

# Social Cognition Mediates the Relation Between Attachment Schemas and Posttraumatic Stress Disorder

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**Objective:** A social–cognitive perspective on posttraumatic stress disorder (PTSD) has been proposed and posits that impaired social cognition, rooted in attachment insecurity, plays a role in the development of PTSD. Support for the role of impaired social cognition in PTSD has been found in adults, but the social–cognitive perspective on PTSD has not been examined in adolescents. This study sought to explore differences in social cognition and PTSD on the basis of attachment security, and it examined social cognition as a mediator in the relation between attachment security and PTSD and with regard to PTSD symptom change during inpatient treatment. **Method:** We recruited 142 adolescents from an inpatient psychiatric hospital, where adolescents and their parents completed assessments at admission and discharge. **Results:** Adolescents with a secure attachment demonstrated better social–cognitive skills than did those with an insecure attachment. Social cognition mediated the relation between adolescents’ maternal attachment representations and PTSD at admission across 3 self- and parent-report measures. Social cognition also mediated the relation between adolescents’ maternal attachment representations at admission and PTSD treatment outcome. **Conclusion:** This study provides the 1st support for the application of Sharp, Fonagy, and Allen’s (2012) social–cognitive perspective of PTSD to adolescents by showing a link between clinically significant symptoms of PTSD and attachment security through social–cognitive impairment. Findings indicate that improvement in PTSD during medium-stay inpatient treatment is partially driven by baseline attachment security and social–cognitive abilities, highlighting the potential of social–cognitive skills as important targets of clinical intervention among adolescents with PTSD.

**Keywords:** attachment, social cognition, PTSD, posttraumatic stress, adolescent

**Supplemental materials:** <http://dx.doi.org/10.1037/tra0000165.supp>

Posttraumatic stress disorder (PTSD) is a highly prevalent, impairing mental health problem in adolescent populations. At the broad diagnostic level, PTSD is characterized by direct and/or indirect exposure to a traumatic event (i.e., threatened death, injury, or violence to the self or others) resulting in a host of functionally impairing, trauma-related symptoms, such as intrusive recollections or reexperiencing of the event, avoidance of trauma-related stimuli, marked physiological arousal, and mood-related changes, which persist for longer than a month following the event(s) (American Psychiatric Association, 2013). In adolescents specifically, PTSD may manifest as difficulties with concentration, separation anxiety, and difficulty communicating with others about traumatic experiences (Perrin, Smith, & Yule, 2000).

PTSD is a major public health concern. National, epidemiologically based studies of adolescents (ages 12–17) have revealed prevalence rates in the United States of 6.3% for female and 3.7% for male adolescents meeting diagnostic criteria for PTSD in the past 6 months (Kilpatrick et al., 2003). One national study estimated the cumulative prevalence of PTSD through the course of childhood and adolescence to be 8% in female and 2.3% in male adolescents (Merikangas et al., 2010). PTSD is also highly prevalent among adolescents seeking inpatient psychiatric care, of which 23%–42% (average age 15) meet diagnostic criteria (Koltek, Wilkes, & Atkinson, 1998). Additionally, PTSD in adolescents can extend for several years—5 to 8 years in approximately one third of teens (Yule et al., 2000)—and is often comorbid with other disorders (Bleich, Koslowsky, Dolev, & Lerer, 1997; Lipschitz, Winegar, Hartnick, Foote, & Southwick, 1999), further highlighting PTSD as a threat to adolescent mental health, especially those in inpatient care.

A key to addressing the high prevalence of PTSD in adolescents is in understanding its etiology. Useful in this regard is Sharp, Fonagy, and Allen’s (2012) social–cognitive basis of PTSD model, which proposes a theoretical framework of the mechanisms by which a traumatic experience develops into PTSD (see Figure S1 in the online supplemental material). Social cognition includes thinking about, perceiving, representing, and understanding infor-

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This article was published Online First June 23, 2016.

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This research was funded in part through the McNair Family Foundation.

