

Cognitive-Behavioral Therapy for PTSD in Children and Adolescents: A Preliminary Randomized Controlled Trial

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ABSTRACT

Objective: To evaluate the efficacy of individual trauma-focused cognitive-behavioral therapy (CBT) for treating posttraumatic stress disorder (PTSD) in children and young people. **Method:** Following a 4-week symptom-monitoring baseline period, 24 children and young people (8–18 years old) who met full *DSM-IV* PTSD diagnostic criteria after experiencing single-incident traumatic events (motor vehicle accidents, interpersonal violence, or witnessing violence) were randomly allocated to a 10-week course of individual CBT or to placement on a waitlist (WL) for 10 weeks. **Results:** Compared to the WL group, participants who received CBT showed significantly greater improvement in symptoms of PTSD, depression, and anxiety, with significantly better functioning. After CBT, 92% of participants no longer met criteria for PTSD; after WL, 42% of participants no longer met criteria. CBT gains were maintained at 6-month follow-up. Effects of CBT were partially mediated by changes in maladaptive cognitions, as predicted by cognitive models of PTSD. **Conclusions:** Individual trauma-focused CBT is an effective treatment for PTSD in children and young people. *J. Am. Acad. Child Adolesc. Psychiatry*, 2007;46(8):1051–1061. **Key Words:** posttraumatic stress disorder, cognitive-behavioral therapy.

Children and adolescents can develop posttraumatic stress disorder (PTSD) after exposure to a variety of traumatic events, including sexual abuse, interpersonal violence, and motor vehicle accidents. PTSD is associated with substantial impairments in social and academic functioning, even at subclinical levels (Giaconia et al., 1995) and, if left untreated, may run a chronic course for at least 5 years in more than one third of children who develop the disorder (Yule et al.,

2000). Effective treatment for pediatric PTSD is needed (see Feeny et al., 2004; Stallard, 2006).

A substantial body of work provides support for the efficacy of cognitive-behavioral therapy (CBT) for children with PTSD and other symptoms following sexual abuse. For example, a series of studies by Deblinger and colleagues (e.g., Cohen et al., 2004; Deblinger et al., 1990, 1999) has demonstrated that trauma-focused CBT (which included anxiety management components such as coping skills training and joint work with parents) with children 3–16 years old is effective in reducing symptoms of PTSD and externalizing symptoms relative to waitlist (i.e., delayed treatment; WL), to child-centered therapy, to parent-only intervention, and to community-care referrals. However, not all of the participants in these studies met criteria for a diagnosis of PTSD, and many presented with additional difficulties (including behavior problems and other anxiety disorders). Indeed, Feeny et al. (2004) note in their recent review that child survivors of sexual abuse may present a different symptom picture from that of children exposed to single-incident

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traumas and may require special treatment programs that differ from those designed for children who have experienced single-incident traumas. That is, given the important differences in etiology and presentation, generalization from these important studies of child survivors of sexual abuse to youths who have been exposed to trauma other than sexual abuse is hazardous (also see American Academy of Child and Adolescent Psychiatry, 1998).

A smaller number of studies has evaluated the effect of CBT for youths with PTSD following single-event traumas such as natural disasters and exposure to violence (e.g., Goenjian et al., 1997; March et al., 1998). To our knowledge, only two randomized controlled trials (RCTs) of group CBT for children who developed PTSD symptoms, but not necessarily the full diagnosis, following natural disasters or exposure to violence have been published (Chemtob et al., 2002; Stein et al., 2003). Both report positive results for CBT relative to no treatment controls. The present study extends these findings by evaluating a new individual CBT in a sample of children, all of whom developed full PTSD following single-event traumas. To our knowledge, this is the first RCT to evaluate individual CBT for young people with a primary diagnosis of PTSD, the first to carry out such an evaluation in a referred clinic sample, and the first child study to use a baseline symptom-monitoring period before randomization.

CBT in the present study shares some features with protocols used in previous studies with children (e.g., March et al., 1998; Stein et al., 2003), but differs in a number of key respects. Intervention was based on the cognitive model of PTSD of Ehlers and Clark (2000), which has generated effective treatments for adults (Ehlers et al., 2003a, 2005). There is now accumulating evidence that, suitably adapted, the model applies well to children and young people (Ehlers et al., 2003b; Meiser-Stedman, 2002; Stallard, 2003). Under the model, persistent PTSD is maintained by disjointed and poorly elaborated trauma memories, idiosyncratic misappraisals of the trauma and trauma-related symptoms, and dysfunctional (behavioral and cognitive) coping strategies. Adaptations of the model for children take account of the important role that parental reactions and coping strategies play in the maintenance of children's PTSD (McFarlane, 1987; Meiser-Stedman et al., 2006; Smith et al., 2001). Treatment targets

these maintaining factors by developing a coherent narrative of the trauma, challenging unhelpful appraisals of the trauma and sequelae, changing maladaptive avoidant coping strategies, modifying parents' unhelpful trauma-related appraisals, and recruiting parents as cotherapists.

