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Impact of Disaster Exposure Severity: Cascading Effects across Parental Distress, Adolescent PTSD Symptoms, as well as Parent-Child Conflict and Communication

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Introduction

Natural disasters, including earthquakes, tsunamis, hurricanes, tornadoes and others, are common occurrences worldwide (Statista, 2018) and can have detrimental effects on the well-being of adolescents, their caregivers, and relationships between family members (Adams et al., 2015; Paul, 2015). Among the potential outcomes following disaster is the development of posttraumatic stress disorder (PTSD), with estimates ranging from 5 to 23%,

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depending on the sample (Neria, Nandi, & Galea, 2008). Following natural disaster exposure, the mental health of individuals, their loved ones, and the relationships between family members are potentially affected. Unlike most forms of traumatic event exposure, exposure to disaster can often impact an entire family; thus, it is imperative to examine inter-relations among offspring-, caregiver/parent-, and family-level variables following an event that is not confounded by characteristics of the perpetrator or victim of the traumatic event (Hasin, Keyes, Hatzenbuehler, Aharonovich, & Alderson, 2007). Among adolescents, a prospective study found only mild improvement in PTSD symptoms five years after the disaster (Goenjian et al., 2005), suggesting that PTSD can be long-lasting if untreated. Although previous research has prospectively examined factors associated with adolescent PTSD symptoms (e.g., Bokszczanin, 2008; Green et al., 1991), to our knowledge, no research to date has examined the longitudinal interrelations of parent-, adolescent-, and family-level outcomes post-disaster.

The impacts of disasters can be quite far-reaching, influencing communities, the individuals within them, and these individuals' relationships with one another. Additionally, although some work suggests that pre-disaster mental health impacts post-disaster symptomatology (Raker, 2019), more work suggests that the processes operating during or after traumatic events, rather than pre-disaster factors are most predictive of outcomes (e.g., Brewin, Andrews, & Valentine, 2000). For instance, individuals who were connected to others who experienced significant loss (Bryant et al., 2017) and those who perceived more threat (Weaver & Clum, 1995) were most adversely impacted following disaster. Additionally, prior work found that communities with less capital/resources (Wind & Komproe, 2012) and those with less connectedness among people and local organizations (Norris, Stevens, Pfefferbaum, Wyche, & Pfefferbaum, 2008) were less resilient following disaster. Therefore, it is especially important to examine the impact of processes operating during or after disasters on parent-, adolescent-, and family-level outcomes.

In terms of key risk factors occurring during disaster, *severity of exposure*—the extent to which individuals worry about themselves or others, or experience the loss of their home or belongings—may be particularly important. Disaster severity impacts community resources and relationships, including family relationships, as well as the well-being of parents and children within these areas themselves. Indeed, those who worry more about their own safety and the safety of close others, and those who experience greater loss (e.g., home, car) following exposure report a higher number of PTSD symptoms and related distress (Lonigan, Shannon, Taylor, Finch, & Sallee, 1994; Thabet & Vostanis, 1999). Additionally, families who face more new stressors, including financial burden, loss of home, and changes in social support report more family conflict post-disaster (Uttervall, Hultman, Ekerwald, Lindam, & Lundin, 2014). Thus, severity of disaster exposure may influence parent and child mental health, as well as family conflict and relationships.

In addition to severity of exposure influencing parent and child post-trauma symptoms and parent-child relationships, parent symptomatology may also influence child mental health. For instance, following disaster exposure, caregiver mental health may impact adolescent symptomatology potentially as social transmission of distress. Several cross-sectional studies have found associations between parent distress and adolescent PTSD symptoms

post-disaster (Chemtob et al., 2010; Green et al., 1991; Kerns et al., 2014; McFarlane, 1987; Trickey, Siddaway, Meiser-Stedman, Serpell, & Field, 2012). Longitudinal work has found that parent post-disaster mental health symptoms are associated with more adolescent PTSD symptoms (Green et al., 1991). Another study found that a mindfulness-based stress reduction program reduced parental stress, which in turn, reduced child PTSD symptoms (Neece, 2014). It may be that adolescent symptoms are exacerbated when they witness their parents struggling, perhaps contributing to an increased sense of uncontrollability or insecurity (Forman & Davies, 2003). Therefore, parent distress following disaster may influence adolescent PTSD symptoms.

In addition to parent symptoms post-trauma impacting child symptoms, child post-trauma mental health may also influence parent symptoms. Although, to our knowledge, there is no work examining how adolescent PTSD symptoms may impact parent distress or mental health, other work finds broadly that child mental health problems impact parent mental health (Weiss, Cappadocia, MacMullin, Viecili, & Lunsky, 2012). It is conceivable that adolescent PTSD symptoms may increase the parent's own distress following a disaster.

In addition to parent and child post-trauma symptoms influencing one another, these factors may also influence and be influenced by parent-child relationship factors, specifically conflict and communication. In terms of the association between child mental health and parent-child relationships, families reporting higher levels of functioning tended to have adolescents with fewer mental health problems after trauma (e.g., depressive and PTSD symptoms; Betancourt, Agnew-Blais, Gilman, Williams, & Ellis, 2010; Wickrama & Kaspar, 2007). When examined prospectively, family atmosphere contributed significantly to adolescent PTSD symptoms (Green et al., 1991). Yet, family communication and cohesion after a natural disaster was not associated with adolescent PTSD (Hafstad, Gil-Rivas, Kilmer, & Raeder, 2010). The presence of more conflictual home environments may increase risk for adolescent PTSD, perhaps via increasing negative cognitions (Andrews, Brewin, Rose, & Kirk, 2000; Brewin et al., 2000). Prior work emphasizes the importance of PTSD re-experiencing symptoms in particular in impacting how individuals make sense of interactions they have with others and learn new information (Brewin, 2015). Given this small, mixed literature, it is unclear if adolescent PTSD symptoms and parent-child conflict-communication may reciprocally impact one another over time.

