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The Role of Anxiety Control and Treatment Implications of Informant Agreement on Child PTSD Symptoms

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Abstract

Objective—To examine parent and child agreement of child posttraumatic stress disorder (PTSD) symptoms pre- and posttreatment, as well as potential moderators of agreement including treatment responder status, child anxiety control, and parent self-reported PTSD symptoms.

Method—We examined child self-reported and parent-reported child PTSD symptoms from the Diagnostic Interview Schedule for Children. Of the 141 parent–child pairs, the mean age of children was 12.72 ($SD = 3.40$), 53 percent were female, and 54 percent were Black. A subsample of participants ($n = 47$) was assessed after completion of a cognitive behavioral therapy treatment for PTSD.

Results—Moderate levels of agreement were found at baseline, though Criterion D (increased arousal) symptoms had lower levels of agreement than the other symptom clusters. Symptom agreement was lower at posttreatment. Treatment responders had higher levels of baseline informant agreement than treatment non-responders. Child perceived anxiety control significantly moderated informant agreement, such that pairs with children who had high levels of perceived control of their anxiety had lower PTSD symptom agreement where children reported lower symptoms relative to their parents. Contrary to expectations, parent self-reported PTSD did not moderate parent–child symptom agreement.

Conclusion—Factors associated with higher parent–child agreement of child PTSD symptoms were being a PTSD treatment responder and children with lower perceived anxiety control. These findings have potential implications for determining those who may benefit from greater symptom monitoring over the course of intervention and potential alternative intervention approaches.

Keywords

informant agreement; informant discrepancies; posttraumatic stress disorder; anxiety control

In assessing child psychopathology, best practice recommendations have been multi-method and multi-informant when possible (Richters, 1992). Clinically, this consists of obtaining both parent and child reports. Agreement between parent and child reports of child psychopathology, or rather the lack of agreement, therefore has received considerable

interest, as determining the most accurate source for measuring symptoms and severity of symptom impairment aids in assessment, diagnosis, and treatment (De Los Reyes, Thomas, Goodman, & Kunder, 2013). The low correlation between various informants who report on child psychopathology is well documented (Achenbach, McConaughy, & Howell, 1987; De Los Reyes & Kazdin, 2004; De Los Reyes et al., 2015). These informant discrepancies call into question the accuracy of specific reporters. However, they may provide valuable information rather than being simply considered “error” (De Los Reyes & Kazdin, 2005), consistent with psychometric models of multiple measures of the same construct.

The limited data on parent–child discrepancies in regards to traumatic stress are consistent with the less-than-perfect agreement found in other syndromes. Many posttraumatic stress disorder (PTSD) symptoms are more abstract and difficult to detect compared to other disorders, including internalizing disorders (Scheeringa, 2011), as some PTSD symptoms are transient and present only in response to triggered cues in the environment. Additionally, flashbacks are unique to PTSD, and may present a challenge for informants of child behavior, as informant discrepancies are influenced by the degree to which symptoms are “observable” (De Los Reyes et al., 2015). Thus, it might be expected that informant discrepancies would be greater in PTSD compared to other disorders. Following a mass motor vehicle accident involving child ages 7-12, both children and parents completed the Child Posttraumatic Stress Reaction Index, based on the child's perceived reactions to the traumatic event (Dyb, Holen, Braenne, Indredavik, & Aarseth, 2003). At the initial assessment, five weeks after the accident, children reported significantly more PTSD symptoms than those reported by parents, as parent-reported child total scores were approximately half that of those provided by children ($M[SE] = 20.4 [13.3]$ vs. $10.6 [4.0]$, respectively), though no significant differences were found at a second assessment six-months later. This change appeared to be driven completely by a reduction in symptoms reported by children, as PTSD symptom report by parents remained consistent across both time points. This evidence of parent “underreporting” of child PTSD scores relative to the child's own report of their PTSD symptoms, at least immediately following the trauma, is consistent with a review by Vogel and Vernberg (1993), which found that children report higher levels of distress following trauma than parents report.

Scheeringa, Wright, Hunt, and Zeanah (2006), using diagnostic interviews, gathered child and parent-reported child PTSD diagnoses on 12-18 year-old children following motor vehicle accidents and other severe accidental injuries. While 8% of adolescents endorsed a PTSD diagnosis, and 4% of parents endorsed a PTSD diagnosis, when the adolescent- and parent-reports of symptoms were combined, such that symptom endorsement by either informant was counted toward the PTSD algorithm, the prevalence of PTSD diagnosis jumped to 38%. This indicates that multi-informant data may produce widely different estimates of PTSD, which merits further study with regards to best practice. Meiser-Stedman, Smith, Glucksman, Yule, and Dalgleish (2008), also using structured diagnostic interviews gathered child- and parent-reported child PTSD diagnoses without the duration requirement on 7-10 year-old children following motor vehicle accidents showed higher prevalence rates reported by parents compared to children. Two to four weeks after the accident, 4% of children endorsed a PTSD diagnosis whereas 23% of parents endorsed a PTSD diagnosis. At six months after the accident, the gap closed to 2% and 13%

respectively. Yet another study of acute stress response demonstrated that rates of endorsement from parents and children varied based on the specific symptom examined (Kassam-Adams, García-España, Miller, & Winston, 2006).

