and avoidance may make it hard for individuals to engage in BA, consequently reducing BA efficacy. However, before I review such explanations a simpler explanation should be addressed. As CPT/BA participants attended more sessions than CPT and BA/CPT participants, one may argue that the advantage of CPT/BA was simply the product of treatment dose. However, as patterns observed in initial analyses were maintained when number of sessions was controlled, the superiority of CPT/BA should not merely be considered a product of treatment dose.

Although the literature examining the direction of PTSD and MDD symptom change during CPT treatment is scarce, preliminary evidence suggests that change in PTSD symptoms drives subsequent change in MDD symptoms to a larger extent than vice versa (Aderka et al., 2011; Erickson, Wolfe, King, King, & Sharkansky, 2001).

[13] As all individuals included in the analysis of mid-phase outcome went on to complete treatment (i.e., no drop-out after Session 6), this reflects completer data.

Thus, CPT/BA may have demonstrated superior outcomes as an initial PTSD focus, and the initial reduction of PTSD symptoms, facilitated subsequent change in MDD and secondary symptoms. Conversely, when BA was presented first, treatment response may have been inhibited as MDD symptom change did not drive PTSD and secondary symptom change to same extent, and that it was not until PTSD symptoms were targeted explicitly that significant change in comorbid MDD was triggered.

The proposition that PTSD symptom change drives subsequent MDD symptom change during trauma-focused therapy is supported by studies that have found anxiety and PTSD symptoms to represent a causal risk factor for MDD development more consistently than vice versa (Aderka et al., 2011; Erickson, et al., 2001; Kessler et al., 1995; Wetherell, Loehach, & Pedersen, 2001; Wittchen, Beesdo, Bittner, & Goodwin, 2003). For example, as noted in Chapter 1, Stander et al. (2014) reviewed the literature surrounding the etiology of comorbid PTSD/MDD in military personnel, finding only inconsistent evidence that pre-existing MDD was a risk factor for PTSD development. In contrast, they found consistent evidence that PTSD was a risk factor for MDD development. They concluded that while some level of bidirectional causality between the two disorders existed, the proposed influence of PTSD on the development of MDD had more support than the reverse.

Turning our attention to the treatment of PTSD, Meyer, Kimbrel, Tull, and Morissette (2011) reviewed research related to the treatment of comorbid PTSD/MDD in military personnel. Consistent with PTSD being a causal risk factor for MDD, they concluded that while treatments for PTSD tended to reduce MDD symptoms, treatments for comorbid conditions such as MDD did not necessarily reduce PTSD symptoms. Aderka et al. (2011) investigated the relationship between PTSD and MDD during prolonged exposure (PE) therapy for children and adolescents. They found a reciprocal relationship between PTSD and MDD symptom change. However, changes in PTSD

symptoms accounted for 64% of change in MDD symptoms, whilst changes in MDD accounted for only 11% of change in PTSD symptoms. This suggests that PTSD plays a more prominent role in mediating subsequent change in MDD compared to MDD mediating change in PTSD. Similarly, in a sample of female adult assault victims Aderka, Gillihan, McLean, and Foa (2013) found that PTSD symptoms fully mediated the effects of PE on MDD symptoms, whereas MDD symptoms only partially mediated the effects of PE on PTSD symptoms. Specifically, during PE change in PTSD accounted for 80.3% of change in MDD, whereas change in MDD accounted for only 45% of change in PTSD. In short, findings suggest that by initially focusing on PTSD symptoms, as is the case in CPT/BA, subsequent change in MDD can be facilitated faster and treatment efficacy improved. Also, by targeting PTSD first, one may be targeting the root of this comorbidity, whereas targeting MDD first may inhibit treatment effectiveness by delaying a focus on more influential PTSD symptoms.

Exactly why PTSD symptoms might be so critical to target first in the presentation of comorbid PTSD/MDD is unknown, but two theoretical propositions provide possible (albeit speculative) explanations. The demoralisation model posits that PTSD evolves into subsequent MDD by a process of demoralisation (Schindel-Allon et al., 2010). The authors suggest that due to difficulties in controlling and coping with anxiety, individuals with PTSD can feel incompetent and helpless following unsuccessful attempts to control anxiety (Mangelli et al., 2005). Continued feelings of helplessness are then proposed to lead to demoralisation that then enhances the risk of developing MDD. As an example, emotional distress in PTSD is exacerbated by a lack of emotional processing and by negative thoughts (Ehring, Ehlers, & Glucksman, 2006; Foa & Kozak, 1986). Unsuccessful attempts to regulate distress may then enhance hopelessness and demoralisation and consequently facilitate the development of MDD. If this is the case and MDD development is a reaction to PTSD, treating PTSD first is

likely to target a key mechanism underlying this comorbidity, thus facilitating secondary MDD symptom change.

The helplessness/hopelessness theory (Alloy, Kelly, Mineka, & Clements, 1990) also provides an explanation. Alloy et al. propose that the experience of helplessness relates to anxiety, whereas feelings of hopelessness relate to depression. According to this theory experiencing helplessness repeatedly, as is the case in PTSD, can lead to hopelessness and subsequently the development of MDD. Thus, as a client begins to cope better with his/her anxiety or PTSD symptoms during treatment, helplessness is reduced and this flows on to subsequently reduce hopelessness and thus depression. In line with my findings, targeting PTSD (and possibly helplessness) first with CPT may have then facilitated subsequent change in hopelessness and therefore MDD. Such a proposition is in line with findings that suggest that hopelessness predicts PTSD and MDD treatment outcome. For instance, in a sample of veterans treated with CPT, Owen, Chard, and Cox (2008) found hopelessness to predict elevated posttreatment PTSD symptom severity. Additionally, Gilman, Schumm, and Chard (2012) found hope, assessed midway through CPT, to predict reductions in PTSD and MDD symptoms from mid- to posttreatment.

Consistent with the notion that change in PTSD drives change in MDD, it may also be the case that PTSD symptoms such as hyperarousal and avoidance frustrate engagement in BA and inhibit subsequent change in MDD. Therefore, if PTSD symptoms are targeted initially and interfering PTSD symptoms reduced, one is placed in a better position to undertake BA and show consequent changes in MDD. In Chapter 1 I suggested that depressive symptoms such as emotional numbing, rumination, and maladaptive cognitions might inhibit PTSD treatment outcomes. However, my findings contradict such a proposition and suggest that the opposite might be true. That is, as emotional numbing, rumination, and maladaptive cognitions are also evident in PTSD,

it may be the case that the relationship is reversed and that these common PTSD symptoms inhibit engagement in BA and changes in MDD.

As an example, in Chapter 1 I suggested that depressive rumination may impede CPT effectiveness by frustrating engagement and capturing mental resources. However, as rumination is also seen in PTSD, the relationship may be reversed, with PTSD-related rumination potentially inhibiting one's ability to become active and gain pleasure from BA-scheduled activities. For example, if an individual is asked to complete a pleasurable, scheduled activity but demonstrates a reduced ability to disengage from trauma-based rumination (e.g., rumination surrounding danger or self blame) they are unlikely to focus on the scheduled task, or find the task pleasurable. This would consequently inhibit change in MDD. Supporting this, PTSD related rumination has been found to be associated with negative mood, heightened arousal, and a reduced ability to feel pleasure. (Ehring, Szeimies, & Schaffrick, 2009; Moore, Zoellner, & Mollenholt, 2008). Thus, PTSD-related rumination and PTSD symptoms may first need to be reduced before one can engage in BA and show reductions in MDD symptoms.

Avoidance is also a hallmark symptom of PTSD that may interfere with the efficacy of BA. During BA individuals are set scheduled tasks as a means of enhancing activity. However, PTSD-related avoidance may have an additive effect that enhances the potency of MDD-related inactivity and increases the difficulty of activating clients during BA. That is, when individuals with comorbid PTSD/MDD present for BA not only must they overcome low mood and reduced motivation when attempting to be active, but they must also overcome habitual patterns of PTSD-related avoidance that have previously helped reduce distress. In line with this, avoidant coping has been found to predict elevated PTSD and MDD symptom severity and reduced treatment outcomes (Badour, et al., 2012; Leiner, Kearns, Jackson, Astin & Rothbaum, 2012).

Thus, as CPT targets PTSD-related avoidance, an initial CPT focus may reduce avoidance and improve one's ability to engage in BA and subsequently reduce MDD.

Hyperarousal symptoms may also inhibit change in MDD symptoms. It is well established that hyperarousal responses are related to elevated levels of arousal, and are indicative of reduced emotion regulation (Etkin & Wagner, 2007; Frewen & Lanius, 2006). Further, hyperarousal responses are often accompanied by maladaptive trauma cognitions whereby individuals with PTSD are likely to catastrophise and perceive objectively safe situations as dangerous (Ehlers & Clark, 2000; Regambal & Alden, 2012). As such, if hyperarousal is not initially targeted, individuals may enter BA with a propensity for overengagement. Further, as many BA activities are likely to occur in situations *perceived* to be dangerous, individuals are also likely to find it difficult to become active. This would of course frustrate BA outcomes and the reduction of MDD. Thus, it may not be until hyperarousal is reduced that meaningful change in MDD can occur.

Studies have begun to demonstrate the prominent role of hyperarousal in PTSD remission. Using a longitudinal design Schell, Marshall, and Jaycox (2004) explored the progression of PTSD symptoms. They found hyperarousal to be the best predictor of symptom change, influencing all other PTSD symptoms across time. Further, individuals who demonstrated higher hyperarousal symptoms at baseline demonstrated poorer 12-month outcomes. Similarly, Marshall, Schell, Gylnn, and Sheety (2006) found hyperarousal symptoms to be a strong predictor of reexperiencing, avoidance, and hyperarousal symptoms at 1, 6, and 12-month post-trauma. These findings provide evidence for the prominence of hyperarousal in the progression of PTSD and suggest that reducing hyperarousal symptoms early on in treatment may be a beneficial treatment approach. This is of course at odds with an initial BA focus.

Although I have posited that PTSD symptom change facilitates MDD symptom change, contradictory findings exist. For instance, although Aderka et al. (2013) found changes in PTSD to predict subsequent change in MDD for PE alone, for PE plus cognitive restructuring, they found that PTSD and MDD symptoms had a more equal effect on one another. Thus changes in PTSD symptoms accounted for 59.6% of changes in MDD symptoms, and changes in MDD symptoms accounted for 50.7% of changes in PTSD symptoms. Additionally, as noted in Chapter 1, Liverant et al. (2012) examined the association between PTSD and MDD during the course of CPT and found changes in PTSD and MDD to occur concurrently. Hampered by the limited number of studies addressing this issue to date, there does not appear to be a consistent pattern that would explain why sometimes change in PTSD has greater explanatory power on MDD than vice versa. One must also remember that the reviewed studies did not examine those with comorbid PTSD/MDD explicitly but rather use mixed samples of individuals with PTSD alone and those with comorbid PTSD/MDD. Further, the reviewed studies used different treatments (e.g., CPT, PE) and different samples (e.g., age, gender, trauma), all of which might partially account for the contradictory findings. Importantly, and consistent with the proposed argument, the one consistency in these studies is that change in PTSD is always shown to be related to change in MDD. Whilst I have argued that CPT/BA demonstrated superior treatment outcome due to PTSD symptom change potentially driving MDD symptom change, other explanations exist. I now discuss emotional engagement as a potential explanation for the superiority of CPT/BA.

Emotional engagement as an explanation of treatment outcome. Emotional engagement findings shed further light onto why CPT/BA was able to achieve superior treatment outcomes (the findings on engagement will also be discussed in more detail in later sections). Results indicated that while underengagement predicted reduced PTSD treatment outcomes for CPT/BA participants, CPT/BA participants were also less likely

to underengage compared with the other groups. On average CPT/BA participants only spent 1.52% of their sessions underengaged compare to 9.50% and 9.80% for CPT and BA/CPT respectively. Thus, even though CPT/BA participants were sensitive to the negative effects of underengagement, because these participants did not spend a substantial proportion of their sessions underengaged the detrimental effects of underengagement were minimised. CPT/BA may have facilitated a lower level of underengagement as CPT sessions promote the expression of feelings and explicitly define emotional expression as a necessity for change. This is of course at odds with a state of underengagement. Alternative explanations are now addressed.

Alternative explanations. As CPT/BA participants reported meaningfully larger pretreatment credibility/expectancy scores than CPT and BA/CPT participants, CPT/BA participants may have demonstrated superior outcomes and may have been more likely to stay engaged in treatment as they viewed their treatment as more credible, or had more hope that their treatment would be effective. This is supported by studies that have consistently found elevated treatment credibility/expectancy ratings to predict better retention, and positive PTSD and MDD treatment outcomes (e.g., Addis & Jacobson, 1996; 200; Fennell & Tesdale, 1987). This finding also suggests that when individuals with comorbid PTSD/MDD present for treatment they may believe that *both* PTSD and MDD symptoms require treatment but, that PTSD should be treated first as PSTD symptoms are of primary importance, or cause the most distress.

Although credibility/expectancy does provide some insight into the superiority of CPT/BA, as differences were nonsignificant and effects only medium, and as effect size confidence intervals included zero, credibility/expectancy does not fully account for the superior efficacy of CPT/BA and findings require replication. Future research must determine the extent to which credibility/expectancy predicts outcome in those with comorbid PTSD/MDD specifically. This would be complimented by research that

explores what clients personally perceive the best treatment of comorbid PTSD/MDD to be.

As recruitment information emphasised the treatment of PTSD, and as participants entered the study requesting ‘trauma-focused’ therapy, one may argue that BA/CPT might not have led to superior outcomes and may have demonstrated enhanced dropout compared with CPT/BA because the initial focus on MDD was not specifically what participants wanted. Although this is partially supported by BA/CPT participants reporting lower pretreatment credibility/expectancy scores than CPT/BA participants, such a proposition is contrary to the finding that BA/CPT and CPT participants reported similar credibility/expectancy scores at pretreatment. Further, such a proposition is contrary to the finding that BA/CPT participants reported higher credible/expectancy scores than CPT participants at posttreatment. Adding to this, as some clinicians have concerns about the anxiety-provoking nature of trauma-focussed therapy, one might expect that BA/CPT would appear to be a more tolerable treatment for participants as their traumas were not explicitly discussed early on in therapy (which should have been associated with lower dropout, although this was not in fact the case).

It could also be suggested that five sessions of BA was simply not enough to facilitate symptom change in a wholly comorbid sample. Although other outcomes studies of BA for MDD deliver a longer course of BA, with some studies providing up to 20 BA sessions (Jacobson et al., 1996), other studies have found smaller doses of BA to still be effective. For instance, in a sample of individuals with comorbid PTSD/MDD, Nixon and Nearmy (2011) found that five sessions of BA led to significant reductions in both MDD and PTSD symptoms. Additionally, one must keep in mind that it was not that five BA sessions was not effective in reducing symptoms, but that the effectiveness of BA sessions was dependent on treatment presentation order. In line with this, for CPT/BA, BA sessions reduced DASS-D scores from 10.09 at mid-phase (just prior to

the initiation of BA) to 4.73 at posttreatment. This represents a clinically significant change. In contrast, for BA/CPT, initial BA sessions changed DASS-D scores from 21.20 at pretreatment to 21.33 at mid-phase (at the end of BA). Consequently, the reduced length of BA does not fully account for the superiority of CPT/BA.

Finally, one may suggest that as CPT/BA participants had a trend towards higher pretreatment symptom severity scores than CPT participants, the elevated effectiveness of CPT/BA may be a reflection of regression to the mean or this condition simply having more room for improvement. While this may be the case, this is an unlikely explanation given that research has consistently found higher pretreatment severity to predict reduced treatment outcomes and elevated rates of dropout (e.g., Blanchard et al., 2003). Further, one must remember that CPT/BA still produced superior effects compared to BA/CPT although pretreatment symptoms scores were more similar for BA/CPT and CPT/BA participants.

In review, results in the ITT sample suggest that a combined treatment that targets both PTSD and MDD results in added benefits relative to CPT alone. However, treatment presentation order is imperative with CPT/BA producing larger effects than BA/CPT. The superiority of CPT/BA is in line with literature that proposes that PTSD symptom reduction drives MDD symptom reduction. I now turn attention to completers.

Completers Derive Good Outcomes

Completers demonstrated very good outcomes regardless of condition. Consistent with previous studies (Galovoski et al., 2012; Resick & Schnicke, 1992, Resick et al., 2002; 2008), completers were very likely to lose their PTSD diagnosis and the proportion of completers who lost their MDD diagnosis by follow-up was larger than that seen in completers in other CPT trials (e.g., Resick et al., 2008). In fact, it was surprising to see that all completers had lost their MDD diagnosis at follow-up. The

proportion of completers who achieved good end-state functioning was smaller than that reported in other trials (Resick et al., 2002). However, this may again be the product of using a wholly comorbid sample and using a very strict definition of good end-state.

Although completers were likely to show very good treatment outcomes, there was a small caveat to this in which CPT completers evidenced very small effects on depressive-rumination (RRS) and only moderate changes on trauma cognitions (PTCI) at follow-up. As CPT has consistently been found reduce trauma cognitions (Forbes et al., 2012; Galovski et al., 2012; Nishith, Nixon, & Resick, 2005), my finding is at odds with a large body of literature. As the cited studies used mixed samples of individuals with PTSD and comorbid PTSD/MDD, it may be the case that when individuals present with comorbid PTSD/MDD negative cognitions are more entrenched and are therefore more resistant to change. In line with this, Nixon et al. (2004) found that those with comorbid PTSD/MDD demonstrated more negative cognitions than those with PTSD or MDD alone. Thus, CPT alone may not be sufficient to change such entrenched cognitions, and an additional BA focus may be needed to facilitate cognitive change.

Less is known about the effect of CPT on depressive-rumination with no CPT randomised control trial reporting on rumination as an outcome (e.g., Monson et al., 2006; Resick et al., 2002; 2008). In Chapter 3 I proposed that lower pretreatment RRS scores and floor effects contributed to CPT completers reporting only a small effect on the RRS. In line with this, it may have also been the case that when CPT completers entered treatment rumination did not cause them clinical impairment (as suggested by lower pretreatment RRS scores) and they were therefore less motivated to change ruminative practices. However, as CPT and CPT/BA demonstrated quite large changes on the RRS, and as CPT/BA reduced RRS scores to a lower point than CPT, lower pretreatment scores and floor effects do not fully explain findings. Interestingly, my results suggested that whilst CPT completers demonstrated large changes on stress-

reactive rumination this was not the case for depressive-rumination. This may indicate that while CPT adequately reduces stress or trauma-related rumination, CPT alone is not sufficient to reduce depressive-rumination and that a specific focus on MDD is needed to reduce such rumination. Due to gaps in the literature, future CPT trials should assess rumination as an outcome.

Finally, although all completers generally demonstrated good outcomes, the superiority of CPT/BA was still evident as CPT/BA participants were more likely to actually be completers (and thus achieve good outcomes), and CPT/BA completers achieved effect sizes larger than that gained by completers in other CPT trials (Forbes et al., 2012; Monson et al., 2006; Resick et al., 2002; 2008). Further, in order to achieve outcomes as good as that demonstrated by BA/CPT and CPT/BA, CPT completers on average attended two extra sessions beyond the typical CPT protocol. This is in line with research (and clinical observations) that suggests that there is variability in the number of sessions required to achieve good outcomes in CPT (Galovski, et al., 2012).

Dropout and Retention

CPT/BA demonstrated superior participant retention with a dropout rate (31.2%) almost half that of CPT (55.6%), and BA/CPT (60%). In light of reported treatment outcomes it is not surprising to see differences in retention. As CPT/BA participants demonstrated larger symptom reductions from pre- to posttreatment than CPT and BA/CPT participants, CPT/BA participants may have been more likely to stay engaged in treatment as they were also more likely to demonstrate symptom change earlier on in therapy, and consequently be more hopeful in the effectiveness of their treatment.

In clinical practice there has at times been concern surrounding the undertaking of trauma-focused therapy (Becker, Zayfert, & Anderston, 2004; Pitman et al., 1991) and Van Minnen, Hendricks, and Olff (2010) suggest that when MDD co-occurs with PTSD

clinicians may view trauma-focused therapy as inappropriate due to elevated symptom severity and reduced emotion regulation. However, such concerns are contradicted by my findings that observed an initial trauma focus to lead to better treatment outcomes and better participant retention. The enhanced retention rate demonstrated by CPT/BA also contradicts the notion that all individuals must be 'prepared' for trauma-focused therapy. Current explorations of combined treatments often suggest that initial sessions should be spent preparing clients for trauma-focused therapy by promoting factors such as emotion regulation or by reducing comorbid symptoms (Cloitre et al., 2002; Falsetti et al., 2001). A possible drawback of using a combined treatment approach to prepare individuals for trauma-focused treatment is that it may inadvertently instil the notion that when individuals present to treatment they are too fragile to discuss their trauma and that their psychological state must be improved before therapy can be undertaken. However, my results suggest that this is not the case and that even when comorbidity is present, many individuals enter treatment able to discuss their trauma. In short, not putting clients in 'cotton-wool' may actually lead to better outcomes.