In addition to parent-child conflict-communication and adolescent PTSD symptoms potentially influencing one another, it is also possible that parent post-disaster symptoms and parent-child conflict-communication may impact each other. Although never examined specifically, a small body of work examining the prospective impact of an individual's mental health on family climate suggests that an individual's symptoms contribute to conflict within a family (Nilsen, Skipstein, & Demerouti, 2016). Yet, in other studies, family functioning and cohesion post-disaster were not prospectively associated with parent psychological well-being (Felix et al., 2015) or parent PTSD symptoms (Hafstad et al., 2010). Given the very small and mixed literature, it is unclear if parent distress and parent-child conflict-communication impact one another post-disaster.

Given the stated gaps in the existing literature and the correlated nature of parent distress, adolescent PTSD, and parent-child conflict and communication post-disaster, it is critically important to test these inter-relations using longitudinal, prospective designs measuring these constructs across time. Dynamic cascade conceptualizations (Haller, Handley, Chassin, & Bountress, 2010; Masten et al., 2005; Obradovic, Burt, & Masten, 2010) examine longitudinal associations of multiple constructs across time. In the context of post-disaster adolescent mental health, a cascade design allows for the associations among parent distress, adolescent PTSD symptoms, and parent-child conflict-communication to be examined across time.

Current Study

We examined the longitudinal associations among parent distress, adolescent PTSD symptoms, and parent-child conflict-communication after exposure to the spring 2011 Alabama and Missouri tornadoes. Two-thousand families with adolescent offspring were recruited from areas affected by the spring 2011 Alabama and Missouri tornadoes for a larger study to investigate the effectiveness of a post-disaster mental health web-based intervention (*Bounce Back Now*) on adolescent mental health. On April 27, 2011, northern Alabama experienced 39 tornadoes with estimated Enhanced Fujita (EF) scale categories 4 (winds 166–200 mph) to EF 5 (winds greater than 200mph). More than 14,000 homes were destroyed, 2,200 people were injured, and 240 individuals were killed ("National Oceanic and Atmospheric Association (NOAA) Tornadoes – annual 2011; NOAA National Climatic Data Center," 2012).

The goal of the current study was to better understand the associations among parent-, adolescent-, and family-level outcomes post-disaster using a cascade model. This approach allows for the interrelations among these constructs to be modeled both longitudinally and concurrently. It was hypothesized that those who experience greater disaster exposure would report more post-disaster parental distress, adolescent PTSD symptoms, and poorer parent-child conflict and communication (Hypothesis 1). This hypothesis is consistent with previous findings that severity of disaster exposure is associated with adolescent PTSD (e.g., Green et al., 1991). It is also consistent with previous findings suggesting that at a cross-sectional level, disaster exposure severity is associated with more negative individual- and family-outcomes (e.g., Uttervall et al., 2014). It was also hypothesized that more parental distress and adolescent PTSD symptoms would be associated with higher levels of the other construct over time (Hypothesis 2). This hypothesis is consistent with previous findings suggesting that there is a longitudinal social transmission of PTSD symptoms from parent to child (e.g., Green et al., 1991). To our knowledge, this study is the first to examine the longitudinal relations among parental distress and adolescent PTSD symptoms, and parent-child conflict and communication post-disaster. Therefore, we did not predict specific directions of associations for the associations between parent-child conflict and communication and parent distress/adolescent PTSD (Exploratory Analyses).

Method

Participants

A targeted address-based sampling (ABS) method, using a sampling of addresses from a nearly universal listing of mail delivery locations, was used. Using household addresses available in the Computerized Delivery Sequence File (CDSF), maintained by the US Postal Service, individuals were targeted in geographic regions affected by these tornadoes.

Inclusion and Exclusion Criteria

Families living at addresses for which phone numbers could be ascertained were contacted via phone and screened for the presence of a child between ages 12 and 17, English language proficiency, and internet access. If multiple adolescents ages 12 to 17 resided in the home, the individual whose birthday was most recent was chosen as a way of randomly selecting one adolescent participant in each home. Present study analyses were limited to individuals present for the tornadoes. Of the 2,000 adolescents in the larger study, 1,271 individuals were included in current study analyses, as they were present at the time of the tornados, and they had non-missing values on predictors/covariates required for the data analyses. Of the 1,271 included, 73% came from Alabama. See Table 1 for tests of differences between the 1,271 adolescents included in and the 729 excluded from analyses (standard guidelines for effect sizes, Cohen's *d*: 0.2 = small, 0.5 = medium; Cramer's *V*: 0.1 = small, 0.3 = medium; Cohen, 1992). Those who were included were more likely to be male, and more likely to be in families experiencing less severity of disaster exposure, higher income, and poorer conflict-communication at the second time point. Notably, all of these significant differences were small effects. There were no significant differences on any other study variables, including age, race, alcohol use, depression, number of prior traumas, adolescent PTSD symptoms or parent distress at any time points, or parent-child conflict-communication at the first or third time points.

Procedure

This study was approved by the Institutional Review Board at the Medical University of South Carolina. APA ethical standards in treatment have been adhered to. Recruitment procedures for this larger project are described in detail elsewhere (Ruggiero et al., 2015). Caregivers were eligible for the study if they were the guardian of an adolescent aged 12 to 17 years and had internet access in their home. Participants were interviewed by phone at a first time point (on average 8-months post-disaster), as well as at a second time point (12-months post-disaster) and also at a third (20-months post-disaster). Additionally, families were exposed to one of three web-based interventions between the first and second time points. During the baseline telephone interview, caregivers provided consent, and adolescents provided assent.

Measures

Demographics.—Age (range 12–17), gender (girls coded as 0 and boys coded as 1), and race were assessed at the first time point. Participants self-identified as Caucasian, African-American or another race. Because of the very small percent of those reporting that they

identify as another race, the covariate race was dichotomized to capture whether individuals were Caucasian or African-American/Other (Martin, Brechbiel, Chaney, Cremeens-Matthews, & Vail-Smith, 2016). Total household income was also assessed at baseline. Caregivers indicated the family's total household income at baseline into one of seven categories, ranging from 0 (under $10,000) to 6 (over $100,000).