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mation in the social environment (Fiske & Taylor, 2013; Higgins & Bargh, 1987; Moskowitz & Tesser, 2005). Existing research has linked PTSD to impairments in social–cognitive processing (Benight & Bandura, 2004; E. A. Carlson, Egeland, & Sroufe, 2009) and other social impairments (e.g., E. A. Carlson et al., 2009; Foa, Hembree, Rothbaum, 2007). In their model, Sharp et al. hypothesized that early experiences with attachment figures produce attachment schemas that inform an individual’s understanding of the self and others. For instance, insecure attachments, reflecting global beliefs of the self as unworthy of care and others as unreliable caregivers (Bowlby, 1969, 1973), negatively impact the development of social cognition, which in turn impairs one’s ability to effectively process social information and reach out for needed social support when faced with a traumatic stressor, thereby contributing to vulnerability for emerging PTSD (Sharp et al., 2012). This study intended to be the first empirical test of this theoretical model in adolescents. Though the Sharp et al. model is causal—insecure attachments underlie social–cognitive deficits and later risk for PTSD—the present study examined links between these constructs within 1 month of inpatient hospitalization as a first step toward the subsequent evaluation of the full model.

Existing empirical research has provided support for the constructs linked within Sharp et al.’s (2012) model; specifically, the links between (a) insecure attachment and PTSD and (b) insecure attachment and social–cognitive impairment have been well studied. First, regarding attachment, insecure attachment has been directly related to PTSD diagnoses and symptoms in youth. Using a longitudinal design, MacDonald et al. (2008) found that insecure attachment status at 12 months predicted greater levels of PTSD symptoms (i.e., reexperiencing, avoidance) in childhood. Joubert, Webster, and Hackett (2012) also found a significant relation between insecure attachment and PTSD symptoms in maltreated adolescents. The aforementioned studies indicate that insecurely attached individuals are not only more likely to have been exposed to trauma (V. Carlson, Cicchetti, Barnett, & Braunwald, 1989; Finzi, Cohen, Sapir, & Weizman, 2000; van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999) but also are more likely to develop PTSD following exposure to trauma.

Second, relations between attachment and social cognition have been demonstrated in adolescents, with relations replicated across multiple domains of social cognition (Dykas & Cassidy, 2011). For example, Cassidy, Ziv, Mehta, and Feeny (2003) found that attachment style was related to attention to social information such that secure adolescents sought more-positive feedback than did insecure adolescents. In studies examining memory of interpersonal interactions, adolescents with insecure attachments had more negatively biased memories of interactions with parents (Feeny & Cassidy, 2003), their negative perception bias of interactions with unfamiliar peers grew stronger over time (Dykas, Woodhouse, Ehrlich, & Cassidy, 2010), and they had slower retrieval of emotional memories from childhood (Dykas & Cassidy, 2010). Multiple studies have found that adolescents with lower attachment security (dimensionally) or an insecure attachment have greater expectations of rejection, less-positive and -flexible expectations of peers, less-positive attributions of friendships, and insecure attachments to peers and romantic partners (Furman, Simon, Shaffer, & Bouchey, 2002; Granot & Mayseless, 2012; Mikulincer & Selinger, 2001; Zimmermann, 2004). Further, attachment style has been linked to adolescents’ performance on theory of mind tasks,

including emotion recognition and complex tasks consisting of inferring thoughts, beliefs, and intentions of characters in vignettes and movies (Hunfress, O’Connor, Slaughter, Target, & Fonagy, 2002; Hünefeldt, Laghi, Ortu, & Belardinelli, 2013; Vanwoerden, Kalpakci, & Sharp, 2015). In all, these findings support the notion that insecure attachment schemas are related to impaired social cognition among adolescents.

As it stands, the third component of the Sharp et al. (2012) model—the degree to which impaired social cognition relates to PTSD in adolescents—is in need of further exploration. The adult literature has revealed that individuals with PTSD perform poorly on social–cognitive tasks (e.g., Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), including tasks involving emotion recognition and responding (Lanius, Frewen, Nazarov, & McKinnon, 2014). Adults with PTSD also experience emotional numbing, which is thought to negatively affect interpersonal functioning (Cook, Riggs, Thompson, Coyne, & Sheikh, 2004). However, to date there have been few investigations of social cognition in youth with PTSD. We are aware of two studies that examined attentional biases to threatening social stimuli in children and adolescents with PTSD (Dalgleish, Moradi, Taghavi, Neshat-Doost, & Yule, 2001; Pine et al., 2005). Pine et al. (2005) found that a strong attentional bias away from threatening faces was positively associated with children’s level of PTSD, such that PTSD predicted higher degrees of attentional avoidance. Using a dot-probe task, Dalgleish and colleagues (2001) found that youth with PTSD selectively attended to socially threatening stimuli and turned away from depression-related stimuli, compared to healthy controls. Another study found social functioning among adolescents with PTSD to be significantly worse than that of healthy peers due to avoidance-type PTSD symptoms (McLean, Rosenbach, Capaldi, & Foa, 2013). Taken together, these studies highlight social biases in the maintenance of PTSD among children and adolescents while underscoring the need for further research.

