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Direction of Influence Between Posttraumatic and Depressive Symptoms during Prolonged Exposure Therapy among Children and Adolescents

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Abstract

Objective—The objective of the present study was to examine the temporal sequencing of posttraumatic and depressive symptoms during prolonged exposure (PE) therapy for posttraumatic stress disorder (PTSD) among children and adolescents.

Method—Participants were 73 children and adolescents (56.2% female) between the ages of 8 and 18. Participants completed self report measures of posttraumatic stress and depression prior to every session. Measures included the Child PTSD Symptom Scale (CPSS), Beck Depression Inventory (BDI), and Children's Depression Inventory (CDI).

Results—Multilevel mediational analyses indicated reciprocal relations during treatment: changes in posttraumatic symptoms led to changes in depressive symptoms and vice versa. Posttraumatic symptoms accounted for 64.1% of the changes in depression, whereas depressive symptoms accounted for 11.0% of the changes in posttraumatic stress.

Conclusions—PE may work primarily by reducing posttraumatic stress which in turn reduces depression.

Keywords

Posttraumatic Stress Disorder; Prolonged Exposure; Anxiety; Depression; Children; Adolescents; Treatment

Posttraumatic stress disorder (PTSD) is a common and chronic disorder among youth (Copeland, Keeler, Angold, & Costello, 2007). PTSD is frequently comorbid with major depressive disorder (MDD) and 48% of individuals with PTSD experience at least one major

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depressive episode (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Among youth, posttraumatic and depressive symptoms frequently co-occur (Copeland et al., 2007).

Evidence regarding the relationship between posttraumatic and depressive symptoms over time is inconsistent. Some studies suggest that posttraumatic symptoms lead to depression. For example, in the National Comorbidity Survey, 78.4% of individuals with both PTSD and MDD reported that the onset of PTSD preceded that of MDD (Kessler et al., 1995). Specifically, among children and adolescents, anxiety symptoms have been found to lead to depressive symptoms over time, but not vice versa (Cole, Peeke, Martin, Truglio, Seroczynski, 1998). Conversely, other studies have suggested that depressive symptoms lead to posttraumatic symptoms more consistently than vice versa (Allon-Schindel, Aderka, Shahar, Stein, & Gilboa-Schechtman, 2010; King, King, McArdle, Shalev, & Doron-LaMarca, 2009). Finally, some studies have found that PTSD and MDD develop simultaneously (e.g., Shalev et al., 1998) and that reciprocal relations exist between posttraumatic and depressive symptoms (Erickson, Wolfe, King, King, & Sharkansky, 2001).

Trauma-focused treatments for PTSD have been repeatedly shown to reduce both posttraumatic and depressive symptoms among adults (Harvey, Bryant, & Tarrier, 2003) and youth (Feeny, Foa, Treadwell, & March, 2004). However, little is known about the relationship between posttraumatic and depressive symptoms along the course of treatment. In a recent study among individuals with social anxiety disorder, changes in social anxiety were found to account for changes in depression during treatment, but not vice versa (Moscovitch, Hofmann, Suvak, & In-Albon, 2005). To our knowledge, no study has examined the anxiety-depression relationship along the course of treatment for PTSD.

The present study addresses this gap by applying hierarchical linear modeling (HLM) techniques to examine the relationship between posttraumatic and depressive symptoms during treatment for PTSD. In addition, the present study is the first to examine this relationship among youth receiving treatment. The relationship between posttraumatic and depressive symptoms during treatment is of paramount importance as it can shed light on both mechanisms of change and psychopathology.

We examined this relationship among children and adolescents during prolonged exposure (PE) therapy for PTSD. PE is a trauma focused cognitive-behavioral treatment rooted in emotional processing theory (Foa & Kozak, 1986). Recently, PE was adapted for treatment of children and adolescents (Foa, Chrestman, & Gilboa-Schechtman, 2008), and found to be effective in an open trial (Foa et al., 2008), as well as a randomized controlled trial (Gilboa-Schechtman et al., 2010).

We examined the four possibilities regarding the relationship between posttraumatic and depressive symptoms over time: (a) changes in posttraumatic symptoms account for changes in depressive symptoms but not vice versa, (b) changes in depressive symptoms account for changes in posttraumatic symptoms but not vice versa, (c) changes in both posttraumatic and depressive symptoms account for changes in each other, and (d) changes in posttraumatic symptoms are unrelated to changes in depressive symptoms.

