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Cognitive therapy for post-traumatic stress disorder: development and evaluation

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Abstract

The paper describes the development of a cognitive therapy (CT) program for post-traumatic stress disorder (PTSD) that is based on a recent cognitive model (Behav. Res. Therapy 38 (2000) 319). In a consecutive case series, 20 PTSD patients treated with CT showed highly significant improvement in symptoms of PTSD, depression and anxiety. A subsequent randomized controlled trial compared CT ($N = 14$) and a 3-month waitlist condition (WL, $N = 14$). CT led to large reductions in PTSD symptoms, disability, depression and anxiety, whereas the waitlist group did not improve. In both studies, treatment gains were well maintained at 6-month follow-up. CT was highly acceptable, with an overall dropout rate of only 3%. The intent-to-treat effect sizes for the degree of change in PTSD symptoms from pre to post-treatment were 2.70–2.82 (self-report), and 2.07 (assessor-rated). The controlled effect sizes for CT versus WL post-treatment scores were 2.25 (self-report) and 2.18 (assessor-rated). As predicted by the cognitive model, good treatment outcome was related to greater changes in dysfunctional post-traumatic cognitions. Patient characteristics such as comorbidity, type of trauma, history of previous trauma, or time since the traumatic event did not predict treatment response, however, low educational attainment and low socioeconomic status were related to better outcome.

Keywords: Cognitive therapy; Cognitive behavioural treatment; Post-traumatic stress disorder; Treatment acceptability; Randomized controlled trial; Predictors of outcome

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1. Introduction

Several versions of cognitive behavioural treatment (CBT) for post-traumatic stress disorder (PTSD) have been described in the literature. In a meta-analysis of controlled and uncontrolled studies, van Etten and Taylor (1998) concluded that CBT for PTSD is effective. The mean observed effect sizes for changes in PTSD symptoms from initial assessment to post-treatment were relatively large, Cohen's $d = 1.27$ for self-report measures of PTSD symptoms, and $d = 1.89$ for assessor ratings.

The most effective programs appear to be those that rely on repeated exposure to the trauma memory (either in imagination or by writing a trauma narrative) and in vivo exposure to situations avoided since the event, on cognitive restructuring of the meaning of the trauma, or a combination of these methods. In a large randomized controlled trial, Resick, Nishith, Weaver, Astin, and Feuer (2002) compared two effective versions of CBT, cognitive processing therapy (Resick & Schnicke, 1992, 1993) and prolonged exposure (Foa et al., 1999; Foa & Rothbaum, 1998; Foa, Rothbaum, Riggs, & Murdock, 1991) with a minimal attention waitlist control. Both CBT programs led to large reductions in PTSD symptoms.

Non-trauma-focused behavioural interventions such as relaxation training are less effective than those that involve systematic exposure to the trauma memory or cognitive restructuring of the meaning of the trauma (Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998). Similarly, trauma-focused CBT is more effective than supportive counseling (Blanchard et al. 2003; Bryant, Moulds, Guthrie, Dang, & Nixon, 2003).

Despite these successes, there is room for improvement. The effect sizes in van Etten and Taylor (1998) meta-analysis are based on *completer* analyses, and thus represent overestimates of treatment efficacy. Recent studies have shown trauma-focused CBT to be effective in *intent-to-treat* analyses, with effect sizes ranging between 1.0 and 1.6 for pre to post-treatment changes in PTSD symptoms (Bryant et al., 2003; Resick et al., 2002).

However, the relatively large proportion of patients who do not complete treatment is a concern. The mean percentage of drop-outs for CBT in van Etten and Taylor (1998) meta-analysis was 15%, but some recent randomized controlled trials (RCTs) have reported higher drop-out rates of around 25% (Blanchard et al., 2003; Bryant et al., 2003; Resick et al., 2002). Furthermore, a subgroup of patients still meet diagnostic criteria for PTSD at the end of treatment. The proportion depends on the severity of initial symptoms, and ranges between 35% and 47% in recent intent-to-treat analyses (Bryant et al., 2003; Foa et al., 1999; Resick et al., 2002).

In the present paper, we describe the development and initial evaluation of a variant of trauma-focused CBT that is based on a recent cognitive model of PTSD (Ehlers & Clark, 2000). The model draws heavily on the writings of other theorists (Brewin, Dalgleish, & Joseph, 1996; Conway, 1997; Foa & Riggs, 1993; Foa & Rothbaum, 1998; Foa, Steketee, & Rothbaum, 1989; Horowitz, 1997; Janoff-Bulman, 1992; Joseph, Williams, & Yule, 1997; Keane, Zimering, & Caddell, 1985; Krystal, Bennett, Bremner, Southwick, & Charney, 1995; Litz & Keane, 1989; Markowitsch, 1996; Resick & Schnicke, 1993; van der Kolk & Fisler, 1995), but provides a unique synthesis. Ehlers and Clark (2000) suggested that PTSD becomes persistent when individuals process the trauma in a way that leads to a sense of serious, current threat. The sense of threat arises as a consequence of (1) excessively negative appraisals of the trauma and/or its sequelae and (2) a disturbance of the autobiographical memory for the trauma characterized by poor

elaboration and contextualization, strong associative memory and strong perceptual priming, which leads to involuntary reexperiencing of aspects of the trauma. Changes in negative appraisals and trauma memory are prevented by a series of problematic behavioural and cognitive strategies. Accordingly, cognitive therapy (CT) for PTSD aims to modify excessively negative appraisals, correct the autobiographical memory disturbance, and remove the problematic behavioural and cognitive strategies.

2. Cognitive therapy for PTSD

For each patient, an individualized version of the Ehlers and Clark (2000) model is developed by identifying the relevant appraisals, memory characteristics and triggers, and behavioural and cognitive strategies that maintain his/her PTSD. These maintaining factors are addressed with the procedures described below. Thus, the relative weight given to different treatment procedures differs from patient to patient.