Before randomized allocation to CBT or WL in the present study, all of the participants completed a 4-week period of symptom monitoring, with minimal therapist contact by telephone. This procedure has been used to good effect with adults, with 12% to 13% of adult PTSD patients improving sufficiently to lose their diagnosis after a 3- to 4-week monitoring phase (Ehlers et al., 2003a; Tarrrier et al., 1999). To our knowledge, the therapeutic effect of such symptom monitoring has yet to be evaluated in young samples.

Our hypotheses were the following: up to 15% of participants would improve sufficiently to lose their diagnosis of PTSD after symptom monitoring; CBT would result in greater improvements in symptoms of PTSD, depression, and anxiety compared to WL; more participants would be free of a PTSD diagnosis following CBT compared to WL; CBT treatment gains would be maintained at 6-month follow-up; and the effect of CBT would be mediated by changes in maladaptive misappraisals.

METHOD

Participants

Sample size was determined in advance by power calculations based on an estimated recovery rate (loss of PTSD diagnosis) of 60% in the CBT group (e.g., March et al., 1998) and 15% in the WL group (e.g., Marks et al., 1998). Assuming these recovery rates, 14 children per group gives 80% power to detect the difference between CBT and WL at a 5% α level (one-tailed). We oversampled at study entry by 20% to take account of the expected loss of participants due to improvement during the symptom-monitoring phase, giving a target of $N = 35$ at study entry. Inclusion criteria were 8–18 years old, main presenting problem is PTSD relating to a single traumatic event, and fluent in English. Exclusion criteria were the presence of organic brain damage, unconscious for more than 15 minutes during the trauma, significant learning difficulty, ongoing trauma-related threat in the environment, recently initiated (within 3 months) treatment with psychotropic medication, and currently receiving another psychological treatment. Participants were recruited from a specialist National Health Service trauma clinic for young people in London. This clinic accepts referrals from community mental health teams, family doctors, and lawyers. Self-referral following attendance at the emergency department of three local hospitals was also possible. Demographic information for the 38 participants who entered the study and for the subsample who were randomized to CBT or WL after symptom monitoring is

TABLE 1
Description of the Sample

	Total Enrolled (<i>N</i> = 38)	Total RCT (<i>n</i> = 24)	CBT (<i>n</i> = 12)	WL (<i>n</i> = 12)	Group Comparison (CBT/WL)
Sex					
Boy	23	12	6	6	
Girl	15	12	6	6	
Age, y					
Mean	13.69	13.89	14.45	13.33	$t_{22} = 0.98, p = .34$
SD	2.67	2.82	2.70	2.95	
Ethnicity					
White British	23	11	6	5	
Black British	10	8	4	4	
Asian British	2	2	0	2	$\chi^2_3 (n = 24) = 2.4, p = .49$
Other/not stated	3	3	2	1	
Referred by					
CAMHS/GP	16	13	6	7	
Hospital ED	18	8	4	4	$\chi^2_2 (n = 24) = 0.41, p = .82$
Lawyer	4	3	2	1	
Traumatic event					
MVA	21	12	7	5	
Assault	12	9	3	6	$\chi^2_2 (n = 24) = 1.7, p = .44$
Witnessed violence	4	3	2	1	
Time since trauma, mo					
Range	2.3–71	3.3–64	3.6–64	3.3–24	
Median	4.90	8.65	5.55	9.67	$U (n = 24) = 54, p = .30$
Attended ED?					
Yes	24	14	9	5	Fisher exact test, $p = .21$
In hospital overnight?					
Yes, no. of nights	10	6	4	2	Fisher exact test, $p = .64$
Range	1–42	1–42	1–42	1	
Median	2.5	2.5	5.5	1	
Days off from school?					
Yes, no.	29	17	9	8	Fisher exact test, $p = 1.0$
Range	2–365	2–365	7–330	2–365	
Median	32.5	36	43	28	
Prior exposure to trauma?					
Yes	11	7	3	4	Fisher exact test, $p = 1.0$
Psychiatric history?					
Yes	10	4	4	0	Fisher exact test, $p = .09$
Ongoing legal case?					
Yes	13	10	7	3	Fisher exact test, $p = .21$
Any comorbidity?					
Yes	29	19	9	10	Fisher exact test, $p = 1.0$

Note: RCT = randomized controlled trial; CBT = cognitive-behavioral therapy; WL = waitlist; CAMHS = child and adolescent mental health service; GP = general practitioner; MVA = motor vehicle accident; ED = emergency department.

summarized in Table 1. Details of patient flow through the trial are presented in the Results section and in Figure 1.

Measures

All of the participants and their parents were interviewed initially using the Anxiety Disorders Interview Schedule (ADIS-C/P; Silverman and Albano, 1996) for PTSD diagnosis and assessment of comorbid disorders (American Psychiatric Association, 1994).