These types of longitudinal studies provide evidence that parent–child discrepancies can change over time, and additionally, that discrepancies at one time point may predict outcomes at a later time point. In a longitudinal study of 1,875 individuals spanning from age 4 to 40 years, including seven assessment waves and multiple informants, parent–child ratings were similarly correlated across different age groupings and symptom domains (van der Ende, Verhulst, & Tiemeier, 2012). Among 15- to 18-year-olds, initial parent–child symptom discrepancies, particularly when children reported higher levels of symptoms than parents, predicted numerous negative outcomes four years later (Ferdinand, van der Ende, & Verhulst, 2004). It may be that discrepancies themselves are clinically meaningful. For example, mother–child discrepancies of parental monitoring, specifically when mothers reported higher levels of parental monitoring relative to their child, predicted higher child delinquency two years later, even after controlling for baseline characteristics (De Los Reyes, Goodman, Kliewer, & Reid-Quñones, 2010).

In the context of treatment, baseline informant discrepancies may have predictive utility regarding treatment response. Using standardized difference scores to assess child self-report and parent-report of child social phobia symptoms from a multi-site randomized controlled trial, greater baseline parent–child discrepancies predicted treatment non-response (De Los Reyes, Alfano, & Beidel, 2010). Children who were non-responders tended to rate themselves relatively lower on anxiety relative to their parents at pretreatment (see Kendall, Panichelli-Mindel, Sugarman, & Callahan, 1997). These studies pointed to the potential value of parent–child discrepancies, but lacked explanatory variables that could account for the discrepancies.

It has been suggested that one explanation for discrepancy is that parents are less attuned to children's PTSD symptoms (Oransky, Hahn, & Stover, 2013). If this were true, it might be expected that discrepancies diminish over time as parents become more attuned, and indeed, two prospective studies have documented this convergence (Meiser-Stedman, Smith, Glucksman, Yule, & Dalgleish, 2007; Schreier, Ladakakos, Morabito, Chapman, & Knudson, 2005). There has been a considerable focus on parents' own traumatic stress reactions as a potential explanation for limited attunement and inflated ratings of their children's symptoms compared to child self-report (e.g., Ghesquire et al., 2008; Ingerski, Shaw, Gray, & Janicke, 2010; Shemesh et al., 2005). Limited attunement may, however, be explained by individual differences within children. Anxiety control, the perception that one can control emotional reactions and frightening events, is an individual difference that may be relevant to reporting of one's own psychopathology. For PTSD symptoms specifically, two studies (with a total of 321 participants, all of whom were adults) found a moderate negative association between anxiety control and PTSD ($r = -.27$, 95% CI $[-.17, -.37]$) (Gallagher, Bentley, & Barlow, 2014).

In the present study, our goal was to advance the understanding of parent–child discrepancies in PTSD symptom reporting by collecting symptom data with semi-structured

diagnostic interviews, examining the contribution of child perceived anxiety control and parent self-reported PTSD symptoms, and exploring these relationships before and after treatment. Aim 1 was to characterize parent–child discrepancies in a trauma-exposed, treatment-seeking sample. We hypothesized moderate positive associations would be found between child self-reported and parent-reported child PTSD symptoms. An exploratory hypothesis was to examine PTSD symptom clusters, as there is mixed evidence regarding agreements by PTSD symptom domains (Charuvastra, Goldfarb, Petkova, & Cloitre, 2010), with one study indicating greatest agreement on the increased arousal symptom cluster (Schreier et al., 2005) and another found that re-experiencing symptoms had the highest parent–child agreement (Stover, Hahn, Im, & Berkowitz, 2010). We examined this aim both at (A) baseline (i.e., pretreatment) in the full sample of trauma-exposed children and their parents, and (B) at posttreatment for the subset of participants who were selected for and completed treatment.

Aim 2 was to examine whether baseline discrepancies predicted treatment responder status (Aim 2.A). Consistent with prior work (De Los Reyes, Alfano, et al., 2010), we expected that greater discrepancies at baseline would predict poorer treatment response. We also examined whether treatment responder status was associated with posttreatment parent–child symptom agreement (Aim 2.B). Aim 3 was to identify potential sources of baseline discrepancies. In order to address this aim, two specific measures were examined as moderators of informant agreement: (A) the child's anxiety control, as greater perceived control of anxiety may be associated with lower parent–child symptom agreement; and (B) parent self-reported PTSD symptoms, given that parent psychopathology may inflate perceptions of the child's symptoms and functioning.

Method

Participants

The participants were recruited for a triple-blind, placebo-controlled, randomized trial of cognitive behavior therapy with adjunctive D-cycloserine (CBT + DCS) versus CBT with placebo (CBT + placebo) (Scheeringa & Weems, 2014). Researchers tried to contact 644 potential participants: 30% were referred by other professionals (clinicians, schools, and social service agencies that were made aware of the project), 14% referred themselves from radio and television advertisements, and 56% were from a Level I Trauma Center registry. Of the 644 potential participants, 243 were not eligible due to exclusion criteria, 195 could not be reached, and 206 were contacted and considered initially eligible. Of the 206 eligible, 65 declined to participate and 141 were evaluated and are included in this study. A portion of the sample ($n = 98$) passed the inclusion and exclusion criteria for the intervention. See Figure 1 for CONSORT diagram.

Inclusion criteria were (1) experienced or witnessed at least one life-threatening event; (2) age 7 through 18 years at time of enrollment; and (3) five or more PTSD symptoms plus functional impairment. Exclusion criteria were (1) head trauma with a Glasgow Coma Scale score of five or less in the emergency room; (2) moderate mental retardation, autistic disorder and related pervasive developmental disorders, blindness, deafness, and foreign language speaking families; (3) suicidal, homicidal, or gravely disabled; (4) children who

wish to remain in concurrent counseling outside of the study; (5) any kidney or liver ailment; (6) children with epilepsy or history of seizures; (7) and children with bipolar disorder or schizophrenia. Psychoactive medications were not an exclusion criterion as long as the dose remained stable. If the dose was changed during the study, treatment was temporarily paused for 4 weeks while the dose stabilized. Figure 1 details study randomization and attrition. The treatment protocol was completed by 47 participants (see Scheeringa & Weems, 2014, for more details).