Goal 1: Modify excessively negative appraisals of the trauma and its sequelae. Excessively negative appraisals *of the traumatic event* are identified by careful questioning, particularly about the meaning of "hot spots" (moments of greatest distress in the trauma memory). "Hot spots" are identified by examining the content of intrusions (see Ehlers, Hackmann, Steil, Wenninger, & Clohessy, 2002) and by a probe imaginal reliving (Foa & Rothbaum, 1998) of the trauma. Socratic questioning and other general cognitive therapy techniques are then used to modify the negative appraisals.

Once an alternative appraisal that the patient finds compelling has been identified, the new appraisal is actively incorporated into the trauma memory. This can be achieved either by adding it to a written account produced by the patient (Blanchard et al., 2003; Resick & Schnicke, 1993) and holding the new appraisal and the "hot spot" in mind simultaneously when reading out the narrative, or by inserting the new appraisal into a subsequent imaginal reliving. For example, a woman who had been raped identified a moment when her assailant said she was ugly and turned her over, as the worst "hot spot". Ever since the rape she had felt unattractive and, more recently, had been engaging in frequent casual sex in an apparent attempt to convince herself that she was attractive. Socratic questioning was used to identify an alternative appraisal, which was that the rapist had identified her because she is attractive and his comment was because he is unable to become aroused without abusing and humiliating women. During a subsequent imaginal reliving, she introduced the new appraisal into the "hot spot" by standing up in the image and saying it to the rapist at the moment that he verbally abused her.

Sometimes introducing corrective information into the trauma memory by verbal means is insufficient. In such instances, performing *actions* that provide information and sensory cues that are incompatible with the original meaning when focusing on the "hot spot" (e.g., walking about if patients thought that they were paralyzed, looking at a recent photograph of themselves if they thought they died during the traumatic event); or *imagery* techniques (e.g., visualizing how their facial wounds healed if patients thought "I will be disfigured forever"; "fast-forwarding" in imagery from the "hot spot" to the point when the patient was safe) can be a useful way of getting the new, less threatening, appraisal to stick.

Disconfirming evidence for the patient's appraisals of the trauma may include information about how the way the event unfolded after the "hot spot". In PTSD, disjointed recall of the trauma contributes to the negative appraisals (Ehlers & Clark, 2000; Ehlers, Hackmann, & Michael, 2004), i.e., while distressing elements of the trauma memory are recalled, it may be difficult for patients to access subsequent information (e.g., "I am still living with my children") that corrected impressions they had or predictions they made at the time (e.g., "I will never see my children again"). In treatment, the new appraisals and updated knowledge about the course of the event are explicitly linked with the "hot spots". Particular attention is given to what aspects of the trauma memory patients use as evidence for their appraisals (e.g., a particular look on a dying person's face meaning that the person is still in a state of eternal suffering), as these aspects of the memory will need to be "updated".

Revisiting the scene of the trauma is used to complement discussion and obtain new information that helps explain why or how an event occurred. This is particularly helpful for patients with appraisals such as "I could have prevented the trauma from happening". For example, a driver involved in an accident with a motor vehicle that unexpectedly emerged from a hidden corner was haunted by feelings of guilt as he thought he should have been able to brake in time to avoid the accident. Revisiting the site, recalling how fast he was travelling, seeing how close he was to the corner, and calculating his theoretically maximum braking time helped him see that there was no possibility of him avoiding the accident.

The integration of the treatment procedures used to modify the appraisals of the trauma and the trauma memory is illustrated in Fig. 1.

Excessively *negative appraisals of trauma sequelae* (not illustrated in Fig. 1), such as the initial PTSD symptoms (e.g., Ehlers, Mayou, & Bryant, 1998; Halligan, Michael, Clark, & Ehlers, 2003; Steil & Ehlers, 2000) and other people's responses after the event (e.g., Dunmore, Clark, & Ehlers, 2001, 1999; Ehlers, Maercker, & Boos, 2000), are modified by information, Socratic questioning and behavioural experiments. For example, a young woman who had always been a "coper" in her family was greatly distressed by the sudden mood swings, periods of tearfulness and the intrusive memories she experienced after a severe road traffic accident. All these symptoms meant to her that she was becoming "like her sister", who was generally acknowledged as the "neurotic" of the family. She tried very hard to suppress her intrusions and emotions when reminded of the accident and believed that if she didn't, she would become a nervous wreck. The alternative view that her symptoms were the normal sequelae of a severe trauma that may be maintained by her attempts to suppress them was discussed. To test her belief that she would become like her sister if she allowed herself to become upset about the trauma, she and her therapist intentionally entered a situation that would provoke flashbacks (sitting in a motor vehicle while it was going through an automatic carwash). She was delighted to discover that, although she became very anxious and briefly dissociated, she was still herself, not her sister, afterwards.

Patients who describe a sense of permanent change since the trauma (e.g., Ehlers et al., 2000) are encouraged to "reclaim" their former lives by reinstating significant activities or social contacts they have given up since the trauma.

Goal 2: Reduce reexperiencing by elaboration of the trauma memories and discrimination of triggers. The therapist helps the patient develop a coherent narrative account, which starts before the trauma begins, ends after the patient is safe again and places the series of events during the trauma in context, in sequence, and in the past. This elaboration of the autobiographical memory