Understanding the etiology of PTSD among inpatient adolescents is of particular importance. Indeed, nearly all adolescents undergoing inpatient care have been exposed to at least one traumatic event (93%: Lipschitz et al., 1999; 96.4%: Havens et al., 2012), whereas estimates of PTSD diagnosis are considerably lower (23%–42%: Koltek et al., 1998). Thus, the prevalence of trauma in inpatient samples can be assumed to be quite high, and comparisons between inpatients with and without PTSD allow for the specific examination of the etiology and correlates of PTSD, apart from the effects of trauma exposure. Research specifically exploring PTSD is essential among inpatients, in whom PTSD, rather than trauma exposure alone, confers risk for increased symptom severity, service use, psychiatric comorbidity, and medication use (Havens et al., 2012). Thus, the present study compared inpatient adolescents with clinically elevated PTSD against a heterogeneous group of inpatients, thereby studying how PTSD relates uniquely to attachment and social cognition in contrast with other forms of psychopathology.

The present study sought to provide the first empirical evaluation of Sharp et al.’s (2012) model in a sample of inpatient adolescents by (a) exploring differences in social cognition and PTSD on the basis of attachment security and (b) determining whether social cognition mediated the relation between attachment security and clinically significant PTSD, as posited in that model. Although some prior research in youth has indicated a relation

between PTSD and attentional biases, one aspect of social-cognitive functioning, little is currently known about relations between PTSD and other social-cognitive abilities. On the basis of research conducted in adults, as well as the theoretical model put forth by Sharp and colleagues, we expected that adolescents with an insecure attachment would demonstrate impaired social-cognitive abilities and be more likely to report clinically significant symptoms of PTSD. We additionally expected that the relation between attachment and PTSD would be mediated by impaired social-cognitive abilities.

It should be noted that Sharp et al.'s (2012) model posits a causal relation between early attachment insecurity, subsequent social-cognitive impairment, and susceptibility to PTSD. Although this model could not be fully evaluated due to the availability of only concurrent data on attachment and social cognition, we sought to evaluate these relations in a pre- and posttreatment (prepost) design by also examining whether social cognition (at admission) mediated the relation between attachment security and PTSD treatment outcome during inpatient care. Although this third aim cannot speak directly to the causal mechanism proposed by Sharp et al., it provides a preliminary evaluation of how attachment and social-cognitive abilities relate to PTSD across time and the degree to which social cognition underlies PTSD symptom expression.

## Method

### Participants

This study was approved by the appropriate institutional review board. Hospital patients between the ages of 12 and 17 who were fluent in English were eligible to participate. Of those approached, 19 declined, two were discharged prior to completion of the assessments, two began assessments and then revoked consent, and 20 were excluded from the study due to psychosis or intellectual disability. The sample was thereby reduced to 250 adolescents.

From this sample, all adolescents whose self- or parent-reported PTSD symptoms were in the borderline clinical range were excluded ( $n = 108$ ). These adolescents were excluded for several reasons. First, this procedure created a dichotomous PTSD outcome variable with which to explore the social-cognitive model of PTSD. Indeed, this model focuses on the emergence of clinical PTSD as a function of attachment and social cognition and highlights these factors as ones that determine whether clinically significant symptoms of PTSD will form in response to trauma. Therefore, the aims of the present study must be examined using an outcome variable that captures clinically significant PTSD rather than subclinical symptoms. Second, these exclusions allowed us to maximize group differences in a highly traumatized sample. Trauma is common among inpatient groups, and therefore trauma symptoms were expected in the self- and parent report of most participants. However, the aims of this study were not to explore PTSD symptoms but rather the emergence of clinically significant PTSD, requiring that a distinction be drawn between the great number of adolescents who have experienced trauma and trauma symptoms and those who developed clinically significant PTSD. Finally, maximizing group differences served to reduce the effects of comorbidity in a complex sample by isolating a group

for whom PTSD was a significant clinical concern and a group for whom it was not.