Measures

National Institute of Mental Health Diagnostic Interview Schedule for Children-IV—(DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The modules for PTSD from the Child and Parent Versions were used to determine the number of child PTSD symptoms. Test-retest reliability with 82 children ranged from $\kappa = 0.25$ (social phobia) to 0.92 (major depressive disorder) (Shaffer et al., 2000). Despite the absence of psychometric data on the PTSD module, we chose it because the DISC is the most widely used diagnostic instrument for children and the questions map in a very straightforward way on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000) criteria. Cronbach's α in this study were 0.87 for the child ratings and 0.83 for the parent-ratings (Scheeringa & Weems, 2014). In addition to total PTSD symptoms, we also examined symptoms within each PTSD symptom cluster (i.e., Criterion B, re-experiencing symptoms; Criterion C, avoidance and numbing symptoms; and Criterion D, increased arousal symptoms).

Child PTSD Symptom Scale—(CPSS; Foa, Johnson, Feeny, & Treadwell, 2001). The CPSS is a self-administered measure that maps onto the 17 DSM-IV symptoms rated on 4-point (0-3) Likert-type scales. This yields a broader range of scores that reflect intensity and frequency, which may be more sensitive to change than the number of PTSD symptoms. This measure was administered weekly prior to the beginning of each CBT session. Previously, moderate test-retest reliability and a κ of 0.55 for PTSD diagnosis were found using this measure (Foa et al., 2001). Cronbach's α in this study was 0.90 (Scheeringa & Weems, 2014).

Responder status was determined via parent-report on the CPSS, such that those who showed a 50% reduction in symptoms from week 1 to week 12 were classified as treatment responders in concert with other treatment research (e.g., Liebowitz, Hollander, Fairbanks, & Campeas, 1990).

Anxiety Control Questionnaire - Child-Short form—(ACQ; Weems, 2005). This 10 item instrument assesses anxiety control using a Likert-type scale with responses ranging from 0 = *none* to 4 = *very very much*, with a range of possible scores from 0 to 40. Example items include: “I am able to change how much nervousness or fear I feel” and “I can make myself feel good again when bad things happen to me.” The ACQ was found to meet acceptable levels of reliability in this sample (Cronbach's $\alpha = .84$) and in prior studies had good convergent validity with anxiety measures (Weems, Silverman, Rapee, & Pina, 2003).

Davidson Self-Rating PTSD Scale—(Davidson et al., 1997). Parent self-reported PTSD symptoms were obtained via this 17 item measure with frequency anchors for each item ranging from 0 = *not at all* to 4 = *everyday*. The items mirror the 17 DSM-IV symptoms of PTSD. Good internal consistency ($r = .99$), test-retest reliability ($r = .86$), and convergent validity have been demonstrated with this measure (Davidson et al., 1997).

Treatment—Both treatment groups received individual CBT treatment with a 12-session manualized protocol, Youth PTSD Treatment (YPT), created for this study. YPT includes traditional components of CBT for pediatric trauma including psychoeducation, skill-building in identification and expression of feelings, relaxation exercises, exploration of negative thoughts, narrative processing of trauma events, graded exposure exercises in and out of the office, safety plans, and involvement of parents in every session. The YPT manual is an older-age extension of the Preschool PTSD Treatment manual that has shown good efficacy in a previous trial with young children (Scheeringa, Weems, Cohen, Amaya-Jackson, & Guthrie, 2011). Therapy was delivered by two master's level therapists trained in CBT. The dosing of DCS was seven doses of 50 mg given before sessions 5-11. The CBT + DCS group did not differ from the CBT + placebo group on PTSD, depression, or anxiety outcomes (Scheeringa & Weems, 2014), so the groups were combined for these analyses.

Procedure

The Tulane University Committee on the Use of Human Subjects approved this study. Written informed consent and assent was obtained from the caregivers and children, respectively. See Scheeringa and Weems (2014) for additional details.

Data Analysis

Data was analyzed using SPSS Version 20. To test Aim 1, we examined agreements about total PTSD symptoms and number of symptoms from each symptom cluster (re-experiencing, numbing/avoidance, and increased arousal). To test Aim 2, treatment responder status was examined as a potential moderator of parent-child PTSD symptom agreement using ordinary least squares linear regression modeling, with parent-reported child PTSD symptoms (centered), and parent-reported child PTSD symptoms squared (centered squared), and treatment responder status on Step 1, and with the interaction between parent-reported child PTSD symptoms \times treatment responder status on Step 2. To test Aim 3, analyses tested child self-reported and parent-reported child PTSD symptom measures (i.e., total PTSD symptoms, Criterion B symptoms, Criterion C symptoms, and Criterion D symptoms) with two potential moderators (i.e., child-reported ACQ and parent self-reported PTSD symptoms). Polynomial regression equations were used consistent with the approach described in Laird and Lafleur (2014) in order to test the interactions between parent-reported child PTSD symptom measures and the proposed moderators in the prediction of child self-reported PTSD symptom measures. Child self-reported PTSD symptoms were regressed on parent-reported child PTSD symptoms and the proposed moderators. Child self-reported and parent-reported child PTSD total symptoms, Criterion B symptoms, Criterion C symptoms, and Criterion D symptoms were tested in four separate regression analyses. Each model included five terms: parent-reported child PTSD symptoms, parent-reported child PTSD symptoms squared, moderator, moderator squared, and a

multiplicative interaction term computed by multiplying parent-reported child PTSD symptoms and the moderator. All predictor variables were mean-centered prior to inclusion in the regression models. In accordance with recommendations by Edwards (1994), four higher order terms (parent-reported child PTSD symptoms cubed, parent-reported child PTSD symptoms \times moderator squared, moderator \times parent-reported child PTSD symptoms squared, and moderator cubed) were included in the model when the addition significantly improved the fit of the model, otherwise these terms were constrained to be equal to zero. Interactions were plotted by calculating simple slopes at high (+1 SD), mean, and low (-1 SD) levels of the moderator.