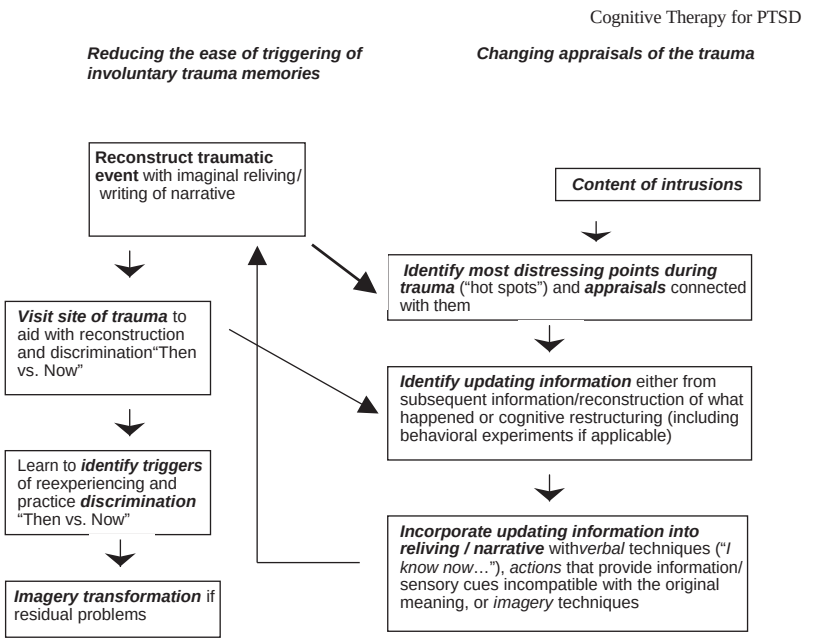

Fig. 1. Cognitive therapy for PTSD: treatment procedures for reducing reexperiencing symptoms and changing appraisals of the trauma. Not shown in the figure are the treatment procedures for changing appraisals of trauma sequelae and for reducing maintaining behaviours and cognitive strategies.

for the event is not only important for identifying and modifying excessively negative appraisals (see Goal 1), but is also thought to inhibit cue-driven retrieval of intrusive memories (Conway, 1997; Ehlers & Clark, 2000).

Three main techniques are used: writing out a detailed account of the event (Blanchard et al., 2003; Resick & Schnicke, 1992), imaginal reliving of the event (Foa & Rothbaum, 1998), and revisiting the site. Each has advantages: writing is particularly useful when aspects of what happened and how it happened are unclear. Reconstructing the event with diagrams and models and a visit to the site can be of further assistance in such instances. Imaginal reliving, in which the patient vividly images the event while simultaneously describing what is happening and what he or she is feeling and thinking, is particularly good at eliciting all aspects of the memory (including emotions and sensory components) and can therefore be very helpful in linking elements together and placing them in context. Revisiting the site of the traumatic event is a particularly helpful way of helping the patient realize that the event is in the past (and hence reducing the sense of "nowness" that characterizes intrusions, see Ehlers et al., 2004). Therapist and patient therefore discuss the way the scene is different from the day of the trauma ("then" versus "now").

Discrimination of triggers usually involves two stages. First, careful analysis of where and when intrusions occur is used to identify triggers. Second, the link between the triggers and the trauma memory is intentionally broken. For example, a man who had been involved in a road traffic accident at night experienced frequent intrusions which sometimes consisted of just

re-experiencing the terror he had felt as he saw that a van was about to plough into the back of his vehicle and sometimes also included images from the crash. He was under the impression that the intrusions came on "out of the blue". A prominent aspect of the trauma memory was the headlights of the van, and it soon became clear the intrusions were often triggered by patches of bright light (such as a patch of sunlight on a lawn or an overhead projector at work). Once this became clear, the patient discriminated between "then" and "now" when the intrusion occurred by telling himself that he was reacting to a past meaning of the light. This point was strengthened by intentionally provoking the memory with bright lights and then behaving in ways that he could not have done at the time (e.g. standing up and moving about).

If reexperiencing symptoms persist, imagery transformation techniques can be useful. For example, a woman who had crashed into a brick pillar was haunted by intrusions in which the pillar flew to within a few inches of her face. Discussion and measuring the distance between her seat and the crumpled front of her vehicle established that in reality the pillar came no closer than 5 ft. This information alone did not stop the intrusion. However, vividly demonstrating to herself that the intrusion was misleading/not real by transforming it into the Microsoft "flying windows" logo did.

Goal 3: Drop dysfunctional behaviours and cognitive strategies. Strategies that have the immediate aim of reducing one's sense of current threat but have the long-term effect of maintaining the disorder are common in PTSD. The strategies maintain the disorder by preventing elaboration of the trauma memory (e.g. avoidance or not talking about the event), by preventing reappraisal (e.g. excessive use of the rear-view mirror after a rear end crash maintaining overestimation of the likelihood of a further crash because the absence of a new accident is attributed to the excessive vigilance), or by increasing PTSD symptoms directly. Treatment usually starts by discussing the problematic consequences of the strategy. The strategy is then dropped/reversed in the context of a behavioural experiment. For example, a young man who believed that he would go mad if he did not try hard to suppress the trauma memory and intrusions was encouraged to test the idea by intentionally allowing intrusions to enter and leave his head without trying to control them. To his surprise, this lead to a subsequent decline in intrusion frequency.

2.1. Similarities and differences to other CBT programs for PTSD

Several of the treatment techniques involved in Cognitive Therapy for PTSD overlap with those of other effective CBT treatments for PTSD. Given this point, one needs to ask in what way the present program differs from existing approaches. Existing approaches are themselves quite variable. However, three general categories of differences can be specified.

First, the way in which some of the overlapping procedures are used differs from how they are used in many, if not all, other CBT programs. Imaginal reliving is not presented as a technique for promoting emotional habituation to a painful memory but instead is used to identify "hot spots" that will be addressed with cognitive restructuring and also to elaborate the trauma narrative. Given this distinction, imaginal reliving is used in a considerably smaller portion of treatment sessions (on average 3 out of 12 sessions) than in repeated and prolonged exposure CBT programs. In some CBT programs (for example, Marks et al., 1998), imaginal reliving and verbal cognitive restructuring are seen as separate procedures with are given in sequence for fixed

amounts of time in a treatment session. By contrast, in the present cognitive therapy program, the two are closely intertwined with the results of cognitive restructuring being actively incorporated into imaginal reliving. Exercises that involve entering feared and avoided situations have a similarly distinct rationale. Rather than aiming to promote emotional habituation by repeated exposure, the exercises are explicitly used to test specific predictions about overgeneralization of danger and to reduce the sense of "nowness" that occurs when a trauma memory is activated. In the latter context, patients are encouraged to activate the trauma memory when revisiting the site of the trauma and focus on what is different between "then" and "now". Cognitive restructuring focuses on the idiosyncratic meanings of the trauma and its sequelae that are identified in sessions. For this reason, unlike some other programs, patients are not taught to use daily thought diaries (Marks et al., 1998) and sessions do not involve discussion of preset cognitive themes (Resick & Schnicke, 1993).