The Clinician Administered PTSD Scale for Children and Adolescents (CAPS-CA; Nader et al., 1994) was used to assess preresponse PTSD caseness and treatment outcome in the second part of the study. Interviews were carried out by doctoral degree-level clinical psychologists who had undergone specific training in assessment. Reliability of PTSD diagnosis was checked on a subset of 30 randomly selected interviews and was satisfactory ($\kappa = .82$). All of the participants completed the following self-report measures at each assessment point: the Child PTSD Symptom

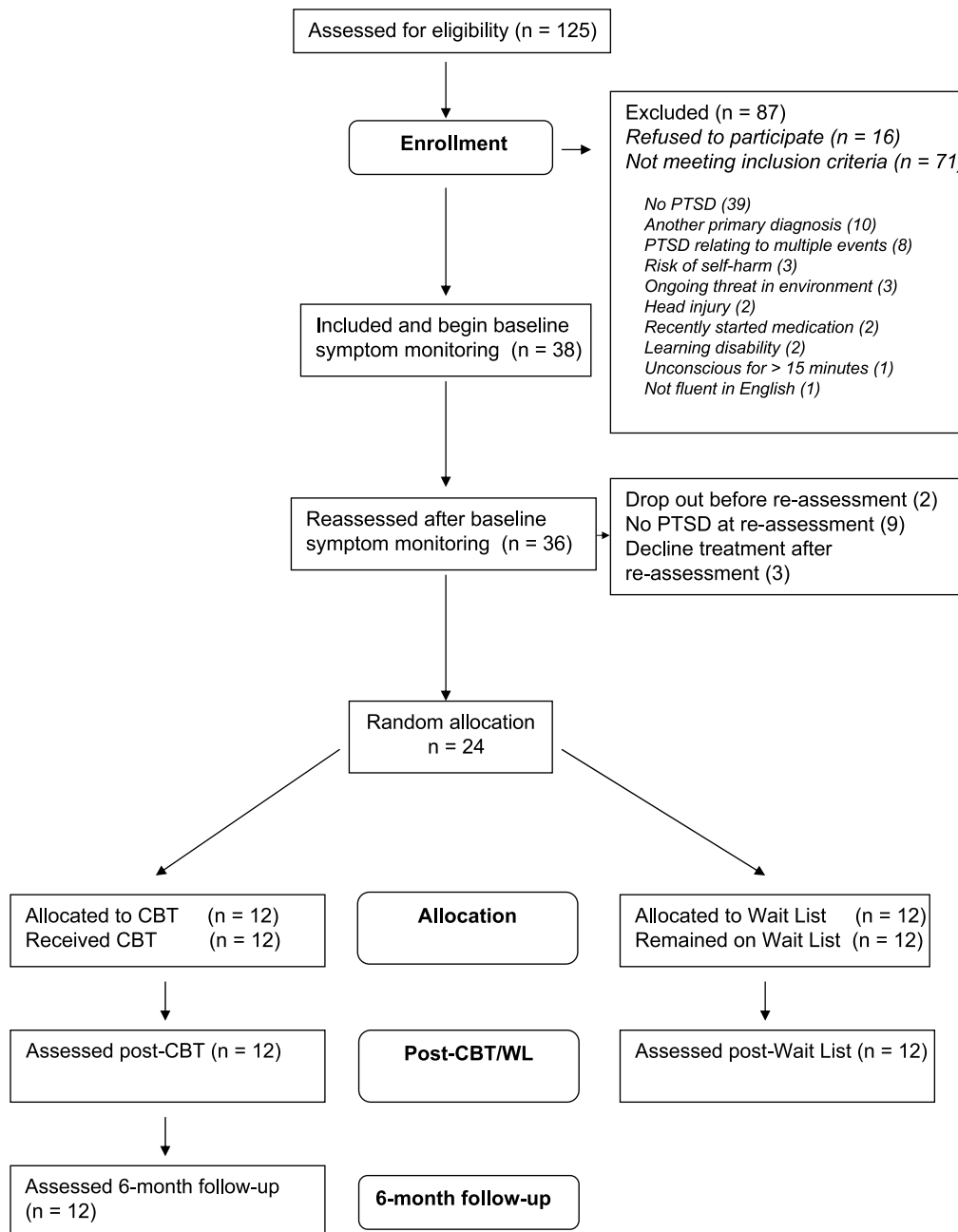


Fig. 1 Participant progress (CONSORT flowchart). CBT = cognitive-behavioral therapy; WL = waitlist; PTSD = posttraumatic stress disorder.

Scale (CPSS; Foa et al., 2001); Children’s Revised Impact of Event Scale (C-RIES; Perrin et al., 2005); the Depression Self-Rating Scale (DSRS; Birlerson, 1981); the Revised Children’s Manifest Anxiety Scale (RCMAS; Reynolds and Richmond, 1978); and the Children’s Post Traumatic Cognitions Inventory (C-PTCI; Meiser-Stedman, 2003). The CPSS was also administered at the beginning of each treatment session to monitor symptom change during the course of treatment. The C-PTCI is a new adaptation of the adult PTCI (Foa et al., 1999) measuring trauma-related beliefs and appraisals. It comprises 25 items rated on a 4-point scale. Unpublished data

from a separate sample of young people exposed to trauma showed that the scale possessed good internal reliability and a meaningful two-factor component structure.