Adolescent Symptoms of PTSD.—The PTSD module from the National Survey on Adolescents (Kilpatrick et al., 2003) was used to assess whether or not (yes/no) 17 DSM-IV PTSD symptoms (e.g., "feeling on guard or very watchful?") were present at all three-time points. For these analyses, the sum of PTSD symptoms in the past month at all three time points was used (eight-months post-tornado: possible [i.e., range for scale in population] and actual range [i.e., range for scale in this dataset]: 0–17 and 0–15; Cronbach'sα: .83; 12-months post-tornado: possible and actual range: 0–17 and 0–15; Cronbach's α: .86; 20-months post-tornado: possible and actual range: 0–17 and 0–14; Cronbach'sα: .87). Higher scores indicated more PTSD symptoms.

Adolescent Alcohol Use.—Using one item from the National Survey of Adolescents (NSA) substance abuse module, which has demonstrated good construct validity (Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997), adolescents reported on the number of days they consumed alcohol in the past month (range: 0–15). Higher scores indicated more past month drinking days.

Adolescent Depressive Symptoms.—Adolescents reported their depressive symptoms using the NSA Depression module (Kilpatrick et al., 2003). Of the nine items to be included in this scale, three overlapped with those on the NSA PTSD Scale (i.e., trouble concentrating, problems sleeping, loss of interest in activities). These items were omitted, and a sum score of the remaining six items was created (range: 0–6; Cronbach's α: .80). Higher scores indicated more self-reported depressive symptoms.

Severity of Tornado Exposure.—At the first time point, caregivers reported whether or not (i.e., yes/no) they were physically injured by the tornado, were concerned about the safety of others, or experienced damage to their home, furniture, sentimental objects, vehicles, pets, land, or any other item not mentioned (i.e., nine items). On average, caregivers reported experiencing two items from this list during/after the tornado (possible and actual range: 0–9; Cronbach's α: .70). Higher scores indicated more disaster severity. This measure was developed for this study, but severity of trauma exposure is a potent predictor of post-trauma symptoms (Lonigan et al., 1994; Thabet & Vostanis, 1999).

Prior Trauma Exposure.—Adolescents reported at the first time point whether (yes/no) they had experienced traumatic events, including a serious accident; another disaster; physical, sexual, or some other type of assault; seen a family member hurt badly, or seen someone in town be hurt badly (i.e., total of five items). On average, adolescents reported experiencing one other trauma in their lifetimes (possible and actual range: 0–5). Higher scores indicated more prior potentially traumatic events. Cronbach's α for this scale was .46; we include this value to be as consistent as possible with our reporting of reliability information for the other variables. Yet, it is not expected that such trauma items will

produce correlations that will result in informative internal reliability values. These questions were adapted from the Traumatic Life Events Questionnaire, which has shown good predictive validity (Kubany et al., 2000).

Parent Distress.—At all three time points, parents reported on their distress using six items from the Kessler-6 Psychological Distress Scale [K6; (Kessler et al., 2002)]. This scale assesses self-reported distress in the past month (e.g., how much of the time did you feel so sad nothing could cheer you up?). Sum score composites were created at each of the three time points (eight-months post-tornado: possible and actual range: 0–24; Cronbach's α: .85; 12-months post-tornado: possible and actual range: 0–24; Cronbach's α: .84; 20-months post-tornado: possible range: 0–24, actual range: 0–20; Cronbach's α: .81). Higher scores indicated greater self-reported parent distress.

Parent-Adolescent Conflict and Communication.—Adolescents reported on their relationship with their parent/caregiver that participated in the study. At all three time points, adolescents were administered 20 (yes/no) items from the Conflict Behavior Questionnaire [CBQ; (Prinz, Foster, Kent, & O'Leary, 1979)] that ask about their relationships with their caregiver (e.g., "parent listens to me").

Intervention Group.—Parents and adolescents who were interviewed at the first time point were asked to participate in activities within an online web portal. Families were randomly assigned to one of three conditions: 1). *An evidence-based behaviorally-focused web-based intervention* (BI), which involved parents and adolescents participating in modules that provided psychoeducation about the impact of trauma, 2). *The behavioral intervention + parent self-help* (BI + self-help), which was identical to the behaviorally-focused intervention condition except that parents were also provided behaviorally-focused skills to improve their own mental health symptoms, and 3). *Assessment only* in which no feedback was provided. Those in the active conditions showed improved PTSD and depressive symptoms, compared to the assessment only condition, in a previously published study using this dataset (Ruggiero et al., 2015). Two dummy variables were created and included as covariates in analyses to compare the BI condition to the assessment only condition and the BI + self-help condition to the assessment only condition.

Statistical Analyses

To examine the structure of parent-child conflict-communication items within and across the time points, confirmatory factor analyses were conducted. To test the main substantive questions posed in this study, an autoregressive, cross-lagged model was developed and fit to the longitudinal response data, using the Weighted Least Squares Mean and Variance Adjusted (WLSMV) estimator. Both the measurement portion of the analyses and structural equation modeling were conducted in Mplus Version 7 (Muthen & Muthen, 2010). Of the 1,271 individuals included in the data analyses, 1,045 (82.2%) were retained at the second time point (i.e., 12-months post-disaster, which was 4 months after the first time point) and 519 (40.8%) were retained at the third time point (i.e., 20-months post-disaster, 12 months after the first time point). Missing data on endogenous variables were estimated as a function

of the observed exogenous variables under the missingness at random assumption (Schafer & Graham, 2002).