Second, a number of fairly novel techniques are utilized. These include: stimulus discrimination procedures to reduce involuntary triggering of re-experiencing symptoms; active incorporation of updating information into the trauma memory; behavioural experiments that demonstrate the way in which various maintaining processes (such as thought suppression and hypervigilance for danger) operate, and imagery transformation techniques.

Third, the treatment has a stronger emphasis than many other CBT programs on identifying and modifying problematic appraisals of trauma sequelae (initial PTSD symptoms, other peoples' responses after the event, etc.) and on the modification of a wide range of behavioural and cognitive maintaining strategies (e.g. rumination, hypervigilance, overt and covert safety behaviours).

3. Consecutive case series

3.1. Methods

3.1.1. Participants

Twenty consecutive patients (10 women, 10 men) who had been referred by their General Practitioners or Community Mental Health Teams for the treatment of PTSD were recruited for the case series. Their ages ranged between 18 and 64. All patients were Caucasian. Patients met DSM-IV diagnostic criteria for PTSD as determined by the Structured Clinical Interview for DSM-IV (SCID, First, Spitzer, Gibbon, & Williams, 1995). Characteristics of the sample are shown in Table 1. Patients suffered from moderately severe PTSD symptoms at the beginning of treatment, as indicated by moderate assessor ratings of distress and interference on the SCID. Patients reported that the current episode of PTSD was linked to one or two discrete traumatic events in adulthood. The time that had elapsed since the trauma varied considerably between four months and 20 years. Two patients (10%) had a delayed onset of PTSD. Half of the patients reported that they had experienced further traumatic events in their lives, including child sexual assault, sexual or nonsexual assault, or severe accidents, but these events were not addressed in treatment.

Four of the patients (20%) met diagnostic criteria for current major depression, one patient (5%) met criteria for dysthymia, and further three patients (15%) reported a history of major

Table 1
Description of the samples: *N* (%) or means (standard deviations)

Variable		Case series	Randomized controlled trial	
		(*N* = 20)	CT group (*N* = 14)	Waitlist (*N* = 14)
Sex	Female	10 (50%)	8 (57%)	7 (50%)
	Male	10 (50%)	6 (43%)	7 (50%)
Age (in years)	Mean (SD)	34.4 (12.8)	35.4 (10.9)	37.8 (11.2)
Type of traumatic event	Accident	13 (65%)	7 (50%)	8 (57%)
	Assault	4 (20%)	5 (36%)	4 (29%)
	Witness death	3 (15%)	2 (14%)	2 (14%)
Time since traumatic event (in months)	Range	4–240	7–120	6–216
	Median	13.2	11.5	10.8
SCID ratings for severity of PTSD	Distress	5.3 (1.0)	5.6 (1.3)	5.6 (1.2)
	Interference	4.9 (1.7)	6.3 (1.1)	5.5 (1.3)
Marital status	Single	9 (45%)	5 (36%)	5 (36%)
	Married	8 (40%)	6 (43%)	5 (36%)
	Cohabitating	1 (5%)	3 (21%)	3 (21%)
	Divorced	2 (10%)	0 (0%)	1 (7%)
Education: exams passed	University	2 (10%)	3 (21%)	2 (14%)
	A levels (17 + years)	4 (20%)	3 (21%)	7 (50%)
	GSCE (15 + years)	11 (55%)	5 (36%)	4 (29%)
	None	3 (15%)	3 (21%)	1 (7%)
Current employment	Unemployed	2 (10%)	3 (21%)	0 (0%)
	On disability	4 (20%)	1 (7%)	1 (7%)
	Part-time[a]	1 (5%)	2 (14%)	2 (14%)
	Full-time[a]	10 (50%)	7 (50%)	6 (43%)
	Student	2 (10%)	0 (0%)	3 (21%)
	Retired	1 (5%)	0 (0%)	0 (0%)
	Homemaker	0 (0%)	1 (7%)	2 (14%)
Profession	Professional	1 (5%)	5 (38%)	4 (29%)
	White collar	9 (45%)	4 (29%)	3 (21%)
	Blue collar	8 (40%)	4 (29%)	4 (29%)
	Student	2 (10%)	0 (0%)	2 (14%)
	Homemaker	0 (0%)	1 (7%)	1 (7%)

[a]This includes patients on sick leave because of their PTSD symptoms.

depression. Four of the patients (20%) had a comorbid anxiety disorder, and two (10%) had borderline personality disorder.

One patient dropped out after two sessions as she was called to a family emergency abroad.

The number of treatment sessions was not fixed in the pilot study. The 19 completers received between 4 and 20 weekly sessions, mean = 8.3, SD = 4.2, and a mean of 2.1 monthly booster sessions (SD = 1.8) in the following 3 months. Initial treatment sessions lasted 90 minutes, and later sessions 60 min. A mean of 3.1 sessions (SD = 2.1) included some form of reliving.

3.1.2. Continuous outcome measures

The main outcome measure was change in the severity of PTSD symptoms. In addition, we assessed changes in anxiety and depression as measures of associated psychopathology and changes in the disability caused by the symptoms.

Severity of PTSD symptoms. Patients completed the Post-traumatic Diagnostic Scale (PDS, Foa, Cashman, Jaycox, & Perry, 1997). The PDS asks patients to rate how often they were bothered by each of the PTSD symptoms specified in DSM-IV ranging from 0 'never' to 3 '5 times per week or more/almost always'. The PDS yields a sum score measuring the overall severity of PTSD symptoms. Foa et al. (1997) demonstrated that the self-report questionnaire has good reliability and concurrent validity with other PTSD measures. The PDS has satisfactory agreement with the SCID (First et al., 1995), kappa = 0.65, agreement = 82%, sensitivity = 0.89, specificity = .75 (Foa et al., 1997). In addition, patients were asked to indicate how distressing they found each of the symptoms, on a scale from 0 'not at all' to 3 'very distressing' (referred to as PDS-distress scale, Dunmore et al., 2001; Steil & Ehlers, 2000), and to rate how disabling the symptoms had been on a scale from 0 'not at all disabling' to 8 'severely disabling', referred to as PTSD-related disability.