Looking at dropout more generally, dropout was high (overall rate of 49%). However, while at the higher end of the spectrum, this rate is still in line with some other treatment trials for PTSD and MDD (Hans & Hiller, 2013; Imel, laska, Jakupcak, & Simpson, 2013; Schottenbauer, Glass, Arnkoff, Tendick, & Gray, 2008). Elevated dropout may have been the result of using a strict definition of dropout in which a dropout was defined as someone who completed less than 12 sessions. Elevated dropout may have also been the result of using inexperienced therapists or because the treated sample was wholly comorbid and exhibited complex trauma histories, with most individuals experiencing sexual assault. These are all factors associated with elevated dropout (Bryant et al., 2003; Hembree, Street, Riggs & Foa, 2004; McDonagh et al., 2005).

It is also possible that the high dropout rate was the product of conducting therapy in a more ecologically valid manner, and conducting the trial within a community setting. Treatment occurred at Flinders University as well as Yarrow Place and Victim Support Services, the latter two being community services for victims of crime and sexual assault. To gain a better sense of attrition in a community setting I reviewed client intake information at Yarrow Place. I examined attrition rates for a random sample of 135 past clients[14] (i.e., clients who had experienced sexual assault more than three months ago) who had contacted the service seeking counselling during the period of the study. Of people who contacted the service 76% never attended a single session or only attended one session. The mean number of sessions attended was 1.08 (*SD* = 1.85) indicating that the majority of clients failed to engage in treatment. Such results are striking and indicate that at Yarrow Place non-engagement is a significant issue. These data may also reflect the client group (sexual assault survivors). Although such a comparison is based on nonrandomised data, it is worth noting that the retention rate for clients entering the study was far better than that achieved in this community setting (mean number of attended sessions for trial = 8.61 [*SD* = 6.47] versus 1.08 [*SD* = 1.85] in the community).

Effect of Order of PTSD and MDD Onset

This thesis provided an initial exploration of the effect of order of PTSD and MDD onset on treatment outcome. My results indicated that PTSD and MDD treatment outcomes did not differ significantly between PTSD/MDD onset, MDD/PTSD onset, and simultaneous PTSD/MDD onset groups. This may suggest that order of onset is not a strong predictor of outcome. However, one must keep in mind that due to the small

[14] A sample was taken from Yarrow Place rather than Victim Support Service as a larger proportion of community clients were recruited from Yarrow Place. The selected sample consisted of clients excluded from the trial due to: a) requiring phone counseling rather than face-to-face counseling, b) being allocated to a non-trial therapists, and c) trial therapist not being available.

sample size and due to collapsing treatment conditions, the effects of order of onset may have been masked. Further, due to the small sample size I was unable to determine if condition interacted with order of onset to affect outcome. Thus, my findings should only be considered preliminary.

Very few studies have examined the relationship between order of disorder onset and treatment outcome. Although no other study to date has examined this relationship in those with comorbid PTSD/MDD, some literature exists in the substance and alcohol abuse field (Brady, Dansky, Sonne, & Saldin, 1998; Schukit, 1985). For example, Nishith, Mueser, Srsic, and Beck (1997) examined whether parolees with primary versus secondary substance abuse disorder (SUD) responded differently to cognitive therapy for SUD. Those with a secondary SUD demonstrated greater decreases in substance-use at posttreatment than those with primary SUD. Currently, only one study has examined the relationship between order of onset and outcome in a PTSD sample. In a sample of individuals with PTSD and alcohol dependence (AD), Back, Jackson, Sonne, and Brady (2005) examined the effect of order of onset on CBT for AD treatment outcomes. They found that those with primary PTSD reported larger improvements in alcohol use than those with primary AD. Thus, some literature suggests that order of onset may affect outcome. As the current literature is scarce and has not examined comorbid PTSD/MDD, and as my findings contradict previous research, additional research is necessary to better understand the effects of disorder onset on PTSD and MDD outcome.

Emotional Engagement Findings

This thesis represents the first instance in which the effects of under-, over-, and optimal emotional engagement on PTSD and MDD outcome have been examined separately. Furthermore, this is the first study to address methodological issues present

in other examinations of engagement by coding every therapy session, by exploring the relationship between peak and modal ratings, and by determining the validity of self-reported SUDS. As a result, we now have a more sophisticated understanding of the relationship between emotional engagement and treatment outcome during the treatment of comorbid PTSD/MDD. This section first explores the general relationship between under-, over-, and optimal engagement and treatment outcome. Condition differences are then reviewed and tentative explanations for such differences provided. Finally, methodological issues surrounding the assessment of engagement are examined.

Under-, over-, and optimal engagement were all found to influence treatment outcome however these effects were less pronounced for BA/CPT. For CPT and CPT/BA, elevated levels of underengagement predicted elevated PTSD (but not MDD) symptoms, whereas elevated levels of optimal engagement were found to predict reduced PTSD and MDD symptoms; as expected, lower levels of optimal engagement also predicted elevated PTSD and MDD symptoms. Further, for all conditions elevated levels of overengagement predicted elevated PTSD and MDD symptoms. The finding that overengagement predicted elevated PTSD and MDD symptoms is in line with past research that has found heightened levels of arousal to predict reduced treatment outcomes (Carryer & Greenberg, 2010; Jaycox et al., 1998, Missirlian et al., 2005; Watson & Bedard, 2006). Under- and optimal engagement findings are more novel as past research has not examined under- and optimal engagement as separate constructs.

Findings provide explicit support for the overarching principles of emotional processing theory. That is, they directly support the detrimental role of under-, and overengagement in the therapeutic process, and the argument that optimal engagement is a prerequisite for optimal PTSD outcome (Foa & Kozak, 1986). Past examinations of emotional processing theory suffer methodological flaws by relying on subjective assessments of emotional engagement (e.g., SUDS) and by failing to separate the effects

of under-, over-, and optimal engagement. By addressing such limitations and by testing the effects of under-, over, and optimal engagement separately using an arguably more objective method (CEAS coding ratings), a more rigorous examination of emotional processing theory has been achieved compared with previous research.

Findings related to underengagement. While underengagement predicted elevated PTSD symptoms for CPT and CPT/BA, underengagement did not significantly predict MDD symptoms. This is at odds with theories of depression as well as emotional processing theory that emphasise engagement as a necessity for positive MDD treatment outcome (Farber et al., 2004, Ferster, 1973; Foa & Kozak, 1986; Greenberg, 2002b; 2008; Greenberg & Korman, 1993). For example, contrary to my findings, several depression theorists have suggested that engagement with negative affect during MDD treatment can facilitate insight and positive growth (Greenberg, 2002a; 2002b; Mackay et al., 1998; Rogers, 1944; Russell & Fosha, 2008), while emotional avoidance (i.e., underengagement) may impede change in MDD (Carryer & Greenberg, 2010). Further, the finding that underengagement was not significantly related to MDD outcome also contradicts previous studies that have found reduced emotional engagement and detachment during MDD treatment to predict poorer MDD outcome (Carryer & Greenberg, 2010; Stringer et al., 2010; Warwar, 2003). Adding to this, the findings are contrary to emotional processing theory's proposition that underengagement predicts reduced treatment outcome during trauma-based therapy. Finally, as reviewed in Chapter 1, PTSD and MDD share many cognitive commonalities and as such it is puzzling to see a significant relationship between underengagement and PTSD, but not underengagement and MDD.

Methodological and conceptualisation differences may partially account for contradictory findings. First, as previous examinations of emotional engagement and MDD outcome have examined engagement as a unified construct and have collapsed

levels of under-, over-, and optimal engagement (e.g., Missirlian et al., 2005; Stringer et al., 2010; Warwar, 2003), differences in the effects of under- and overengagement may have been masked in previous studies. Second, although the findings contradict predictions made by emotional processing theory, one must keep in mind that while emotional processing theory discusses underengagement in the context of processing trauma memories (i.e., during trauma account exposure sessions) (Foa & Kozak, 1986), I examined underengagement across all sessions and as a more general processing style. As such, contradictory findings may in part be the result of assessing emotional engagement more broadly than the theory intended.

Contradictory findings may also relate to the low rate of underengagement observed in this thesis. Depression is defined by the flattening of affect, reduced emotion responding, and emotional numbing. This is of course very similar to conceptualisations of underengagement. Conversely, PTSD is defined by elevated levels of anxiety and hyperarousal which are marked by heightened (i.e., overengagement), rather than reduced arousal (i.e., underengagement). Therefore, as PTSD symptoms are more at odds with underengagement, PTSD symptoms may be more sensitive to the effects of underengagement. Alternatively, as MDD symptoms may already represent a certain degree of underengagement, for underengagement to have an effect on MDD outcomes, proportions of underengagement may need to be more pronounced. Thus, the failure of underengagement to predict MDD treatment outcome may relate to the somewhat low levels of underengagement demonstrated by clients. This is a salient point given that proportions of underengagement were small (i.e., $M = 6.27\%$) in this thesis. As past research has not examined under- and overengagement separately it is difficult to determine if such low levels of underengagement are typical. However, as the mean modal rating was slightly higher in this thesis compared to other examinations

of engagement (e.g., Missirlian et al., 2005),[15] it may be the case that underengagement was reduced in this study (or in comorbid samples where one disorder is associated with over-arousal such as PTSD) and that larger proportions of underengagement were needed for underengagement to have an effect.

Although methodological differences and small proportions of underengagement may in part explain contradictory findings, one cannot discount the possibility that underengagement simply does not influence MDD symptoms and that theoretical propositions are incorrect. Thus, future research must examine this relationship further and determine how, or if, underengagement effects MDD symptoms. Initial examinations of this relationship may benefit from taking an experimental approach, as this might ensure that greater levels of underengagement are induced.

Findings related to overengagement. While condition differences existed for under- and optimal engagement (discussed in detail below), no such differences emerged for overengagement with higher proportions of overengagement seen to predict elevated PTSD and MDD symptoms for all conditions. One explanation is that irrespective of the therapy approach and symptom target, overengagement is universally an unhelpful state to be in. When individuals enter a state of overengagement during therapy it is often accompanied by incredible distress and likely results in a significantly reduced ability to process concepts discussed in therapy. For example, if an individual is crying hysterically their ability to listen to and process discussed concepts is likely to be hindered as they are overcome by emotion and the cognitive resources necessary for the processing of such information are drained. Alternatively, when individuals are underengaged they may still have some ability to process incoming information as they are not flooded by emotion. In line with this, research has consistently found disorders of emotion dysregulation (e.g., borderline personality disorder, PTSD) and states of

[15] At times it is difficult to determine mean modal ratings of engagement in past research as researchers have failed to report descriptive statistics, and fluctuate between the use of peak and modal scores.

high arousal to be associated with memory, learning, attention, and cognitive flexibility deficits (Dinn et al., 2004; Driskell & Salas, 1996; Judd & Ruff, 1993; Ruocco, 2005).

My results confirm that the interplay between emotional engagement and outcome is complex and paint the challenging situation that clinicians face in managing client's emotional engagement in session. It is clear that this requires more than merely enhancing or encouraging the expression of emotion. Rather my findings highlight the important role clinicians have in assisting clients to regulate their emotions in order to achieve an optimal state of engagement during treatment, and the need for clinicians to safeguard clients against maladaptive levels of emotional engagement. Condition differences in the effects of emotional engagement are now discussed.

Why Did the Effect of Emotional Engagement Differ By Treatment Condition?

The relationship between under-, over-, and optimal engagement and PTSD and MDD treatment outcome differed between conditions. While heightened levels of underengagement predicted elevated PCL scores for CPT and CPT/BA, this was not the case for BA/CPT. Further, for CPT and CPT/BA, elevated levels of optimal engagement predicted reduced PTSD and MDD symptoms, and reduced levels of optimal engagement predicted heighted PTSD and MDD symptoms. However, for BA/CPT, optimal engagement did not significantly predict outcome. I found no evidence that the model obtained when the relationship between emotional engagement and outcome was allowed to vary over sessions provided a significantly better fit than the model obtained when the relationship remained the same across sessions. This indicates the relationship between engagement and symptom was relatively stable (and did not vary to a large degree at different times in therapy). Possible explanations for interactions are now provided. Due to the dearth of research of this type and the fact that the efficacy of the tested treatments requires replication, explanations are speculative.

As the relationship between under- and optimal engagement and outcome functioned similarly for the CPT and CPT/BA conditions, differences between these conditions and BA/CPT might relate to the structure of therapy, specifically, that BA sessions were presented initially. As CPT and BA are theorised to exert their effects by different means, initial CPT and initial BA sessions are likely to provide clients with very different expectations and understandings of therapy. As highlighted in the following sections, it may be the case that compared with initial CPT sessions, initial BA sessions facilitate different processes or perspectives that are carried throughout therapy and that subsequently reduce sensitivity to under-, and optimal engagement.

CPT adopts an internal focus whereby individuals are taught to identify and feel emotions. CPT underscores emotional awareness and expression as a necessity for optimal treatment outcome and explicitly teaches individuals that if they do not connect with their emotions recovery will be inhibited. Alternatively, BA emphasises activity as the mechanism underlying treatment change. In BA individuals are taught to take an 'outward-in' focus and act in line with their schedule rather than acting in line with their emotions. BA fosters an external focus on one's context and promotes an action and goal oriented form of processing. In essence, BA fosters an external goal focus whereby activity is conceptualised as the mechanism driving change while CPT emphasises the expression of emotion (as well as developing alternatives to unhelpful trauma thoughts).

By placing an external focus on action and goals, and by explicitly emphasising activity rather than thoughts and emotion as key mechanisms underlying symptom change, initial BA sessions may teach individuals to maintain an external focus and allow them to remain active and complete certain therapy tasks irrespective of how they feel. This may subsequently reduce the importance BA/CPT participants place on the expression of emotion throughout therapy and may subsequently *minimise* the impact under-, and optimal engagement have on outcome. Alternatively, as CPT posits that

without emotional engagement change is not possible, an initial CPT focus may enhance the importance individuals place on engagement and the openness they have to feel emotion. This may therefore translate to an *increased* sensitivity to the effects of under-, and optimal engagement. Overengagement however may have an effect on outcome irrespective of condition, as it is a more pervasive negative state that hijacks processing regardless of approach taken. Simplistically, the way BA/CPT participants start therapy and the BA treatment rationale that emphasises activity as the mechanism underlying change, may reduce the importance BA/CPT participants place on emotional expression and reduce sensitivity to under-, and optimal engagement during treatment.

Supporting such a proposition, research underlines the importance of initial sessions and treatment rationales in predicting posttreatment outcome (Addis & Jacobson, 1996; 2000; DeRubeis et al., 1990; Hayes et al., 2007). For example, during CBT for depression Fennell and Teasdale (1987) found that those who showed larger reductions during the first two weeks of treatment had significantly better long-term outcomes than those who responded more slowly. They found that rapid responders more strongly endorsed cognitive conceptualisations of depression offered early on in therapy, and reported a more positive response to initial homework assignments. Such results not only illustrate the importance of initial sessions in predicting later treatment outcomes but also highlight the importance of providing acceptable treatment rationales.

I have so far proposed that a focus away from emotion onto activity may reduce sensitivity to under- and optimal engagement in BA/CPT. I now discuss the focus initial BA sessions may foster more explicitly, and highlight the potential impact of taking an action orientation. By focusing heavily on activity and goals, initial BA sessions are likely to facilitate an ‘action orientation’ in individuals that is carried throughout therapy. This is of relevance to the current discussion as an action orientation has been found to reduce sensitivity to affect and emotional arousal (Brunstein & Olbrich, 1985;

Koole & Jostmann, 2004; Rholes, Michas, & Shroff, 1989). An action orientation relates to a maintained focus on goals and activity. When an individual subscribes to an action orientation, information processing mechanisms such as the allocation of attention and the inhibition of extraneous or maladaptive cognitions, along with analytic strategies such as problem solving and behaviour routines are used to regulate affect and maintain attempts to achieve goals in the presence of maladaptive affective states (Gollwitzer & Brandstatter, 1997; Kuhl, 1994). That is, an action orientation allows for greater flexibility and self-regulation that then reduces sensitivity to emotion and allows individuals to maintain a focus on goals and actions irrespective of the level of arousal or emotion experienced. For example, research and theory implies that if an individual takes an action orientation they are more likely to stay focused on the task at hand, accomplish goals, and gain good outcomes even in the presence of low or maladaptive affect as an action orientation reduces sensitivity to affect and changes in arousal (Koole & Jostmann, 2004). An action orientation is at odds with a state orientation. State oriented individuals respond to stressful conditions by turning attention inwards and focusing more so on negative affect (Koole & Jostmann, 2004).

The validity of the action orientation construct and its ability to reduce sensitivity to emotion has consistently been supported. Research has found action oriented individuals to report less unpleasant feelings in response to repeated failures, and less depressive symptoms compared to state oriented individuals (Rholes et al., 1989). Experimental research provides further supports for a link between action orientation and reduced negative affect sensitivity (Koole & Jostmann, 2004). Brunstein and Olbrich (1985) showed that repeated failure inductions led state oriented participants to report more negative affect and self blame whilst action oriented participants reported reduced periods of negative affect and increased use of motivating self-instructions. The results of this body of research indicate that the outcomes and activities of action

oriented individuals may be less affected by levels of emotion as their enhanced ability to direct attention towards external objects and goals reduces sensitivity to emotion. As BA facilitates an action orientation by emphasising activity, it is possible that BA/CPT participants may have developed and maintained an action orientation that reduced their sensitivity to affect and potentially their sensitivity to the negative effects of underengagement, while preventing them from maximising the benefits of optimal engagement.

In a similar vein, research suggests that taking an external focus and turning attention towards one's environment rather than one's internal cognitions and emotions is likely to also reduce sensitivity to affect (Nix, Watson, Pyszcynski, & Greenberg, 1995; Nolen-Hoeksema, Morrow, & Fredrickson, 1993; Strack, Blaney, Ganellen, & Coyne, 1985). Relevant to the current discussion, major self-awareness theories propose that an internal self-focus can produce and intensify affective states and enhance sensitivity to emotion (see Pyszczynski & Greenberg, 1992). Theorists assume that an internal, self-focus can influence affect and increase sensitivity to emotion by: (a) increasing awareness of emotion (Scheier & Carver, 1977), (b) increasing awareness of discrepancies between one's current state and goals (Hull & Levy, 1979), and (c) increasing access to self-referent information (Hull & Levy). Instead, an external focus may reduce sensitivity to affect as less attention is paid to emotion, and as goals and one's external environment are the focus of attention. Accordingly, an external focus, as emphasised by BA, may allow BA/CPT participants to achieve reasonable outcomes even when affect is reduced as attention is turned towards external factors that then reduces sensitivity to affect. This may then allow a focus to remain on the achievement of activity irrespective of emotional state. Alternatively, as CPT facilitates an internal focus, CPT and CPT/BA may show heightened sensitivity to emotion and therefore be more affected by under- and optimal levels of engagement. It is of course important to

note that CPT equips clients in this regard, by teaching them skills (most notably cognitive therapy skills) with which to manage distressing thoughts and emotions.

In sum, by emphasising a focus on activity and goals initially, and by providing a treatment rationale that first emphasises activity rather than emotion, initial BA sessions are likely to promote an action orientation that is carried throughout treatment. This orientation might then allow BA/CPT participants to remain focused on activity irrespective of level of affect, and reduce sensitivity to under- and optimal engagement. As research to date has not examined emotional engagement during BA/CPT future research must replicate such findings and determine if BA/CPT does indeed lead to a reduced sensitivity to under-, and optimal engagement. Research must then endeavour to determine why this is the case. In Chapter 1 I argued that methodological limitations of past research have hindered our understanding of the role of engagement. Accordingly, in this thesis I attempted to address such methodological limitations and examine emotional engagement in a more valid way. Methodological issues related to the assessment of engagement are now addressed.

Methodological Issues Related to the Assessment of Emotional Engagement

This is the first study to explicitly correlate peak and modal scores. While peak and modals score were inter-related with one another, compared to modal ratings, peak ratings appeared to minimise proportions of underengagement and overestimate proportions of overengagement. This suggests that modal scores are less sensitive to fluctuations in engagement and thus provide a better estimate of underengagement. As emotional processing theory, along with my findings, emphasise the detrimental impact of underengagement, it is important that future assessments of engagement do not conceal proportions of underengagement. Thus, my findings advance knowledge surrounding the assessment of engagement and advocate for the use of modal scores.

Although emotional processing theory defines under- and overengagement as distinct constructs, past research has not determined if under- and overengagement are separate states. Correlations of under- and overengagement in this thesis generally produced small and nonsignificant results suggesting that under- and overengagement were indeed separate states. However, further explorations of correlations provided some suggestion that the relationship between under- and overengagement was not straightforward. Correlations suggested that for some sessions participants demonstrated a tendency for both under- and overengagement while in other sessions they demonstrated a tendency towards either under- or overengagement. Adding to the complexity of this relationship, the relationship between under- and overengagement differed by condition. At the start of therapy CPT individuals presented with a small tendency for either under- or overengagement and as therapy progressed, this tendency generally disappeared. This was not the case for BA/CPT and CPT/BA. However, one must keep in mind that as results were nonsignificant and not always consistent, this could at best only be considered a modest pattern of findings. Further, as changes in the tendency towards under- and overengagement were confined to CPT, and as it was not my primary aim to examine the connection between under- and overengagement for the different conditions, it is hard to determine how CPT had such an effect on engagement.