In sum, these exclusions resulted in a sample of 142 adolescents. Parent report data was missing for eight of these adolescents. Therefore, a sample of 142 adolescents had complete self-report data, and 134 adolescents had complete parent-report data. Analyses related to Aims 1 and 2 were based on  $n = 142$  and  $n = 134$ , depending upon the outcome measure used: Youth Self Report (YSR) and Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). Of these ( $N = 142$ ), 66.9% ( $n = 95$ ) were female, and the average age was 15.53 years ( $SD = 1.41$ ). Ethnically, 7.1% of the sample were Hispanic, and the racial breakdown was as follows: 89.9% Caucasian, 5.9% Asian, 3.4% African American, and .8% Multiracial. Self-reported PTSD symptoms (YSR) were used to create dichotomous PTSD groups ( $n = 59$ , or 41.5% of adolescents, with PTSD;  $n = 83$ , or 58.5% adolescents, without PTSD). Clinical characteristics of the sample and descriptive data on study variables are presented in Table S1 in the online supplemental material.

Analyses related to Aim 3 required discharge data in order to evaluate treatment outcome. There were 48 (33.8%) adolescents for whom discharge data were not available due to sudden decisions to discharge made by the adolescents' parents or treatment team. There were no significant differences between these 48 adolescents and those who completed discharge assessments with regard to age, sex, or psychopathology. These 48 adolescents were excluded from analyses related to the Aim 3, leaving  $n = 94$  adolescents. Of these, 70.2% ( $n = 66$ ) were female, and the average age was 15.56 years ( $SD = 1.43$ ). Ethnically, 10.0% were Hispanic, and the racial breakdown was as follows: 86.9% Caucasian, 8.3% Asian, 3.6% African American, and 1.2% Multiracial.

### Procedure

All assessments were conducted in private on the unit by doctoral clinical psychology students and trained clinical research assistants. Assessments were conducted within 1 week of admission and then again 1 week or less prior to discharge. The average length of stay on the adolescent unit in this sample was 34.18 days ( $SD = 12.74$ , range = 11–77). During this time, adolescents participated in a milieu-based, inpatient treatment emphasizing improvement of social-cognitive capacity and forming close relationships with clinicians who provide individualized attention in resolving and processing the emotional and behavioral problems adolescents face throughout the day. This treatment integrates cognitive-behavioral and family systems approaches; the primary framework is interpersonal-psychodynamic (Sharp et al., 2009).

### Measures

**Posttraumatic stress disorder.** A categorical rating of PTSD symptoms was used on the basis of self- and parent report: the Youth Self Report (YSR) and the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). The categorical rating was used in order to detect the presence of clinically significant PTSD and maximize differences between adolescents who had experienced trauma symptoms and those who had clinically significant symptoms of PTSD. Both the YSR and the CBCL are appropriate

for use with adolescents between the ages of 12 and 17 or their parents. They each contain 112 problem items, each scored on a 3-point scale: 0 (*not true*), 1 (*somewhat or sometimes true*), and 2 (*very or often true*). Of these, 13 items make up the PTSD scale. *T* scores on this scale higher than 70 made up the clinical PTSD group ( $n = 59$ ; 41.5%); scores less than 65 made up the non-PTSD group ( $n = 83$ ; 58.5%). This measure has demonstrated adequate reliability and validity (Achenbach & Rescorla, 2001).

In order to supplement analyses with the main outcome variables (YSR and CBCL PTSD subscale), we also used the PTSD scale from the Trauma Symptom Checklist (TSCC; Briere, 1996). The TSCC is a 54-item self-report measure that evaluates post-traumatic symptoms in children and adolescents. This assessment does not measure whether actual trauma has occurred. Instead, it measures potential pathological reactions in response to trauma. It therefore includes six clinical subscales, including anxiety, depression, posttraumatic stress, sexual concerns, dissociation, and anger. The frequency of each symptom is rated on a 4-point scale ranging from 0 (*never*) to 3 (*almost all of the time*). In this study, the TSCC PTSD scale was used as a continuous measure of PTSD that could bolster main analyses (i.e., YSR and CBCL).

**Attachment schemas.** The Child Attachment Interview (CAI; Target, Fonagy, Shmueli-Goetz, Data, & Schneider, 2007) is an interview-based measure used to assess attachment security. In this study, attachment schemas were operationalized as categorical ratings of attachment security on the basis of this interview. The interview consists of 15 open-ended questions that ask about attachment figures of particular importance to the child and the qualities of the people and relationship described. All CAIs were completed in private, videotaped, transcribed, and coded by clinical research assistants or doctoral students who had completed training with the measure's authors. During coding, interviews are first rated on the basis of continuous subscales: emotional openness, balance of positive and negative reference to attachment figures, use of examples, preoccupied anger, idealization, dismissal, resolution of conflicts, and overall coherence. These ratings are then used to make a categorical classification, secure versus insecure, for each adolescent's attachment representation about his or her mother. Adequate reliability and validity for the CAI was demonstrated by the authors (Shmueli-Goetz, Target, Fonagy, & Datta, 2008) and in an inpatient sample (Venta, Shmueli-Goetz, & Sharp, 2014). Interrater reliability has been established and indicates consistency between raters ( $\kappa = .64$ ; percentage agreement = 84.2%; Venta et al., 2014).