Baseline Symptom Monitoring

Symptom-monitoring followed the procedure described by Tarrier et al. (1999). Participants completed a single sheet diary once per day for 4 weeks, noting how frequently intrusive memories or nightmares had occurred that day or the previous night, how

upsetting they were (i.e., two frequency scales with two Likert ratings of distress), and what efforts were made to manage them (open-ended question). Therapist contact during this period was limited to one brief weekly telephone call to remind participants to complete the forms. No psychoeducation about the nature of PTSD or advice regarding symptom management was provided at this stage.

Cognitive-Behavioral Therapy

CBT was based on the model of PTSD of Ehlers and Clark (2000) with suitable adaptations for use with children and young people (Yule et al., 2005). Treatment was manual based, piloted before the start of the trial, and then delivered in 10 weekly individual sessions. The majority of sessions were individual CBT with the young person; parents were always seen (if available) after the individual session with their child; and joint parent-child sessions were carried out as necessary. Treatment components included psychoeducation, activity scheduling/reclaiming life, imaginal reliving (including writing and drawing techniques), cognitive restructuring followed by integration of restructuring into reliving, revisiting the site of the trauma, stimulus discrimination with respect to traumatic reminders, direct work with nightmares, image transformation techniques; behavioral experiments, and work with parents at all stages. The close integration of cognitive restructuring with reliving and the use of stimulus discrimination techniques are components unique to this particular child treatment program. The remaining components overlap with those used in previous child treatment programs (e.g., March et al., 1998; Stein et al., 2003). The current program did not include relaxation training or other anxiety management techniques. CBT was delivered by doctoral-level clinical psychologists with at least 10 years postqualification experience of working with traumatized children. Monthly supervision (provided by D.M.C. and W.Y.) included viewing videotapes of clinical sessions and addressed treatment adherence as well as clinical issues.

Waitlist

Immediately after randomization, participants allocated to WL were given an appointment for 10 weeks later. An appointment letter and one reminder telephone call was made before the reassessment; otherwise, there was no contact with the clinic during the waiting period.

Procedure

The study was approved by the Research Ethical Committee of the Institute of Psychiatry University of London (reference number 011/01). Written informed consent to participate was obtained from young people and their parents. Initial assessment was carried out using the ADIS-C/P with young people and parents separately. All of the participants completed 4 weeks of symptom monitoring. Reassessment was carried out using the ADIS-C/P with parents and the CAPS-CA with young people. Participants who retained their PTSD diagnosis were randomized using a computer program (MINIM) (Evans et al., 1990) to CBT or WL. Randomization was carried out using a minimization procedure (Pocock, 1983) with stratification according to age (two groups: younger than 14 years and 14 years and older), sex, and initial symptom severity (two groups: scores of <70 and ≥ 70 on the CAPS-CA). Participants were reassessed at week 11 by assessors (T.D., T.T.) who were blind to condition using CAPS-CA with young people and ADIS-CA with

parents. Blind assessors did not deliver treatment and were not involved in any other aspect of the trial (e.g., recruitment, supervision, administration). Participants who received CBT were also assessed at 6 months posttreatment, using the same semistructured diagnostic interviews.

Data Analysis

Independent t tests and χ^2 tests were used to examine differences between those who responded to symptom monitoring and those who did not. A series of parametric and nonparametric tests was used to detect differences in background characteristics and initial symptom severity levels between CBT and WL. Multivariate analyses of covariance (MANCOVAs) were carried out on post-CBT/WL data to detect any differences between the groups at week 11, controlling for initial symptom or disability levels. Repeated-measures multivariate analyses of variance (MANOVAs) were used to detect pre/post changes in symptoms and disability in the CBT group (including 6-month follow-up data) and in the WL group. All of the analyses were intent to treat. For categorical (diagnostic) data, χ^2 tests were carried out. Controlled and uncontrolled effect sizes are reported on the main outcome measures (Cohen, 1988). Mediation analysis used bootstrap procedures appropriate for small samples (Preacher and Hayes, 2004) to test the magnitude of any indirect effects as well as the more traditional series of regression analyses recommended by Baron and Kenny (1986).

RESULTS

Patient Flow

Figure 1 illustrates patient flow. Between March 2002 and February 2005, 125 young people were assessed for possible inclusion in the trial. Fifty-four were eligible, of whom 38 (70%) consented to take part. Those who refused to take part did not differ from those who consented in terms of age, gender, type of trauma, time since exposure, or on any of the self-report symptom measures (all t values <0.80 , all p values $>.37$). However, compared with those who refused to take part, participants showed greater impairment in functioning (child-rated disability, $t_{49} = 2.45$, $p < .05$; parent-rated disability, $t_{47} = 2.07$, $p < .05$). Of the 38 participants who started symptom monitoring, 36 were reassessed, of whom 9 no longer met criteria for PTSD (and therefore took no further part in the trial), and 3 met PTSD criteria but declined treatment. The remaining 24 children were randomly allocated to either CBT ($n = 12$) or WL ($n = 12$). No patient dropped out of CBT, and so 12 were reassessed after treatment and at 6 months. No patient dropped out of WL, and thus 12 participants on the WL were also reassessed at the end of the waiting period.