The above findings demonstrate that when engagement is treated as an unified construct valuable information is lost. Future examinations of engagement must separate under- from overengagement and researchers must also recognise that individuals can move between the states of under- and overengagement during and over treatment sessions. Further, while this thesis provides some preliminary evidence to suggest that therapy can reduce one's tendency towards either under- or overengagement, future research must explore this in more detail.

As psychometric data for the SUDS is lacking I also examined the validity of SUDS and CEAS ratings. The SUDS was found to reliably covary with CEAS ratings of overengagement thus demonstrating good convergent validity. Also, the SUDS and CEAS ratings of underengagement were not significantly correlated with one another thus demonstrating good discriminant validity. This suggests that the SUDS provides a good snapshot assessment of engagement and demonstrates adequate discriminant and convergent validity with a more objective assessment of engagement. As the SUDS is easily administered, my findings suggest that a reliable assessment of engagement, particularly overengagement, can be gained without spending extensive amounts of time coding sessions. However, the SUDS is not without limitation. Although the SUDS provide a valid assessment of overengagement and may provide a good snapshot of engagement when distress is high (e.g., in trauma account sessions), the SUDS does not provide an assessment of underengagement and is less useful when arousal is low (e.g., during psychoeducation sessions). Thus, researchers interested in assessing underengagement should therefore consider using other methods (e.g., CEAS ratings).

My findings have identified several directions for future research. First, researchers must develop clearer conceptualisations of engagement and determine whether they are interested in modal or peak levels of engagement. Second, as under-, over-, and optimal engagement reflect distinct states, researchers must examine their effects separately. Third, although SUDS provide a good snapshot of overengagement, in order to gain a more comprehensive picture of engagement (i.e., underengagement) more in depth assessments should be used. Fourth, arousal scores should not be summarised with averages and proportion scores should be used to allow under-, over-, and optimal engagement to be analysed separately. Finally, as there are multiple ways to assess engagement researchers should assess engagement with both subjective and more objective methods and continue to assess the validity of these measures.

Limitations

This study is not without limitation. First and foremost, the attrition rate was large. As noted, the high attrition rate may have been the product of undertaking therapy within a community setting that was already characterised by a high rate of dropout. As dropout is elevated within community settings and still remains an issue in randomised control trials for PTSD, future treatment modifications should consider targeting attrition. This is a critical point as my results suggested that if individuals completed treatment they were likely to show good treatment outcomes. As a means of enhancing retention future studies may attempt to replace traditional CPT with CPT-Cognitive (CPT-C). CPT-C is a variant of CPT in which trauma accounts are excluded from the CPT protocol and more emphasis is placed on challenging cognitions (Resick et al., 2008). Current studies suggest that CPT-C is able to reduce PTSD and MDD symptoms to a comparable level as CPT while demonstrating better retention (McCarthy & Pertrakis, 2011; Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). Future research may also attempt to alter the frequency of sessions to determine if this affects retention. Although biweekly sessions have been used in CPT trials with individuals with PTSD alone and those with comorbid PTSD/MDD (e.g., Ehlers et al., 2014; Resick et al., 2002), and while daily and twice-weekly exposure sessions have been shown to result in reduced dropout rates in the treatment of OCD (Abramowitz, Foa, & Franklin, 2003), the utility of daily or biweekly sessions requires further examination in wholly comorbid PTSD/MDD samples. Further, as CPT/BA had a lower rate of dropout, research is needed to determine if CPT/BA reliably predicts better retention.

Second, sample size was small. It is well established that small sample size can mask significant results while also inflating the risk of mistakenly finding significant results where they do not exist. Although this thesis was underpowered and the sample size was small, the consistency of results should be highlighted. Exploration and

analysis of different outcome measures (e.g., CAPS, PCL, DASS-D, PTCI, RRS) consistently supported the efficacy of CPT/BA. Further, analyses conducted using available-case analysis also produced similar results. As the results were incredibly consistent we can be relatively confident in these findings, even in the presence of a small sample size.

The third limitation relates to data retention and the use of multiple imputation I used multiple imputation as this approach is well-studied and has been shown to provide adequate results in the presence of low sample size and high rates of missing data (Graham, et al., 1997; Rubin. 1987; Wayman, 2003). For instance, Rubin (1987) suggests that even with 50% missing data, multiple imputation predicts/imputes values at 95% efficiency. Further, multiple imputation is able to overcome problems seen in other approaches to missing data (e.g., complete case analysis, substituting missing values with the mean of that value, last observation carried forward). For instance, complete-case analysis can reduce sample size, and available-case analysis can lead to bias as it does not take into account that non-responders may differ from responders. Additionally, these methods are based on stronger and more unrealistic assumptions including that the probability of dropout does not depend on anything, dropout is purely random. Alternatively, multiple imputation accounts for uncertainty in missing data and maintains the original variability of the missing data by creating imputed values that are based on variables correlated with the missing data and correlated with causes of missingness. Further, multiple imputation assumes that the probability of a participant having missing values may depend on observed values (such as covariates and pretreatment measures) but not on missing ones (i.e., the values of the posttreatment measures had they been recorded).

However, while multiple imputation is a well-studied and effective means of working with missing data, the high rate of missing data in this thesis should still be

considered a limitation. Missing data at posttreatment and follow-up ranged from 4-55%. Missing data occurred as a proportion of 6-month follow-up data was not due at the time of writing, and as a proportion of participants failed to complete posttreatment and follow-up assessments. While multiple imputation extends beyond other strategies for dealing with missing data, given that the rate of missing data was quite pronounced, it is possible that missing data influenced or skewed results and that different results may have been found if data retention had been greater. As such, results, and in particular 6-month follow-up results, should be interpreted with this in mind. Future research should endeavour to replicate such findings and specifically attempt to enhance data retention.

Fourth, due to time and financial constraints treatment adherence and therapist competence was not independently assessed. Thus, I cannot comprehensively comment on treatment adherence or rule out the possibility that condition differences were the product of differing levels of therapist competence. However, as I conducted most therapy sessions across all conditions, it is unlikely that adherence and competence differed significantly between conditions.[16] Also, as Reg Nixon and myself routinely reviewed therapy tapes, issues in competence and adherence are likely to have been detected. None-the-less, there is a need for replication with a larger sample and for treatment fidelity and therapist competence to be assessed. Finally, while I used pre-defined cut-offs of under-, over-, and optimal engagement that were based on theory and past research, clinicians would benefit from future research that identifies the typical proportion of engagement that is reliably associated with good or poor outcomes. Developing such clinical cut-offs would provide clearer guidance for clinicians to identify problematic engagement in session.

[16] Although I acknowledge that it is possible that I had a bias towards a particular treatment which influenced how a treatment was delivered, given the results were not as hypothesised, this suggests that this was not the case.

Implications of Treatment and Process Findings

This study abided by CONSORT guidelines while still achieving a high level of ecological validity. By including participants who had a range of comorbidities and who reported complex trauma histories my sample represented the clients typically seen in clinical practice. Further, as my study took place in two community services, research was conducted in a naturalistic setting and abided by the procedures seen in typical community practices. Other strengths included the randomised, crossover design and the assessment of outcome and process measures throughout therapy.

While this study joins a growing body of evidence that supports the efficacy of CPT in the treatment of PTSD and MDD, my thesis builds upon previous research and allows three explicit conclusions surrounding the treatment of comorbid PTSD/MDD to be made. First, a combined focus on PTSD *and* MDD symptoms leads to *superior* outcomes compared to CPT alone. However, treatment presentation order is important and influences the efficacy of treatment modifications. Second, although CPT and BA/CPT showed some evidence of catch-up at follow-up, CPT/BA should be considered the treatment of choice for comorbid PTSD/MDD. Third, optimal levels of engagement are critical to the CPT process.

As most CPT treatment trials use mixed samples of individuals with PTSD and comorbid PTSD/MDD it has previously been hard to determine whether CPT is sufficient when MDD is significantly elevated, or if a combined treatment is necessary. This thesis therefore contributes significantly to the field and suggests that while CPT is an effective treatment for individuals with comorbid PTSD/MDD, clinicians can gain superior outcomes with a combined CPT/BA approach, and suggests that CPT/BA should be offered as the treatment of choice for individuals who present with comorbid PTSD/MDD. As CPT/BA produced superior outcomes compared with BA/CPT, my findings also illustrate the ability of treatment presentation order to inhibit or enhance

treatment outcomes. This suggests that clinicians should be cautious when considering supplementing CPT with additional treatment components. When comorbidity exists clinicians must not only consider what treatment components should be added but also how and when such components will be incorporated (i.e., sequential vs. combined treatments). In order to better inform clinical practice randomised control trials must incorporate comparison conditions that compare altered treatment presentation orders. This would be complemented by research investigating the role of case formulation in clinicians' decision-making when it comes to combining treatment approaches and targeting specific symptoms, a need that has recently been highlighted in clinical domains (Eells, 2013; Persons, 2008).

Emotional engagement findings also emphasise the need for clinicians to be attentive to levels of emotional engagement during treatment. Clinicians should gently enhance engagement and the expression of affect when underengagement occurs and should attempt to down regulate emotion and facilitate more moderate or optimal levels of arousal when overengagement occurs. Perhaps most importantly, my findings suggest that clinicians should move away from the belief of the benefits of unregulated catharsis or the notion that 'more emotion is better'. In line with this, researchers and clinicians must attempt to determine useful means of facilitating optimal levels of engagement during treatment. Techniques that enhance arousal when individuals are underengaged and techniques that reduce engagement when individuals are overengaged are of specific importance. Techniques such as grounding and distress tolerance are well established as techniques that reduce distress or overengagement (Linehan, 1993). However, less is known about how clinicians can help individuals overcome habitual patterns of underengagement. Dialectical behaviour therapy (DBT) may serve as a useful starting point as DBT not only aims to facilitate distress tolerance and the dampening of overwhelming affect, but also aims to facilitate connection with, and the

mindfulness of emotion (Dimeff & Koerner, 2007; Linehan, 1993). Strategies employed within DBT that may reduce underengagement include: 1) psychoeducation surrounding emotion, 2) tasks to help individuals identifying, monitor, and label their emotions, and 3) the teaching of mindfulness skills to allow individuals to be open to the "emotional mind" and current emotional experiences.

The reduced sensitivity that BA/CPT showed to the effects of under-, and optimal engagement also has implications for clinical practice. One could argue that BA/CPT's reduced sensitivity to emotional engagement is a potential strength of this treatment as a reduced sensitivity to engagement in essence safeguards participants from the detrimental effects of under- or non-optimal engagement. Further, one may argue that if BA/CPT reduces sensitivity to the effects of under- and optimal engagement, BA/CPT may be a good treatment for new therapists who are not yet well equipped at regulating clients' emotional expression. However, one must remember that although lower levels of optimal engagement did not predict reduced treatment outcomes for BA/CPT participants, it was also the case that higher levels of optimal engagement did not predict enhanced treatment outcomes. In other words, BA/CPT participants missed out on the beneficial effects of optimal engagement. This is a noticeable point given that a large proportion of session time for clients was spent in an optimally engaged state and very little session time was spent underengaged. One must also keep in mind that BA/CPT did not achieve effects as large as CPT/BA. Thus, although BA/CPT participants were less sensitive to under-, and optimal engagement, CPT/BA should still be considered the treatment of choice. Areas for future research are now addressed.

Future Research

In addition to the need for replication and suggestions already made, I now propose areas for future research. Although BA was used to target MDD, other effective

treatments for MDD exist and these different treatments may contribute to different outcomes. Therefore, future examinations of combined PTSD/MDD treatments should explore the utility of other MDD treatments. Mindfulness based cognitive therapy (MBCT) may prove a viable treatment to combine with CPT as it lends itself well to a reduced treatment length and has been found to have good outcomes on MDD (see Hofmann, Sawyer, Witt, & Oh, 2010). Specifically, by teaching individuals to better recognise and feel their emotions, and by halting the escalation of negative thoughts by emphasising a focus on the present moment, MBCT may reduce MDD symptoms and enhance the efficacy of CPT. Additionally, while combined treatments may be used to target comorbid PTSD/MDD, transdiagnostic treatments that target a variety of disorders simultaneously may also prove fruitful in the treatment of comorbid PTSD/MDD. Although the efficacy of unified protocols has been empirically supported in the treatment of emotional disorders (e.g., Mansell, Harvey, Watskins, & Shafran, 2008), no study to date has explored transdiagnostic treatments for comorbid PTSD/MDD and studies seldom make head-to-head treatment comparisons whereby transdiagnostic approaches are compared to single or combined treatments.

In this thesis level of emotional engagement was the primary variable of interest. However, future exploration should not only look at the strength of the emotion displayed (i.e., based on a CEAS rating) but should also explore the *type* of emotion displayed. Specific attempts should be made to explore the nature and type of emotion expressed in session, and the interplay between emotion type, level of arousal, and outcome. Theoretically such exploration is warranted as the influence of emotional arousal is likely to be dependent on the type of emotion displayed (e.g., are highly aroused ratings of anxiety better predictors of poorer outcomes than highly aroused ratings of hopelessness?), and because studies have consistently found distinct emotion states such as shame, anger, and hopefulness to predict PTSD and MDD symptom

severity (Andrews, 1995; Andrews, Brewin, Rose & Kirk, 2000; Andrews & Hunter, 1997). Additionally, as emotional processing is predicted to occur both in and between treatment sessions it is important that research begin to explore the importance of emotional engagement *outside* of the therapy room. An initial starting point may be to explore the relationship between outcome and self-reported SUDS during homework tasks, especially when reading and completing trauma account homework assignments.

The influence of culture should also be touched on. In my thesis the cultural homogeneity of my sample (i.e., middle class, Caucasian) precluded the exploration of culture. However, as culture influences the expression of emotion, it is possible that culture may have an influential role in the predictive ability of under-, over-, and optimal engagement. Cultural display rules are culturally prescribed rules that are learnt through socialisation and relate to the management and modification of emotional displays (Ekman & Friesen, 1969; Hofstede, McCrae, 2004; Matsumoto et al., 2008). Cultural display rules influence one's expression of emotion whereby one expresses emotion in light of what their culture defines as acceptable or unacceptable (Matsumoto, Kasri, & Kooken, 1999). In other words, cultural display rules determine how, when, and to whom people will express their emotional experiences. Within individualistic cultures personal feelings, and their free expression confirm the importance of the individual. Alternatively, collectivistic cultures focus on groups, contexts, and relationships, and personal feelings and their free expression are relatively less important. In line with this research suggests that emotions have relatively greater intrapersonal meaning in individualistic cultures (Matsumoto et al., 2008; Suh, Diener, Oishi, & Triandis, 1998) and that individuals from individualistic cultures tend to endorse greater overall emotional expressivity (Matsumoto et al., 2008; Safdar et al., 2009).

As different cultures have different norms regarding the display of emotion, the predictive value of level of emotional engagement may differ depending on culture. It may be the case that the predictive value of emotional engagement is circumscribed to individuals from individualistic or western backgrounds who more freely express emotion. Future research is needed to explore this. It is interesting to note however, that although culture may influence emotional expression and processing, the effectiveness of CPT have been observed in multicultural samples including Kurds in Northern Iraq, Bosnian refugees, and trauma survivors from the Democratic Republic of the Congo (e.g., Kaysen, Lindgrem, Zangana, Murray, Bass, & Bolton, 2013; Kruse, Joksimovic, Cavka, Woller, & Schmitz, 2009; Schulz, Resick, Huber, & Griffith, 2006).

Concluding Remarks

I have shown that there is indeed benefit in targeting both PTSD and MDD symptoms in the treatment of comorbid PTSD/MDD. Specifically, I have demonstrated the superior efficacy of a combined CPT/BA treatment, and recommend this as a viable and effective treatment for those with comorbid PTSD/MDD. My results also suggest that achieving optimal engagement is critical to the CPT process. While CPT is proving to be an extremely versatile protocol for the treatment of PTSD, the challenge for future research rests in determining when a single PTSD-focused treatment is sufficient and when a combined treatment is required. Put simply, research must determine how treatment outcomes can be maximised when comorbidity exists. As ‘pure’ PTSD is rarely seen in clinical practice, research must continue to explore methods that maximise the efficacy of CPT in the presence of the complexities that often accompany our clients.

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Appendix A: Completer and Non-Completer Participant Demographics

Table A1

Participant Demographic and Baseline Characteristics for Completers and Non-Completers

Characteristics Mean (SD) or number (%)	Completers (n = 25)	Non-Completers (n = 24)	Test	p	*ES* *d or phi*
Age years, mean (SD)	34.84 (13.60)	30.29 (11.46)	t = 1.26	.21	0.36[a]
Female	24 (52.2%)	22 (47.8)%	χ^2 = 0.40	.52	0.09[b]
Caucasian ethnicity	21 (47.7%0	23 (52.3%)	χ^2 =5.07	.28	0.32
Total years of education, mean (SD)	14.16 (2.67)	12.52 (2.50)	t = 2.21	.03	0.63
Currently employed	15 (53.6%)	13 (54.2%)	χ^2 = 0.17	.68	0.06
Income					
Less than $10,000	4 (16%)	2 (12.5%)			
$10,000 – 30,000	5 (20%)	7 (29.2%)			
$30,001 – 50,000	7 (28%)	9 (37.5%)	χ^2 = 3.31	.51	0.26
$50,001 – 70,000	8 (32%)	3 (12.5%)			
$70,001 – 90,000	0 (0%)	0 (0%)			
More than $90,000	1 (4%)	2 (8.3%)			
Marital status					
Single	14 (56.0%)	11 (44%)			
Married/cohabiting	5 (20%)	7 (29.2%)	χ^2 = 2.17	.54	0.21
Divorced/separated/widower	1 (4%)	3 (12.5%)			
Relationship not living together	5 (20%)	3 (37.5%)			

Characteristics Mean (SD) or number (%)	Completers (n = 25)	Non-Completers (n = 24)	Test	p	*ES* *d or phi*
Index Trauma					
Adult physical assault	4 (16%)	5 (20.8%)			
Adult sexual assault	14 (56%)	12 (50%)			
Child physical abuse	0 (0%)	1 (4.2%)	$\chi^2 = 4.05$	.54	0.29
Child sexual abuse	1 (4%)	3 (12.5%)			
Armed hold up or home invasion	4 (16%)	1 (4.2%)			
Motor vehicle accident	2 (8%)	2 (8.3%)			
Years since index trauma, mean, (SD)	5.31 (7.59)	4.02 (6.32)	$t = 0.65$	.52	0.33
Current co-morbid diagnoses					
Additional mood disorder	9 (36%)	4 (16.7%)	$\chi^2 = 2.35$	.13	0.22
Additional anxiety disorder	14 (56%)	14 (58.3%)	$\chi^2 = 0.03$	.87	0.02
Substance abuse or dependence	4 (16%)	8 (33.3%)	$\chi^2 = 1.99$	.16	0.20
Total number of disorders, mean, (SD)	2.32 (1.15)	3.13 (1.42)	$t = -2.19$	.03	0.63
Currently on psychotropic medication	9 (36%)	13 (54.2%)	$\chi^2 = 1.63$	.20	0.18
Order of PTSD/MDD Onset					
PTSD onset prior to MDD	5 (20%)	4 (16.7%)	$\chi^2 = .20$	.91	0.06
MDD onset prior to PTSD	12 (48%)	13 (54.2%)			
PTSD and MDD onset concurrently	8 (32%)	7 (47%)			

Characteristics Mean (SD) or number (%)	Completers (n = 25)	Non-Completers (n = 24)	Test	p	*ES* *d or phi*
Baseline Assessment Measures					
CAPS	76.88 (20.43)	81.91 (16.59)	t = -0.95	.35	0.23
PCL	57.64 (10.58)	60.38 (9.40)	t = -0.96	.34	0.27
DASS –Depression	23.36 (9.90)	22.17 (10.71)	t = 0.41	.69	0.12
PTCI	140.52 (33.85)	140.63 (37.43)	t = -0.01	.99	0.00
RRS	55.92 (12.62)	56.38 (15.21)	t = -0.11	.91	0.03
SRRS	1329.80 (437.28)	1333.96 (442.64)	t = -0.03	.97	0.01
Emotional Numbing Questionnaire	27.56 (6.39)	27.42 (6.20)	t = 0.08	.94	0.02
TAS	60.12 (9.79)	58.55 (12.93)	t = 0.50	.62	0.14

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; ES = Effect Size; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

[a.] Effect size conventions for Cohen's *d*: 0.2 = small, 0.5 = medium, and 0.8 = large.

[b.] Effect size conventions for phi: 0.1 = small, 0.3 = medium, and 0.5 = large.