Associated psychopathology. Symptoms of anxiety and depression were assessed with the Beck Anxiety Inventory (BAI, Beck & Steer, 1993a) and the Beck Depression Inventory (BDI, Beck & Steer, 1993b), standard 21-item self-report measures of high reliability and validity.

3.1.3. Measures of treatment response

Several dichotomous measures of treatment response were calculated for comparability with previous studies.

Percentage of patients with PTSD. Patients were considered to meet DSM-IV criteria for PTSD on the PDS if they reported the minimum number of symptoms in each symptom cluster with a score of at least 1, reported a disability score of greater than 2, and had a total score of at least 14 (similar to Ehlers et al., 2003, and Foa, 1998).

Treatment responder. Following Foa and Meadows (1997), patients were classified as treatment responder if they showed a reduction in PTSD symptom severity of 50% or greater on the PDS.

High end state functioning. High end-state functioning was defined as a PDS score below 14, a disability rating below 3, and BDI and BAI scores below 12 (similar to Ehlers et al., 2003).

3.1.4. Data analysis

We report both completer and intent-to-treat data, using the last available assessment to estimate scores for those patients that did not attend the respective assessment. Changes in symptoms over time were assessed with multivariate repeated measures analyses of variance

(MANOVA). The first MANOVA compared the pre and post-treatment scores on the three scales assessing the severity of PTSD symptoms, i.e., PDS, PDS-distress, and PTSD-disability. The second MANOVA compared the pre- and post-treatment scores on measures of associated psychopathology, i.e., depression (BDI) and anxiety (BAI). Corresponding analyses were performed for the comparison of baseline scores with the 3- and 6-month follow-ups, and for the comparison of post-treatment and follow-up scores. If multivariate time effects were significant, univariate analyses followed. We report the statistical results for the conservative intent-to-treat analyses.

Treatment effect sizes for changes in PTSD symptom scores were calculated using Cohen's d statistic (Cohen, 1988), following the formula used in van Etten and Taylor (1998) meta-analysis of PTSD treatments: $d = M_{\text{initial}} - M_{\text{post}}/\text{SD}_{\text{pooled}}$, where $\text{SD}_{\text{pooled}} = \sqrt{[]}(\text{SD}^2_{\text{initial}}+\text{SD}^2_{\text{post}})/2$. The original PDS score was used for comparability with other studies.

3.2. Results

Table 2 shows the results for the consecutive cases series. CT significantly reduced the severity of PTSD symptoms, $F(3, 17) = 37.26$, $p < .0005$, all univariate tests $p < .0005$. Treatment also led to highly significant reductions in symptoms of anxiety and depression, $F(2, 18) = 26.21$, $p < .0005$, univariate tests $p < .0005$. The effect sizes for pre to post-treatment changes in PTSD symptoms (PDS) were $d = 2.81$ for completers, and $d = 2.70$ for the intent-to-treat analysis.

At post-treatment, seventeen patients (90% of the completers, 85% of the intent-to-treat sample) no longer met DSM-IV criteria for PTSD. Eighteen patients (95% of the completers, 90% of the intent-to-treat sample) were classified as treatment responders, and 16 patients (84% of the completers, 80% of the intent-to-treat sample) had achieved high end-state functioning.

The positive outcome was maintained at the 3-month and 6-month follow-ups. PTSD symptom severity remained much reduced compared to initial assessments, $F(3, 17) = 38.15$, $p < .0005$ at 3 months and $F(3, 17) = 38.13$, $p < .0005$ at 6 months, all univariate tests $p < .0005$; as did anxiety and depression, $F(2, 18) = 26.73$, $p < .0005$ at 3 months and $F(2, 18) = 24.76$, $p < .0005$ at 6

Table 2
Treatment outcome for the consecutive case series: means (standard deviations)

Measure	Before treatment	Completer			Intent-to-treat		
		After treatment	3-month follow-up	6-month follow-up	After treatment	3-month follow-up	6-month follow-up
N	20	19	19	14	20	20	20
PDS (original scale)	27.5 (9.7)	5.0 (5.2)	4.7 (5.8)	5.6 (7.5)	5.7 (5.9)	5.5 (6.5)	7.0 (7.9)
PDS (distress scale)	28.4 (10.2)	3.7 (4.8)	3.8 (1.8)	4.5 (7.0)	5.1 (7.5)	5.3 (8.1)	6.8 (9.2)
Self-reported disability due to PTSD symptoms	3.9 (1.8)	0.9 (1.2)	1.2 (1.5)	1.0 (1.4)	1.2 (1.6)	1.4 (1.8)	1.6 (2.0)
Depression (BDI)	21.9 (13.0)	5.2 (5.5)	4.6 (7.1)	5.0 (6.7)	6.3 (7.3)	5.8 (8.5)	7.6 (8.9)
Anxiety (BAI)	20.4 (10.7)	3.3 (4.0)	2.3 (3.5)	3.3 (5.4)	3.6 (4.1)	2.6 (3.7)	4.3 (5.3)

months, univariate tests $p < .0005$. There were no changes between the post-treatment and follow-up assessments, all multivariate p's $> .54$.