Response to Symptom Monitoring

Of the 38 participants who started symptom monitoring, 9 (24%) no longer met criteria for PTSD at the end of monitoring ("responders"). Responders and nonresponders (those still with PTSD) did not differ on the majority of baseline variables. However, responders had less severe initial PTSD symptoms (CPSS scores; $t_{33} = 2.20, p < .05$) and better initial functioning ($t_{34} = 2.30, p < .05$) and were less likely to have a comorbid diagnosis at initial assessment ($\chi^2 = 10.3, df = 1, n = 36, p < .01$) than nonresponders. The magnitude of changes in PTSD symptoms (CPSS scores) was greater in responders compared with nonresponders, as indicated by a significant group \times time interaction ($F_{1,33} = 13.61, p < .005$) using repeated-measures ANOVA.

Randomized Controlled Trial

Pre-CBT/WL Comparisons. The CBT and WL groups were well matched on salient background characteristics, with no significant differences on variables assessed at RCT entry (Tables 1 and 2).

Effects of CBT versus WL

PTSD Symptoms. At posttreatment, MANCOVA (with initial PTSD symptom severity scores on all three measures as covariates) revealed that the CBT group scored significantly lower than WL for measures of PTSD symptomatology, on the CPSS, RIES, and CAPS ($F_{3,16} = 15.8, p < .0005$) (Fig. 2). Univariate statistics were also significant for each measure, all in favor of CBT (Table 2). In the CBT group repeated-measures MANOVA revealed significant pre-/post-treatment improvement ($F_{3,9} = 35.4, p < .0005$), with significant univariate statistics for each PTSD measure (CPSS: $F_{1,11} = 81.2, p < .0005$; RIES: $F_{1,11} = 122.7, p < .0005$; CAPS: $F_{1,11} = 75.0, p < .0005$). In the WL group repeated-measures MANOVA also revealed significant improvement over time on PTSD measures ($F_{3,8} = 4.4, p < .05$). Univariate tests showed significant pre-/posttreatment improvement in RIES scores ($F_{1,10} = 6.1, p < .05$) and CAPS scores ($F_{1,10} = 13.3, p < .005$), but nonsignificant change in CPSS scores ($F_{1,10} = 4.2, \text{not significant}$).

Associated Psychopathology (Anxiety and Depression). At posttreatment, MANCOVA (with initial anxiety and depression symptom severity scores as covariates)

revealed that the CBT group scored significantly lower than WL on measures of anxiety and depression ($F_{2,17} = 9.0, p < .005$). Univariate statistics were also significant for each measure, both in favor of CBT (Table 2). In the CBT group repeated-measures MANOVA revealed significant pre-/posttreatment improvements in depression and anxiety ($F_{2,9} = 23.6, p < .0005$), with significant univariate tests for each measure (DSRS: $F_{1,10} = 43.0, p < .0005$; RCMAS: $F_{1,10} = 21.3, p < .005$). In the WL group the multivariate effect was not significant ($F < 1$).

Impact on Functioning. At posttreatment, MANCOVA (with initial impact on functioning scores as covariates) revealed significantly better functioning in the CBT group compared to WL for measures of impact on functioning rated by the child, parent, and assessor ($F_{3,15} = 5.3, p < .05$). Univariate tests were also significant for each measure, indicating better functioning in the CBT group (Table 2). In the CBT group repeated-measures MANOVA revealed significant pre-/posttreatment improvements in functioning ($F_{3,7} = 12.5, p < .005$), with significant univariate statistics for child-rated disability ($F_{1,9} = 22.6, p < .005$), parent-rated disability ($F_{1,9} = 25.9, p < .005$), and assessor-rated disability ($F_{1,9} = 18.6, p < .005$). In the WL group repeated-measures MANOVA to detect pre-/posttreatment changes over time functioning was not significant ($F_{3,9} = 1.5, \text{not significant}$).

Diagnoses. Categorical analyses with respect to diagnostic status also revealed significant differences between the CBT and WL groups, with 11 of 12 (92%) young people in the CBT group free of PTSD diagnosis posttreatment; and 5 of 12 (42%) young people in the WL group free of PTSD diagnosis at the end of the waiting period ($\chi^2 = 6.8, df = 1, n = 24, p < .01$).