Appendix B: Raw Cognitive Emotion Regulation Questionnaire Descriptive Statistics

Table B1

CERQ Descriptive Statistics: Raw Data for Intent-to-Treat Sample

	CPT			BA/CPT			CPT/BA	
CERQ Subscale	*n*	M	SD	n	M	SD	*n*	M	SD
				Pretreatment					
Self blame	18	4.22	1.80	15	5.13	2.23	16	4.44	2.12
Acceptance	18	6.39	2.17	15	5.73	2.25	16	7.25	2.51
Rumination	18	5.44	2.45	15	6.07	2.25	16	7.63	1.78
Positive refocusing	18	4.94	2.29	15	3.80	1.61	16	4.56	1.59
Refocus on planning	18	5.17	1.86	15	4.73	1.67	16	5.06	1.81
Positive reappraisal	18	5.28	2.56	15	5.33	2.74	16	6.63	2.53
Putting into perspective	18	4.50	2.01	15	5.33	2.55	16	5.56	2.22
Catastrophising	18	5.94	2.75	15	7.20	2.39	16	6.50	2.10
Other blame	18	5.29	2.70	15	4.00	2.59	16	4.44	2.58
Total	18	47.06	11.53	15	47.00	10.21	16	52.13	8.75
				Postreatment					
Self blame	18	3.89	2.37	12	3.50	1.68	12	2.83	1.11
Acceptance	18	6.89	2.11	12	7.42	2.19	12	7.58	2.71
Rumination	18	5.61	2.00	12	5.92	1.73	12	4.67	1.50
Positive refocusing	18	5.61	2.64	12	4.42	2.02	12	6.00	1.95

	CPT			BA/CPT			CPT/BA	
CERQ Subscale	*n*	M	SD	*n*	M	SD	*n*	M	SD
Refocus on planning	18	5.78	2.24	12	4.58	2.02	12	6.67	2.34
Positive reappraisal	18	6.72	2.49	12	6.75	2.49	12	8.75	1.60
Putting into perspective	18	5.61	2.15	12	5.00	2.41	12	5.08	2.75
Catastrophising	18	4.94	2.60	12	4.45	2.23	12	3.58	1.56
Total	18	51.22	10.62	12	48.45	8.32	12	51.33	9.66
				6-Month Follow-up					
Self blame	11	3.72	1.68	6	3.60	2.07	6	3.67	2.34
Acceptance	11	7.00	2.37	6	6.20	1.92	6	7.83	2.48
Rumination	11	5.45	2.21	6	3.60	2.05	6	3.83	1.47
Positive refocusing	11	5.72	1.68	6	6.20	2.05	6	5.17	2.63
Refocus on planning	11	5.45	2.07	6	5.40	2.88	6	6.83	2.79
Positive reappraisal	11	6.63	2.94	6	7.20	2.28	6	8.17	2.86
Putting into perspective	11	5.00	2.19	6	6.00	2.00	6	6.67	2.50
Catastrophising	11	4.45	2.21	6	3.20	2.17	6	3.17	0.98
Other blame	11	5.00	2.72	6	5.60	3.36	6	4.17	1.17
Total	11	48.45	10.85	6	47.00	10.07	6	49.50	9.07

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CERQ = Cognitive Emotion Regulation Questionnaire.

Appendix C: Correlations of CERQ with Other Outcome Measures

Table C1

Correlation of Pretreatment CERQ Total and Subscales With Other Related Pretreatment Measures

CERQ Subscale	PTCI	PTCI - NS	PTCI - NW	PTIC - SB	RRS	SRRS	EN	TAS	PCL	DASS-D
CERQ: Self blame	.20	.10	-.09	.58**	.15	.16	.31	.30	.25	.19
CERQ: Acceptance	-.16	-.15	.15	-.15	.19	.15	-.28	-.23	-.09	-.07
CERQ: Rumination	.26	.24	.41*	.06	.68**	.63**	.08	.17	.10	.22
CERQ: Positive refocus	-.15	-.20	.02	-.25	-.03	-.07	-.21	-.17	-.10	-.27
CERQ: Refocus on planning	-.27	-.25	-.13	-.18	-.11	-.05	-.22	-.28	.02	-.04
CERQ: Positive reappraisal	-.09	-.23	.15	.09	.15	.13	-.25	-.11	.04	-.07
CERQ: Putting into perspective	-.06	-.12	.14	-.13	.03	.05	-.28	-.17	-.05	-.00
CERQ: Catastrophising	.18	.18	.29*	-.08	.62**	.67**	.38*	.32*	.38*	.39*
CERQ: Other blame	.03	.11	.25	-.52**	.14	.12	.08	.13	.05	.15
CERQ: Total score	.01	-.04	.29*	-.15	.43*	.42*	-.09	-.09	.13	.12

Note. CERQ = Cognitive Emotion Regulation Questionnaire; PTCI = Posttraumatic Cognitions Inventory Total; PTCI – NS = Posttraumatic Cognitions Inventory – Negative Self Subscale; PTCI – NW = Posttraumatic Cognitions Inventory – Negative World Subscale; PTCI – SB = Posttraumatic Cognitions Inventory – Self Blame Subscale; RRS = Ruminative Response Scale of The Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale.

* $p < 0.05$; ** $p < 0.01$.

Appendix D: Emotional Numbing Questionnaire

Please answer each of these questions about how you have felt in the **past week**. Circle one number next to each question to indicate how you have felt over the past week.

	Not at all	Mildly	Medium	Quite a bit	Very much
1. Did you feel numb or distant from your emotions over the past week?	1	2	3	4	5
2. Did you ever feel in a daze over the past week?	1	2	3	4	5
3. Did things around you ever feel unreal or dreamlike over the past week?	1	2	3	4	5
4. Did you ever feel distant from your normal self over the past week?	1	2	3	4	5
5. Have you been less interested in activities you used to enjoy over the past week?	1	2	3	4	5
6. Have you felt distant from other people over the past week?	1	2	3	4	5
7. Have there been times when you felt emotionally numb or had trouble experiencing feeling like love of happiness over the past week?	1	2	3	4	5

Appendix E: Client Emotional Arousal Scale-III

Introduction

The Emotional Arousal Scale-III is a 7-point process measure that was developed to assess the intensity of observable, expressed emotional intensity, based on the evaluation of audio or videotapes of psychotherapy sessions. Using this scale, an emotional response is indicated when a person acknowledges having experienced an emotion (e.g., I feel afraid) or when a person demonstrates an emotion action tendency (e.g., covering one's head in shame or shrinking back in fear). The higher levels of the scale indicate higher emotional arousal intensities. The quality of emotional arousal at the same levels of the scale can vary depending on the type of emotion being expressed. For example, one would expect higher levels of anger to be reflected by increasing assertiveness and loudness in voice, while fear may be characterized by a more timid, shaky, and softer sounding vocal quality.

Depending on the context, emotional intensity in therapy can be helpful or unhelpful and sometimes even disruptive (e.g. when a client experiences a panic attack). This scale measures the intensity of emotional expression regardless of its therapeutic value. Therefore, there are no assumptions or judgments made concerning the therapeutic or non-therapeutic nature of the level of expressed emotional intensity.

This scale is an elaboration and revision of an original Emotional Arousal Scale by Daldrup, Beutler, Engle, and Greenberg (1988) developed to measure the presence, intensity, and function of anger in Focused Expressive Psychotherapy. As this original scale showed promise in its application to verbal and non-verbal material, particularly anger, it was first revised by Machado (1992) and later expanded by Machado, Beutler, and Greenberg (1999) to include six primary emotions (love, anger, fear, joy, surprise, and sadness) and their intensities. This scale also incorporates aspects of Rice, Koke, Greenberg and Wagstaff's (1979) Client Vocal Quality Classification System.

Key terms in manual

Emotional Voice

We use one of the vocal quality styles called "emotional voice" described in Rice, Koke, Greenberg, & Wagstaff's (1979) Manual for Client Vocal Quality which was designed to assess the quality of the client's involvement in the therapy process.

Emotional voice is characterized by "an overflow of an emotion into a speech pattern" or a "disruption of ordinary speech patterns (vol 2, p.14)." Their emotional category refers to when statements in the speech pattern are disrupted or distorted to some extent by emotional overflow (Rice and Kerr, 1986). Ordinary speech patterns may change in different ways depending on the emotion being expressed.

Aspects of Emotional Voice

Aspects of speech patterns identified by Rice et al. related to emotional arousal are:

1. Accentuation pattern;
2. Regularity of pace;
3. Terminal contours; and
4. Whether there has been a disruption of speech patterns.

1. Accentuation pattern refers to emphasis patterns in sentences. In the English language, accentuation of words tends to occur in particular ways in sentences. This can either give the effect of a regular beat that can be more pronounced than usual for the English language, analogous to a sermon, e.g., "we are gathered here today to…". Conversely, accentuation patterns can also be more irregular than usual.
2. Regularity of pace refers to the variation of pace within a particular utterance. For example, a person may begin speaking quickly and continue the last half of their phrase in a slower manner.
3. Terminal contours involve aspects of pitch like evenness, rises or drops in pitch. Contours can be used in an accentuating speech-making way, or they can give the total intonation pattern a more ragged, unexpected sound.

4. Finally, disruption of speech pattern refers to the extent to which the regular speech pattern is disrupted or distorted by emotional overflow.

When emotional arousal is present, one would expect the emotion being expressed to overflow into the speech pattern. The clearest sign of emotional arousal is a disruption of ordinary speech patterns (e.g. A person's voice may break or tremble). When emotional arousal occurs, the accentuation is usually irregular, the regularity of pace is uneven, the terminal contours are unexpected, and there is a disruption of normal speech patterns.

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Paralinguistics

- Paralanguage refers to the non-verbal elements of communication used to modify meaning and convey emotion.
- It is important to understand that as emotions become more active and as the person becomes more emotionally engaged in therapy, you should see that speech becomes louder, faster, higher pitched, less resonant and clipped.
- Paralanguage may be expressed consciously or unconsciously, and it includes the pitch, volume, and, in some cases, intonation of speech.

Pitch:

- The distinctive quality of a sound, dependent primarily on the frequency of the sound waves produced by its source.
- Studies exploring pitch have found that contempt was expressed by low pitch, a wide pitch range and extreme variations in inflection. Anger had the greatest shift in pitch, generally downwards inflection, and the greatest variability. Fear was high pitched, with the widest pitch range and few pauses. Grief was slowly spoken with the least variability among features (Fairbanks & Pronovost, 1939).
- Pitch is found to increase during times of high arousal, fear and deception (Ekman et al., 1976; Streeter, Krauss, Geller, Olson, & Apple, 1977)

Volume:

- The fullness or intensity of tone or sound

Tone:

- Any sound considered with reference to its quality, pitch, strength, source
- Accentuation and inflection:
- To place strength or emphasis on something.
- It is based on stress and varies from a monotone to rising and falling or irregular pattern.
- Accentuation pattern refers to emphasis patterns in sentences. In the English language, accentuation of words tends to occur in particular ways in sentences. This can either give the effect of a regular beat that can be more pronounced than usual for the English language, analogous to a sermon, e.g., "we are gathered here today to…". Conversely, accentuation patterns can also be more irregular than usual.

Regularity:

- Relates to the consistency of the rate and intensity of communications
- Regularity of pace refers to the variation of pace within a particular utterance. For example, a person may begin speaking quickly and continue the last half of their phrase in a slower manner.
- Articulation control:
- Relates to the degree of control the individual exercises over their pronunciation of words (i.e., precise articulation vs. slurred)

Clipping:

- Clipping occurs when syllables are squeezed into shorter time intervals and the transition between elements is precise.
- Frequency or Rate:
- Relates to the speed at which words are spoken

Speech Disturbances

- Disruption of speech pattern refers to the extent to which the regular speech pattern is disrupted or distorted by emotional overflow.
- This is based on errors in speech which increase during the expression of certain emotions. There is evidence to show that speech disturbances are associated with higher levels of anxiety and emotional experience.

Speech disturbances can be separated into the following eight categories (Mahl, 1956)

1. Filled pauses
   E.g., "ah," "er", "um" and so on
2. Sentence corrections
   Related to when a client corrects the form or content of a given statement
3. Sentence incompletions
   Sentences or phrases are left incomplete without correction
4. Repetition of words
   Unnecessary repetition of one or more words.
5. Stuttering
   Repetition of consonants or syllable of words or,
   Frequent pauses or the drawing out of sounds
6. Intruding incoherent words
   Relates to when incomprehensive or incoherent words are added to certain phrases, sentences of statements
7. Tongue slips
   Conscious or unconscious deviations from the apparently intended form of an utterance.
8. Partial or complete omission of words
   Words or parts of words omitted or passed over;
   Generally end syllables of words

Individuals' Baselines and the More General Nature of Arousal

This scale measures emotional intensity as it occurs in verbal interactions between people. When making decisions about levels of arousal, it is important to consider both the general nature of arousal, as well as individual differences in expression of arousal. Thus, when rating emotional arousal, we are interested in noting and considering deviations from a person's baseline level of arousal as represented by his or her normal speech in therapy. Using this scale a rating of approximately 3 and 4 would indicate a normal level of emotional expression (i.e., arousal we would expect to see in an everyday conversation).

Ratings of 1 and 2 indicate a withholding of emotion; levels 3 and 4 would be considered slightly elevated arousal (optimal therapeutic arousl) and levels 5 to 7 indicate heightened to uncontrolled arousal. These levels/ratings should be weighed against a client's baseline level of arousal (baseline being a level 3).

Emotion Categories

We have found the following emotion categories most relevant to psychotherapy sessions. Before a segment can be rated on arousal, it first must be categorized according to the following emotion list. If

the segment does not fit into any of the categories, it should be classified as an unspecified bad emotion. This 16th category has been added to avoid losing information about emotionally aroused moment

1. Pain/Hurt
2. Sadness
3. Hopelessness/Helplessness
4. Loneliness
5. Anger/Resentment
6. Contempt/Disgust
7. Fear/Anxiety
8. Love
9. Joy/Excitement
10. Contentment/Calm/Relief
11. Shame/Guilt
12. Pride/Self-confidence
13. Anger and Sadness
14. Pride (Self-assertion) and Anger
15. Surprise/Shock
16. Unspecified bad emotion

Ratings

Units for Rating

The smallest unit that can be rated is a client statement. Segment size can be determined depending on the specific needs of the investigator. For example, videotapes can be segmented according to significant therapy themes, or units of time in a therapy session or episodes of emotions. Within the current study units of rating were one minute.

Types of Ratings

Both modal and peak ratings can be given to segment rated on this arousal scale.

- A modal rating represents the overall or average level of the segment being rated.
- A peak rating characterizes the highest level of arousal attained in the segment being rated.

The Current Study

For the purpose of this study we are concerned with the peak and modal rating of each therapeutic minute. Each successive minute of a session should be given an emotional label from the above list. Then a modal and peak expressed arousal rating should be given. If an emotion it not recognizable, it should be named as unspecified and an arousal rating still given.

Coding should start when the video starts (i.e., 0:00secs). Please record the exact minute/second coding begins. Coding should cease either when the therapist starts to schedule the time for the next session or when the recorder is turned off. Every consecutive 5 minute segment should be given the emotion category name and arousal rating of the minute with the highest arousal appearing within the 5 minutes.

- In cases in which more than one of the 5 minute segments has an equally high arousal rating, the segment should be categorized according to the emotion category with the greatest number of minutes of high arousal.
- If the number of highest arousal minutes is equal in the different categories, then the 5 minutes segment should be given more than one emotion category rating.
- However, one exception exists: If the unspecified bad category conflicts with another unpleasant emotion from a general bad feeling category consisting of pain/hurt, sadness, hopelessness/helplessness, loneliness, fear/anxiety, and shame/guilt, then the rating of unspecified bad should be dropped in favor of the more specific category

In sum, coding should start when the video starts. Each minute should be given an emotional label and a peak and modal arousal rating. Then, every 5 minute segment should also be labeled with the emotion category name and arousal rating of the minute with the highest arousal. Each minute is given a peak rating, a modal rating and an emotional label. Scores of 1 and 2 should be considered restricted engagement or under-engagement. Scores of 3 and 4 should be considered optimal engagement. Scores of 5 and above should be considered to indicate increased or over-engagement.

Level 1 – No emotional expression, extreme withholding or numbing of feelings

Summary of category:

Client does not acknowledge emotion. There is **no** arousal in voice or body, emotion appears numbed or withheld.

- The client does not express emotions (verbally or physically). To gain a rating of one, the client should not express or label feelings.
- There appears to be a void of emotional expression.
    - There should be a sense of significant emotional numbness or withholding or avoidance of feelings
    - Emotion expression may not appear congruent with topics discussed
- The person appears **distant or not present** within the session; may appear numb, detached or avoidance.
- There may even appear to be a lack of understanding surrounding feelings and emotions.

This rating is evidenced by:

1. Voice **<u>does not</u>** disclose any emotional arousal
    - There are **<u>no changes</u>** in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
    - Speech **<u>does not</u>** appear to become louder, faster, higher pitched, less resonant or clipped.
    - Speech should appear almost dull and flat and absent of change. It appears duller or more monotone than an everyday conversation.
    - Below characteristics should be void or flat – tone, volume, pitch, accentuation, articulation control, clipping and frequency should be consistent, controlled or changes absent.
    - ❖ **Notes: There should be no significant changes in any of the above paralinguistic categories. Speech should almost appear monotone.**
      All the below categories should appear flat or normal. Speech should almost appear monotone and dull.

    Tone:
    - <u>Tone must appear normal and controlled. Tone should remain consistent.</u>
    - The tone of communication should not waiver and change as emotional concepts are discussed. The speech pattern does not appear monotone or flat.
    - Even if the content being expressed is emotional the tone of communication remains consistent and level.

    Volume:
    - <u>Volume must appear normal, and controlled. Volume should remain consistent and well controlled.</u>
    - Speech is neither loud nor silent.
    - The volume of speech reflects that evident in an everyday communication.
    - The volume of speech should not be elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
    - The volume of speech should not waiver between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)

    Pitch:
    - <u>Pitch must appear normal, and controlled for the coded sequence.</u>
    - Speech pitch remains consistent and does not waiver. The pitch is neither elevated nor lowered. Pitch does not change and remains consistent
    - Speech pitch does not appear notably elevate or lower than baseline (i.e., pitch appears to be higher or lower than would occur in an everyday conversation)
    - Anxiety, fear or depression does not appear evident in voice.

    Accentuation and inflection:

- Accentuation and inflection must appear normal, and controlled for the coded sequence.
- There does not appear to be any accentuation evident in communications. The client does not place emphasis on certain words or phrases.
- Speech seems monotone or without emphasis (i.e., droid like).
- Accentuation of certain words or phrases is not significantly evident. The individual does not place emphasis on certain words or phrases.
- Speech does not appear more animated and appears monotone.

Articulation control:

- Articulation should appear normal, and controlled for the coded sequence.
- Words do not appear to be slurred.
- Stuttering or difficulty getting words out is not evident.
- There is no evidence of poor articulation control.

Clipping:

- Clipping should not be present.
- Words do not appear shortened or squeezed or,
- Words do not appear to be an effort to say

Frequency and Rate:

- Frequency and rate should appear normal, and controlled for the coded sequence.
- The rate of speech does not change. That is, the rate at which the client speaks remains the same regardless of the topic being discussed.
- Frequency and rate are consistent.
- Speech appears smooth and does not appear jerky
- Speech should not appeared rushed and somewhat frantic or,
- The rate of speech does not appear to change. That is, the rate at which the client speaks does not alter between fast, moderate or slow (i.e., speech does appears appear jerky)

2. To gain a rating of 1 speech disturbances should be **absent**.
   - ❖ **Note: To meet this criterion there should be a near-absence of speech disturbances and if speech disturbances exist they should not appear to be connected to emotional expression (i.e., memory or concentration related)**

   Speech disturbances include:
   - Sentence corrections
   - Sentence incompletions
   - Repetition of words
   - Stuttering
   - Intruding incoherent words
   - Tongue slips
   - Partial or complete omission of words

3. To gain a rating of 1 body language **does not** disclose any emotional arousal.
   - ❖ **Note: For body language to be considered closed there should be significant evidence of closed body language (see below) and a near-absence of engaged body language (see below).**
     Body language is completely closed or withheld and the individual should not appear open or interested in the topics being discussed.
   - Individual should appear closed or disinterested in topics discussed. The person may not appear present in the session.

   Closed body language includes:

- Head resting in hand for majority of coded sequence (i.e., > 50% of coded sequence)
- Eyes downcast for majority of coded sequence
- Head down for majority of coded sequence
- Body physically turned away from therapist for majority of coded sequence
- Arms crossed across body for majority of coded sequence
- Consistently looking down or away for majority of coded sequence
- Gaze aversion for majority of coded sequence

Examples of engaged body language (i.e., evidence against this rating) would include:

- Sitting forward in seat for majority of coded sequence (i.e., > 50% of coded sequence)
- Consistent eye contact for majority of coded sequence
- Head movements (e.g., quickly tilted head, nodding) present for majority of coded sequence
- Biting nails for majority of coded sequence
- Fidgeting hands for majority of coded sequence

4. There should be an **absence of gestures and body movement**

- ❖ **Feet and body should remain still almost without movement for the majority of coded sequence (i.e., > 50% of coded sequence)**

- The person should not have significant arms movements or gestures whilst talking.
- There should be no presence of fidgeting or difficulty sitting still.
- Gestures should not disclose any sense of not being comfortable, anxiety, guilt or sadness.