Method

Participants

Participants were 73 children and adolescents who sought treatment for PTSD at the Schneider Children's Medical Center in Israel. Participants were diagnosed with primary PTSD according to DSM-IV criteria using a semi-structured interview - the Schedule of

Affective Disorders and Schizophrenia for School-Age Children- Revised for DSM-IV (K-SADS - Kaufman et al., 1997). Participants were included in the present study if they were between 8 and 18 years of age, received a primary diagnosis of PTSD, and were fluent in Hebrew. Participants were excluded if they suffered head injuries resulting in permanent brain damage, changed medication or dosage during treatment, had current substance abuse, or attended an additional psychotherapeutic treatment. Table 1 presents demographic, trauma-related, and pretreatment clinical measures.

Procedure

This study was approved by the Schneider Children's Medical Center ethics review board. Participants arrived at the clinic and were interviewed by experienced psychologists, who received training prior to the study. Following the intake, interviewers invited parents and children to participate in the study. Both parents and children signed informed consent forms. During treatment, participants filled out self-report measures before every session.

Treatment and therapists

Treatment in the present study was based on the PE manual for children and adolescents (Foa et al., 2008). Treatment consisted of 12-15, 90 minute sessions, and for younger children, sessions were shorter and breaks were allowed. PE included 3 modules. The first module was psychoeducation and treatment planning, and consisted of 2-3 sessions. The second module, exposure, consisted of 8-10 sessions. These sessions included in-session imaginal exposure as well as assigning in vivo exposure exercises as homework. For younger children, the imaginal exposure was aided by using a visual modality (e.g., drawing). The third module, relapse prevention, consisted of 2 sessions and included a discussion of triggers for relapse and appropriate coping strategies. A detailed account of the treatment can be found in Foa et al. (2008), and Gilboa-Schechtman et al. (2010). Therapists were clinical psychologists, trained in the administration of pediatric PE prior to the study by the second author. During treatment, all therapists received weekly supervision sessions from the last author to ensure treatment integrity.

Measures

Participants and their parents were interviewed using the Schedule of Affective Disorders and Schizophrenia for School-Age Children – Revised for DSM—IV (K-SADS - Kaufman et al., 1997), in order to establish diagnoses. The K-SADS is a highly reliable and valid semi-structured interview, covering both current and lifetime disorders (Kaufman et al., 1997). Before each treatment session participants were administered the Child PTSD Symptom Scale (CPSS – Foa, Johnson, Feeny, & Treadwell, 2001), which is a well-validated self-report inventory with 17 items that assess PTSD symptoms according to the DSM-IV. In addition, before each session participants were administered the Beck Depression Inventory (BDI - Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) if they were 13 years old and over, or the Children's Depression Inventory (CDI - Kovacs, 1982) if they were under 13 years old. In order to facilitate comparison between individuals who filled out the CDI and BDI, we unified them to create a single depression variable. We divided CDI scores by the total range (54) to express each score as a percentage of the total range. We then used the percentage to assign a BDI score by multiplying it by the total BDI range (63). In this way we converted all CDI scores into corresponding BDI scores. This facilitates comparison between the age groups and allows for a unified examination of the entire sample¹.

¹To ensure that the conversion did not influence our results we reran all analyses among children and adolescents separately using original, non-converted BDI and CDI scores. Results obtained among children and adolescents separately were identical to those reported for the entire sample. Thus, we report only analyses conducted on the entire sample.

Analytic Strategy

Our data formed a multilevel, hierarchical nested structure: The lower level, or Level 1 data, consisted of the repeated measures that were collected during treatment sessions (i.e., posttraumatic and depressive symptoms). Level 1 data were nested within Level 2 units (i.e., participants). These data are appropriate for hierarchical linear modeling (HLM) techniques (Raudenbush, 2001) which allow the number of observations to vary between participants, and are capable of dealing with missing data effectively. We followed the procedures reported by Kenny, Korchmaros, and Bolger (2003) to conduct multilevel mediational analyses. In order to perform a more rigorous test for mediation, we ‘lagged’ the mediator variable in all analyses. Thus, we examined whether changes in the mediator variable at Time t accounted for changes in the outcome variable at Time $t + 1$. Conducting the lagged mediation analyses builds on, and extends previous studies that have examined the relationship between anxiety and depression (e.g., Moscovitch et al., 2005).