Treatment Effect Sizes

Controlled (between group) and uncontrolled (within group) effect sizes were calculated for the main outcome measures of continuous PTSD symptoms on the CPSS, RIES, and CAPS. Conventional interpretations of Cohen's effect sizes are as follows: between 0.2 and 0.5 is a small effect size, between 0.5 and 0.8 is a medium effect size, and >0.8 is a large effect size (Cohen, 1988). For CPSS scores the uncontrolled effect size for CBT was 3.43, the uncontrolled effect size for WL was 0.27, and the controlled effect size was 2.48. For RIES scores the uncontrolled effect size for

TABLE 2
Outcome Measures at Each Assessment

Assessment	CBT (<i>n</i> = 12)		WL (<i>n</i> = 12)		Group Effect ^a
	Mean	SD	Mean	SD	
CPSS					
Pre	28.1	8.8	28.3	10.5	$F_{1,22} = 0.01$
Post	3.0	5.4	25.25	11.5	$F_{1,18} = 48.3^{***}$
6-mo FU	2.3	2.9			
C-RIES					
Pre	47.5	11.5	41.6 ^b	11.7	$F_{1,21} = 1.5$
Post	8.5	9.4	35.3	14.5	$F_{1,18} = 36.8^{***}$
6-mo FU	6.2	7.0			
CAPS					
Pre	60.9	9.6	54.7	14.6	$F_{1,22} = 1.5$
Post	12.0	17.4	40.3	18.3	$F_{1,18} = 20.2^{**}$
6-mo FU	6.8	7.6			
DSRS					
Pre	18.3	5.2	13.9 ^b	5.6	$F_{1,21} = 3.7$
Post	8.0	8.7	13.3	5.4	$F_{1,18} = 19.1^{***}$
6-mo FU	6.3	5.2			
RCMAS					
Pre	19.8	5.6	16.3 ^b	5.7	$F_{1,21} = 2.3$
Post	7.4 ^b	9.2	16.5	7.3	$F_{1,18} = 14.3^{**}$
6-mo FU	6.2 ^b	7.4			
Child-rated disability (CAPS)					
Pre	6.3	1.6	6.9	2.6	$F_{1,22} = .44$
Post	1.6	2.0	5.8	2.9	$F_{1,17} = 9.5^*$
6-mo FU	0.8	1.7			
Parent-rated disability (ADIS)					
Pre	5.2 ^b	1.7	5.3	1.4	$F_{1,21} = .06$
Post	1.0 ^b	1.4	4.4	2.4	$F_{1,17} = 15.7^{**}$
6-mo FU	1.1 ^b	1.3			
Assessor-rated disability (CAPS)					
Pre	2.5	0.5	2.2	0.4	$F_{1,22} = 3.1$
Post	0.8	0.8	1.9	0.8	$F_{1,17} = 7.5^*$
6-mo FU	0.8	1.1			

Note: CBT = cognitive-behavioral therapy; WL = wait list; Pre = pre-treatment; Post = post-treatment; FU = follow-up; CPSS = Child PTSD Symptom Scale; C-RIES = Children's Revised Impact of Event Scale; CAPS = Clinician Administered PTSD Scale; DSRS = Depression Self-Rating Scale; RCMAS = Revised Children's Manifest Anxiety Scale; ADIS = Anxiety Disorders Interview Schedule.

^a At pretreatment, 1-way analysis of variance; at posttreatment, 1-way analysis of covariance with pretreatment scores as covariates.

^b *n* = 11.

* $p < .05$; ** $p < .005$; *** $p < .001$.

CBT was 3.71, the uncontrolled effect size for WL was 0.46, and the controlled effect size was 2.20. For CAPS-CA scores the uncontrolled effect size for CBT was 3.47, the uncontrolled effect size for WL was 0.44, and the controlled effect size was 1.59.

Follow-up (6 Months)

Table 2 shows that treatment gains were well maintained at 6-month follow-up. All 12 of the CBT

group had lost their PTSD diagnosis at follow-up and consequently remained very much improved compared to pretreatment on continuous measures of PTSD (CPSS, RIES, CAPS-CA total, all p values $< .0005$), associated psychopathology (DSRS, RCMAS, both p values $< .005$), and impact on functioning (child-rated, assessor-rated, and parent-rated, all p values $< .005$). Between posttreatment and follow-up, the direction of change on all symptom and functioning measures

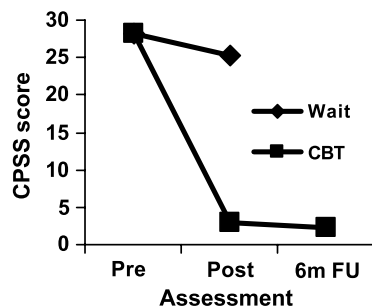


Fig. 2 Posttraumatic stress disorder symptoms. CPSS = Child Posttraumatic Stress Scale; CBT = cognitive-behavioral therapy; Wait = waitlist control condition.

was toward further improvement, albeit statistically non-significant (F values <3.3 , p values $>.10$; Table 2).