5. The person should appear **rigid or tense**

- People can tend to tense their muscles, rigidify their movements and over-elevate their shoulders in an attempt to block the expression and in some cases the experience, of an unpleasant or threatening emotional state.
- In extreme cases this defence may develop into an enduring patter of muscular rigidity which prevents people form interacting in a free and easy way with their environment and those around them.
  - ❖ **Note: The client should appear very rigid and tense. The majority of the below examples of rigid expression should be present for most of the coded sequence.**

Examples of rigid expression include:

- Sitting still without moving for the majority of coded sequence (i.e., > 50% of coded sequence)
- Not relaxing into one's chair for the majority of the coded sequence
- Staring straight ahead for the majority of coded sequence
- Gaze aversion for the majority of coded sequence
- Rigid chin for the majority of coded sequence
- Clenched jaw for the majority of coded sequence
- Firm voice for the majority of coded sequence

6. The client does not **lean forward** to meet the therapist.
   - ❖ **Note: The proportion of leaning forwards towards the therapist should not exceed 40% of the coded sequence.**
   - You may notice that the individual has a lowered head, tilting it to one side, running the head away from the therapist, supporting the head on one hand, leaning back and stretching out the legs

7. **Eye contact** is void or **incredibly minimal**. There is a sense of not wanting to meet the therapist's gaze

- ❖ **Note: The proportion of eye contact towards the therapist should not exceed 50% of the coded sequence (i.e.,. should avoid eye contact for more than half of the time).**

***Note*:** Refusal to discuss certain topics may be evident but is not necessary for this rating

To gain this rating:

- All 7 categories must be met
- The individual **should not name emotions** (current and past)
  - If the client names emotions being expressed but all categories are covered, a level 2 should be given

Level 2 – Very little arousal, evidence of some withholding or numbing of feeling

Summary of category:

Client shows a limited acknowledgement of emotion. The client may acknowledge emotion but there is **very little** arousal in voice or body.

- A level 1 corresponds to the absence of emotion or total withholding. Alternatively, a level 2 is where the client demonstrates **minimal or some emotional expression** but there is **still some evidence of withholding of emotion.**
- Arousal appears significantly limited (but not completely void). There still appears to be some numbing or withholding of emotion.
- Emotional should appear duller than what would be expected from a typical conversation. The client may appear uninterested or distance within session.

This rating is evidenced by:

1. Voice discloses only very small amounts of emotional arousal.
   - There are **very slight changes** in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
   - Speech does not appear to become significantly louder, faster, higher pitched, less resonant or clipped. However, there may be some slight changes evident.
   - Speech mimics that used in everyday settings or may appear slightly duller.

   - ❖ **Notes: There should be no significant changes in most of the below paralinguistic categories. There should only be slight changes evident – voice should still appear somewhat flat and monotone**
     The majority of the categories should appear flat or normal. However, speech does not necessarily need to be absent of any change (i.e., only minimal change).

   Tone:

   - Tone must appear relatively normal and controlled. Tone should remain relatively consistent however, some changes may be evident.
   - The tone of communication should not waiver and change as emotional concepts are discussed. The speech pattern does not appear monotone or flat.
   - Even if the content being expressed is emotional the tone of communication remains consistent and level.

   Volume:

   - Volume must appear relatively normal and controlled; it should remain relatively consistent however, some changes may be evident.
   - Speech is neither loud nor silent and reflects that evident in an everyday communication.

- The volume of speech should not be significantly elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
- The volume of speech should not waiver significantly between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)

Pitch:

- Pitch must appear normal, and controlled for the majority of the coded sequence. There may be some changes in pitch; however, change is not significant.
- Speech pitch remains consistent and does not waiver significantly. There may be slight evidence of wavering.
- Speech pitch does not appear notably elevated or lower than baseline (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
- Pitch does not change significantly and remains consistent
- Anxiety, fear or depression not apparently evident in voice.

Accentuation and inflection:

- Accentuation and inflection must appear normal, and controlled for the majority of the coded sequence.
- There does not appear to be significant accentuation evident in communications. The client does not place strong emphasis on certain words or phrases.
- Speech seems slightly monotone or without emphasis (i.e., droid like).
- Accentuation of certain words or phrases is not significantly evident. The individual does not place emphasis on certain words or phrases.
- Speech does not appear more animated than an everyday conversation.

Articulation control:

- Articulation should appear normal, and controlled for the majority of the coded sequence.
- Words do not appear to be slurred.
- Stuttering or difficulty getting words out is not evident.
- There is no significant evidence of poor articulation control.

Clipping:

- Significant clipping should not be present within the coded sequence.
- Words do not appear shortened or squeezed or,
- Words do not appear to be a significant effort to say

Frequency and Rate:

- Frequency and rate should appear normal, and controlled for the majority of the coded sequence.
- The rate of speech does not change significantly and appear consistent.
- Frequency and rate are consistent.
- Speech appears smooth and does not appear jerky
- Speech should not appeared rushed and somewhat frantic
- The rate at which the client speaks does not significantly alter between fast, moderate or slow (i.e., speech does appears appear jerky)

2. Speech disturbances are **only minimal.**
   - **Note: Only minimal speech disturbances should be present. There should not be a pronounced presence of disturbances.**
     Disturbance should appear to be more related to memory or concentration issues than emotional expression.

Speech disturbances include:

- Sentence corrections
- Sentence incompletions
- Repetition of words
- Stuttering
- Intruding incoherent words
- Tongue slips
- Partial or complete omission of words

3. Body language **does not** disclose any significant emotional arousal and appears relatively closed.
    - ❖ **Note: For body language to be considered closed there should be evidence of at least three below indicators of closed body language (or similar) and there should be a near-absence of engaged body language.**
      Whilst a rating of 1 requires an absence of engaged body language, a rating of 2 is gained if only minimal engaged indicators are present (i.e., only one or two indicator of engaged body language present)
      Body language appears relatively closed or withheld and the individual does not appear open or significantly interested in the topics being discussed
    - There should be a sense of the client being disconnected within the session (however, this does not need to be completely absent as there can be some minimal indicators

    Closed body language can include:
    - Head resting in hand for the majority of coded sequence (i.e., > 50% of coded sequence)
    - Eyes downcast for the majority of coded sequence
    - Head down for the majority of coded sequence
    - Body physically turned away from therapist for the majority of coded sequence
    - Arms crossed across body for the majority of coded sequence
    - Consistently looking down or away for the majority of coded sequence
    - Gaze aversion for the majority of coded sequence

    Examples of engaged body language include:
    - Sitting forward in seat for the majority of coded sequence (i.e., > 50% of coded sequence)
    - Consistent eye contact for the majority of coded sequence
    - Head movements (e.g., quickly tilted head, nodding) for the majority of coded sequence
    - Biting nails for the majority of coded sequence
    - Fidgeting hands for the majority of coded sequence

4. There should be a near absence of gestures and body movement
    - ❖ **Note: Feet and body should remain still almost without movement for a significant proportion of the coded sequence (i.e., 40%)**
    - The person should not have significant arms movements or gestures whilst talking; movements should seem restricted and minimal
    - There should be *minimal* fidgeting. Whilst there may be small amounts of fidgeting, arms should remain relatively still for the majority of the coded sequence.

5. The person should appear somewhat rigid or tense
    - ❖ **Note: The client should appear rigid or tense. There should be a presence of some of the below examples.**
      Overall, the client still appears somewhat tense and reserved however there may be some appearance of slight relaxing
    - Examples of rigid expression include:

- Sitting still without moving for the majority of coded sequence (i.e., > 50% of coded sequence)
- Not relaxing into one's chair for the majority of the coded sequence
- Staring straight ahead for the majority of coded sequence
- Gaze aversion for the majority of coded sequence
- Rigid chin for the majority of coded sequence
- Clenched jaw for the majority of coded sequence
- Firm voice for the majority of coded sequence

6. The client is not leaning forward to meet the therapist
   - ❖ **Note: The proportion of leaning forwards towards the therapist should not exceed 50% of the coded sequence**

7. Eye contact is void or incredibly minimal; there is a sense of not wanting to meet the therapist's gaze
   - ❖ **Note: The proportion of eye contact towards the therapist should not exceed 60% of the coded sequence (i.e.,. should avoid eye contact for more than 40% of coded sequence).**

To gain this rating:

- Approximately five categories should be met (not strict)
- The individual may label emotions but there is a sense that they are not being outwardly expressed in their voice or body language
- Note: level 2 reflects a more effortful withholding of emotion or more significant presence of numbing compared to a level 3.

Level 3 – Mild arousal - typical of everyday conversation

Summary of category:

Person acknowledges emotions, but displays in voice and body are **mild**. Arousal is **mild** in voice and body and reflects the level of arousal expected for a **typical everyday conversation**.

- There should be very little emotional overflow in body and voice
- Whilst levels 1 and 2 reflect withholding and numbing, level 3 involves minimal arousal (i.e., typical in everyday) with a near-absence or numbing or active withholding.
- Usual speech patterns are only **mildly disrupted** and there is evidence of mild arousal or emotion expression.

This rating is evidenced by:

1. Voice discloses **only mild levels** of emotional arousal. There are **only slight changes** in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
   - Speech mimics that used in everyday settings possibly with a minimal level of restriction
   - There are **mild changes** in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
   - Speech slightly appears to waiver in volume, pitch etc. Slight changes in paralinguistic cues are evident and appear to be typical for what one would expect in an everyday conversation.
     - ❖ **Notes: To meet this rating, there should be slight changes in most of the below paralinguistic categories.**
       Speech should reflect that of everyday speech only with mild changes or flatness
       Speech should not be significantly monotone or flat – this should only be considered minor/mild
       Speech should appear only slightly restricted

     Tone:

- Tone must appear somewhat normal and controlled however, some changes should appear evident (i.e., not completely monotone)
- The tone of communication should waiver slightly as emotional concepts are discussed. The speech pattern should not appear completely monotone or flat.

Volume:

- Volume must appear somewhat normal and controlled however, some changes should be evident.
- The volume of speech may be slightly elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
- Volume may slightly waiver between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)
- There should be mild changes in volume – as would be expected in everyday conversation

Pitch:

- Pitch must appear relatively however there should be some changes in pitch (not significant; only mild).
- Mild evidence of wavering.
- Speech pitch appears slightly elevated or lower than baseline (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
- Pitch changes mildly and does not remain consistent
- Anxiety, fear or depression may appear evident in voice to a mild degree (this should not be overly dominant).

Accentuation and inflection:

- Accentuation and inflection appears relatively normal, and controlled; there are some instance of mild changes.
- Mild accentuation evident in communications. The client places small emphasis on certain words or phrases.
- Speech does not seem monotone or without emphasis (i.e., droid like).
- Mild accentuation of certain words or phrases is evident. The individual places some emphasis on certain voices or phrases.
- Speech appears slightly more animated and does not appear monotone.

Articulation control:

- Articulation should appear relatively normal, and controlled with only mild disturbances present
- Some words may appear to be slurred but this is not significant
- Some (mild) level of stuttering or difficulty getting words out is evident.
- There is no significant evidence of poor articulation control – typical of everyday

Clipping:

- Clipping should not be present to a mild extent within the coded sequence.
- Words appear shortened or squeezed to a mild extent
- Words appear to be a slightly difficult to say

Frequency and Rate:

- Frequency and rate should appear normal, and controlled for the majority of the coded sequence.
- The rate of speech does changes mildly
- Speech should appeared slightly rushed and somewhat frantic or,

- The rate of speech changes slightly. That is, the rate at which the client speaks will alter between fast, moderate or slow (i.e., speech appears appear jerky)

2. Speech disturbances are **minimal**. Speech disturbances include:
   - ❖ **Note: Minimal speech disturbances should be present. There should a slight presence of disturbances.**
     Disturbances reflect that which would be expected to be seen in everyday conversation.

   Speech disturbances include:
   - Sentence corrections
   - Sentence incompletions
   - Repetition of words
   - Stuttering
   - Intruding incoherent words
   - Tongue slips
   - Partial or complete omission of words

3. Body language only reflects **mild** emotional arousal
   - Body language is minimally closed and the individual appears slightly interested in the topics being discussed
     - ❖ **Note: For body language to be considered mild there should only be one indicator of closed body language present. There should be some evidence of engaged body language (i.e., at least one example)**
       Body language should still appear somewhat closed and the individual should only appear slightly interested in the topics being discussed
   - Client should not appear completely absent or disconnected

   Closed body language can include:
   - Head resting in hand for the majority of coded sequence (i.e., > 50% of coded sequence)
   - Eyes downcast for the majority of coded sequence
   - Head down for the majority of coded sequence
   - Body physically turned away from therapist for the majority of coded sequence
   - Arms crossed across body for the majority of coded sequence
   - Consistently looking down or away for the majority of coded sequence
   - Gaze aversion for the majority of coded sequence

   Examples of engaged body language include:
   - Sitting forward in seat for the majority of coded sequence (i.e., > 50% of coded sequence)
   - Consistent eye contact for the majority of coded sequence
   - Head movements (e.g., quickly tilted head, nodding) for the majority of coded sequence
   - Biting nails for the majority of coded sequence
   - Fidgeting hands for the majority of coded sequence

4. Gestures and body movement should only be **mild or infrequent**
   - The person should not have significant arm or body movements or gestures whilst talking; movement should appear relatively normal and what would be expected in everyday
   - Need to consider client's baseline – i.e., some clients are more expressive than others
   - There should only be *minimal* fidgeting. Whilst there may be small amounts of fidgeting, arms should remain relatively still for at least 50% of the coded sequence

5. The person should appear **only mildly rigid or tense**

- ❖ **Note: The client's rigidity and tenseness should only appear minimal or slight. There should only be a slight a presence of some of the below examples. There should be some appearance of slight relaxing, but overall, the client still appears a little tense.**

Examples of rigid expression include:

- Sitting still without moving for the majority of coded sequence (i.e., > 50% of coded sequence)
- Not relaxing into one's chair for the majority of the coded sequence
- Staring straight ahead for the majority of coded sequence
- Gaze aversion for the majority of coded sequence
- Rigid chin for the majority of coded sequence
- Clenched jaw for the majority of coded sequence
- Firm voice for the majority of coded sequence

6. The client only **slightly leans forward** to meet the therapist
   - ❖ **Note: The proportion of leaning forwards towards the therapist should not exceed 60% of the coded sequence**

7. **Eye contact is minimal;** there is a sense of not wanting to meet the therapist's gaze
   - ❖ **Note: The proportion of eye contact towards the therapist should not exceed 70% of the coded sequence (i.e., should avoid eye contact for more than 30% of coded sequence).**

To gain this rating:

- Approximately four categories should be met (not strict)
- The individual may label emotions but there is a sense that they are not being outwardly expressed in their voice or body language

Level 4 – Moderate/Slightly elevated Arousal

Summary of category:

Person acknowledges emotions and arousal appears to be **slightly elevated** as **compared to baseline or everyday conversation**. Arousal is **moderate** in voice and body.

- There is a small levels of emotional overflow in body and voice
- There is some freedom from control and restraint. Arousal appears slightly higher than what would be expected for everyday conversation.
- No distinct numbing or withholding of emotion present - Person appears to be somewhat aware of emotion
- Whilst level 3 involves minimal arousal (i.e., typical in everyday), level 4 involves slightly more elevated arousal with more changes and disturbances present.
- Usual speech patterns are **moderately to mildly disrupted** and there is evidence of moderate arousal or emotion expression. Emotional voice is present: Ordinary speech patterns are **moderately** disrupted by emotional overflow as represented by changes in accentuation patterns, unevenness of pace, changes in pitch
- Emotional voice is present: This involves the "turning inward of attentional energy

This rating is evidenced by:

1. Voice discloses **moderate levels of emotional arousal.**

- There should be **moderate changes** in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
  - ❖ **Notes: There should be moderate changes in most of the below paralinguistic categories.**

Speech should not be significantly monotone or flat
Speech should deviate slightly from normal everyday speech
Whilst there should be some deviations and disruptions in speech, speech should still have a small level of restriction or consistency (i.e., emotion should not seem overly intense)

Tone:

- Tone must show some slight to moderate changes and should not appear monotone. Tone may waiver from what would be considered normal. Tone appear somewhat normal and controlled however, some changes should appear evident
- The tone of communication seems to waiver and change as emotional concepts are discussed.
- The speech pattern does not appear monotone or flat.

Volume:

- Volume must waiver or change slightly. Volume should appear slightly louder or softer than would be expected in everyday or should waiver more than expected.
- The volume of speech may be elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
- The volume of speech waivers between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)

Pitch:

- There should be some changes in pitch (mild-moderate).
- Speech pitch appears notably elevate or lower than baseline (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
- Pitch changes and does not remain consistent or,
- Anxiety, fear or depression appears evident in voice.

Accentuation and inflection:

- Accentuation and inflection demonstrates moderate changes.
- Accentuation of certain words or phrases is evident. The individual places emphasis on certain voices or phrases.
- Speech appears more animated.

Articulation control:

- Mild to moderate disturbances present
- Words may appear slurred or,
- Some level of stuttering or difficulty getting words out is evident.

Clipping:

- Clipping should be present to a mild extent within the coded sequence.
- Words appear shortened or squeezed or,
- Some words may appear to be an effort to say

Frequency and Rate:

- Frequency and rate should waiver slightly
- Speech should appeared somewhat rushed and somewhat frantic or,
- The rate of speech should change. That is, the rate at which the client speaks will alter between fast, moderate or slow (i.e., speech appears appear jerky)

2. Speech disturbances are **slight/moderate.**

- ❖ **Note: Slight speech disturbances should be present.**
  Disturbances slightly exceed that which would be expected to be seen in everyday conversation.

Speech disturbances include:

- Sentence corrections
- Sentence incompletions
- Repetition of words
- Stuttering
- Intruding incoherent words
- Tongue slips
- Partial or complete omission of words

3. Body language reflects **moderate** emotional arousal.
   - Body language is relatively open.
   - The individual appears open and interested in the topics being discussed
     - ❖ **Note: There should the presence of approximately 5 indicators of open/engaged body language and a near-absence of closed body language cues.**
       To gain this rating body language appears relatively open and the individual should appear interested in the topics being discussed
       The individual does not appear frozen or lifeless

Examples of closed body language include:

- Head resting in hand for the majority of the coded sequence (i.e., > 50% of coded sequence)
- Eyes downcast for the majority of the coded sequence
- Arms crossed across body for the majority of the coded sequence
- Looking down or away for the majority of the coded sequence
- Gaze aversion for the majority of the coded sequence

Examples of engaged body language would include:

- Sitting forward in seat for the majority of the coded sequence
- Learning forward to meet the therapist the for majority of the coded sequence
- Consistent eye contact with the therapist for the majority of the coded sequence
- Head movements (e.g., nodding, quickly tilted head) for the majority of the coded sequence
- Fidgeting hands for the majority of the coded sequence. Can include:
  - Playing with ring
  - Moving hand to nose or chin,
  - Touching hair,
  - Putting hands in pockets,
  - Tapping table,
  - Moving feet,
  - General fidgeting
- Biting or picking nails for the majority of the coded sequence
- Fidgeting with worksheets for the majority of the coded sequence
- Swinging feet for the majority of the coded sequence
- Changes in facial expressions – smile, frowns, squinting etc.
- Observable increase in breathing rate for the majority of the coded sequence
- Observable increase in perspiration for the majority of the coded sequence
- Crying or evidence of eyes welling up for the majority of the coded sequence
- Complexion changes such as in colour; red in face or neck area for the majority of the coded sequence
- Rocking or shifting in seat for the majority of the coded sequence

4. Gestures and body movement should be **<u>moderate and relatively frequent</u>**
    - The person should have significant arms, leg or body movements or gestures whilst talking; movements should not seem overly restricted
    - There should be *moderate* fidgeting.
    - Client should be moving for at least 60% of coded sequence (approximately)

5. The person should **<u>not appear significantly rigid or tense</u>**, there should be a sense of feeling more relaxed and freed
    - ❖ **Note: The client's rigidity and tenseness should only appear very slight. There should be an appearance of relaxing. Overall, the client should not appear tense.**

    Examples of rigid expression include:
    - Sitting still without moving or relaxing for the majority of the coded sequence
    - Staring straight ahead for the majority of the coded sequence
    - Gaze aversion for the majority of the coded sequence
    - Rigid chin for the majority of the coded sequence
    - Clenched jaw for the majority of the coded sequence
    - Firm voice for the majority of the coded sequence

6. The client should lean forward to meet the therapist
    - ❖ **Note: The proportion of leaning forwards towards the therapist should be 60% or greater of the coded sequence**

7. Eye contact relatively consistent; There is no sense of not wanting to meet the therapist's gaze
    - ❖ **Note: The proportion of eye contact towards the therapist should exceed 60% of the coded sequence**

To gain this rating:
- <u>Four categories</u> should be met (not strict)
- There should be some level of emotional acknowledgment although the client does not need to state what emotion they are feeling or make note of specific emotions felt

<u>Level 5 – Fairly Intense and Full Arousal but Control Still Present</u>

Summary of category:

Person acknowledges emotions. Arousal appears to be **fairly elevated** as **compared to baseline or everyday conversation**. Arousal is **full and fairly intense** in voice and body.