Factor Analyses of Parent-Child Conflict-Communication Items.—Separate confirmatory factor analyses (CFA) were carried out for the 20 items in the parent-child conflict-communication inventory at each of the three time points to evaluate the dimensionality of the measurement structure. Consistent with prior research examining the unidimensional structure of the binary parent-child conflict-communication (e.g., Andrews, Hops, Duncan, 1997; Fuligni, 1998), a single common factor adequately accounted for the associations among the 20 parent-child conflict-communication items at each of the three time points (time point 1: CFI = 0.97, RMSEA = 0.04 [*CI*: 0.041–0.05]; time point 2: CFI = 0.98, RMSEA = 0.05 [*CI*: 0.04–0.053]; time point 3: CFI = 0.97, RMSEA = 0.05 [*CI*: 0.043–0.058]). One item, item number two, was found to function relatively poorly displaying weak discrimination (i.e., a relatively small factor loading) when defining individual differences on the parent-child communication-conflict latent scale. This item ("We end arguments calmly") was dropped from further modeling.

To evaluate the equivalence of the single factor parent-child conflict-communication construct defined by the 19-item set across the 3 time points, measurement invariance (MI) testing was conducted. The details of the longitudinal measurement invariance testing can be found in Appendix A.

Data Analytic Plan for Testing Cascading Effects.—To test the specified study hypotheses, a cascade model (Masten et al., 2005) was developed and fitted to the longitudinal data. Autoregressive paths (i.e., regressing a downstream variable assessed at a later point in time on the same variable assessed at an earlier time point) for each of the constructs were included. The autoregression coefficient and its effect size is a type of "stability" index indicating how predictive individual differences assessed at one point in time are of the same variables assessed at a later point in time. Cross-lagged paths (i.e., regression a downstream variable at a later point in time on a different construct at an earlier time point) for each of the constructs were also modeled. The robust limited information WLSMV estimator was used to obtain parameter estimates. After estimating the full cascade model, additional restrictive model tests were carried out to determine whether all auto-regressive and cross-lagged paths were needed. These tests fixed the respected paths to zero one at a time to examine the overall changes in fit.

Building on the established invariant measurement model, severity of disaster exposure was added as an exogenous predictor of parent distress, adolescent PTSD symptoms, and parent-child conflict-communication at the first time point. Covariates were included when testing all of the paths predicting parent distress, adolescent PTSD symptoms, and parent-child conflict-communication at all time periods, regardless of their statistical significance. Figure 1 depicts the application of the cascade model to test our research questions.

Results

Descriptive Statistics

Table 2 provides the descriptive summary of the zero-order correlations among the primary model variables. In terms of zero-order correlations, greater disaster exposure was associated with more parental distress and more adolescent PTSD symptoms at the first time point. Higher levels of parental distress, adolescent PTSD symptoms, and more parent-child conflict-communication at the first time point were all associated with higher levels of these constructs at the second time point. Higher levels of parental distress, adolescent PTSD symptoms, and more parent-child conflict-communication at the second time point also tended to be associated with higher levels of these constructs at the third time point. In terms of descriptive information for study covariates, the mean age of participants was 14.54 years, with 48.1% of participants being female. Participants were 71.6% Caucasian, 24.9% African-American, and 3.5% Other. On average, individuals had experienced 1.06 prior traumas, meaning they had experienced about one traumatic event prior to experiencing this tornado. Individuals reported drinking alcohol on 0.15 days in the past month, meaning they reported using alcohol less than once day per month. On average, individuals experienced 0.45 depressive symptoms, meaning about one out of every two people reported one of these symptoms. On average, families reported ~$40,000-$60,000 a year for income.

Final Study Model

Several nested restrictive models were specified and tested to evaluate central directional path features of the cascade model. First, a model with all six of the autoregressive path coefficients set to zero was fit. Second, a model with all 12 cross-lagged paths set to zero was also fit. The full restricted autoregressive model would not converge even after increasing the number of iterations, which was due to attempting to constrain the strong autoregressive parent-child conflict-communication factor pathways to zero. Nevertheless, a model in which parent distress and adolescent PTSD symptoms autoregressive paths were set to zero did converge. The χ^2 difference test, comparing this model the full cascade model, produced a statistically significant χ^2 difference test ($\Delta\chi^2_{(4)} = 684.8$, $p < .001$, ΔCFI = .018, ΔRMSEA = .004), indicating that the autoregressive paths are a central feature of the model. Likewise, the χ^2 difference test was also statistically significant for the model fixing all the cross-lagged parameters to zero ($\Delta\chi^2_{(12)} = 57.9$, $p < .001$, ΔCFI = .004, ΔRMSEA = .001), indicating that at least some of the cross-lagged paths are needed.

The final study model included covariates in all paths predicting parental distress, adolescent PTSD symptoms, and parent-child conflict-communication at all three time points. Over and above covariate effects, all stability paths across all three constructs were positively and significantly associated. Additionally, greater severity of disaster exposure was associated with increased parental distress and more adolescent PTSD symptoms at the first time point. Higher levels of initial parental distress were associated with more adolescent PTSD symptoms at the second time point. Initial adolescent PTSD symptoms predicted more parent distress and parent-child conflict-communication at the second time point. Parent-child conflict-communication at the first and second time point were associated with adolescent PTSD symptoms at the second and third time points, respectively. Of note, parent

distress and parent-child conflict-communication were not associated with each other in either direction at any time points. The final study model showed good fit to the data: CFI = .97 and RMSEA = .01 (95% CI: .01-.014). R^2 values provide a common metric indexing the percent of the total variance accounted for in an outcome variable by all predictors that have an asymmetric arrow pointing at it. Specifically, the r^2 for parent distress, adolescent PTSD symptoms, and parent-child conflict-communication were .27, .49, .20 (1), .53, .29, .75 (2), and .60, .46, and .76 (3) at the first second and third time points, respectively. For example, all predictors together explained 27% of the variance in parent distress at the first time point. Standardized betas and the standard errors for the coefficients for the final model results are presented in Tables 3 and 4. See Figure 1 for significant, hypothesized effects for the final study model.