Mediation Analysis

The Pearson correlation coefficient between changes in PTSD symptoms (CAPS-CA) and changes in appraisals (C-PTCI) after CBT was large and significant ($r_{10} = 0.86$, $p < .005$). To test the hypothesis that the effect of CBT (versus WL) on PTSD symptom change was mediated by change in maladaptive appraisals, we applied the criteria of Baron and Kenny (1986). First, there was a significant effect of therapy both on changes in PTSD symptoms ($\beta = .74$, $p < .001$) and on changes in misappraisals ($\beta = .64$, $p < .005$). There was also a significant effect of changes in misappraisals on changes in PTSD symptoms ($\beta = .81$, $p < .001$). Critically, after controlling for the effect of changes in misappraisals, the direct effect of therapy on changes in PTSD symptoms was reduced ($\beta = .38$, $p < .05$). This attenuation in the magnitude of the direct effect of therapy on changes in symptoms when changes in misappraisals are taken into account indicates a significant mediating role for changes in misappraisals. To test whether this mediation effect was itself statistically significant, we employed a bootstrapping procedure designed for small samples (Preacher and Hayes, 2004). This procedure provides a formal test of the hypothesis that the effect of CBT on changing symptoms is operating indirectly via changing appraisals. The magnitude of this indirect (mediation) effect (estimated using 3,000 bootstrap resamples, with replacement) was indeed significant, (bootstrap coefficient = 19.08, SE 7.91), with a 95% confidence interval of 5.40 to 36.16 (significance is indicated by the 95% confidence interval not crossing zero).

DISCUSSION

The main findings of this study are summarized as follows. First, following a 4-week symptom monitoring period, 24% of young people with a primary diagnosis of PTSD improved such that they no longer met criteria for the disorder. Second, relative to a WL control condition, trauma-focused CBT resulted in significant reductions in symptoms of PTSD, depression, and anxiety; significantly greater recovery from PTSD (92% compared with 42% in the WL group); and significant improvement in functioning. The treatment was acceptable to young people and families in that no patient dropped out and there were no adverse effects of CBT. In particular, although the treatment includes discussing highly traumatic events, no patient showed an overall deterioration in PTSD symptoms. The posttreatment gains of CBT were maintained across patients at 6-month follow-up. To our knowledge, this is the first RCT to demonstrate the effectiveness of individual CBT for young people who have developed PTSD following a variety of traumatic events such as motor vehicle accidents, assaults, and witnessing violence. Finally, changes in PTSD symptoms as a function of CBT appeared to be mediated by changes in maladaptive appraisals about the trauma and its aftermath, as predicted by cognitive theories of PTSD (Dalgleish, 2004; Ehlers and Clark, 2000). As far as we are aware, this is the first demonstration that treatment gains following CBT for PTSD are mediated by changes in cognitive variables and one of a small number of such demonstrations in the CBT literature as a whole (see Teasdale et al., 2001).

The rate of improvement following the symptom-monitoring procedure was unexpectedly high, being around twice that found in adult studies of symptom monitoring (Ehlers et al., 2003a; Tarrier et al., 1999). Children who were least impaired initially (in terms of PTSD symptoms, impact on functioning, and comorbidity) were more likely to respond to the procedure. The mechanism underlying improvement during this phase is not clear. It may represent spontaneous remission over time; alternatively, symptom monitoring itself may have had a therapeutic effect. This hypothesis will be tested in future research, using a monitoring versus no monitoring comparison design and incorporating more detailed analysis of the content of self-monitoring. Irrespective of the underlying mechanism(s),

clinical use of this sort of symptom-monitoring procedure is indicated because for a substantial minority of young people it appears to provide a low-cost intervention, requiring little therapist time or training. For example, symptom-monitoring procedures may be usefully incorporated into routine clinical practice as part of a stepped care approach for children and young people who present with mild PTSD symptoms and little impairment in functioning.

Treatment was relatively brief in the present trial, with fewer sessions on average (median number of sessions, 9) compared to previously reported group CBT protocols (18 sessions, March et al., 1998; 11 sessions, Stein et al., 2003). Treatment components also differed somewhat. For example, relaxation training was not used. Formal relaxation training does not therefore appear necessary for therapeutic effect or for patients' acceptance of treatment. Some form of exposure to the trauma memory (imaginal exposure through talking, writing, or drawing and in vivo exposure) was used in all cases and was closely integrated with cognitive restructuring aimed at modifying misappraisals of the trauma and symptoms (see Grey et al., 2002 for a detailed clinical account of this procedure with adults). As predicted by the cognitive model of PTSD on which treatment was based (Ehlers and Clark, 2000), mediation analysis suggested that the effect of CBT on PTSD symptoms was partially mediated by changes in these trauma-related misappraisals. The implication is that identifying and modifying the sorts of misappraisals that have been shown to maintain PTSD in children (Ehlers et al., 2003b) is an important active component of therapy.