**Social cognition.** The Movie for the Assessment of Social Cognition (MASC; Dziobek, Fleck, Kalbe, et al., 2006) test is a computer-based measure of social-cognitive abilities needed to navigate social situations in daily life. To that end, each adolescent is asked to watch a short film (15 min) about four characters planning and getting together for a dinner party. As in daily life, this experience elicits emotions and mental states including anger, affection, gratefulness, jealousy, fear, ambition, embarrassment, and disgust from the characters. At 45 points throughout the film, an interviewer pauses to ask questions concerning the characters' mental states (e.g., "What is Betty feeling?" "What is Cliff thinking?"). Correct responses are scored as 1 point and added to an overall score that reflects total mentalizing—representing the adolescent's social-cognitive capacity. This task has proven a reliable and sensitive means of detecting subtle social-cognitive

deficits in adults (Dziobek et al. 2006), young adults (Smeets, Dziobek, & Wolf, 2009), and inpatient groups (Montag et al., 2010).

## Results

### Preliminary Analyses

Independent-samples *t* tests and chi-square analyses were used to compare adolescents with a secure attachment and those with an insecure attachment on the basis of age and sex in order to identify possible confounds. There were no group differences in age,  $t(df = 140) = .89$ ,  $p = .38$ , or sex,  $\chi^2(df = 1, N = 142) = .55$ ,  $p = .46$ . This procedure was repeated in order to identify PTSD group differences, and there were no group differences in age,  $t(df = 140) = 1.11$ ,  $p = .27$ , or sex,  $\chi^2(df = 1, N = 142) = .284$ ,  $p = .594$ , using self-reported PTSD on the YSR. Continuously self-reported PTSD (TSCC) was not correlated with age ( $r = -.13$ ,  $p = .13$ ) and did not differ by sex,  $t(df = 140) = -.85$ ,  $p = .39$ . Therefore, it was not necessary to control for sex or age in subsequent analyses.

### Are There Differences in Social Cognition and PTSD on the Basis of Attachment?

Independent-samples *t* tests were used to compare adolescents on the basis of social cognition. Adolescents with a secure attachment performed significantly better on the MASC, with a mean score of 34.19 ( $SD = 3.83$ ), compared with a mean score of 31.30 ( $SD = 5.85$ ) for adolescents with an insecure attachment,  $t(114.95, \text{equality of variances not assumed}) = 3.47$ ,  $p = .001$ ,  $d = .585$ . Chi-square analyses were used to compare adolescents with a secure attachment to those with an insecure attachment on the basis of categorical PTSD status but did not reveal any group differences in self-reported PTSD status on the YSR,  $\chi^2(df = 1, N = 142) = .84$ ,  $p = .36$ . Independent-samples *t* tests were used to compare adolescents with secure and insecure attachment on the basis of continuous PTSD symptoms (TSCC). No group differences were noted ( $M_{\text{secure}} = 51.67$ ,  $SD_{\text{secure}} = 10.60$ ;  $M_{\text{insecure}} = 52.53$ ,  $SD_{\text{insecure}} = 10.72$ ),  $t(df = 140) = -.44$ ,  $p = .66$ .

### Does Social Cognition Mediate the Relation Between Attachment and PTSD?

Before testing for mediation, we used formal detection tolerance and the variance inflation factor (VIF) to assess multicollinearity. Because multicollinearity was not a problem, with tolerance greater than .2 and a VIF less than 4, centering the predictor variable was not necessary (Aiken & West, 1991; Holmbeck, 2002). Preacher and Hayes's (2008) test of the indirect effect was used to assess whether social cognition (MASC total score) mediated the relation between adolescents' maternal attachment representations (CAI) and self-reported PTSD. This test was used instead of a traditional Sobel test because it provides a bootstrap test of the indirect effect (confidence interval) and permits the use of a binary outcome (Preacher & Hayes, 2008). In this study, 5,000 bootstrap samples were used to create 95% bias-corrected and accelerated bootstrap confidence intervals (CIs) of the indirect effect. Mediation models are presented in Figure S2 of the online