In terms of notable covariate effects, family income was a significant predictor of parental distress at the first two time points, such that those with higher incomes reported less parent distress. Additionally, depressive symptoms predicted parent distress at the first and third time points, adolescent PTSD symptoms at all time points, and parent-child conflict-communication at the first time point. Specifically, more depressive symptoms were predictive of more parent distress, more adolescent PTSD symptoms, and poorer parent-child conflict-communication. Adolescent alcohol use was largely not associated with constructs of primary interest at any time point, with the exception of baseline adolescent PTSD symptoms. Specifically, those consuming more alcohol also reported more PTSD symptoms at baseline.

Post-hoc, Supplemental Analyses

Although we view alcohol and depression as key covariates, given their associations with PTSD in the larger literature, we wanted to determine to what extent their inclusion in the model contributed to the findings of the original specification. Of note, all key findings remained the same, with one exception. Specifically, while greater disaster severity was significantly associated with more adolescent PTSD symptoms at baseline initially ($p < .05$), this effect fell below the conventional p-value cutoff level when alcohol use and depression were omitted ($p = 08$). Thus, this effect is weakened when the partial associations between depression and alcohol use with PTSD are not considered.

Discussion

The current study used a dynamic cascade conceptualization to examine the associations among disaster exposure, parental distress, adolescent PTSD symptoms, and parent-child conflict-communication in a sample of disaster-exposed youth. Our hypotheses were partially supported. The dynamic cascade approach helped explicate findings regarding post-disaster adolescent mental health.

The first hypothesis stated that greater disaster exposure would be associated with more parental distress, adolescent PTSD symptoms, and poorer conflict-communication at baseline. Although we cannot discern the direction of effect, as hypothesized, disaster exposure was significantly associated with parental distress and adolescent PTSD symptoms. Yet, there was no association between disaster exposure and parent-child

conflict-communication (see Table 3, Figure 1). This finding is perhaps because post-trauma reactions are impacting each member of the family individually and have not yet disrupted conflict-communication, as individuals may be pooling resources to support the family. Indeed, some findings indicate improved adolescent-caregiver relations following disaster (Uttervall et al., 2014). Although others find post-disaster families report more conflict, irritability, and withdrawal (McFarlane, 1987), this study did not include measures of offspring mental health in their models, so it may be that the impact of disaster on conflict/less communication is explained by child symptomatology. Our findings underscore the importance of including measures at the parent-, youth-, and family-level of analysis.

The second hypothesis involved testing whether more adolescent PTSD symptoms were associated with subsequent adolescent PTSD symptoms, and, conversely, whether more parental distress was associated with subsequent parental distress, across all three timepoints. More parental distress at the first time point was associated with more adolescent PTSD symptoms at the second time point, and greater adolescent PTSD symptoms at the first time point were associated with more parental distress at the second. Yet, neither construct predicted the other between the second and third time points (see Tables 3 and 4, Figure 1). The effect of parent distress on adolescent PTSD is consistent with prior work finding that parents' functioning post disaster impacts adolescent PTSD symptoms (Green et al., 1991; Hafstad et al., 2010), and that improving parent mental health has positive, distal effects on child behavior (Neece, 2014). The effect of adolescent PTSD on parent distress is consistent with work suggesting that child psychopathology exerts significant effects on parental mental health functioning (Raposa, Hammen, & Brennan, 2011), although the body of research focusing on child effects on parent mental health is much smaller. Based on these findings, it appears that these inter-relations are most pronounced more soon after disaster. It may also be that the associations between parent distress and adolescent PTSD symptoms weaken over time because individuals adopt various coping strategies (e.g., either active such as seeking out new sources of support, or passive, such as avoidance) in an attempt to adapt to their new circumstances.

Findings from the exploratory analyses indicated that poorer parent-child conflict-communication prospectively predicted more adolescent PTSD symptoms at both time points, and that adolescent PTSD symptoms at the first time point predicted parent-child conflict-communication at the second time point (see Tables 3 and 4, Figure 1). This work adds to a small cross-sectional (Bokszczanin, 2008) and longitudinal literature (Boney-McCoy & Finkelhor, 1996) finding that parent-child relationships impact adolescent PTSD symptoms following disaster and interpersonal trauma, respectively. To our knowledge, there are no studies to date examining the prospective impact of adolescent PTSD symptoms following disaster on family level constructs. Thus, our findings add to work suggesting that the mental health of adolescents impacts their relationships they have with their caregivers, and that adolescent mental health and adolescent-caregiver relationships reciprocally influence one another following disaster.

Of note, parent distress and parent-child conflict-communication were not associated with one another at any time points (see Tables 3 and 4, Figure 1). As these constructs were significantly associated with one another in the zero-order correlations (see Table 2), it may

be that their inter-relations are better explained by adolescent PTSD symptoms post-disaster and/or covariates. More research is warranted examining the associations between parent symptomology, distress, and parent-child conflict-communication, particularly taking into account additional pre-disaster risk factors (e.g., anxiety symptoms, health status; Cao, Jiang, Li, Lo, & Li, 2013; McDermott & Cobham, 2012).

In terms of covariate effects, individuals who reported more depressive symptoms reported more PTSD symptoms. Individuals with more depressive symptoms also had parents who reported more distress, and who reported poorer parent-child conflict-communication (see Tables 3 and 4). These findings extend prior work (Goenjian et al., 2005; Vrana & Lauterbach, 1994) by suggesting that depression is a risk factor for future PTSD symptoms and parent distress. Additionally, adolescents with more depressive symptoms at the first time point had parents reporting poorer parent-child conflict-communication also at the first time point. Yet, as this effect did not extend to later time points, it is unclear how robust this effect is. Additionally, those with higher reported family incomes had parents reporting less distress. It may be that financial resources serve to buffer the effect of a stressor on parent mental health, but because we do not have longitudinal data on family income, we cannot confirm the direction of this effect. More research in this area is needed to further clarify these associations.