Results

Multilevel-Mediational Analyses

We began by examining a model in which time² was the predictor, posttraumatic symptoms were the mediator and depressive symptoms were the outcome. In other words we examined to what extent changes in posttraumatic symptoms account for changes in depressive symptoms during treatment. Consistent with the guidelines of Kenny et al. (2003), we first regressed depressive symptoms on time (path C in Figure 1 and Table 2). Results indicated that depressive symptoms significantly decreased during treatment. We then regressed posttraumatic symptoms on time (path A in Figure 1 and Table 2). Results indicated that posttraumatic symptoms also significantly decreased during treatment. Next, we regressed depressive symptoms on both time and posttraumatic symptoms simultaneously. The effect of posttraumatic symptoms (path B in Figure 1 and Table 2) was significant indicating that reductions in posttraumatic symptoms significantly predicted subsequent reductions in depressive symptoms. The effect of time (path C' in Figure 1 and Table 2) was not significant, indicating that time did not have an additional affect on depressive symptoms above and beyond the effect accounted for by posttraumatic symptoms. As can be seen in Figure 1 and Table 2, the original pathway between time and depressive symptoms (path C) was reduced from -0.23 to -0.07 when posttraumatic symptoms were entered (path C').

We also examined multilevel mediation using the program Prodcin (MacKinnon, Fritz, Williams, & Lockwood, 2007). This program provides a powerful test for multilevel mediation that can adjust for covariance between paths A and B. We found clear evidence for multilevel mediation as the confidence intervals for the indirect effect did not include zero (-0.21 to -0.11). Posttraumatic symptoms accounted for 64.1% of the total effect of time on depressive symptoms³.

²Time was modeled using session number (i.e., 1, 2, 3...). Prior to running analyses we compared a model with a linear time variable, and a model with a non-linear, natural log, time variable. As these models were non-nested we compared them using Akaike's Information Criterion (AIC; Akaike, 1987) and the deviance statistic. Results indicated that a non-linear model fit the data better. Thus, we used a non-linear time variable in all analyses.

³We calculated percent mediation according to the guidelines of Kenny, Korchmaros, & Bolger (2003), that have been previously applied to multilevel mediational analyses of anxiety and depression by Moscovitch et al. (2005). According to Kenny et al. (2003), the total effect in lower level mediation models is the sum of the direct effect (C'), the indirect effect (AB), and the covariance between A and B (the covariance between the ordinary-least-squares estimates for paths A and B). Thus, the formula used to calculate percent mediation was: $100 * \{ [C' + AB + \text{Cov}(AB)] - C' \} / [C' + AB + \text{Cov}(AB)]$, or $100 * (\text{total effect} - \text{direct effect}) / (\text{total effect})$. The covariance between the ordinary-least-squares estimates for paths A and B was 0.026 for the mediational model, and 0.040 for the reverse model.

We also examined a reverse model in which changes in depressive symptoms accounted for changes in posttraumatic symptoms during treatment. Table 3 and Figure 2 present the results of these analyses. In the reverse model, the effect of time on posttraumatic symptoms (path C') remained significant after regressing posttraumatic symptoms on time and depressive symptoms simultaneously. We found evidence for multilevel mediation (confidence intervals = -0.13 to -0.05), with depressive symptoms accounting for 11.0% of the total effect of time on posttraumatic symptoms.

Discussion

The present study examined the relationship between posttraumatic and depressive symptoms during prolonged exposure therapy for PTSD among youth. Consistent with previous studies (e.g., Gilboa-Schechtman et al., 2010), PE significantly reduced both posttraumatic and depressive symptoms (Paths A and C in Table 1). Moreover, we found support for the reciprocal influence model (model C). However, changes in posttraumatic symptoms accounted for 64.1% of the changes in depressive symptoms, whereas changes in depressive symptoms accounted for only 11.0% of the changes in posttraumatic symptoms. Our findings indicate that reciprocal relations exist between posttraumatic and depressive symptoms during treatment, but changes in posttraumatic symptoms lead to changes in depressive symptoms to a greater extent than vice versa.

According to the tripartite model (Clark & Watson, 1991), anxiety and depression share a higher-order factor of negative affect. Although it is possible that trauma-focused treatment would target shared posttraumatic and depressive factors leading to reciprocal, simultaneous reductions, our findings suggest that such treatment primarily affects posttraumatic symptoms, which in turn lead to reductions in depressive symptoms. This has important implications for treatments of PTSD, and indicates that treatments do not need to target shared posttraumatic and depressive factors in order to reduce depressive symptoms.