supplemental material. Adolescents' maternal attachment representation served as the independent variable, social cognition served as the mediator, and self-reported PTSD served as the dependent variable. First, binary self-reported PTSD status on the YSR served as the dependent variable, and the test of the indirect effect indicated that social cognition mediated the relation between adolescents' maternal attachment representations and self-reported PTSD, with the mean of the indirect effect across all bootstrap samples estimated at .22 and a resulting confidence interval that did not include 0 (CI [.02, .53]; Preacher & Hayes, 2008). There was a significant direct effect of attachment on social cognition ( $B = -2.89$ ,  $SE = .98$ ),  $t(df = 140) = -2.94$ ,  $p = .004$ , and a significant direct effect of social cognition on PTSD ( $B = -.07$ ,  $SE = .03$ ,  $Z = -2.16$ ,  $p = .03$ , Wald = 4.68). The direct effect of attachment on PTSD was not significant ( $B = .14$ ,  $SE = .40$ ,  $Z = .35$ ,  $p = .72$ , Wald = .12). Second, continuously self-reported PTSD symptoms on the TSCC was used as the outcome variable, and the test of the indirect effect indicated that social cognition mediated the relation between adolescents' maternal attachment representations and PTSD, with the mean of the indirect effect across all bootstrap samples estimated at 1.08 and a resulting confidence interval that did not include 0 (CI [.14, 2.67]; Preacher & Hayes, 2008). There was a significant direct effect of attachment on social cognition ( $B = -2.89$ ,  $SE = .98$ ),  $t(df = 140) = -2.94$ ,  $p = .004$ , and of social cognition on PTSD ( $B = -.37$ ,  $SE = .17$ ),  $t(df = 139) = -2.20$ ,  $p = .03$ . The direct effect of attachment on PTSD was not significant ( $B = -.20$ ,  $SE = 1.99$ ),  $t(df = 139) = -.10$ ,  $p = .92$ .

The same procedure was used to confirm these findings utilizing parent-reported PTSD status (CBCL). This model is presented in Figure S3 in the online supplemental material and confirms the self-report findings, showing that social cognition mediated the relation between adolescents' maternal attachment representations and PTSD, with the mean of the indirect effect across all bootstrap samples estimated at .27 and a confidence interval that did not include 0 (CI [.05, .67]; Preacher & Hayes, 2008). There was a significant direct effect of attachment on social cognition ( $B = -2.57$ ,  $SE = 1.01$ ),  $t(df = 132) = -2.54$ ,  $p = .01$ , and a significant direct effect of social cognition on PTSD ( $B = -.10$ ,  $SE = .05$ ,  $Z = -2.08$ ,  $p = .04$ , Wald = 4.31). The direct effect of attachment on PTSD was not significant ( $B = .41$ ,  $SE = .44$ ,  $Z = .91$ ,  $p = .36$ , Wald = .83).

### Does Social Cognition Mediate Between Attachment and PTSD Treatment Outcome?

PTSD treatment outcome was calculated by subtracting self-reported PTSD group status (0 = no PTSD, 1 = clinically significant PTSD) at admission from PTSD group status at discharge. Therefore, the PTSD treatment outcome variable had the following possible values: 0 = *no change in diagnostic status*, 1 = *participant reported clinically significant PTSD at discharge but not at admission*, and -1 = *participant reported clinically significant PTSD at admission but not at discharge*. In our sample, no adolescents reported increased PTSD after treatment, 19.1% ( $n = 18$ ) changed group status from clinically significant PTSD symptoms at admission to nonclinically significant symptoms at discharge, and 80.9% ( $n = 76$ ) did not change group status.

Preacher and Hayes's (2008) test of the indirect effect was used to test the hypothesis that social cognition (MASC) mediates the relation between attachment and changes in self-reported PTSD status from admission to discharge. In this model, presented in Figure S4 of the online supplemental material, social cognition served as the mediator, attachment served as the independent variable, and PTSD treatment outcome served as the dependent variable. The test of the indirect effect indicated that social cognition mediated the relation between adolescents' maternal attachment representations and PTSD treatment outcome, with the mean of the indirect effect across all bootstrap samples estimated at  $-.28$  and a resulting confidence interval that did not include 0 (CI  $[-.73, -.02]$ ; Preacher & Hayes, 2008). There was a significant direct effect of attachment on social cognition ( $B = -3.32$ ,  $SE = 1.31$ ),  $t(df = 92) = -2.53$ ,  $p = .01$ . The direct effects of social cognition on treatment outcome ( $B = .08$ ,  $SE = .04$ ,  $Z = 1.78$ ,  $p = .08$ , Wald = 3.16) and of attachment on treatment outcome ( $B = -.55$ ,  $SE = .71$ ,  $Z = -.78$ ,  $p = .43$ , Wald = .61) were not significant.