Results

Sample Characteristics

The sample demographics and descriptive statistics are presented in Table 1.

Aim 1.A: Symptom agreements at baseline

Child self-reported and parent-reported child PTSD symptoms were significantly positively correlated for total PTSD and cluster B (re-experiencing), C (avoidance and numbing), and D (increased arousal) symptoms (Table 2). The correlations were large in magnitude for total PTSD, B, and C symptoms, whereas for Criterion D symptoms the parent-child correlation was in the low to moderate range (Cohen, 1988). Paired sample t-tests revealed that parent-reported child PTSD symptoms at baseline were higher than child self-reported total PTSD, C, and D symptoms ($ps < .02$).

When these analyses on *pretreatment* measures were conducted with only those who qualified for the study and eventually completed treatment ($n = 47$), no significant differences were found for parent-child correlations in the subsample compared to the full sample ($ps > .05$). Additionally, parent-reported child PTSD symptoms at baseline were higher than child self-reported PTSD, C, and D symptoms in the subsample ($ps > .02$).

Aim 1.B: Symptom agreements at posttreatment

The correlations between child self-reported and parent-reported child PTSD symptoms at posttreatment are presented in Table 3 for the subset of participants that completed the treatment. The correlations for child self-reported and parent-reported child total PTSD symptoms and Criterion B symptoms were significantly positively correlated and in the moderate range. The correlations between parent and child report of Criterion C and D symptoms were small and did not reach statistical significance.

Aim 2.A: Symptom agreement at baseline and treatment responder status

We tested whether treatment responders ($n = 26$) and non-responders ($n = 17$) differed in baseline symptom agreement. Models were constructed in a manner similar to those following expert recommendations (Laird & Lafleur, 2014), which are neutral to what is conceptually considered an independent and dependent variable. Treatment responder status (i.e., yes vs. no) was treated as a binary moderator variable. There was a significant interaction ($t(37) = 2.78, p = .009$) for treatment responder status and the relationship of

child self-reported and parent-reported child PTSD symptoms. As showed in Figure 2A, decomposing the interaction indicated that for treatment responders, there was a significant linear association between parent-reported child PTSD symptoms and child self-reported PTSD symptoms ($\beta = .88$, $t(23) = 2.90$, $p = .008$). However, among treatment non-responders, parent-reported child PTSD symptoms were unrelated to child self-reported PTSD symptoms ($\beta = -.31$, $t(14) = -0.83$, $p = .42$).

These analyses were repeated within each symptom cluster domain. The interaction for treatment responder status and the relationship of child self-reported and parent-reported did not reach statistical significance for B (Figure 2B), C (Figure 2C), or D symptoms (Figure 2D).

Aim 2.B: Symptom agreement at posttreatment and treatment responder status

We then tested whether parent–child symptom agreement at posttreatment was moderated by treatment responder status (50% symptom reduction per parent-report on the CPSS). No significant interactions were found for total PTSD symptoms or Criterion B, C, or D symptoms.

Aim 3.A: Child anxiety control and baseline parent–child PTSD symptom agreement

At baseline (*pretreatment*) we examined whether child anxiety control moderated the association between baseline child self-reported and parent-reported child PTSD symptoms. Significant interaction terms were found in two of the four polynomial regression models (Table 4). Parent-reported child total PTSD symptoms and Criterion C symptoms interacted with child self-reported anxiety control to predict child self-reported total PTSD symptoms and Criterion C symptoms, respectively. There was no significant interaction between parent-reported Criterion B or D symptoms with child reported anxiety control on child self-reported Criterion B or D symptoms, respectively.

Parent-reported child total PTSD symptoms were a stronger predictor of child self-reported total PTSD symptoms at low levels of ACQ (Figure 3A). A similar pattern was evident for Criterion C (Figure 3C) and D symptoms (Figure 3D). In addition to the interaction effect, there were significant main effects of child self-reported anxiety control for total PTSD, C, and D symptoms. Greater perceived anxiety control predicted lower levels of child self-reported symptoms. No association between Criterion B symptoms and ACQ was found (Figure 3B).

When these analyses on *pretreatment* measures were conducted with the subsample that completed treatment, the interaction between total PTSD symptoms with ACQ remained significant ($p = .004$) while the interaction for Criterion C symptoms and ACQ was not statistically significant ($p = .07$).

When these analyses were repeated on *posttreatment* measures, there were no significant interaction terms found in the four polynomial regression models. Strikingly, for all symptom measures there was no significant main effect of parent-reported child posttreatment symptoms predicting child self-reported posttreatment symptoms (see Figures 4A-D). There was a significant main effect of ACQ on child self-reported Criterion D

symptoms at posttreatment, with greater perceived anxiety control predicting lower levels of child self-reported symptoms. The quadratic effect of ACQ on child self-reported Criterion D symptoms was also significant.