The helplessness/hopelessness theory (Alloy, Kelly, Mineka, & Clements, 1990) may account for the effect of posttraumatic symptoms on subsequent depressive symptoms during treatment. This theory posits that feelings of helplessness are primarily related to anxiety, whereas feelings of hopelessness are primarily related to depression. According to this theory, experiencing helplessness repeatedly or for long periods of time, can lead to hopelessness (Alloy et al., 1990). Thus, as individuals begin to better cope with their symptoms during treatment, their helplessness is reduced, which results in subsequent reductions in hopelessness. Alternatively, the anhedonia model of depression (Loas, 1996) may also account for the findings of the present study. It is possible that diminishing anxiety and avoidance levels during treatment, may lead to the increased experience of pleasurable life events, thus reducing anhedonia and subsequent depression (Loas, 1996).

Our findings are congruent with those of Moscovitch et al. (2005), who found that social anxiety reduction leads to depression reduction to a greater extent than vice versa during treatment for social anxiety disorder. However, it remains unclear if the relationship found in the present study holds in other treatments. For example, since cognitive biases play an important role in both PTSD and depression, treatments that are more cognitively-focused and less exposure-based than PE may lead to a more reciprocal relationship between posttraumatic and depressive symptoms. Future studies may examine the relationship between posttraumatic and depressive symptoms in various treatments for PTSD in order to elucidate whether different treatments for PTSD work through similar mechanisms. In addition, future studies may also examine the relationship between anxiety and depressive symptoms in individuals with primary depression. Examining such a group of patients can

indicate whether the relationship found in the present study is a result of primary PTSD or whether it represents a general anxiety-depression relationship during treatment.

In closing, we would like to note several limitations of the present study. First, we did not include a control condition and therefore cannot assess whether our findings are specific to processes that occur during PE or capture more naturalistic processes underlying changes in PTSD and depression. Second, the present study focused solely on the interplay between posttraumatic and depressive symptoms and did not assess mechanisms that could account for the findings. Future studies may investigate mechanisms by which posttraumatic symptoms can affect depressive symptoms (e.g., cognitions, hopelessness, anhedonia, positive and negative affect, physiological arousal) in order increase our understanding of the pathways connecting posttraumatic symptoms and depressive symptoms.

Despite these limitations, the present study represents the first multilevel mediational study of the longitudinal relationship between posttraumatic and depressive symptoms, during the treatment of PTSD. Our findings extend previous research on treatment for PTSD, by indicating that PE affects depressive symptoms primarily through its effect on posttraumatic symptoms.

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Figure 1. Mediation model

Figure 2. Reverse mediational model

Table 1
Demographic, trauma related, and pre-treatment clinical measures ($n = 73$)

Measure	
Gender	
Male	43.8%
Female	56.2%
Age	12.6 (3.0)
Family status	
Both parents live at home	54.8%
One parent lives at home	45.2%
Type of Trauma	
Motor Vehicle Accidents	35.6%
Terror Attacks	27.4%
Non-sexual assault	4.1%
Sexual assault	15.1%
Other	17.8%
Time since trauma (years)	1.4 (1.4)
Additional AXIS-I disorder	
MDD	12.3%
Anxiety disorders	16.4%
ADHD	11.0%
Medication	12.3%
CPSS	25.8 (10.0)
Converted BDI ^a	16.0 (9.0)

Note. MDD = Major depressive disorder; ADHD = Attention-deficit hyperactivity disorder; CPSS = Child posttraumatic stress disorder symptom scale; BDI = Beck depression inventory.

^aTo facilitate comparison we converted CDI scores into BDI scores. First, we divided CDI scores by the total possible score (54) and turned them into percentages. Second, we multiplied these percentages by the total BDI score (63) to assign a converted BDI score.

Table 2
Summary of multilevel regression analyses for the mediational model

Step	Path	Predictor Variable	Outcome Variable	B	SE B	T	p
1	C	Time	Depression	-0.23	0.04	-5.87	<0.001
2	A	Time	Posttraumatic Symptoms	-0.30	0.04	-8.05	<0.001
3	B	Posttraumatic Symptoms	Depression	0.51	0.05	9.66	<0.001
	C'	Time	Depression	-0.07	0.05	-1.50	0.14

Table 3
Summary of multilevel regression analyses for the reverse mediational model

Step	Path	Predictor Variable	Outcome Variable	B	SE B	T	p
1	C	Time	Posttraumatic Symptoms	-0.34	0.04	-9.30	<0.001
2	A	Time	Depression	-0.19	0.04	-5.17	<0.001
3	B	Depression	Posttraumatic Symptoms	0.44	0.06	7.85	<0.001
	C'	Time	Posttraumatic Symptoms	-0.32	0.05	-7.03	<0.001