## Discussion

The present study sought to provide the first empirical evaluation of Sharp et al.'s (2012) model in a sample of inpatient adolescents by (a) exploring differences in social cognition and PTSD on the basis of attachment security and (b) determining whether social cognition mediated the relation between attachment security and clinically significant PTSD symptoms. As expected, adolescents with an insecure attachment demonstrated impaired social cognition. Contrary to our expectation, attachment insecurity was not directly associated with clinically significant symptoms of PTSD. Indeed, across three measures (CBCL, YSR, and TSCC) completed by adolescents and their parents, the effect of attachment insecurity on PTSD was exerted *through* impaired social cognition (with significant mediation in all cases). Consistently across measures and reporters, there was a negative relation between attachment and social cognition; a negative relation between social-cognitive abilities and PTSD, indicating impaired social cognition in association with clinically significant PTSD symptoms; and no evidence of a significant, direct relation between attachment and PTSD. Although the latter stands in contrast to the results of prior studies documenting increased PTSD symptoms among individuals with insecure attachments (e.g., Joubert et al., 2012), this finding is consistent with Sharp et al.'s (2012) hypothesis that attachment insecurity confers risk for PTSD *through* impaired social-cognitive functioning. Specifically, attachment-related internal working models have been conceptualized by Bowlby (1969) and others (see Fonagy, Gergely, & Target, 2008) as relational schemas, which are unconscious and not always directly accessible to the individuals themselves. Consistent with attachment-based models of social cognition (Fonagy, Gergely, & Target, 2007), the clinical utility of attachment influences is enhanced when the effects of attachment on clinical outcomes are assessed through more readily accessible here-and-now social-cognitive constructs.

We also sought to examine relations between attachment, social cognition, and PTSD pre- and posttreatment by examining whether social cognition (at admission) mediated the relation between attachment security and PTSD treatment outcome during inpatient

treatment in a naturalistic setting. Treatment outcome in this study was based upon adolescent self-report collected at admission and discharge from an inpatient unit. Parent-reported PTSD symptoms at discharge were not considered because, in this inpatient facility, adolescents spend little time with their parents during hospitalization due to heavy scheduling of therapeutic activities on the unit as well as geographic constraints related to the fact that families travel widely in order to seek treatment for their children at this facility. On the basis of adolescent self-report, 19.1% of adolescents changed group status from clinically significant PTSD at admission to nonclinically significant PTSD at discharge, and 80.9% did not change group status. Mediation analyses indicated that social-cognitive capacity at admission explained the relation between attachment and PTSD treatment outcome. As in prior analyses, there was a significant relation between attachment and social cognition; however, no evidence of significant evidence of a relation between social cognition and PTSD treatment outcome was noted ( $p = .08$ ). The test of the indirect effect used to assess mediation in this study is statistically powerful, more so than are stepwise approaches to assessing mediation (Kenny, 2014), and may explain why the overall mediational effect was significant despite nonsignificant direct effects. The prepost design of this study preliminarily suggests that social-cognitive impairment underlies the presence of PTSD symptoms, such that impairment in social cognition is associated with PTSD risk just as adequate social cognition is associated with PTSD recovery. Still, the finding that social cognition mediated the relation between attachment and PTSD treatment outcome should be reevaluated in a larger sample for two primary reasons. First, sample size constraints in the present study precluded conducting mediational analyses that included only individuals with clinically significant PTSD at admission, and thus, the analyses of the present study conflated inpatients who never had clinically significant PTSD (i.e., no symptom change) with those who maintained clinically significant PTSD symptoms (i.e., no symptom change). In this study, excluding the former would have reduced the sample size ( $n = 38$ ), underpowering the current study analyses. Second, the findings of the present study should be replicated in a sample with tighter treatment evaluation, in order to more robustly establish this finding and determine specific aspects of treatment (e.g., focus on PTSD symptoms specifically or enhancement of social-cognitive skills generally) that drive symptom reduction.