Aim 3.B: Parent self-reported PTSD and baseline parent–child PTSD symptom agreement

Parent self-reported PTSD was positively correlated with child self-reported PTSD symptoms and moderate in effect ($r(139) = .35, p = .001$). At baseline (*pretreatment*), we examined whether parent self-reported PTSD symptoms moderated the association between baseline child self-reported and parent-reported child PTSD symptoms. No significant interaction terms were found in the four polynomial regression models when the higher order terms were excluded, given their inclusion did not improve the model fit. There was no linear effect of parent self-reported PTSD symptoms on child self-reported PTSD symptoms in the model. However, there was a significant effect for the quadratic term of parent self-reported PTSD symptoms in the model predicting child self-reported Criterion C symptoms, where the peak of child self-reported symptoms was found in moderate levels of parent self-reported PTSD symptoms.

When these analyses on *pretreatment* measures were conducted with the smaller subsample consisting of those who completed treatment, there were no significant interaction terms between parent self-reported PTSD symptoms and parent-reported child PTSD symptoms in the prediction of child self-reported PTSD symptoms.

When these analyses were repeated on *posttreatment* measures, no significant interaction terms were found for the linear terms and higher order effects were not found to add significantly to the model. There was a main effect of parent self-reported PTSD symptoms on child self-reported posttreatment Criterion B symptoms, such that higher levels of parent self-reported PTSD symptoms predicted greater levels of Criterion B symptoms after accounting for parent-reported child Criterion B symptoms.

Discussion

This was the first study to examine discrepancies between parent and child informants on child PTSD symptoms in the context of a treatment study, and the first to examine potential explanatory factors for the source of these discrepancies in this population. As parental attunement and potential bias have received considerable attention as sources of discrepancy in parent–child reporting (Ghesquire et al., 2008; Ingerski et al., 2010; Shemesh et al., 2005), several important findings from this study are worth highlighting. First, the positive correlation between parent and child informants on total PTSD symptoms measured at pretreatment appeared to be relatively stronger than those previously found in non-clinical samples. In a study of children ages 8-17, within one month of child injury, parent–child symptom agreement for acute stress disorder was examined (Kassam-Adams et al., 2006) and parent–child ratings of symptom severity were moderately positive ($r = .35$), and lower than those in the current sample ($r = .53$). Greater parent–child agreement was found in two high-risk samples (Lauth et al., 2010; Phipps, Long, Hudson, & Rai, 2005), with levels similar to those observed in the present study.

Second, the positive correlation between parent and child informants on total PTSD symptoms at pretreatment appeared to be explained by agreement on Criterion B and C symptoms (using DSM-IV criteria). This is consistent with prior studies that found agreement between informant reports to vary by the type of PTSD symptoms. Previously, moderate parent–child agreement was found for reports of nightmares, physical reactions, and sleep, but lower agreement was found for thoughts/memories and being upset by traumatic reminders (Charuvastra et al., 2010). Conversely, no agreement was found for flashbacks, irritability, concentration, and hypervigilance. Similarly, poor diagnostic agreement and “inconsistent” patterns of parent–child agreement were found in another study of PTSD, with moderate agreement for avoidance and arousal symptom criteria being met, but no association for the re-experiencing criterion (Meiser-Stedman et al., 2008). Our finding of lower agreement for Criterion D (i.e., increased arousal) symptom scores is, however, consistent with other literature that caregivers are more likely than children to report child externalizing problems, as these symptoms are more externally observed than those from the other symptom clusters. Studies that examined parent–child agreement in domains outside of PTSD may provide additional insight into potential reasons for informant discrepancies. Using a binary approach for clinical significance in a large sample of children with at least one psychiatric disorder and their parents, agreement was relatively high (69%) (Van Der Meer, Dixon, & Rose, 2008). However, when only one party reported that the child's problems were clinically significant, it was much more frequently the parent (27% of the sample) than the child (4% of the sample). Thus, it may be that some children are less willing to acknowledge their own difficulties.

Third, contrary to expectations, we found that the magnitude of parent–child symptom correlations were lower at posttreatment. Intuition would have expected that caregivers and children would become more jointly attuned to the presence of symptoms as symptoms were discussed during the course of therapy. Greater agreement might also have been expected due to the more limited range in symptom reports given the general success of the intervention (Scheeringa & Weems, 2014). This seems to be an important finding that attunement does not necessarily increase as a result of treatment, though it should be noted that only a subsample of the original trauma-exposed study participants completed the treatment and therefore were available to provide information on posttreatment symptom levels. We speculate that the parent–child discrepancy widened at posttreatment because either caregivers or youth developed different perceptions of the magnitude of symptom improvement, but this awaits empirical confirmation.

Fourth, when children perceived that they had greater control of their anxiety, there was greater discrepancy between parent and child reports of symptoms. This represents the first known child-level cognitive factor that can potentially explain informant discrepancies with caregiver report. Because children who reported high levels of perceived control of their anxiety also reported significantly lower levels of PTSD symptoms relative both to their parents and to those children with lower perceived anxiety control, it may be that children who perceived greater control under-reported their symptoms. While higher anxiety control typically is associated with lower reported symptoms in anxiety disorders (Cannon & Weems, 2010; Frala, Leen-Feldner, Blumenthal, & Barreto, 2010; Pereira, Barros, & Mendonça, 2012; Weems et al., 2003), causality is not clear. It may be that the high levels of

perceived control by children result in misperceptions of their own symptom severity, where parents may have greater insight into the child's actual symptom expression. A direction for future research would be to investigate whether perceptions of anxiety control are a cause or consequence of symptom severity.