- Arousal in voice and body is **fairly intense and full.** Arousal in voice obviously **exceeds what would be typical of an everyday conversation** or **exceeds baseline level of arousal**. However, whilst arousal is elevated there is still some level of control (i.e., there is still a line that the person will not cross; there is still some constraint in emotional expression)
- There is a slight freedom from control and restraint. Arousal appears obviously higher than what would be expected for everyday conversation. However, speech is still understandable and demonstrates a sense of control.
- There is **still a certain level of control evident**; the person does not appear to be lost in their emotion.
- Emotional voice if full and there is an overflow of emotion into voice and body. Emotion overflows into speech pattern to a **moderate extent**: speech patterns deviate from the client's baseline, and are fragmented or broken.
- Usual speech patterns are **notably disrupted** and there is evidence of moderate arousal or emotion expression.

This rating is evidenced by:

1. Voice discloses **full levels of emotional arousal and appears fairly intense.**
   - Voice discloses **higher levels of emotional arousal** and appears to **exceed baseline levels of arousal**
   - There should be changes in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
   - Speech should deviate from normal everyday speech due to deviations in paralinguistic cues. That is, speech should not mimic that used in everyday life
   - Whilst there should be some deviations and disruptions in speech, speech should still have an appearance of control (i.e., emotion should not seem overly intense; emotions do not seem to be out of control or speech so disturbed that it is hard to understand)

     ❖ **Notes: There should be fairly significant changes in the majority of the below paralinguistic categories.**
     Speech should not be significantly monotone or flat
     Speech should deviate from normal everyday speech – appear more emotional or highly aroused
     Whilst there should be some deviations and disruptions in speech, speech should still have a small level of restriction or consistency (i.e., emotion should seem slightly intense, not seem overly intense)

Tone:

- Tone must show some moderate to distinct changes and should not appear monotone. Tone should waiver from what would be considered normal but not cause voice/communication to be incomprehensible
- The tone of communication waivers moderately and changes as emotional concepts are discussed.
- The speech pattern does not appear monotone or flat.

Volume:

- Volume waivers to a moderate extent and should slightly louder or softer than would be expected in everyday or should waiver more than expected. There is a distinct disturbance in voice volume.
- The volume of speech is elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
- The volume of speech waivers between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)

Pitch:

- There should be a moderate and distinct change in pitch
- Speech pitch appears notably higher or lower than baseline (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
- Pitch changes and does not remain consistent.
- Anxiety, fear, or depression appears evident in voice.

Accentuation and inflection:

- Accentuation and inflection demonstrates distinct changes.
- Accentuation of certain words or phrases is evident. The individual places emphasis on certain voices or phrases.
- Speech seems more animated and does not appear monotone.

Articulation control:

- **Articulation demonstrates moderate disturbances** present
- Some words may appear to be slurred or,
- There is some level of stuttering or difficulty getting words out is evident.

Clipping:

- Clipping should be present to a moderate extent, there is some appearance of finding it hard to articulate words
- Words appear shortened or squeezed or,
- Some words may appear to be an effort to say.

Frequency and Rate:

- Frequency and rate should waiver moderately
- Speech should appeared rushed and somewhat frantic or,
- The rate of speech changes. That is, the rate at which the client speaks will alter between fast, moderate or slow (i.e., speech appears appear jerky)

2. Speech disturbances are **notable and distinct** – more than would be expected for everyday speech or baseline.
   - ❖ **Note: Speech disturbances should be moderately present.**
     Disturbances exceed every day or baseline communication

Speech disturbances include:

- Sentence corrections
- Sentence incompletions
- Repetition of words
- Stuttering
- Intruding incoherent words
- Tongue slips
- Partial or complete omission of words

3. Body language reflects **moderate** emotional arousal. There is a sense of being more aroused than baseline or what would be expected in everyday. Body language is **open and unrestricted.**
   - Body language is open and seems unrestricted. Individual appears open to conversation and does not appear defensive.
   - ❖ **Note: Approximately 5 indicators of open/engaged body language should be present and there should be a near-absence of closed body language cues.**
     Body language appears open and the individual appear interested in the topics being discussed
     The individual does not appear frozen or lifeless – there is an openness to their body language

Examples of closed body language include:

- Head resting in hand for the majority of the coded sequence (i.e., > 50% of coded sequence)
- Eyes downcast for the majority of the coded sequence
- Arms crossed across body for the majority of the coded sequence
- Looking down or away for the majority of the coded sequence
- Gaze aversion for the majority of the coded sequence

Examples of engaged body language would include:

- Sitting forward in seat for the majority of the coded sequence
- Learning forward to meet the therapist the for majority of the coded sequence
- Consistent eye contact with the therapist for the majority of the coded sequence
- Head movements (e.g., nodding, quickly tilted head) for the majority of the coded sequence
- Fidgeting hands for the majority of the coded sequence. Can include:
  - Playing with ring
  - Moving hand to nose or chin,

    - Touching hair,
    - Putting hands in pockets,
    - Tapping table,
    - Moving feet,
    - General fidgeting
- Biting or picking nails for the majority of the coded sequence
- Fidgeting with worksheets for the majority of the coded sequence
- Swinging feet for the majority of the coded sequence
- Changes in facial expressions – smile, frowns, squinting etc.
- Observable increase in breathing rate for the majority of the coded sequence
- Observable increase in perspiration for the majority of the coded sequence
- Crying or evidence of eyes welling up for the majority of the coded sequence
- Complexion changes such as in colour; red in face or neck area for the majority of the coded sequence
- Rocking or shifting in seat for the majority of the coded sequence

4. Gestures and body movement distinctively show emotional arousal and body movements are **frequent** through coded sequence. Movements should **exceed baseline and slightly exceed** what would be expected for **everyday conversation**.
    - The person should move arms, feet and body whilst talking, movements should not seem restricted and should seem slightly exaggerated.
    - There should be distinct fidgeting or movement
    - Client should be moving for at least 70% of coded sequence (approximately)

5. The person should **not appear rigid or tense**, the client should appear freed and movement apparent
    - ❖ **Note: The client should not at all appear rigid. There should be an appearance of being free. Overall, the client should not appear tense.**

    Examples of rigid expression include:
    - Sitting still without moving or relaxing for the majority of the coded sequence
    - Staring straight ahead for the majority of the coded sequence
    - Gaze aversion for the majority of the coded sequence
    - Rigid chin for the majority of the coded sequence
    - Clenched jaw for the majority of the coded sequence
    - Firm voice for the majority of the coded sequence

6. The client should lean forward to meet the therapist
    - ❖ **Note: The proportion of leaning forwards towards the therapist should be 70% or greater of the coded sequence**

7. Eye contact relatively consistent; There is no sense of not wanting to meet the therapist's gaze
    - ❖ **Note: The proportion of eye contact towards the therapist should exceed 60% of the coded sequence**

To gain this rating:
- Five categories should be met (not strict0
- There should be some level of emotional acknowledgment although the client does not need to state what emotion they are feeling or make note of specific emotions felt

Level 6 – Very Intense and Extremely Full Arousal with Reduced Control

Summary of category:

Arousal is **very intense and extremely full**, the person is freely expressing emotion in voice and body.

- Arousal is very intense and full, distinctively exceeds baseline and what would be considered typical.
- There is a small sense of the person being unable to control their emotions.
- Emotion overflows into speech pattern to an extreme extent: speech patterns deviate **markedly** from the client's baseline, and are fragmented or broken.
- Overflow of emotion into voice and body is obviously apparent and observable. Usual speech patterns are **extremely disrupted** as indicated by changes in accentuation patterns, unevenness of pace, changes in pitch, and volume or force of voice.

This rating is evidenced by:

1. Voice discloses **high and extremely full levels of emotional arousal.**
    - There should be extreme changes in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
    - There should be significant deviations and disruptions in speech and there should be an appearance of limited control (i.e., emotion seem overly intense and slightly out of control; speech may be hard to understand)
    - Speech should deviate from normal everyday speech
    - Voice discloses **high/extreme levels of emotional arousal** and appears to **significantly exceed baseline levels of arousal**
    - Sense that person cannot control the effect emotion has on voice.
    - There should be **no evidence of emotion being restricted**. The individual should not appear to be holding back or masking emotion
        - ❖ **Notes: There should be extreme changes in most of the below paralinguistic categories.**
          Speech should not be monotone or flat
          Speech should deviate significantly from normal everyday speech and appear less controlled. Emotion/arousal should appear to be slightly overwhelming and spill into voice without client's control
          Restriction and consistently should be nearly absent

    Tone:

    - Tone shows distinct changes and does not appear monotone. Tone waiver significantly from what would be considered normal. Limited sense of control over tone should be evident.
    - Tone waivers and changes significantly as emotional concepts are discussed.
    - The speech pattern does not appear monotone or flat but had specific and there are significant changes in tone.

    Volume:

    - Volume waivers significantly. Volume appears extremely louder or softer than would be expected in everyday or should waiver significantly more than expected.
    - The volume of speech is elevated (i.e., appears to be louder than would occur in an everyday conversation) or,
    - The volume of speech waivers significantly between being loud and silent (i.e., volume is not consistent and there seems to be a reduced ability to control the volume of one's speech)

    Pitch:

    - There should be significant changes in pitch
    - Speech pitch appears notably higher or lower than used in everyday (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
    - Pitch changes and does not remain consistent.
    - Anxiety, fear or depression appears evident in voice.
    - There is a wavering quality to the voice

    Accentuation and inflection:

- Accentuation and inflection demonstrates significant changes and accentuation or inflection may at times appear odd.
- Accentuation of certain words or phrases is evident.
- The individual places emphasis on certain voices or phrases. Speech seems very animated or forceful and does not appear monotone.

Articulation control:

- Articulation demonstrates extreme disturbances
- Some words may appear to be slurred or,
- Some level of stuttering or difficulty getting words out is evident

Clipping:

- Clipping should be significantly present
- Words appear shortened or squeezed or,
- Some words may appear to be an effort to say.

Frequency and Rate:

- Frequency and rate should waiver extremely – it should be slightly hard to understand what is being said at times
- Speech should appear rushed and somewhat frantic. Speech is at times so rushed it is incoherent or,
- The rate of speech changes significantly. That is, the rate at which the client speaks will alter between fast, moderate or slow (i.e., speech appears appear jerky)

2. Speech disturbances are **extreme.**
   - ❖ **Note: Extreme speech disturbances should be present**
     Disturbances significantly exceed that which would be expected to be seen in everyday conversation.

Speech disturbances include:

- Sentence corrections
- Sentence incompletions
- Repetition of words
- Stuttering
- Intruding incoherent words
- Tongue slips
- Partial or complete omission of words

3. Gestures disclose **extreme** emotional arousal.
   - Body language is relatively open. Individual appears open and interested in discussed topics
   - The individual does not appear frozen or lifeless
   - ❖ **Note: There should the presence of approximately 5 indicators of open/engaged body language and a near-absence of closed body language cues.**
     To gain this rating body language appears relatively open and the individual should appear interested in the topics being discussed. Or, body language may contradict topic or feelings discussed.
     The individual does not appear frozen or lifeless

Examples of closed body language include:

- Head resting in hand for the majority of the coded sequence (i.e., > 50% of coded sequence)
- Eyes downcast for the majority of the coded sequence
- Arms crossed across body for the majority of the coded sequence
- Looking down or away for the majority of the coded sequence

- Gaze aversion for the majority of the coded sequence

Examples of engaged body language would include:

- Sitting forward in seat for the majority of the coded sequence
- Learning forward to meet the therapist the for majority of the coded sequence
- Consistent eye contact with the therapist for the majority of the coded sequence
- Head movements (e.g., nodding, quickly tilted head) for the majority of the coded sequence
- Fidgeting hands for the majority of the coded sequence. Can include:
    - Playing with ring
    - Moving hand to nose or chin,
    - Touching hair,
    - Putting hands in pockets,
    - Tapping table,
    - Moving feet,
    - General fidgeting
- Biting or picking nails for the majority of the coded sequence
- Fidgeting with worksheets for the majority of the coded sequence
- Swinging feet for the majority of the coded sequence
- Changes in facial expressions – smile, frowns, squinting etc.
- Observable increase in breathing rate for the majority of the coded sequence
- Observable increase in perspiration for the majority of the coded sequence
- Crying or evidence of eyes welling up for the majority of the coded sequence
- Complexion changes such as in colour; red in face or neck area for the majority of the coded sequence

4. Gestures and body movement should be **frequent and exaggerated.** Movements should definitely **exceed baseline and** what would be expected for **everyday conversation**.
    - The person should move arms, feet and body whilst talking, movements should not seem restricted and should seem exaggerated.
    - The person should have significant arms, leg or body movements or gestures whilst talking; movements should not seem overly restricted
    - There should be high levels of fidgeting
    - There should be *moderate* fidgeting.
    - Client should be moving for at least 60% of coded sequence (approximately)

5. The person **should not** appear rigid or tense
    - ❖ **Note: The client should not appear rigid.**

    Examples of rigid expression include:

    - Sitting still without moving or relaxing for the majority of the coded sequence
    - Staring straight ahead for the majority of the coded sequence
    - Gaze aversion for the majority of the coded sequence
    - Rigid chin for the majority of the coded sequence
    - Clenched jaw for the majority of the coded sequence
    - Firm voice for the majority of the coded sequence

6. The client should lean forward to meet the therapist
    - ❖ **Note: The proportion of leaning forwards towards the therapist should be 70% or greater of the coded sequence**

7. Eye contact relatively consistent; There is no sense of not wanting to meet the therapist's gaze
    - ❖ **Note: The proportion of eye contact towards the therapist should exceed 60% of the coded sequence**

To gain this rating:

- **Approximately six categories** should be met and **category 1 must be met**. If six categories are met, but category one is not, assign a level 5 rating

Level 7 – Extremely Intense and Extremely Full Arousal and Uncontrolled

Summary of category:

Arousal is **extremely intense and full** in voice and body. Emotion appears **uncontrolled** and spills into voice and body significantly.

- Arousal is extremely intense and full, distinctively exceeds baseline and what would be considered typical. There is a strong sense of the person being unable to control their emotions.
- Speech patterns are **completely disrupted** by emotional overflow
- The expression is **completely spontaneous and unrestricted**
- Arousal appears uncontrollable and enduring.
- There is a falling apart quality: Although arousal can be a completely unrestricted therapeutic experience, it may also be a disruptive negative experience in which the clients feels like they are falling apart
- May be under-regulated i.e. overwhelming.
- Overflow of emotion into voice and body is obviously apparent and observable.

Control = containment in contrast to control = restriction

* The distinguishing feature between level 6 and level 7 is that in level 6 there is the sense that although a person's expression may be fairly unrestricted, this individual would be able to contain or control his or her arousal, whereas in level 7, a person's expression is completely unrestricted and there is the sense that emotional arousal would not be within this person's control.

This rating is evidenced by:

1. Voice discloses **high and extremely full levels of emotional arousal**. Emotion appears **overwhelming** and the expression of emotion almost seems **too much for the person to bear.**
   - There should be significant changes in paralinguistic cues such as tone, volume, accentuation pattern, or regularity of speech.
   - Speech should deviate from everyday speech. Appears to fall apart or be incoherent in parts.
   - Speech should appear under-regulated or hard to understand
   - No evidence of emotion being restricted and speech should be heavily uncontrolled
   - The individual should not appear to be holding back or masking emotion. Rather, it may appear as though the individual is showing too much emotion
   - ❖ **Notes: There should be extreme changes in the below paralinguistic categories and speech should appear very disorganized and uncontrolled.**
     Speech should not be monotone or flat and may be hard to understand
     Speech should deviate significantly from normal everyday speech. Strong sense of speech being uncontrolled
     Emotion/arousal should appear to be significantly overwhelming and spill into voice without client's control
     Restriction and consistently should be completely absent

   Tone:
   - Tone shows distinct changes and does not appear monotone. Tone waiver significantly from what would be considered normal. There is very limited control over tone
   - The tone of communication waivers and change significantly as emotional concepts are discussed.
   - The speech pattern does not appear monotone or flat but had specific and significant changes in tone.

Volume:

- Volume waivers extremely and appears extremely louder or softer than would be expected in everyday or should waiver significantly more than expected. There is limited volume control
- The volume of speech is significantly elevated any may have a yelling or hostile like quality (i.e., appears to be louder than would occur in an everyday conversation) or,

Pitch:

- Extreme and uncontrolled changes in pitch
- Speech pitch appears notably high or lower than used in everyday (i.e., pitch appears to be higher or lower than would occur in an everyday conversation) or,
- Pitch changes and does not remain consistent. Anxiety or depression appears evident in voice. There is a wavering quality to the voice

Accentuation and inflection:

- Significant and uncontrolled disturbances are present
- Accentuation of certain words or phrases is evident. The individual places emphasis on certain voices or phrases.
- Speech seems very animated or forceful and does not appear monotone.
- Accentuation may at time not make sense

Articulation control:

- Accentuation and inflection demonstrates significant changes and accentuation or inflection may at times appear odd. Limited control is apparent
- Some words may appear to be slurred or,
- Some level of stuttering or difficulty getting words out is evident.

Clipping:

- Present and uncontrolled
- Words appear shortened or squeezed or,
- Some words may appear to be an effort to say

Frequency and Rate:

- Waiver extremely and appears uncontrolled - hard to understand what is being said at times
- Speech should appear rushed and somewhat frantic. Speech is at times so rushed it is incoherent or,
- The rate of speech changes significantly. That is, the rate at which the client speaks will alter between fast, moderate or slow (i.e., speech appears appear jerky)

2. Speech disturbances are **significant and uncontrolled** .
    - ❖ **Note: Extreme speech disturbances should be present**
      Disturbances significantly exceed that which would be expected to be seen in everyday conversation.

Speech disturbances include:

- Sentence corrections
- Sentence incompletions
- Repetition of words
- Stuttering
- Intruding incoherent words
- Tongue slips
- Partial or complete omission of words

3. Gestures disclose **extreme** emotional arousal.
   - Body language is very open and not restricted. May appear odd.
   - Should not appear closed
   - The individual does not appear frozen or lifeless
   - ❖ **Note: There should the presence of approximately 5 indicators of open/engaged body language and a near-absence of closed body language cues.**
     Body language appears very unrestricted. Or, body language may contradict topic or feelings discussed.
     The individual does not appear frozen or lifeless

   Examples of closed body language include:
   - Head resting in hand for the majority of the coded sequence (i.e., > 50% of coded sequence)
   - Eyes downcast for the majority of the coded sequence
   - Arms crossed across body for the majority of the coded sequence
   - Looking down or away for the majority of the coded sequence
   - Gaze aversion for the majority of the coded sequence

   Examples of engaged body language would include:
   - Sitting forward in seat for the majority of the coded sequence
   - Learning forward to meet the therapist the for majority of the coded sequence
   - Consistent eye contact with the therapist for the majority of the coded sequence
   - Head movements (e.g., nodding, quickly tilted head) for the majority of the coded sequence
   - Fidgeting hands for the majority of the coded sequence. Can include:
     - Playing with ring
     - Moving hand to nose or chin,
     - Touching hair,
     - Putting hands in pockets,
     - Tapping table,
     - Moving feet,
     - General fidgeting
   - Biting or picking nails for the majority of the coded sequence
   - Fidgeting with worksheets for the majority of the coded sequence
   - Swinging feet for the majority of the coded sequence
   - Changes in facial expressions – smile, frowns, squinting etc.
   - Observable increase in breathing rate for the majority of the coded sequence
   - Observable increase in perspiration for the majority of the coded sequence
   - Crying or evidence of eyes welling up for the majority of the coded sequence
   - Complexion changes such as in colour; red in face or neck area for the majority of the coded sequence

4. Gestures and body movements are **frequent and may appear exaggerated.** Movements should definitely **exceed baseline and slightly** what would be expected for **everyday conversation**. Gestures may appear odd
   - The person should have significant arms movements or gestures whilst talking; movements should not seem overly restricted and should appear exaggerated.
   - Gestures and movements should not be that observed in normal everyday conversation; they should appear inflated or blown up.
   - There should be *extreme* fidgeting.

5. The person should **not appear rigid or tense**. Rather the person should appear very free to the experience of emotion and emotional expression should appear overwhelming.
   - ❖ **Note: The client should not appear rigid.**

Examples of rigid expression include:

- Sitting still without moving or relaxing for the majority of the coded sequence
- Staring straight ahead for the majority of the coded sequence
- Gaze aversion for the majority of the coded sequence
- Rigid chin for the majority of the coded sequence
- Clenched jaw for the majority of the coded sequence
- Firm voice for the majority of the coded sequence

6. The individual seems to **be unable to sit still** in their seat for the majority of the coded sequence
   - The client appears to rock in their seat, fidget significantly, or continually move their feet for the majority of the coded sequence.