3.3. Discussion

The results of the consecutive case series were encouraging. Cognitive Therapy for PTSD was found to be very acceptable. Only one patient (5%) dropped out, and this appeared to be for reasons unrelated to treatment. CT was highly effective in reducing PTSD symptoms as well as symptoms of anxiety and depression. Treatment gains were maintained during follow-up. The effect size for pre to post-treatment changes in self-reported PTSD symptoms (PDS) of 2.81 was twice as high as that reported in the meta-analysis of CBT treatments (1.27) by van Etten and Taylor (1998). The results also compare favorably with the effect sizes observed for cognitive restructuring without imaginal reliving of the trauma (Marks et al., 1998; Tarrier et al., 1999). The results are comparable to those achieved in *completer* analyses of recent trials of prolonged exposure (PE, Foa et al., 1999; Resick et al., 2002), although much less reliving was used, possibly contributing to the high acceptability of the treatment.[1]

The results remain preliminary as no control group was included, and there were no independent assessor ratings. The preliminary data supported the evaluation of the treatment in a randomized controlled trial.

4. Randomized controlled trial

4.1. Method

4.1.1. Patients

Twenty-eight patients were recruited from consecutive referrals from General Practitioners and Community Mental Health Teams. To be accepted into the trial, patients had to meet the following inclusion criteria: 18–65 years old; meeting diagnostic criteria for PTSD as determined by the SCID (First et al., 1995); the current episode of PTSD was linked to discrete traumatic events in adulthood; PTSD was the main problem; and time since the trauma was at least 6 months. Half of the patients reported an earlier trauma meeting the A criterion of DSM-IV, but these events were not addressed in treatment.

Exclusion criteria were: unconsciousness for more than 15 min or no memory for the trauma; history of psychosis; current alcohol or drug dependence; borderline personality disorder; severe depression needing immediate treatment in its own right (i.e., suicide risk); and assessment and treatment could not be conducted without the aid of an interpreter.

A total of 51 SCID interviews with consecutive referrals were conducted to recruit the sample. A random sample of 40 SCID interviews (by eight interviewers) from the present and a related study (Ehlers et al., 2003) was also coded by a second clinician (six raters). Interrater reliabilities

[1] In the initial development stage of the treatment, we tried to identify these appraisals through verbal discussion techniques and obtained smaller effect sizes, comparable to those in Tarrier et al.'s (1999) study who, for the purposes of their research design, did not use imaginal reliving to identify these appraisals.

were very good (PTSD diagnosis: kappa = .95; distress: $r = .84$; interference: $r = .96$). Thirteen patients did not meet DSM-IV criteria for PTSD, one did not complete the assessment, and two reported that their symptoms stemmed from prolonged childhood trauma. Of the patients who met inclusion criteria, one did not agree to participate in the trial, and 6 were excluded because they met one or more exclusion criterion (1 patient with borderline personality disorder, 1 patient with substance dependence and psychosis, and 4 patients with high suicide risk requiring immediate treatment).

Patients were randomly allocated to either immediate cognitive therapy (CT, $N = 14$) or a 13-week waitlist (WL, $N = 14$) condition. Table 1 presents demographic and trauma characteristics. With the exception of one black patient in immediate CT, all patients were Caucasian. One patient in the CT group and two patients in the WL group reported delayed onset of their PTSD. The groups were comparable in demographic and trauma characteristics (all p's > .26). As shown in Table 3, self-reported symptom severity was also similar on all measures (all p's > .17). However, independent assessors rated the CT group as more severe on the Clinician-Administered PTSD Scale (CAPS-SX, Blake et al., 1995); CAPS-frequency, $F(1, 26) = 10.55$, $p = .003$, CAPS-intensity, $F(1, 26) = 4.92$, $p = .036$, and overall CAPS-severity, $F(1, 26) = 8.37$, $p = .008$. There was also a trend for a greater proportion of patients with comorbid anxiety disorders in the CT group, 4 (43%) versus 2 (14%), $\chi^2(1, 28) = 2.80$, $p = .094$. Seven of the CT patients (50%) and 4 of the waitlist patients (29%) met diagnostic criteria for current major depression, and further 4 (29%) of the CT patients and 3 (21%) of the waitlist patients reported a history of major depression.

4.2. Treatment conditions

Cognitive therapy. CT followed the treatment model described above. Each case was discussed in weekly group supervision and selected videotapes from treatment sessions were watched to ensure that therapists adhered to therapy protocol. The design allowed up to 12 weekly treatment sessions (of 90 min for the initial sessions, and 60 minutes thereafter), which could be offered within 13 weeks (i.e., sessions were lost if patients did not attend) and up to 3 monthly booster sessions. Patients received between 4 and 12 weekly sessions, mean = 10.0, SD = 2.9, and a mean of 2.4 monthly booster sessions, SD = 1.1. A mean of 3.0 sessions, SD = 1.4, contained some imaginal reliving; with a total mean duration of 98.0 min of reliving for the whole course of treatment, SD = 56.5.

Waitlist. Patients assigned to the 13-week waitlist condition were told that they would receive a course of CT in 13 weeks time, after completing a second assessment. They were told to contact the research group if they felt they could no longer wait for treatment or felt suicidal, but no patient used this option.

4.3. Outcome measures

Self-report measures of PTSD symptoms, depression and anxiety were the same as for the case series. Additional measures included:

Clinician-rated PTSD symptoms. Independent assessors who were not aware of the treatment condition (trained psychologists) gave the CAPS-SX (Blake et al., 1995). The CAPS assesses the

Table 3
Treatment outcome for the randomized controlled trial: means (standard deviations)