Strengths, Limitations, and Implications

This study has several important strengths. First, it utilized a cascade methodology, meaning that constructs are measured at multiple time points, improving our ability to understand the relative strength of these interrelations across time. We incorporated measures of parent-, adolescent-, and family-level symptomatology, to test whether these interrelated, but unique factors may impact the other areas of functioning. Because post-disaster associations were examined, we are more confident that pre-disaster risk factors were not driving the associations between trauma exposure and parent, adolescent, and conflict-communication.

Despite methodological strengths, limitations are important to note. Although PTSD symptoms since the baseline interview were assessed, these symptoms may not be a direct result of the tornado. Second, although parents reported on their distress and the severity of disaster exposure experienced by the family, adolescents reported on conflict-communication and PTSD symptoms. There may be shared reporter bias between severity of disaster and parent distress, and adolescent PTSD symptoms and parent-child conflict-communication. The larger study did not collect data on youth report of severity of exposure, so it is not possible to compare parent and youth perception of disaster severity. One potentially unmeasured confounder is parent PTSD status (or other psychopathology). Specifically, it may be that parents with PTSD, for example, perceived greater disruption following the disaster, were more likely to create/escalate conflictual situations and withdraw from adolescents, as well as pass on some inherited vulnerability for adolescent PTSD. Although parental distress and PTSD are likely related, because we did not measure parent PTSD, we unfortunately cannot rule out this explanation. More work is needed to test this alternative explanation. In addition, the current study included reports from adolescents and one parent/caregiver who participated in the study. It is possible that interactions

involving the unmeasured parent may have impacted the processes being studied. Additionally, the initial time point was 8 months after the tornado. It is certainly possible that the associations among study constructs would have been stronger had the study began closer in time to the disaster. In addition, these findings may not generalize to studies of other types of disaster exposure. Additionally, as there was considerable overlap in the ages of adolescents at the different assessments, we are unable to discuss how these constructs are associated for individuals across specific age ranges. The current study only examined individual- and family-level factors, and did not consider the social community and other collective-level events or impacts. There is innovative research now examining social networks within the study of disasters (e.g., Brewin, Andrews, & Valentine, 2000; Norris et al., 2008; Varda, 2009; Wind & Komproe, 2012), and future work would benefit from examining these individual- and familial-level factors within the context of the community and other collective-level factors. Finally, while it is possible that in addition to the mean levels of these constructs being associated across time, intercept and growth rate in these parameters may also be interrelated.

This study helps to clarify the associations between parent distress, adolescent PTSD symptoms, and conflict-communication following a natural disaster. In particular, severity of tornado exposure has adverse effects on parent distress and adolescent PTSD symptoms, and parent distress and adolescent PTSD impact one another within the first year after a disaster. While adolescent PTSD symptoms only impact parent-child conflict-communication within the first year, family functioning predicts adolescent PTSD symptoms up to 20 months later.

Results of this study suggest that, eight to 20 months following a natural disaster, efforts aimed at providing tangible supplies and supports (e.g., housing, food, transportation, and essential possessions) to those who have experienced greater exposure may be beneficial at reducing parents' distress and adolescent PTSD symptoms. These findings bode well for non-immediate disaster relief efforts. Results also suggest possible revisions to existing interventions that already exist for post-disaster communities (e.g., Jose, 2019; Kar, 2009), for example, providing instruction for parents regarding ways to manage their own distress and thus possibly help to reduce the likelihood that their children are aware of the distress they themselves are experiencing. Such proposed changes to existing interventions may enhance treatment effects related to reducing parent and adolescent post-disaster stress responses.

Conclusions

These findings have important substantive and methodological conclusions. First, the results highlight the inter-related nature of parent distress, adolescent PTSD symptoms, and parent-child conflict-communication. Interestingly, the associations between parent and adolescent functioning, and adolescent functioning and parent-child conflict-communication were stronger than those between parent functioning and parent-child conflict-communication. Additionally, the impact of severity of disaster exposure was most detrimental for adolescent and parent functioning, and was not associated with parent-child-conflict-communication. Finally, as this study employed a longitudinal design, measuring multiple constructs on three occasions, one has more confidence that there are temporal relations among study

constructs. Additional work investigating the aftermath of natural disasters would benefit from the use of this cascade methodology so that the unique and prospective associations at these different levels of analysis (i.e., parent, child, parent-child relationship) can be determined.

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Appendix A

An important set of parameters in the full longitudinal cascade model are the autoregressive paths between the same variables and constructs at the different assessment waves. To be able to interpret these autoregressive effects unambiguously, it is important that the constructs are being equivalently defined at each of the different assessments. Because the conflict-communication construct was modeled as a latent common factor, it was possible to evaluate measurement invariance (MI). Because all the conflict-communication indicator variables were coded as binary (0 = no, 1 = yes), only a joint test of both weak/metric (factor loadings) and strong/scalar (item thresholds) invariance could be assessed (Liu et al., 2017). Both forms of measurement invariance should hold to be assured that individiual differences for the conflict-communication latent factor are defined in the “same way” at each time point. If MI cannot be demonstrated, the interpretation of the autoregressive coefficients can be compromised due to confounding introduced by the non-invariance characteristics of the measurement models.

An initial baseline model was fit allowing all factor loadings and item thresholds (one per conflict-communication indicator) to be freely estimated for each of the factor indicators at each of the three time points. The necessary factor model identification specifications were satisfied by fixing the factor mean and variance to 0 and 1, respectively. This adjustment allows for a full invariance test of the item factor loadings, which is not possible if the factor model identification is specified by fixing one of the factor loadings at each time point to 1. Correlations among the residuals for the same item across the three waves were allowed. In this application, strict MI (i.e., forcing the item uniquenesses to be invariant for the same items at the different time points) was not tested. Since item uniquenesses in theory are made up of both reliable item-specific variance as well as random measurement error, forcing invariance on these components of the measurement model is extreme and very restrictive and only makes sense in certain types of applications (e.g., when such measurement models are used to make high stakes decsions such as admitting students in to college or job hiring situations). For purposes in this application to assure that the autoregressive paths could be interpreted as predictive effects of the “same” construct at different time points, tests of metric/weak and strong/scalar invariance were adequate.