7. Eye contact relatively consistent; There is no sense of not wanting to meet the therapist's gaze.
   - ❖ **Note: The proportion of eye contact towards the therapist should exceed 60% of the coded snippet**

8. Speech should appear **uncontrolled, unrestricted and not contained.**
   - The emotion should appear to be too much for the individual to generally handle
   - The emotion should appear overwhelming and something that would be considered anxiety provoking

9. Uncontrolled crying or outburst of anger for the majority of the coded sequence
   - ❖ **Note: The client should be openly teary or sobbing or should have observable expressions of anger for the majority of the coded sequence (i.e., > 50%)**

To gain this rating:

- Approximately six categories should be met and category 1, 2 and 3 must be met. If six categories are met, but categories 1, 2, and 3 are not, assign a level 6 rating

Appendix F: Client Emotional Arousal Scale (CEAS) Coding Example

ID: #1 Session: 1 Start time: 0:00 Total session length: 30 minutes

Minute	1	2	3	4	5
Emotion	Content	Content	Content	Content	Content
Rate - Peak	3	3	4	4	4
Rate - Modal	3	3	4	4	3

Minute	6	7	8	9	10
Emotion	Fear/anxiety	Fear/anxiety	Fear/anxiety	Fear/anxiety	Fear/anxiety
Rate - Peak	4	5	5	5	5
Rate - Modal	4	5	5	5	5

Minute	11	12	13	14	15
Emotion	Pain/hurt	Pain/hurt	Pain/hurt	Sadness	Sadness
Rate - Peak	5	4	4	4	4
Rate - Modal	5	4	4	3	3

Minute	16	17	18	19	20
Emotion	Content	Content	Content	Content	Fear/anxiety
Rate - Peak	3	3	3	3	3
Rate - Modal	3	3	3	3	2

Minute	21	22	23	24	25
Emotion	Fear/anxiety	Sadness	Sadness	Content	Content
Rate - Peak	2	2	2	2	2
Rate - Modal	2	2	2	2	2

Minute	26	27	28	29	30
Emotion	Content	Content	Content	Content	Content
Rate - Peak	3	3	4	4	4
Rate - Modal	3	3	4	4	4

Calculation of modal underengagement proportion score:

Minutes spent underengaged (CEAS rating ≤ 2) = 6

Proportion underengagement $= \left(\frac{\text{Minute spent underengaged}}{\text{Total session length (minutes)}}\right) \times 100$

$= \left(\frac{6}{30}\right) \times 100$

$= 20\%$

Calculation of modal overengagement proportion score:

Minutes spent overengagement (CEAS rating ≥ 5) = 5

Proportion underengagement $= \left(\frac{\text{Minutes spent oveengaged}}{\text{Total session length (minutes)}}\right) \times 100$

$= \left(\frac{5}{30}\right) \times 100$

$= 16.67\%$

Calculation of modal optimal engagement proportion score:

Minutes spent at optimal engagement (CEAS rating 3 and 4) = 19

Proportion optimal engagement $= \left(\frac{\text{Minutes spent optimally engaged}}{\text{Total session length (minutes)}}\right) \times 100$

$= \left(\frac{19}{30}\right) \times 100$

$= 63.33\%$

Appendix G: Multiple Imputation Overview

Imputations for Missing Data

Missing data are unavoidable in clinical research and can undermine the validity of results. In order to tackle problems that arise from missing data I used multiple imputation to make predictions for missing data. In this appendix I provide a brief review of multiple imputation procedures. More in depth explanations are provided by Graham, Cumsille, and Elek (2003), Graham and Hofer (2000), and Schafer (1997).

Numerous approaches exist to deal with missing data. These include replacing missing values with values observed in the data (e.g., substituting missing values with the mean of that value), and replacing missing values with the last measured value (last observation carried forward). Some researchers have suggested the use of complete-case analysis in which participants with missing data are excluded from analyses. Others suggest available-case analysis whereby all available data points are included in analysis and attempts are not made to predict missing values. However, these approaches can lead to serious bias (Wayman, 2003; Little & Schenker, 1995; Graham & Hofer, 2000). For instance, complete-case analysis can reduce sample size, and available-case analysis can lead to bias as it does not take into account that non-responders may differ from responders. Alternatively, multiple imputation accounts for uncertainty in missing data and maintains the original variability of the missing data by creating imputed values that are based on variables correlated with the missing data and correlated with causes of missingness. Multiple imputation is well-studied and has been shown to provide adequate results in the presence of low sample size or high rates of missing data (Graham, et al., 1997; Wayman, 2003). Consequently, multiple imputation is an attractive solution to missing data problems.

Brief Overview of Multiple Imputation

In multiple imputation missing data are predicted using existing values from other variables. Imputed (or predicted) values are then substituted for the missing values and this allows a full, imputed dataset to be created. In multiple imputation this process is performed multiple times thereby producing multiple imputed datasets. It should be noted that imputed values produced from multiple imputation are not intended to be "guesses" of what a particular missing value might be, rather, multiple imputation creates an imputed dataset which maintains the overall variability in the sample while maintaining relationships with other variables. Once imputed datasets are created they are then analysed using standard procedures normally used on complete datasets and results are combined.

A note should be made about the importance of running multiple, rather than single imputations. As any estimation procedure produces error one cannot simply use one estimate to reliably predict missing data. Specifically, single imputation procedures produce standard error estimations that are too small and inferences about missing data that are therefore over-confident (Wayman, 2003). Thus, the use of multiple or numerous imputed datasets is paramount. By creating multiple versions of the data, one produces different plausible versions of the imputed values, and thus, different plausible versions of how the data might appear in the sample. By creating multiple feasible versions of the data one is then able to collate these versions to produce a better estimate for missing values. More explicitly, by running the imputation multiple times and averaging these imputations one can determine which results are "real" and which are due to the "error" in the imputation process.

To recap, multiple imputation can be thought to involve three phases. First, missing data are filled in *n* times to generate *n* complete datasets that are plausible representations of the data. Second, the chosen statistical analysis is performed on each

of the *n* imputed datasets. And third, the results from the *n* complete datasets are combined or pooled, according to complex formulae (see Raghunathan & Dong, 2013) to allow for meaningful inferences. These steps are now briefly reviewed.

Creating imputed datasets. The first step in multiple imputation is to create values or imputations for the missing data. In this thesis missing data was imputed using the statistical package, 'Multivariate Imputation by Chained Equations' (MICE) (van Buuren & Groothuis-Oudshoorn, 2011). The MICE package is used in R and generates multiple imputations for incomplete multivariate data. In the following section I discuss the procedures underlying the creation of imputed datasets using the MICE package.

To create imputed values a model (or, regression line) is identified which allows one to create imputed values based on other variables (or predictors) in the dataset. This is done multiple times to create multiple imputed datasets that can be considered similar but different from each other. A simple way of thinking of it is that each imputed dataset represents a different version of what the data may look like if missing data follow the same patterns as observed data. The number of imputed datasets is determined by the researcher but generally between three and ten datasets are produced.

Created models require predictor variables to help preserve and estimate relationships in the data. Predictors are selected based on their correlation to missing variables, their correlation to the reasons for missingness, or both. Van Buuren and Groothuis-Oudshoorn (2011) suggest that the increased explained variance in linear regression is small after the 15 best predictive variables have been added. Therefore, they suggest that for imputations a subset of 15 to 25 predictors should be selected.

Analysis and interpretation of imputed datasets. Once the imputed datasets have been created the chosen analysis (e.g., ANOVA, regression) is conducted on each imputed dataset. Following the analysis of each imputed dataset results of the analyses are combined to provide an overall set of estimates. That is, results are pooled to allow

for a single interpretation. Pooling can be thought of as averaging across the results, but is technically much more complicated than that (see Rubin 1987).

Multiple Imputation and Data Analysis in This Thesis

In this thesis imputations were made for missing posttreatment and 6-month follow-up data. Pretreatment values did not require imputation as all pretreatment values were collected. In selecting predictors I abided by the principles suggested by van Burren and Groothuis-Oudshoorn (2011) and included all variables that appeared in the complete dataset, variables that were related to non-response, and variables that explained a considerable amount of variance in missing values. Imputations followed the procedures discussed above. Ten imputed datasets were created. Once imputed datasets were created I analysed pre- to posttreatment, and pre- to 6-month follow-up changes on all symptom measures using mixed, repeated-measures ANOVAs. Results were then pooled according to the specifications of Raghunathan and Dong (2013) to allow for single and meaningful interpretations.

Appendix H: Imputation and Predictor Summary

Table H1

Summary of Imputed and Predictor Variables

Variable Name	Predictor, imputed, or calculation status
Total number of sessions	Predictor
Treatment condition	Predictor
Pretreatment CAPS severity	Predictor
Pretreatment CAPS diagnosis met	Predictor
Pretreatment total number of disorder assessed by MINI	Predictor
Pretreatment PCL	Predictor
Pretreatment DASS	Predictor
Pretreatment PTCI	Predictor
Pretreatment TAS	Predictor
Pretreatment EN	Predictor
Pretreatment RRS	Predictor
Pretreatment SRRS	Predictor
Pretreatment CERQ	Predictor
Posttreatment CAP severity	Predictor and imputed
Posttreatment CAPS diagnosis met	Predictor and imputed
Posttreatment PCL	Predictor and imputed
Posttreatment EN	Predictor and imputed
Posttreatment RRS	Predictor and imputed
Posttreatment SRRS	Predictor and imputed
Follow-up CAPS severity	Predictor and imputed
Follow-up CAPS diagnosis met	Predictor and imputed
Follow-up MINI MDD diagnosis met	Predictor and imputed
Follow-up total number of disorder assessed by MINI	Predictor and imputed
Follow-up PCL	Predictor and imputed
Follow-up EN	Predictor and imputed
Follow-up RRS	Predictor and imputed
Follow-up SRRS	Predictor and imputed
Posttreatment PTSD good end-state functioning met?	Predictor and calculated
Posttreatment depression good end-state functioning met?	Predictor and calculated
Posttreatment PTSD and depression good end-state functioning met?	Predictor and calculated
Posttreatment DASS	Predictor and calculated
Posttreatment PTCI	Predictor and calculated
Posttreatment CERQ	Predictor and calculated
Follow-up PTSD good end-state functioning met?	Predictor and calculated
Follow-up depression good end-state functioning met?	Predictor and calculated
Follow-up PTSD and depression good end-state functioning met?	Predictor and calculated

Variable Name	Predictor, imputed, or calculation status
Follow-up DASS	Predictor and calculated
Follow-up PTCI	Predictor and calculated
Follow-up CERQ	Predictor and calculated
Posttreatment MINI MDD diagnosis met	Imputed
Posttreatment DASS depression subscale	Imputed
Posttreatment DASS anxiety subscale	Imputed
Posttreatment DASS stress subscale	Imputed
Posttreatment PTCI negative self subscale	Imputed
Posttreatment PTCI negative world subscale	Imputed
Posttreatment PTCI self blame subscale	Imputed
Posttreatment TAS	Imputed
Posttreatment CERQ self blame subscale	Imputed
Posttreatment CERQ acceptance subscale	Imputed
Posttreatment CERQ rumination subscale	Imputed
Posttreatment CERQ positive refocusing subscale	Imputed
Posttreatment CERQ refocus on planning subscale	Imputed
Posttreatment CERQ positive reappraisal subscale	Imputed
Posttreatment CERQ putting into perspective subscale	Imputed
Posttreatment CERQ catastrophising subscale	Imputed
Posttreatment CERQ other blame subscale	Imputed
Follow-up MINI additional mood disorder present	Imputed
Follow-up MINI additional anxiety disorder present	Imputed
Follow-up MINI substance/alcohol abuse present	Imputed
Posttreatment DASS depression subscale	Imputed
Follow-up DASS anxiety subscale	Imputed
Follow-up DASS stress subscale	Imputed
Follow-up PTCI negative self subscale	Imputed
Follow-up PTCI negative world subscale	Imputed
Follow-up PTCI self blame subscale	Imputed
Follow-up TAS	Imputed
Follow-up CERQ self blame subscale	Imputed
Follow-up CERQ acceptance subscale	Imputed
Follow-up CERQ rumination subscale	Imputed
Follow-up CERQ positive refocusing subscale	Imputed
Follow-up CERQ refocus on planning subscale	Imputed
Follow-up CERQ positive reappraisal subscale	Imputed
Follow-up CERQ putting into perspective subscale	Imputed
Follow-up CERQ catastrophising subscale	Imputed
Follow-up CERQ other blame subscale	Imputed

Note: CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Checklist; DASS = Depression Anxiety and Stress Scale; PTCI = Posttraumatic Cognition Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaires; TAS = Twenty- Item Toronto Alexithymia Scale.; CERQ = Cognitive Emotion Regulation Questionnaire.

Appendix I: Available Case Analysis Descriptive Statistics

Table I1

Available Case Analysis: CPT, BA/CPT, and CPT/BA Means, Standard Deviations, Sample Sizes Over Time on All Measures: Intent-to-Treat Sample

		Posttreatment			Posttreatment			6-Month Follow-up		
Measure	Condition	*N*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
CAPS	CPT	18	72.50	23.09	16	35.94	16.15	10	37.50	13.24
	BA/CPT	15	81.60	14.79	11	44.18	11.03	6	36.17	14.21
	CPT/BA	16	84.94	14.39	12	20.08	10.56	6	16.5	8.87
PCL	CPT	18	56.06	11.28	18	36.33	9.12	12	37.67	8.71
	BA/CPT	15	60.20	7.49	15	44.20	7.83	9	44.00	9.44
	CPT/BA	16	61.12	10.35	14	26.14	5.59	6	22.00	3.72
DASS – D	CPT	18	21.11	10.68	18	11.00	6.67	12	14.83	5.96
	BA/CPT	15	21.20	9.91	15	15.87	5.11	9	14.44	5.86
	CPT/BA	16	26.12	9.76	14	8.57	5.52	6	0.50	1.88
PTCI	CPT	18	130.67	36.62	18	103.89	28.84	11	101.00	26.22
	BA/CPT	15	139.27	39.87	12	103.83	29.23	6	99.00	25.73
	CPT/BA	16	152.94	26.36	12	64.50	14.83	6	60.67	12.80
RRS	CPT	18	51.94	13.97	18	46.56	9.02	11	45.18	8.40
	BA/CPT	15	57.07	13.71	12	47.17	7.16	6	39.83	4.72
	CPT/BA	16	61.88	11.28	12	32.67	3.95	6	31.83	4.46

		Posttreatment			Posttreatment			6-Month Follow-up		
Measure	Condition	*n*	*M*	*SD*	*N*	*M*	*SD*	*n*	*M*	*SD*
SRRS	CPT	18	1159.17	462.47	18	865.00	327.58	11	757.73	261.69
	BA/CPT	15	1310.33	439.30	12	820.42	284.55	6	611.67	267.34
	CPT/BA	16	1546.25	313.98	12	565.17	207.44	6	454.42	266.67
EN	CPT	18	25.67	7.93	18	15.72	4.30	11	15.72	4.18
	BA/CPT	15	28.47	4.67	12	19.08	3.38	6	15.33	3.00
	CPT/BA	16	28.62	5.15	12	11.25	3.31	6	13.17	4.57
TAS	CPT	18	55.28	12.41	18	49.44	8.04	11	43.00	8.09
	BA/CPT	15	62.47	9.80	12	53.42	6.54	6	48.67	5.15
	CPT/BA	16	60.94	10.67	12	36.58	4.26	6	36.00	5.06

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; ES = Effect Size; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Appendix J: Inferential Statistics and Effect Sizes for ANCOVAs Controlling for Number of Sessions

Table J1

Pooled Inferential Statistics from Imputed Datasets for Intent-to-Treat Sample on All Measures Controlling for Number of Sessions: Pre- to Posttreatment, and Pre- to 6-Month Follow-Up

	Posttreatment				6-Month Follow-up			
Measure	*df1*	*df2*	*F*	*p*	*df1*	*df2*	*F*	*p*
CAPS								
Time (T)	0.99	31.62	162.58	< .001	0.95	21.69	106.48	< .001
Condition (C)	1.71	38.21	1.84	.18	0.73	33.80	0.42	.46
C × T	1.10	31.62	2.03	.16	0.89	21.69	1.45	.24
PCL								
T	1.00	40.23	187.08	< .001	0.92	17.47	93.17	< .001
C	2.00	44.43	1.72	.19	0.66	19.11	0.67	.37
C × T	1.73	40.23	2.84	.08	1.09	17.47	0.94	.35
DASS – D								
T	0.99	37.20	53.62	< .001	0.78	22.81	23.74	< .001
C	1.65	44.21	0.58	.53	0.39	21.11	0.11	.50
C × T	1.55	37.20	1.70	.20	0.42	22.81	0.34	.39
PTCI								
T	0.98	35.61	58.29	< .001	0.86	19.45	56.30	< .001
C	1.09	43.66	0.11	.76	0.21	24.08	0.07	.38
C × T	1.77	35.61	3.76	.04	1.02	19.45	2.73	.11
RRS								
T	0.98	31.11	30.70	< .001	0.96	27.00	39.02	< .001
C	0.93	42.59	0.25	.60	0.83	26.21	0.12	.69
C × T	1.78	31.11	4.24	.03	1.39	27.00	3.57	.054
SRRS								
T	0.98	35.89	65.39	< .001	0.93	25.77	81.05	< .001
C	0.56	44.46	0.59	.36	0.96	36.23	0.65	.42
C × T	1.67	35.89	3.78	.04	1.62	25.77	3.89	.04
EN								
T	0.98	39.38	68.10	< .001	0.93	30.47	50.01	< .001
C	1.64	42.06	1.49	.24	0.52	26.01	0.42	.40
C × T	1.22	39.38	0.50	.52	0.16	30.47	0.13	.28
TAS								
T	0.96	28.16	30.83	< .001	0.71	26.03	24.01	< .001
C	1.79	43.46	1.65	.21	1.66	24.28	0.91	.40
C × T	1.23	28.16	2.02	.17	0.93	26.03	0.41	.51

Note. CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table J2

Pooled Effect Sizes (Cohen's d) [and 95% Confidence Intervals for Effect Sizes] from Pre- to Posttreatment, and Pre- to 6-month Follow-Up on All Symptom Measures Controlling for Number of Sessions: Intent-to-Treat Sample Controlling for Number of Sessions

	CPT		BA/CPT		CPT/BA	
Measure	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up	Posttreatment	6-Month Follow-Up
CAPS	1.67	1.41	2.43	3.16	3.75	3.33
	[0.89, 2.42]	[0.67, 2.13]	[1.46, 3.37]	[2.05, 4.23]	[2.57, 4.91]	[2.23, 4.40]
PCL	1.90	1.70	2.43	2.50	2.76	2.45
	[1.09, 2.68]	[0.93, 2.46]	[1.46, 3.37]	[1.52, 3.46]	[1.76, 3.73]	[1.51, 3.36]
DASS – D	1.05	0.76	0.69	0.81	1.44	1.41
	[0.35, 1.74]	[0.08, 1.43]	[-0.06, 1.42]	[0.06, 1.55]	[0.65, 2.21]	[0.63, 2.18]
PTCI	0.81	0.93	0.88	1.00	2.54	2.65
	[0.12, 1.48]	[0.24, 1.62]	[0.13, 1.63]	[0.23, 1.75]	[1.59, 3.48]	[1.67, 3.60]
RRS	0.44	0.57	0.76	1.17	2.01	2.31
	[-0.23, 1.10]	[-0.10, 1.23]	[0.01, 1.50]	[0.38, 1.94]	[1.15, 2.87]	[1.40, 3.20]
SRRS	0.69	0.93	1.20	1.50	2.51	3.04
	[0.02, 1.36]	[0.23, 1.61]	[0.41, 1.97]	[0.67, 2.30]	[1.56, 3.43]	[2.00, 4.06]
EN	1.34	1.14	1.95	2.25	2.49	2.31
	[0.61, 2.06]	[0.42, 1.84]	[1.06, 2.82]	[1.31, 3.16]	[1.54, 2.41]	[1.39, 3.20]
TAS	0.55	0.92	0.97	1.78	1.63	1.69
	[-0.12, 1.22]	[0.23, 1.61]	[0.20, 1.72]	[0.92, 2.62]	[0.82, 2.42]	[0.86, 2.49]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; ES = Effect Size; PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Appendix K: Inferential Statistics for Pretreatment Symptom Severity - Completers

Table K1

Inferential Statistics for One-Way ANOVAs Comparing Pretreatment Symptom Severity on all Measures between CPT, BA/CPT, and CPT/BA: Completers

Measures	*df*	*F*	*p*
CAPS	2, 22	6.41	.01
PCL	2, 22	2.08	.15
DASS-D	2, 22	2.63	.09
PTCI	2, 22	2.87	.08
RRS	2, 22	4.71	.02
SRRS	2, 22	4.50	.02
EN	2, 22	0.84	.45
TAS	2, 22	1.33	.29

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Checklist; DASS-D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognition Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaires; TAS = Twenty- Item Toronto Alexithymia Scale.

Where Did Significant Pretreatment Differences Occur in the Completer Sample?