Measure	Cognitive therapy group			Waitlist group		Statistical results		
	Initial assessment	After 3 months of treatment	6-month follow-up	Initial assessment	After 3-month wait	Group difference post treatment	Changes in CT group	Changes in wait group
N	14	14	14	14	14	MANCOVA and univariate tests	MANOVA and univariate tests	MANOVA and univariate tests
PTSD symptoms						*F(4,19)=15.09, p<.0005*	*F(4,10)=26.34, p<.0005*	*F(4,10)=2.36, p=.124*
PDS (original scale)	32.4 (6.5)	10.3 (8.9)	12.4 (9.9)	31.2 (6.3)	29.8 (8.4)	$p<.0005$	$p<.0005$	n.s.
PDS (distress scale)	33.8 (7.1)	9.7 (10.1)	11.0 (9.9)	34.4 (7.1)	30.5 (9.3)	$p<.0005$	$p<.0005$	($p=.031$)
CAPS (frequency)[a]	42.0 (8.5)	16.0 (15.3) 11.4 adj. *M*	16.0 (14.4)	31.6 (8.4)	35.5 (11.4) 40.2 adj. *M*	$p<.0005$	$p<.0005$	n.s.
CAPS (intensity)[a]	36.5 (9.4)	13.7 (13.4) 10.4 adj. *M*	15.5 (14.8)	29.0 (8.5)	30.9 (9.6) 34.2 adj. *M*	$p<.0005$	$p<.0005$	n.s.
Disability						*F(2,23)=18.38, p<.0005*	*F(2,12)=30.12, p<.0005*	*F(2,12)=.67, p=.531*
Disability (self-report, Sheehan scale)	7.6 (1.9)	3.0 (2.6)	3.0 (2.6)	6.7 (1.9)	6.3 (1.8)	$p<.0005$	$p<.0005$	n.s
Disability (CAPS)	3.1 (.6)	1.4 (1.1)	1.3 (1.2)	2.4 (.6)	2.5 (.7)	$p<.0005$	$p<.0005$	n.s.
Associated psychopathology						*F(2,23)=8.52, p=.002*	*F(2,12)=18.15, p<.0005*	*F(2,12)=3.68, p=.056*
Depression (BDI)	23.7 (9.0)	10.6 (8.6)	11.2 (9.6)	23.2 (8.0)	19.3 (7.2)	$p=.003$	$p<.0005$	($p=.025$)
Anxiety (BAI)	24.1 (11.1)	8.2 (10.8)	7.5 (9.7)	19.2 (7.2)	21.2 (11.2)	$p<.0005$	$p<.0005$	n.s.

Legend: PDS—Posttraumatic Diagnostic Scale; CAPS—Clinician Administered PTSD Scale; BDI—Beck Depression Inventory; BAI—Beck Anxiety Inventory; n.s.—non-significant; ( ) univariate effect in the absence of a significant multivariate effect.

[a]As there was a significant baseline difference between the CT and waitlist groups on the CAPS, we report adjusted means (adj. *M*) at 3 months.

frequency and severity of each of the symptoms specified in DSM-IV. A random sample of 38 CAPS interviews (eight different interviewers) from the present and a related study (Ehlers et al., 2003) was rated by a second clinician (seven different raters). Results indicated very good reliability for the PTSD diagnosis, kappa = .94, and total severity score, $r = .96$. Patients were considered to meet DSM-IV criteria for PTSD on the CAPS-SX if they reported the minimum number of symptoms in each symptom cluster with a score of at least 1 (on both the frequency and intensity scales) and the global severity rating was 2 or greater "definite distress or functional impairment".

Disability measures. Patients completed the Sheehan Disability Scale (American Psychiatric Association, 2000). Patients rated the interference caused by the PTSD symptoms in their (a) work, (b) social life/ leisure activities, and (c) family life/home responsibilities on three Likert scales from 0 'not at all' to 10 'very severe'. The disability score was the mean of these ratings. The overall CAPS (Blake et al., 1995) severity rating (item 24) ranging from 0 'No clinically significant symptoms, no distress or functional impairment' to 4 'Extreme, marked distress or marked impairment in two or more areas of functioning' was used as a global measure of clinician-rated disability.

4.4. Data analysis

Data analysis followed a hierarchical approach. To test whether CT and WL led to differential outcome at 3 months (the end of weekly CT sessions), we performed multivariate analyses of covariance (MANCOVA) for each of the three sets of continuous outcome measures (PTSD symptoms, associated psychopathology, disability), using scores at initial assessment as covariates. Reported multivariate F values are based on Pillais coefficients. If multivariate effects of condition were significant, we conducted univariate comparisons. Dichotomous criteria of treatment response were compared with χ^2-tests.

Controlled effect sizes are the differences between the mean 3-month PTSD symptom scores for CT versus waitlist, divided by the pooled SD of the two conditions compared (Sherman, 1996). The original PDS score and the CAPS-total score (adjusted means from analysis of covariance, due to baseline difference) were used for this analysis.

Changes in symptoms with time were assessed with multivariate repeated measures analyses of variance (MANOVA) comparing scores at initial assessment with those at 3 months (end of weekly CT sessions, end of WL) and follow-up (6 months after the end of weekly CT sessions). Stability of treatment effects was assessed by MANOVAs comparing post-treatment and follow-up scores.

4.5. Results

4.5.1. End of treatment

Table 3 presents the results of the randomized controlled trial. No patient dropped out. On all measures, the CT group had better outcome at 3 months than the WL group. CT led to highly significant changes on all measures. The WL group did not show significant changes. None of the patients waiting for treatment, but 10 (71.4%) of the patients who received immediate CT did not meet diagnostic criteria for PTSD (CAPS and PDS) at the 3-month assessment, $\chi^2(1, 28) = 15.56$,

$p < .0005$. Similarly, none of the waitlist patients, but 11 (78.6%) of the CT group met criteria for treatment response $\chi^2(1, 28) = 18.12$, $p < .0005$. Nine CT patients (64.3%) and no WL patients (0%) achieved high end state functioning, $\chi^2(1, 28) = 13.26$, $p < .0005$.

4.6. Maintenance of treatment effects

Table 3 shows that the treatment gains in the CT group were well maintained at follow-up six month after the end of treatment (9 months after beginning of treatment). On all measures, CT patients remained very much improved compared to initial assessments, all p's $< .0005$. There were no changes in the scores from post-treatment to follow-up assessments on any measure, all p's $> .24$.

4.7. Effect sizes

The effect size for pre to post-treatment changes in PDS scores with CT was $d = 2.82$, and for the CAPS $d = 2.07$. The controlled effect size for post-treatment scores on the PDS for CT versus WL was $d = 2.25$, and $d = 2.18$ (adjusted means: CT: $M = 21.58$, SD = 28.56; WL: $M = 74.55$, SD = 19.12) on the CAPS.