An important negative finding was that we found no evidence that parent self-reported PTSD symptoms predicted parent-child discrepancies. Frequently, parents own symptomatology has been considered as a potential factor that may bias parent-report of child psychopathology. Indeed, several studies have linked parent self-reported PTSD symptoms with their report of their child's symptoms (e.g., Ghesquire et al., 2008; Ingerski et al., 2010; Shemesh et al., 2005), perhaps reflecting that traumatic events are often experienced by multiple members of a family. Given our findings, caution is urged before attributing informant discrepancies to parental bias.

Lastly, our finding that greater discrepancy between informants at baseline predicted treatment non-response represents the first exploration of this issue in children being treated for PTSD. In a previous study of social phobia, comparable levels of discrepancies were found between responders and non-responders at pretreatment, and informant discrepancies were still evident posttreatment only for the non-responders, which was determined by external informant judgment (De Los Reyes, Alfano, et al., 2010). Our findings suggest that pretreatment parent-child discrepancies may be a particularly sensitive indicator of treatment response for PTSD, though the use of a clinician-rated or other methods to determine treatment response may have yielded different results.

These present findings should be considered in light of important limitations. It might have been informative to try to resolve discrepancies through consensus discussions between parents and children, however our method of using separate parent and child reports more accurately reflects realistic clinical practice. Another limitation is the relatively small size of the sample that completed the treatment, resulting in different size samples available for examining pre- and posttreatment informant discrepancies. Though the subgroup that completed treatment did not differ in important demographic characteristics (i.e., age, sex, race) from those who did not, their selection to the treatment indicated more severe PTSD and potentially other unassessed differences, which may limit the ability to directly compare the findings from this subgroup to the full sample. Additionally, while we obtained child report of anxiety control, and the construct appears to be promising in examining how parents may perceive their child's symptoms and the potential functional impairment of their anxiety, we did not obtain a similar marker of the parent's perceived control that their child has over anxiety. Obtaining both parent and child reports of anxiety control may help to elucidate the association between perceptions of control and endorsements of functionally impairing anxiety and PTSD symptoms. Lastly, we were unable to determine temporally whether levels of anxiety control and parental PTSD preceded the identified traumatic event(s) experienced by the children. Identifying whether these domains change in response to the children's identified trauma may be useful in advancing our understanding of the way in which they might moderate parent-child symptom agreement. Lastly, our sample consisted of a racially and socioeconomically diverse group drawn from a metropolitan area

characterized by high levels of trauma exposure, and therefore it is unclear how the findings may generalize to other populations.

In conclusion, we sought to examine levels of parent–child PTSD symptoms agreement in a large sample of trauma-exposed children at pre- and posttreatment for PTSD. Though moderate to large positive levels of symptom agreement were found, informant agreement was moderated by two important factors: treatment responder status and child's perceived control over their anxiety. Pairs characterized as non-responders as well as those with children who reported high levels of anxiety control had lower levels of agreement where children self-reported lower levels of symptoms than parent-report of child symptoms. These findings have potential implications for determining those families that may benefit from greater symptom monitoring over the course of intervention and potential alternative intervention approaches.

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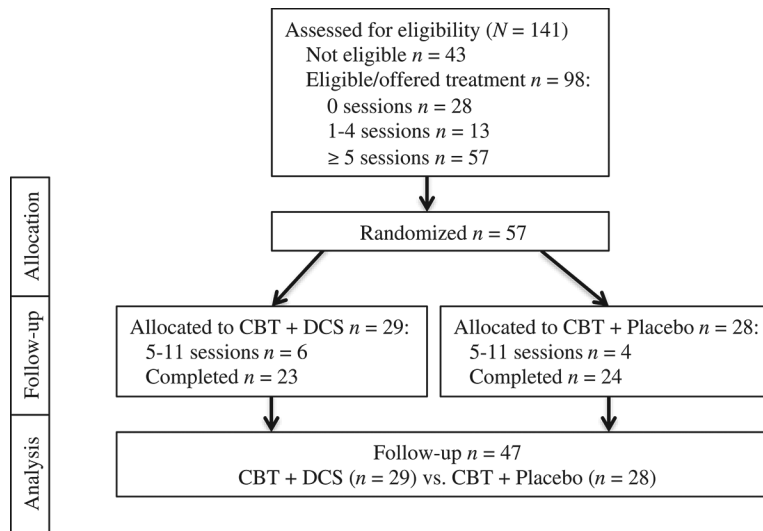


Figure 1. Flow chart of group assignment, attrition, treatment completers, and three-month follow-up assessment.