The overarching finding of this study, that attachment insecurity relates to PTSD in adolescents through impaired social cognition, is consistent with aforementioned research cited by Sharp et al. (2012) and with evidence-based approaches to the treatment of PTSD in children, which include social-cognitive and attachment-related constructs in treatment. Indeed, trauma-focused cognitive-behavioral therapy (TF-CBT), the gold-standard psychosocial treatment for PTSD in youth (Cohen, Mannarino, & Deblinger, 2006), is based partially on attachment models (Child Sexual Abuse Task Force and Research & Practice Core, National Child Traumatic Stress Network, 2008) and contains intervention components that target social-cognitive abilities. Likewise, the attachment, self-regulation, and competency (ARC) framework (Blaustein & Kinniburgh, 2010) for the treatment of trauma symptoms in youth has been identified as a promising practice by the National Child Traumatic Stress Network and explicitly places trauma treatment within an attachment context.

ARC specifically addresses a child's need to develop a clear understanding of others' communication via facial expression, vocalizations, and actions and places this development within an attachment context. Together, both ARC and TF-CBT include components designed to address social-cognitive impairments (e.g., hypervigilant, negative interpretations) in the treatment of PTSD. The findings of the present study add to this research base by explicitly tying together attachment, social cognition, and PTSD in adolescents for the first time and demonstrating improvement in PTSD symptoms during a naturalistic treatment that aimed to enhance social-cognitive abilities, pointing to the potential role of social cognition as a mechanism of PTSD recovery—as is suggested by ARC and TF-CBT models that target similar abilities in their treatment protocols.

Several important limitations to the present study should be noted. First, the mediational models linking attachment, social cognition, and PTSD were conducted on cross-sectional data, and therefore, the developmental hypothesis put forth by Sharp et al. (2012) could not be explored. Moreover, it is important to note that within TF-CBT and ARC models, social-cognitive impairments (such as difficulty correctly identifying emotions or overattributing negative intentions to others) are conceptualized as consequences of a traumatic experience, not predisposing factors. The present study cannot speak to the directionality of the identified relation between social cognition and PTSD, although that remains an important area of research.

Second, the present study did not assess traumatic events but, rather, relied upon three measures capturing PTSD symptoms. To that end, the present study cannot speak to the type (e.g., interpersonal vs. noninterpersonal), number, or chronicity of trauma endured by participants and, thus, could not examine whether identified relations between attachment, social cognition, and PTSD are generalizable across multiple types of traumatic events. The absence of information about the types of traumatic events in question may also explain why previously documented relations between attachment and PTSD in youth were not replicated in the current study. Indeed, most prior studies documenting a link between insecure attachment and PTSD did so in maltreated samples exposed to physical abuse or neglect (e.g., V. Carlson et al., 1989; Finzi et al., 2000; Joubert et al., 2012; van IJzendoorn et al., 1999), and perhaps the chronicity of maltreatment-related trauma could explain why these findings were not replicated in this study. It is well known that child maltreatment is a common cause of trauma symptoms in youth and that affected children are disproportionately insecurely attached—unsurprising in light of the parent commonly being the source of maltreatment. However, the present study did not focus on a maltreated sample, and moreover, the absence of information about prior traumatic events makes it possible that many adolescents were suffering with symptoms related to traumatic events perpetrated by a nonattachment figure or related to noninterpersonal events (e.g., automobile accident). Further research elucidating the relation between attachment security and PTSD, with specific analyses relating to the type, number, and chronicity of trauma endured, is needed.

Additional limitations include that the study was conducted in a naturalistic setting, preventing tightly controlled treatment evaluation and the absence of clinician-rated diagnostic measures, and that the study was conducted in a private psychiatric hospital in which adolescents and their families represented a predominantly

Caucasian, socioeconomically advantaged group. Future research should expand this study to include ethnically and socioeconomically diverse samples. Notwithstanding these limitations, the present study takes an important step toward examining attachment, social cognition, and clinically significant symptoms of PTSD in an adolescent sample for the first time. Moreover, the present study is strengthened by consideration of PTSD treatment outcome and is the first, to our knowledge, to point to social cognition as an important factor in PTSD treatment outcome among adolescents. Finally, this study makes use of a large inpatient sample, multiple informants, and multiple measures, further strengthening its findings.

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Received October 27, 2015

Revision received May 17, 2016

Accepted May 23, 2016 ■