5. Predictor analysis

Data for patients treated the consecutive case series and in the RCT (immediate and post-wait treatment) were combined for a predictor analysis of treatment outcome. A further nine consecutive patients with chronic PTSD that met RCT criteria and were subsequently treated with CT by our group were added to increase sample size ($N = 57$). The measure of treatment outcome was the residual gain score from the regression of pre-treatment composite PDS and PDS-distress scores onto composite scores at the end of treatment (composite scores were used to generate a single measure). The relationship between 10 possible demographic and clinical predictors and outcome was either tested by Pearson correlations (continuous measures), Spearman correlations (rank ordered variables), biserial correlations (dichotomous measures), or analysis of variance (categorical variables with more than two categories).

The following variables were not related to treatment outcome: age, sex, living alone/with a partner, time since trauma, trauma type, presence of previous traumas, comorbid major depression, and comorbid anxiety disorders. The only significant predictors of outcome were lower educational attainment, $\rho = .316$, $p = .017$, and lower socioeconomic status, Rho = .281, $p = .034$, both of which were associated with *good* outcome.

CT for PTSD is based on the assumption that changing appraisals that lead to a sense of current threat is a necessary condition for improvement. A theoretically motivated analysis therefore tested whether changes in the PTCI (Foa et al., 1999), which measures a range of appraisals typical of PTSD patients, correlated with outcome. The correlation was highly significant, $r = .561$, $p < .0005$. Better outcome was related to greater changes in the PTCI.

6. Discussion

The RCT showed that CT for PTSD was superior to a 3-month waitlist condition on measures of PTSD symptoms, disability, and associated symptoms of anxiety and depression. As in the consecutive case series, the effect size for changes in PTSD symptoms with CT was very large. The effect size of 2.82 for pre to post-treatment changes with CT in the PDS in our *intent-to-treat* analysis compares favorably to the mean effect size of 1.27 for treatment *completers* for cognitive behavioural treatments of PTSD reported in van Etten and Taylor (1998) meta-analysis, and is in the range of the effect sizes reported for *completer* analyses in recent controlled evaluations of other CBT programs for PTSD (Blanchard et al., 2003; Bryant et al., 2003; Resick et al., 2002; Foa et al., 1999). The effect size exceeds the effect sizes of intent-to-treat analyses reported in other studies. The controlled intent-to-treat effect sizes (CT versus wait) of 2.25 for the PDS and 2.18 for the CAPS also compare favorably with the mean of .52 reported for completer analyses of psychotherapeutic treatments for PTSD in another meta-analysis (Sherman, 1996).

These results are encouraging. However, comparisons of effect sizes between with studies using different populations of PTSD patients are problematic so that no firm conclusions can be drawn. Although the inclusion and exclusion criteria of the present studies were comparable to those of other randomized controlled trials of CBT treatments for PTSD, future randomized controlled trials will have to compare CT for PTSD against alternative treatments to test whether it is a specific treatment, and whether it offers any advantages over other existing CBT programs.

As in the pilot study, acceptability of the treatment was good. None of the patients who received immediate CT dropped out. If the pilot study and the RCT are considered together, the dropout rate for CT can be estimated at 3%. This is in line with the results of an early intervention study using our CT program that yielded a dropout rate of 0% (Ehlers et al., 2003). These rates compare favorably with the results for CBT approaches in general. One may argue that the low dropout rates may be a reflection of the particular way health services are organized in the UK. We do not think that this is the case as other UK trials (Marks et al., 1998; Tarrier et al., 1999) using different versions of CBT have found drop out rates as high as those in North American or Australian studies.

As expected, change in cognitive appraisals characteristic of patients with PTSD (Foa et al., 1999) was positively related to treatment outcome. This is in line with the hypothesis that changing these appraisals is one of the mechanisms of change in CT, although the correlational data do not allow causal conclusions. The Ehlers and Clark (2000) model suggests two other pathways of change: change in the autobiographical memory for the trauma, and dropping of maintaining behaviours and cognitive strategies. Although the treatment addressed these other maintaining factors, changes were not systematically measured in the present study and their role in predicting treatment outcome will need to be investigated in future research. A more comprehensive test of the model will include mediation analyses of treatment outcome in which the three pathways of change are considered simultaneously.

Demographic, trauma and diagnostic variables largely did not predict treatment outcome, suggesting that the treatment is applicable to a wide range of trauma survivors. However, the degree of variation in the sample was limited and it is possible that future studies will detect differences. It was encouraging to see that comorbid depression and previous trauma history did not negatively affect outcome, as these are common features of patients with PTSD. Similar

results were obtained in the Gillespie, Duffy, Hackmann & Clark (2002) study. The significant association of lower educational attainment and socioeconomic status with good outcome should not be over-interpreted as this may present a chance finding. However, the findings contradict the hypothesis that CT only works in highly educated patients.

The present studies had several limitations. First, the sample size in the RCT was modest. However, there was no problem with power as the effect sizes were very large. Furthermore, the results replicated well from the consecutive cases series to patients treated in the RCT. Thus, it is highly unlikely that the large effects occurred by chance. Finally, another RCT using CT as an early intervention had a larger sample size and showed equivalent results (Ehlers et al., 2003). Second, the treatment was conducted by therapists specialized in CT for anxiety disorders who developed the treatment. As the treatment is quite complex, the question arises of whether the treatment can be also used effectively by other clinicians. A recent study suggests that this is the case. Clinicians working in the National Health Service who had received training in CT for PTSD treated an unselected community sample of 91 survivors of the terrorist bomb attack in Omagh, Northern Ireland, and found a similarly high effect size of $d = 2.47$ for pre to post-treatment changes in self-reported PTSD symptoms (Gillespie et al., 2002).

Overall, the results support the efficacy of CT for PTSD. The treatment had good acceptability and led to large improvements that were maintained in follow-up. The treatment appears to be a viable alternative to other CBT programs for PTSD that involve more extensive exposure.

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