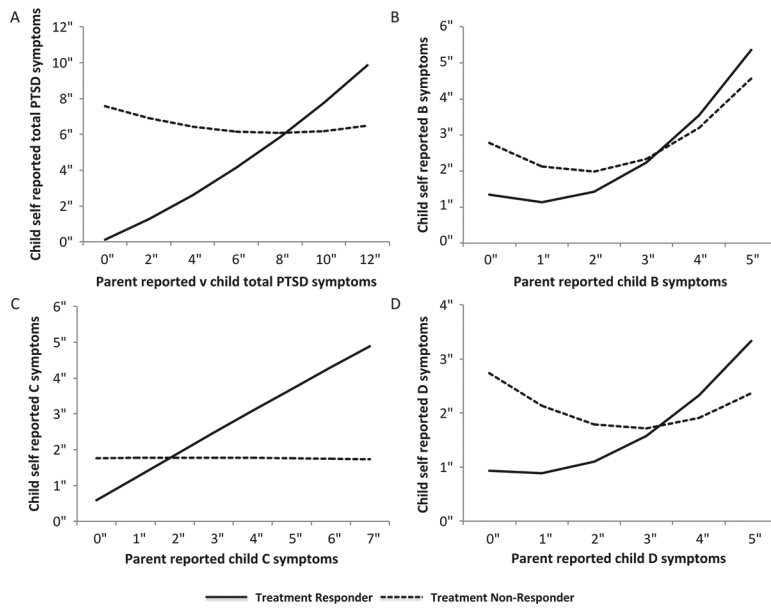


Figure 2. Treatment responder status by baseline parent-reported child PTSD in the prediction of baseline child self-reported PTSD for (A) Total PTSD symptoms, (B) Criterion B symptoms, (C) Criterion C symptoms, and (D) Criterion D symptoms. PTSD = posttraumatic stress disorder.

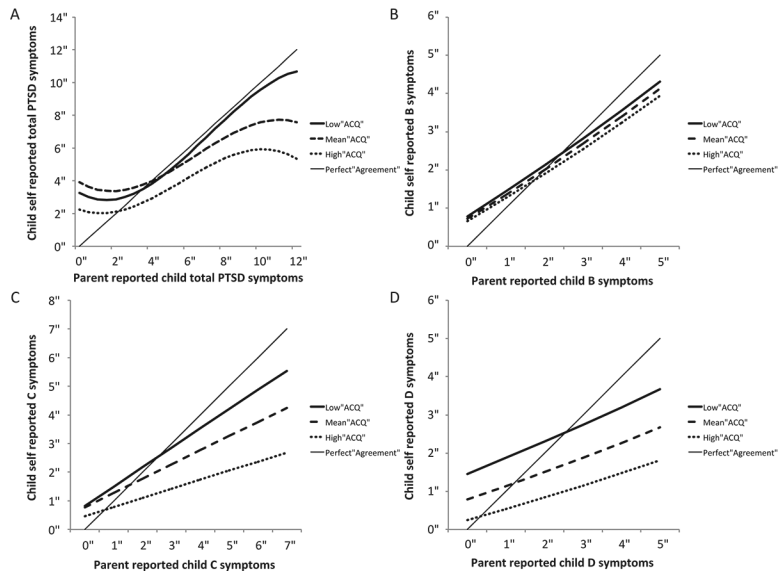


Figure 3. Anxiety control by baseline parent-reported child PTSD in the prediction of baseline child self-reported PTSD for (A) Total PTSD symptoms, (B) Criterion B symptoms, (C) Criterion C symptoms, and (D) Criterion D symptoms. PTSD = posttraumatic stress disorder. ACQ = anxiety control questionnaire. Low and High refer to values at $-1SD$ and $+1SD$ from the ACQ mean, respectively.

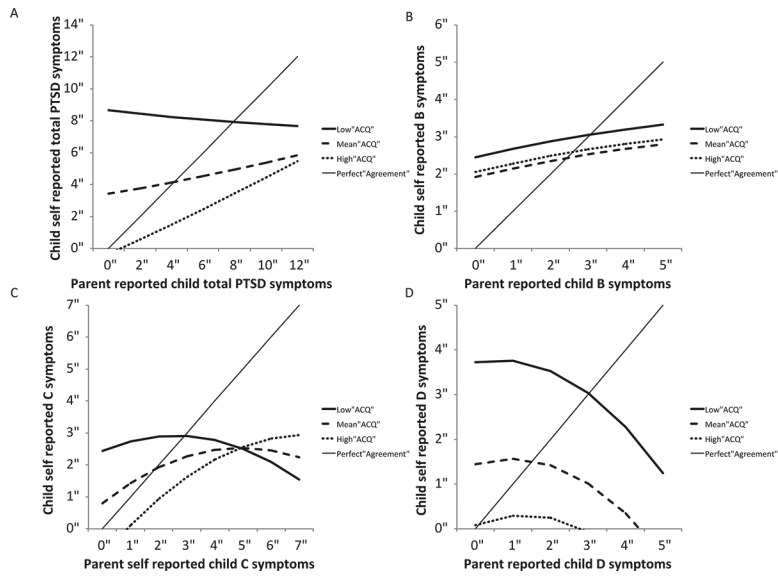


Figure 4. Anxiety control by posttreatment parent-reported child PTSD in the prediction of posttreatment child self-reported PTSD for (A) Total PTSD symptoms, (B) Criterion B symptoms, (C) Criterion C symptoms, and (D) Criterion D symptoms. PTSD = posttraumatic stress disorder. ACQ = anxiety control questionnaire. Low and High refer to values at $-1SD$ and $+1SD$ from the ACQ mean, respectively.

Table 1

Demographics and Sample Characteristics at Baseline

Variable	<i>M (SD) or % (n)</i>
Age	12.72 (3.40)
Sex (% female)	53% (74)
Race	
Black	54% (76)
White	33% (46)
Mixed	11% (15)
Other	2% (3)
Maternal age	42.37 (10.10)
Maternal education	13.71 (2.69)
Maternal employed	57% (79)
Paternal age	41.68 (8.92)
Paternal education	12.65 (2.56)
Paternal employed	70% (74)
Father lives with child	25% (32)
Step- or boyfriend lives with child	25% (32)
Child reported anxiety control	21.25 (8.13)
Parent self-report of PTSD symptoms	19.62 (16.24)
Months since last trauma	23.99 (25.38)

Note. PTSD = posttraumatic stress disorder.