The baseline joint longitudinal model for the parent-child conflict-communication factor at the 3 different time points fit the data well (CFI = 0.98, RMSEA = 0. 016 (95% CI: 0.014–

0.018)). To test for invariance of the 19 parent-child conflict-communication items across the three time points, the following restrictive constraints were imposed. All factor loadings and item thresholds were forced to be equal for each item. Factor means, variances and scaling factors for the second and third time points were allowed to be freely estimated. This specification provides estimates of any mean and variance differences at time points 2 and 3 compared to the fixed 0 and 1 mean and variance at the first time point. This specification isolates and removes possible confounding due to non-invariance functioning in the measurement model (i.e., factor loadings and item thresholds) part from legitimate changes in the common factor means and variance as well as from differences in the underlying unobserved response liabilities used to obtain estimates of the single thresholds for each of the binary indicator items. A χ^2difference test appropriate for the limited information WLSMV estimators in Mplus was used to evaluate this restricted MI model testing the joint invariance of the factor loadings and thresholds ($\Delta\chi^2(34) = 37.5$, $p = 0.3138$, CFI = 0.98, RMSEA = 0. 016 (95% CI: 0.014–0.018)). Based on these MI restrictive model test results, there was no statistical evidence indicating that the parent-child conflict-communication common factor was being differentially defined due to change in the measurement properties (i.e., factor loadings and item thresholds) at the three different time points.

References

Adams ZW, Danielson CK, Sumner JA, McCauley JL, Cohen JR, & Ruggiero KJ (2015). Comorbidity of PTSD, Major Depression, and Substance Use Disorder Among Adolescent Victims of the Spring 2011 Tornadoes in Alabama and Joplin, Missouri. Psychiatry, 78(2), 170–185. doi:10.1080/00332747.2015.1051448 [PubMed: 26168094]

Andrews B, Brewin CR, Rose S, & Kirk M. (2000). Predicting PTSD symptoms in victims of violent crime: the role of shame, anger, and childhood abuse. J Abnorm Psychol, 109(1), 69–73. doi:10.1037//0021-843x.109.1.69 [PubMed: 10740937]

Andrews J, Hops H, Duncan S. (1997). Adolescent modeling of parent substance use: The moderating effect of the relationship with the parent. Journal of Family Psychology, 11(3), 259–270.

Betancourt TS, Agnew-Blais J, Gilman SE, Williams DR, & Ellis BH (2010). Past horrors, present struggles: the role of stigma in the association between war experiences and psychosocial adjustment among former child soldiers in Sierra Leone. Soc Sci Med, 70(1), 17–26. doi:10.1016/j.socscimed.2009.09.038 [PubMed: 19875215]

Bokszczanin A. (2008). Parental support, family conflict, and overprotectiveness: predicting PTSD symptom levels of adolescents 28 months after a natural disaster. Anxiety Stress Coping, 21(4), 325–335. doi:10.1080/10615800801950584 [PubMed: 18686053]

Boney-McCoy S, & Finkelhor D. (1996). Is youth victimization related to trauma symptoms and depression after controlling for prior symptoms and family relationships? A longitudinal, prospective study. J Consult Clin Psychol, 64(6), 1406–1416. [PubMed: 8991327]

Brewin CR (2015). Re-experiencing traumatic events in PTSD: new avenuesin research on intrusive memories and flashbacks. Eur J Psychotraumatol, 6, 1–5.

Brewin CR, Andrews B, & Valentine JD (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol, 68(5), 748–766. doi:10.1037//0022-006x.68.5.748 [PubMed: 11068961]

Brewin CR, Andrews B, & Valentine JD (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol, 68(5), 748–766. [PubMed: 11068961]

Bryant RA, Gallagher HC, Gibbs L, Pattison P, MacDougall C, Harms L, … Lusher D. (2017). Mental Health and Social Networks After Disaster. Am J Psychiatry, 174(3), 277–285. doi:10.1176/appi.ajp.2016.15111403 [PubMed: 27838935]

Cao X, Jiang X, Li X, Hui Lo MC, & Li R. (2013). Family functioning and its predictors among disaster bereaved individuals in China: eighteen months after the Wenchuan Earthquake. PLoS One, 8(4), e60738. doi:10.1371/journal.pone.0060738

Chemtob CM, Nomura Y, Rajendran K, Yehuda R, Schwartz D, & Abramovitz R. (2010). Impact of maternal posttraumatic stress disorder and depression following exposure to the September 11 attacks on preschool children's behavior. Child Dev, 81(4), 1129–1141. doi:10.1111/j.1467-8624.2010.01458.x [PubMed: 20636686]

Cohen J. (1992). A power primer. Psychol Bull, 112(1), 155–159. [PubMed: 19565683]

Felix E, Afifi T, Kia-Keating M, Brown L, Afifi W, & Reyes G. (2015). Family functioning and posttraumatic growth among parents and youth following wildfire disasters. Am J Orthopsychiatry, 85(2), 191–200. doi:10.1037/ort0000054 [PubMed: 25822609]

Forman EM, & Davies PT (2003). Family instability and young adolescent maladjustment: the mediating effects of parenting quality and adolescent appraisals of family security. J Clin Child Adolesc Psychol, 32(1), 94–105. doi:10.1207/s15374424jccp3201_09 [PubMed: 12573935]

Fuligni AJ (1998). Authority, autonomy, and parent-adolescent conflict and cohesion: a study of adolescents from Mexican, Chinese, Filipino, and European backgrounds. Dev Psychol, 34(4), 782–792. [PubMed: 9681270]

Goenjian AK, Walling D, Steinberg AM, Karayan I, Najarian LM, & Pynoos R. (2005). A prospective study of posttraumatic stress and depressive reactions among treated and untreated adolescents 5 years after a catastrophic disaster. Am J Psychiatry, 162(12), 2302–2308. doi:10.1176/appi.ajp.162.12.2302 [PubMed: 16330594]