The controlled posttreatment effect size for PTSD symptoms of 2.48 (based on the CPSS scores) compares favorably with that of 1.08 reported in the previous WL-controlled RCT for group CBT using the same self-report measure with young participants (Stein et al., 2003). It is possible that the current individual treatment format allows for more intensive CBT, resulting in more substantial clinical improvement. The additional procedures used in this particular form of CBT (e.g., cognitive restructuring followed by integration of restructuring with reliving, stimulus discrimination with respect to traumatic reminders, behavioral experiments) may also have been helpful. Current results showing significant improvement in self-reported depression when PTSD is targeted are

also consistent with the findings of Stein et al. (2003). Current findings of improvement in self-reported general anxiety following trauma-focused CBT have not been reported previously in an RCT for this population, but they are consistent with findings from the quasiexperimental evaluation of group CBT for pediatric PTSD of March et al. (1998). The suggestion from this and previous studies is that clinical levels of symptoms of depression and anxiety (and/or comorbid diagnoses) in young people exposed to trauma may often be secondary to PTSD (Bolton et al., 2000) and that treatment of PTSD first is indicated because it is likely to lead to symptom improvement in other domains. This suggestion requires testing in further large-scale treatment evaluation studies with broad inclusion criteria.

The WL group also improved significantly over time on two measures of PTSD symptoms, although none of them had received treatment (from elsewhere) during the waiting period. Forty-two percent of participants in the WL condition no longer met criteria for a diagnosis of PTSD at the end of the waiting period. Such improvement in the untreated control group is broadly consistent with previous child (Stein et al., 2003) and adult (e.g., Ehlers et al., 2003a) RCTs and may be due to spontaneous recovery over time and/or to the therapeutic effect of repeated assessments. If this improvement was due to spontaneous recovery over time, then we would expect WL participants' recovery to be inversely related to time elapsed since the trauma, but the study was insufficiently powered to test this hypothesis. However, prospective follow-up studies of trauma-exposed young people have found that there is substantial natural recovery over the first 6 months posttrauma (Meiser-Stedman et al., 2005), and early intervention studies with adults (Mayou et al., 2000) and children (Stallard et al., 2006) have highlighted the need to include an untreated comparison group to control for this natural recovery. Indeed, because natural recovery can be substantial, it is possible that a treatment that is associated with significant pre-/posttreatment improvement may be ineffective or may impede natural recovery (see Ehlers and Clark 2003). Future RCTs of treatments for pediatric PTSD would benefit from comparing trauma-focused CBT with other active interventions. In addition, given the current finding of substantial improvement in the WL control group, it appears important to also include a delayed

treatment control group in future trials so that possible harmful effects of therapy can be detected and so as not to overestimate the apparent effect of therapy.

Limitations

A limitation of the present trial was the relatively small number of participants who eventually entered treatment, despite initial recruitment into the trial exceeding the level indicated by our power calculations. This was due to the unexpectedly good clinical response to symptom monitoring. The number of participants who continued to the second (RCT) stage of the study was therefore slightly fewer than had been indicated by our initial power calculations. However, this was offset by the fact that the effect sizes for CBT were higher than we anticipated, ensuring that none of the analyses relating to our a priori hypotheses were underpowered. Despite the relatively small numbers in the study, inclusion criteria were deliberately broad (e.g., participants with comorbid diagnoses were included) and initial symptom severity levels were comparable to those of the participants in Stein et al. (2003). Nevertheless, replication with a larger sample is indicated. Although about one third of participants reported previous exposure to trauma, the extent to which this intervention is effective for young people presenting with PTSD symptoms relating to multiple traumatic events is unknown. Some adaptations to the protocol (including for example additional sessions to process multiple trauma memories) may be needed for youths presenting with PTSD following multiple exposure, and this will be tested in a future trial.

Clinical Implications

There are two broad clinical implications. First, results suggest that structured symptom monitoring, as part of a stepped-care approach, may be indicated for young people who present with relatively mild symptoms and little comorbidity or impairment in functioning. Second, the study provides empirical support for the efficacy of individual, short-term, trauma-focused CBT for the treatment of pediatric PTSD that has arisen as a result of a variety of relatively common single-incident traumatic events. Further RCTs with larger samples, including WL and supportive counseling control groups, would allow identification of specific therapy components and treatment response predictors. Finally, evaluation of the effective-

ness of the protocol in regular clinical practice (in contrast to specialist university-based clinics) is needed to test the generalizability of the present positive findings.

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Legal and Ethical Considerations: Risks and Benefits of Postpartum Depression Screening at Well-Child Visits Linda H. Chaudron, MD, MS, Peter G. Szilagyi, MD, MPH, Amy T. Campbell, JD, MBE, Kyle O. Mounts, MD, Thomas K. McInerney, MD

Pediatric professionals are being asked to provide an increasing array of services during well-child visits, including screening for psychosocial and family issues that may directly or indirectly affect their pediatric patients. One such service is routine screening for postpartum depression at pediatric visits. Postpartum depression is an example of a parental condition that can have serious negative effects for the child. Because it is a maternal condition, it raises a host of ethical and legal questions about the boundaries of pediatric care and the pediatric provider’s responsibility and liability. In this article we discuss the ethical and legal considerations of, and outline the risks of screening or not screening for, postpartum depression at pediatric visits. We make recommendations for pediatric provider education and for the roles of national professional organizations in guiding the process of defining the boundaries of pediatric care. **Pediatrics** 2007;119:123–128.