A significant main effect emerged on the CAPS, RRS, and SRRS in which CPT completers reported lower pretreatment scores than BA/CPT and CPT/BA completers. Pairwise comparisons demonstrated that CPT completers reported significantly lower pretreatment CAPS scores than BA/CPT (p = .02, d = 1.54, 95% CI d = [0.29, 2.74]) and CPT/BA completers (p = .01, d = 1.45, 95% CI d = [0.40, 2.47]). Pretreatment CAPS scores did not significantly differ between BA/CPT and CPT/BA completers (p = 1.00, d = 0.18, 95% CI d = [-0.82, 1.17]). In regards to RRS scores, CPT completers

reported significantly lower pretreatment RRS scores than BA/CPT (p = .05, d = 1.45, 95% CI d = [0.22, 2.63]) and CPT/BA completers (p = .04, d = 1.25, 95% CI d = [0.23, 2.24]). RRS scores did not differ significantly between BA/CPT and CPT/BA completers (p = 1.00, d = 0.18, 95% CI d = [0.82, 1.17]). Further, CPT completers reported significantly lower SRRS scores than CPT/BA completers (p = .04, d = 1.48, 95% CI d = [0.43, 2.50]). SRRS scores did not differ significantly between CPT and BA/CPT (p = .06, d = 1.24, 95% CI d = [0.05, 2.39]) and BA/CPT and CPT/BA (p = 1.00, d = 0.11, 95% CI d = [-0.89, 1.10]).

Appendix L: Treatment Outcomes - Supplementary Analyses

I undertook two supplementary analyses. First, I explored credibility/expectancy and working alliance as a potential explanation for the superiority of CPT/BA. I then examined if order of PTSD and MDD onset influenced treatment outcomes in participants.

Credibility/Expectancy of Treatment and Working Alliance

I examined condition differences in pretreatment and posttreatment credibility/expectancy and working alliance to determine if such therapeutic processes provided an explanation for observed treatment differences. For example, did CPT/BA participants report reduced dropout and better treatment outcomes compared to CPT and BA/CPT participants, because CPT/BA participants viewed their treatment as more credible or due to having a better relationship with their therapist (i.e., greater working alliance)? Imputations were not made for missing data and only collected data was analysed. Analyses were conducted in SPSS. It should be highlighted that pretreatment credibility/expectancy scores were collected at Session 1 and pretreatment working alliance scores collected at Session 2. Thus, if participants dropped out of treatment prior to such sessions questionnaires were not collected. Posttreatment credibility/expectancy and working alliance scores were also collected during the participants' last treatment sessions. If participants did not complete treatment posttreatment credibility/expectancy and working alliance scores were collected during posttreatment assessment. Descriptive statistics for the ITT sample are presented in Table L.1.

Table L.1

Descriptive Statistics for Credibility/Expectancy and Working Alliance Scales at Pre- and Posttreatment for Intent-to-Treat Sample

Measure	Condition	Pretreatment			Posttreatment		
		n	*M*	*SD*	*n*	*M*	*SD*
		Intent-to-Treat Sample					
Credibility/ Expectancy	CPT	14	42.64	7.70	17	39.18	10.49
	BA/CPT	12	44.33	3.63	8	44.75	5.75
	CPT/BA	15	50.33	15.32	13	48.92	5.68
WA – Task	CPT	13	24.15	3.00	12	25.42	3.15
	BA/CPT	11	25.09	2.39	9	25.44	2.74
	CPT/BA	14	25.71	2.30	13	26.37	2.60
WA – Bond	CPT	13	23.46	3.33	12	25.17	3.01
	BA/CPT	11	25.18	3.03	9	26.00	1.41
	CPT/BA	14	25.71	3.02	13	26.54	2.22
WA – Goal	CPT	13	24.46	2.57	12	25.08	3.68
	BA/CPT	11	25.90	2.30	9	25.67	2.60
	CPT/BA	14	25.21	3.00	13	26.31	3.07

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; WA = Working Alliance.

For the ITT sample at pretreatment there were no significant differences between conditions on the credibility/expectancy questionnaire $F(2, 38) = 2.14$, $p = .13$, working alliance task $F(2, 35) = 1.24$, $p = .30$, working alliance bond $F(2, 35) = 1.86$, $p = .17$, and working alliance goal scores $F(2, 35) = 0.88$, $p = .42$. All pairwise comparisons were also nonsignificant. However, given the small sample size I examined effects sizes and descriptive statistics to determine if nonsignificant results were the product of reduced power. Descriptive statistics and pairwise comparison effects sizes suggested that at pretreatment, (although nonsignificant) CPT/BA participant tended to perceive their treatment to be more credible than CPT ($p = .17$, $d = 0.62$, 95% CI d = [-0.13, 1.36]) and BA/CPT participants ($p = .45$, $d = 0.53$, 95% CI d = [-0.25, 0.53]). Meaningful difference did not emerge between CPT and BA/CPT participants ($p = 1.00$,

d = 0.27, 95% CI *d* = [-0.51, 1.04]). Effect sizes from pairwise comparisons were not substantial for working alliance task, bond, and goal scores. Results indicate that during the initial stages of therapy and after treatment rationales were provided (i.e., during Session 1), CPT/BA participants tended to perceive their treatment to be more credible than CPT and BA/CPT participants. However, working alliance did not appear to meaningfully differ between conditions.

For the ITT sample at posttreatment a significant difference was found on the credibility/expectancy scale $F(2, 35) = 5.23$, $p = .01$. Pairwise comparisons demonstrated that CPT/BA participants reported significantly higher credibility/expectancy scores than CPT participants ($p = .01$, $d = 1.11$, 95% CI d = [0.32, 1.88]), and meaningfully higher credibility/expectancy scores than BA/CPT participants ($p = .80$, $d = 0.73$, 95% CI d = [-0.19, 1.63]). Further, effect sizes suggested that BA/CPT participants reported meaningfully larger credibility/expectancy scores than CPT participants. ($p = .37$, $d = 0.60$, 95% CI d = [-0.26, 1.45]). This suggests that at posttreatment, compared to CPT and BA/CPT participants, CPT/BA participants viewed their treatment as more credible and had higher expectancies that their treatment would reduce symptoms. No significant differences emerged on posttreatment working alliance task $F(2, 31) = 0.46$, $p = .64$, working alliance bond $F(2, 31) = 1.05$, $p = .36$, and working alliance goal scores $F(2, 31) = 0.46$, $p = .64$, and pairwise comparison effect sizes were not substantial.

Although nonsignificant, results suggested that CPT/BA participants viewed their treatment as more credible than CPT and BA/CPT participants at pretreatment. Further, at posttreatment CPT/BA participants viewed their treatment as significantly more credible than CPT participants, and meaningfully more credible than BA/CPT participants. Thus, differences in credibility/expectancy may in part explain CPT/BA superiority. However, as effects were not large (and in most cases nonsignificant), as the

sample size was small (especially at posttreatment), and as confidence intervals often included zero, such findings require replication. As working alliance did not appear to meaningfully differ between conditions, working alliance does not account for treatment differences.

Order of PTSD and MDD Onset

Research to date has seldom examined the influence of order of PTSD and MDD onset on outcome. The paucity of research in this area is problematic as a deeper understanding of the relationship between order of PTSD and MDD onset and outcome could enhance clinical practice and allow clinicians to potentially use order of onset as a predictor of treatment outcome. Consequently, I conducted exploratory analyses to examine if order of PTSD and MDD onset influenced treatment outcome. Specifically, I was interested in determining if treatment outcomes differed between participants who reported PTSD onset prior to MDD onset (PTSD/MDD onset), participants who reported MDD onset prior to PTSD onset (MDD/PTSD onset) or, participants who reported simultaneous PTSD and MDD onset (simultaneous PTSD/MDD onset).

Due to the small sample size I was unable to determine if the effect of order of PTSD and MDD onset differed between treatment conditions. Examining the interaction between condition, time, and order of PTSD and MDD onset would require nine cells and with a sample of only 49 participants this analysis would be considerably underpowered. Consequently, to enhance power and to allow an initial exploration of the effects of order of PTSD and MDD onset, conditions were combined and only the interaction of time and order of PTSD and MDD onset was examined. I acknowledge that collapsing conditions is problematic given that significant condition differences existed, and further, analysing data in such a way does not allow one to determine if the relationship between order of onset and outcome differed by condition. However, such

analysis provides an initial exploration of the effects of order of PTSD and MDD onset on outcome.

To determine the effect of order of PTSD and MDD onset on posttreatment and 6-month follow-up outcomes I conducted two, 2 (Time: pretreatment, posttreatment *or* 6-month follow-up) × 3 (Order of onset: PTSD/MDD onset, MDD/PTSD onset, simultaneous PTSD/MDD onset) mixed, repeated-measures ANOVAs on CAPS, PCL, DASS-D, PTCI, RRS, SRRS, TAS, and EN scores for the ITT sample. Missing posttreatment and 6-month follow-up scores were imputed using multiple imputation and results were pooled. Pooled statistics are presented throughout. As I was interested in determining if treatment outcome differed by order of onset group, main effects of order of onset group as well as Time × Order of Onset interactions were of primary interest. I now report relevant results from the undertaken ANOVAs. Imputed descriptive statistics, pooled inferential statistics, and pooled effect sizes are presented in Tables L.2 to L.4. Results suggested that outcome did not differ meaningfully between order of onset groups.

For the ITT sample, no significant main effects of order of onset emerged on any measure from pre- to posttreatment, or pre- to 6-month follow-up. When examining pre- to posttreatment changes, most Time × Order of Onset interactions were nonsignificant. Significant Time × Order of Onset interactions only emerged on the SRRS and TAS. Effect sizes suggested that compared to those who report PTSD/MDD onset, and those who reported MDD/PTSD onset, those who reported simultaneous PTSD/MDD onset demonstrated greater reductions on the SRRS and TAS. For SRRS and TAS scores respectively the effect size for simultaneous PTSD/MDD onset was 2.78 and 2.33 times larger than that reported by PTSD/MDD onset, and 1.81 and 1.55 times larger than that reported by MDD/PTSD onset. A similar pattern of results emerged for pre- to 6-month follow-up changes. Again, most Time × Order of Onset interactions were nonsignificant

and the only significant interaction to emerge was for the SRRS. Effect sizes suggested that those who reported simultaneous PTSD/MDD onset reported an effect on the SRRS 2.42 times larger than those who reported PTSD/MDD onset.

As significant main effects of order of onset were not found on any measure, and as significant Time × Order of onset interactions did not emerge for primary PTSD and MDD treatment outcomes, results suggest that order of onset did not meaningfully influence treatment outcome (aside from SRRS and TAS scores). Further, as all effect sizes were medium to large results suggest that participants were able to achieve moderate to large symptom reductions irrespective of order of PTSD and MDD onset.

Table L.2

Order of PTSD and MDD Onset Imputed Means, Standard Deviations, Sample Sizes, and Proportion of Imputed Data Over Time on All Measures: Intent-to-Treat Sample

Measure	Order of Onset	*n*	Pretreatment			Posttreatment			6-Month Follow-up		
			M	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
CAPS	PTSD then MDD	9	78.89	24.03	0 (0)	48.53	33.14	11.11 (1)	43.14	26.90	66.67 (6)
	MDD then PTSD	25	76.60	18.18	0 (0)	34.34	24.33	64.00 (16)	39.05	27.29	40.00 (10)
	PTSD and MDD	15	84.20	15.95	0 (0)	33.19	28.15	20.00 (3)	31.45	27.52	73.33 (11)
PCL	PTSD then MDD	9	57.44	10.76	0 (0)	39.56	21.44	0 (0)	42.32	16.58	55.56 (5)
	MDD then PTSD	25	59.20	9.59	0 (0)	36.43	13.64	4.00 (1)	38.07	17.79	28.00 (7)
	PTSD and MDD	15	59.53	10.85	0 (0)	33.90	17.36	6.67 (1)	34.99	17.16	66.67 (10)
DASS - D	PTSD then MDD	9	19.33	11.45	0 (0)	14.00	15.33	0 (0)	16.47	14.09	55.56 (5)
	MDD then PTSD	25	23.44	10.27	0 (0)	11.87	10.77	4.00 (1)	12.87	12.27	28.00 (7)
	PTSD and MDD	15	23.73	9.62	0 (0)	10.96	11.10	6.67 (1)	10.61	12.18	66.67 (10)
PTCI	PTSD then MDD	9	131.67	37.23	0 (0)	95.75	59.69	11.11(1)	94.30	64.79	66.67 (6)
	MDD then PTSD	25	143.28	30.73	0 (0)	105.04	38.89	12.00 (3)	93.81	35.42	36.00 (9)
	PTSD and MDD	15	141.40	42.31	0 (0)	84.36	47.05	20.00 (3)	90.94	37.09	73.33 (11)
RRS	PTSD then MDD	9	50.67	14.83	0 (0)	44.78	15.43	11.11(1)	42.47	11.62	66.67 (6)
	MDD then PTSD	25	55.48	14.02	0 (0)	45.21	14.84	12.00 (3)	40.04	12.16	36.00 (9)
	PTSD and MDD	15	62.53	9.80	0 (0)	41.11	16.75	20.00 (3)	39.79	12.06	73.33 (11)

Measure	Order of Onset	*n*	Pretreatment			Posttreatment			6-Month Follow-up		
			M	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)	*M*	*SD*	% of data imputed (*n*)[a]
SRRS	PTSD then MDD	9	1084.44	400.17	0 (0)	789.50	500.92	11.11(1)	732.50	365.16	66.67 (6)
	MDD then PTSD	25	1332.40	426.42	0 (0)	898.24	447.95	12.00 (3)	710.24	436.76	36.00 (9)
	PTSD and MDD	15	1479.33	428.74	0 (0)	629.83	513.20	20.00 (3)	535.83	430.01	73.33 (11)
EN	PTSD then MDD	9	24.56	8.62	0 (0)	17.04	11.12	11.11(1)	16.59	8.88	66.67 (6)
	MDD then PTSD	25	26.68	5.81	0 (0)	17.26	8.98	12.00 (3)	17.84	9.59	36.00 (9)
	PTSD and MDD	15	30.60	3.98	0 (0)	15.23	9.73	20.00 (3)	16.12	9.22	73.33 (11)
TAS	PTSD then MDD	9	55.89	11.98	0 (0)	47.54	14.64	11.11(1)	43.46	18.01	66.67 (6)
	MDD then PTSD	25	57.52	11.03	0 (0)	51.11	14.26	12.00 (3)	43.98	18.92	36.00 (9)
	PTSD and MDD	15	64.40	10.39	0 (0)	43.78	17.04	20.00 (3)	44.05	20.20	73.33 (11)

Note. PTSD = Posttraumatic Stress Disorder; MDD = Major Depressive Disorder; PTSD Then MDD = PTSD Onset Preceded MDD Onset; MDD Then PTSD Onset = MDD Onset Preceded PTSD Onset; PTSD And MDD = PTSD And MDD Presented Simultaneously; CAPS = Clinician Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Checklist; DASS-D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognition Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale; EN = Emotional Numbing Questionnaires; TAS = Twenty- Item Toronto Alexithymia Scale.

[a] Five PTSD/MDD onset, five MDD/PTSD onset, and eight simultaneous PTSD/MDD onset, 6-month follow-up assessments were not due at the time of writing and were therefore not included in reported analyses.

Table L.3

Pooled Inferential Statistics for Imputed Datasets for the Effect of Order of PTSD and MDD Onset on All Measures: Intent-to-Treat Sample

	Posttreatment				6-Month Follow-up			
Measure	*df1*	*df2*	*F*	*p*	*df1*	*df2*	*F*	*p*
CAPS								
Time (T)	0.99	36.46	108.5	< .001	0.95	20.14	101.20	< .001
Order of onset (O)	1.60	43.01	0.65	.50	0.10	33.93	0.01	.32
O × T	1.69	36.46	1.40	.26	1.30	20.14	1.45	.25
PCL								
T	1.00	45.00	94.91	< .001	0.92	28.54	69.80	< .001
O	1.59	44.98	0.08	.88	0.46	28.50	0.11	.54
O × T	1.94	45.02	0.63	.53	0.29	28.85	0.42	.30
DASS – D								
T	0.99	43.00	35.72	< .001	0.78	29.03	19.48	< .001
O	1.43	44.36	0.04	.91	1.01	22.07	0.19	.67
O × T	1.88	43.00	1.07	.35	1.19	29.03	1.40	.25
PTCI								
T	0.98	37.31	41.88	< .001	0.86	29.76	47.13	< .001
O	1.64	42.52	0.64	.50	0.70	23.54	0.15	.61
O × T	0.71	37.31	0.38	.37	0.98	29.76	0.25	.62
RRS								
T	0.98	34.14	25.82	< .001	0.96	29.86	35.94	< .001
O	1.18	43.04	0.31	.62	0.92	27.47	0.97	.33
O × T	1.59	34.14	2.55	.10	1.05	29.86	1.47	.24
SRRS								
T	0.98	38.12	57.37	< .001	0.93	27.01	78.74	< .001
O	1.81	44.09	0.75	.47	0.79	37.78	0.33	.52
O x T	1.78	38.12	4.49	.02	1.62	27.01	3.76	.05
EN								
T	0.98	35.12	60.23	< .001	0.92	29.36	53.39	< .001
O	1.38	44.76	0.32	.65	0.23	27.02	0.25	.31
O × T	1.65	35.12	2.30	.12	.97	29.36	1.56	.22
TAS								
T	0.96	42.804	27.78	< .001	0.71	18.63	24.61	< .001
O	1.26	2.80	0.20	.72	0.65	23.47	0.46	.43
O × T	1.60	33.01	4.18	.03	0.29	18.63	0.46	.29

Note. CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale.

Table L.4

Pooled Effect Sizes (Cohen's d) [and 95% Confidence Intervals for Effect Sizes] over Time for Order of PTSD and MDD Onset: Intent-to-Treat Sample

	PTSD onset prior to MDD		MDD onset prior to PTSD		PTSD and MDD simultaneously	
	Posttreatment	6-Month Follow-up	Posttreatment	6-Month Follow-up	Posttreatment	6-Month Follow-up
CAPS	1.05 [0.04, 2.03]	1.40 [0.34, 2.42]	1.97 [1.29, 2.65]	1.63 [0.98, 2.27]	2.24 [1.30, 3.15]	2.36 [1.41, 3.29]
PCL	1.06 [0.05, 2.03]	1.09 [0.08, 2.07]	1.93 [1.25, 2.60]	1.48 [0.85, 2.11]	1.77 [0.91, 2.61]	1.79 [0.93, 2.63]
DASS – D	0.39 [-0.55, 1.32]	0.22 [-0.71, 1.14]	1.10 [0.50, 1.69]	0.94 [0.35, 1.52]	1.24 [0.44, 2.01]	1.25 [0.45, 2.02]
PTCI	0.73 [-0.24, 1.67]	1.06 [0.06, 2.04]	1.10 [0.50, 1.69]	1.51 [0.87, 2.14]	1.28 [0.48, 2.06]	1.30 [0.50, 2.08]
RRS	0.39 [-0.55, 1.32]	0.61 [-0.34, 1.55]	0.71 [0.14, 1.28]	1.18 [0.57, 1.77]	1.57 [0.74, 2.39]	2.08 [1.17, 2.96]
SRRS	0.65 [-0.31, 1.59]	0.92 [-0.07, 1.88]	1.00 [0.40, 1.58]	1.44 [0.81, 2.06]	1.81 [0.94, 2.65]	2.23 [1.30, 3.14]
EN	0.76 [-0.21, 1.71]	0.93 [-0.06, 1.90]	1.25 [0.63, 1.85]	1.13 [0.52, 1.72]	2.09 [1.18, 2.97]	2.08 [1.17, 2.97]
TAS	0.63 [-0.33, 1.57]	0.84 [-0.14, 1.79]	0.50 [-0.06, 1.07]	0.88 [0.29, 1.46]	1.47 [0.65, 2.76]	1.30 [0.50, 2.08]

Note. CPT = Cognitive Processing Therapy; BA = Behavioural Activation; CAPS = Clinician-Administered PTSD Scale; PCL = Posttraumatic Stress Disorder Check List; DASS – D = Depression Anxiety and Stress Scale – Depression Subscale; PTCI = Posttraumatic Cognitions Inventory; RRS = Ruminative Response Scale of the Response Style Questionnaire; SRRS = Stress-Reactive Rumination Scale. EN = Emotional Numbing Questionnaire; TAS = Twenty-Item Toronto Alexithymia Scale

Appendix M: Correlation of Subjective Units of Distress Scores and CEAS Ratings

Table M1

Correlations for Subjective Units of Distress Scores with Under-, Over- and Optimal Engagement assessed by Client Expressed Emotional Arousal: First Written Trauma Account Session

	Subjective units of distress scores
Proportion underengagement	-.12
Proportion overengagement	.48*
Proportion optimal engagement	-.39*

* $p < 0.05$; ** $p < 0.01$.

Table M2

Correlations for Subjective Units of Distress Scores with Under-, Over- and Optimal Engagement assessed by Client Expressed Emotional Arousal: Second Written Trauma Account Session

	Subjective units of distress scores
Proportion underengagement	-.12
Proportion overengagement	.44*
Proportion optimal engagement	-.43*

* $p < 0.05$; ** $p < 0.01$.