Table 2

Bivariate Correlations among Child Self-report and Parent-report of Child PTSD Symptoms Obtained from the Diagnostic Interview Schedule for Children at the Baseline Assessment

	<i>M (SD)</i>	1.	2.	3.	4.	5.	6.	7.
Child self-report								
1. Total PTSD symptoms	5.34 (4.24)	1						
2. Criterion B symptoms	1.91 (1.63)	.91 ^{***}	1					
3. Criterion C symptoms	1.81 (1.53)	.91 ^{***}	.74 ^{***}	1				
4. Criterion D symptoms	1.65 (1.54)	.89 ^{***}	.69 ^{***}	.71 ^{***}	1			
Parent-report of child symptoms								
5. Total PTSD symptoms	6.14 (4.03)	.52 ^{***}	.51 ^{***}	.44 ^{***}	.43 ^{***}	1		
6. Criterion B symptoms	1.83 (1.45)	.48 ^{***}	.54 ^{***}	.39 ^{***}	.35 ^{***}	.83 ^{***}	1	
7. Criterion C symptoms	2.13 (1.77)	.51 ^{***}	.47 ^{***}	.49 ^{***}	.42 ^{***}	.85 ^{***}	.53 ^{***}	1
8. Criterion D symptoms	2.23 (1.52)	.28 ^{***}	.27 ^{***}	.19 [*]	.29 ^{***}	.84 ^{***}	.61 ^{***}	.54 ^{***}

Note. PTSD = posttraumatic stress disorder.

* $p < .05$.

*** $p < .001$.

Table 3

Bivariate Correlations among Child Self-report and Parent-report of Child PTSD Symptoms Obtained from the Diagnostic Interview Schedule for Children at the Posttreatment Assessment

	<i>M (SD)</i>	1.	2.	3.	4.	5.	6.	7.
Child self-report								
1. Total PTSD symptoms	5.57 (5.87)	1						
2. Criterion B symptoms	2.22 (2.50)	.84 ^{***}	1					
3. Criterion C symptoms	1.70 (2.38)	.75 ^{***}	.40 ^{**}	1				
4. Criterion D symptoms	1.73 (2.42)	.83 ^{***}	.60 ^{***}	.40 ^{**}	1			
Parent-report of child symptoms								
5. Total PTSD symptoms	6.62 (6.34)	.31 [*]	.44 ^{**}	-.03	.31 [*]	1		
6. Criterion B symptoms	2.31 (2.17)	.21	.42 ^{**}	-.09	.15	.88 ^{***}	1	
7. Criterion C symptoms	2.18 (2.52)	.40 ^{**}	.42 ^{**}	.14	.27 [*]	.86 ^{***}	.69 ^{***}	1
8. Criterion D symptoms	2.18 (2.65)	.18	.30 [*]	-.15	.26	.85 ^{***}	.64 ^{***}	.54 ^{***}

Note. PTSD = posttraumatic stress disorder.

* $p < .05$.

*** $p < .001$.

Table 4

Polynomial Regression Analyses Predicting Baseline Child Self-report of PTSD Symptoms from Baseline Parent-report of Child PTSD Symptoms and Baseline Child Self-reported Anxiety Control

Parameter	Child self-reported total PTSD symptoms		Child self-reported B symptoms		Child self-reported C symptoms		Child self-reported D symptoms	
	B (SE)	<i>p</i>	B (SE)	<i>p</i>	B (SE)	<i>p</i>	B (SE)	<i>p</i>
Parent-report	0.70 (0.15)	.001	0.66 (0.20)	.001	0.50 (0.11)	< .001	0.37 (0.02)	.064
Parent-report squared	0.01 (0.02)	.68	0.01 (0.08)	.87	-0.001 (0.07)	.99	0.01 (0.06)	.91
Parent-report cubed	-0.01 (0.004)	.003	-0.05 (0.04)	.28	-0.03 (0.02)	.12	-0.05 (0.04)	.22
ACQ	-.16 (0.07)	.03	-0.01 (0.03)	.67	-0.07 (0.03)	.013	-0.09 (0.03)	.002
ACQ squared	-0.01 (0.004)	.24	0.00 (0.002)	.99	-0.002 (0.001)	.24	0.001 (0.002)	.50
ACQ cubed	-0.01 (0.001)	.14	0.00 (0.00)	.63	0.00 (0.00)	.18	0.00 (0.00)	.25
Parent-report × ACQ	-.02 (0.01)	.024	-0.003 (0.12)	.77	-0.02 (0.01)	.029	-0.01 (0.01)	.48
Parent-report squared × ACQ	-0.003 (0.002)	.20	-0.01 (0.01)	.26	0.00 (0.004)	.98	-0.001 (0.01)	.92
Parent-report × ACQ squared	0.002 (0.001)	.092	0.001 (0.001)	.40	0.001 (0.001)	.12	0.001 (0.001)	.30
Model R^2	.43	< .001	.37	< .001	.37	< .001	.25	< .001
Parameter Constraints	$\chi^2(4)$	<i>p</i>	$\chi^2(4)$	<i>p</i>	$\chi^2(4)$	<i>p</i>	$\chi^2(4)$	<i>p</i>
Higher Order Terms Removed	14.28	.006	3.73	.44	5.53	.24	3.62	.46

Note. PTSD = posttraumatic stress disorder. ACQ = anxiety control questionnaire. PTSD scores were obtained from the Diagnostic Interview Schedule for Children. Higher order terms were removed when tests of parameter constraints showed that they could be removed without significantly worsening the fit of the model.