Green BL, Korol M, Grace MC, Vary MG, Leonard AC, Gleser GC, & Smitson-Cohen S. (1991). Children and disaster: age, gender, and parental effects on PTSD symptoms. J Am Acad Child Adolesc Psychiatry, 30(6), 945–951. doi:10.1097/00004583-199111000-00012 [PubMed: 1757444]

Hafstad GS, Gil-Rivas V, Kilmer RP, & Raeder S. (2010). Parental adjustment, family functioning, and posttraumatic growth among Norwegian children and adolescents following a natural disaster. Am J Orthopsychiatry, 80(2), 248–257. doi:10.1111/j.1939-0025.2010.01028.x [PubMed: 20553518]

Haller M, Handley E, Chassin L, & Bountress K. (2010). Developmental cascades: linking adolescent substance use, affiliation with substance use promoting peers, and academic achievement to adult substance use disorders. Dev Psychopathol, 22(4), 899–916. doi:10.1017/s0954579410000532 [PubMed: 20883589]

Hasin DS, Keyes KM, Hatzenbuehler ML, Aharonovich EA, & Alderson D. (2007). Alcohol consumption and posttraumatic stress after exposure to terrorism: effects of proximity, loss, and psychiatric history. Am J Public Health, 97(12), 2268–2275. doi:10.2105/ajph.2006.100057 [PubMed: 17971553]

Jose R, Holman EA, & Silver RC (2019). Community organizations and mental health after the 2013 Boston Marathon bombings. Social Science & Medicine, 222.

Kar N. (2009). Psychological impact of disasters on children: review of assessment and interventions. World J Pediatr, 5(1), 5–11. doi:10.1007/s12519-009-0001-x [PubMed: 19172325]

Kerns CE, Elkins RM, Carpenter AL, Chou T, Green JG, & Comer JS (2014). Caregiver distress, shared traumatic exposure, and child adjustment among area youth following the 2013 Boston Marathon bombing. J Affect Disord, 167, 50–55. doi:10.1016/j.jad.2014.05.040 [PubMed: 25082114]

Kessler RC, Andrews G, Colpe LJ, Hiripi E, Mroczek DK, Normand SL, … Zaslavsky AM (2002). Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol Med, 32(6), 959–976. [PubMed: 12214795]

Kilpatrick DG, Acierno R, Resnick HS, Saunders BE, & Best CL (1997). A 2-year longitudinal analysis of the relationship between violent assault and substance abuse in women. J Consult Clin Psychol, 65, 834–847. [PubMed: 9337502]

Kilpatrick DG, Ruggiero KJ, Acierno R, Saunders BE, Resnick HS, & Best CL (2003). Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: results from the National Survey of Adolescents. J Consult Clin Psychol, 71(4), 692–700. [PubMed: 12924674]

Kubany ES, Leisen MB, Kaplan AS, Watson SB, Haynes SN, Owens JA, & Burns K. (2000). Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: the Traumatic Life Events Questionnaire. Psychological assessment, 12(2), 210. [PubMed: 10887767]

Liu Y, Millsap RE, West SG, Tein JY, Tanaka R, & Grimm KJ (2017). Testing measurement invariance in longitudinal data with ordered-categorical measures. Psychol Methods, 22(3), 486–506. doi:10.1037/met0000075 [PubMed: 27213981]

Lonigan CJ, Shannon MP, Taylor CM, Finch AJ Jr., & Sallee FR (1994). Children exposed to disaster: II. Risk factors for the development of post-traumatic symptomatology. J Am Acad Child Adolesc Psychiatry, 33(1), 94–105. [PubMed: 8138526]

Martin RJ, Brechbiel K, Chaney BH, Cremeens-Matthews J, & Vail-Smith K. (2016). Alcohol-related injuries, hazardous drinking, and BrAC levels among a sample of bar patrons. Am J Addict, 25(2), 132–137. doi:10.1111/ajad.12340 [PubMed: 26824504]

Masten AS, Roisman GI, Long JD, Burt KB, Obradovic J, Riley JR, … Tellegen A. (2005). Developmental cascades: linking academic achievement and externalizing and internalizing symptoms over 20 years. Dev Psychol, 41(5), 733–746. doi:10.1037/00121649.41.5.733 [PubMed: 16173871]

McDermott BM, & Cobham VE (2012). Family functioning in the aftermath of a natural disaster. BMC Psychiatry, 12, 55. doi:10.1186/1471-244x-12-55 [PubMed: 22647086]

McFarlane AC (1987). Posttraumatic phenomena in a longitudinal study of children following a natural disaster. J Am Acad Child Adolesc Psychiatry, 26(5), 764–769. doi:10.1097/00004583-198709000-00025 [PubMed: 3667509]

Muthen B, & Muthen L. (2010). Mplus Version 6.0. Los Angeles, CA: Muthen & Muthen. National Oceanic and Atmospheric Association (NOAA) Tornadoes – annual 2011. NOAA National Climatic Data Center (2012). Retrieved from http://www.ncdc.noaa.gov/sotc/tornadoes/2011/13.

Neece CL (2014). Mindfulness-based stress reduction for parents of young children with developmental delays: implications for parental mental health and child behavior problems. J Appl Res Intellect Disabil, 27(2), 174–186. doi:10.1111/jar.12064 [PubMed: 23813562]

Neria Y, Nandi A, & Galea S. (2008). Post-traumatic stress disorder following disasters: a systematic review. Psychol Med, 38(4), 467–480. doi:10.1017/s0033291707001353 [PubMed: 17803838]

Nilsen W, Skipstein A, & Demerouti E. (2016). Adverse trajectories of mental health problems predict subsequent burnout and work-family conflict - a longitudinal study of employed women with children followed over 18 years. BMC Psychiatry, 16(1), 384. doi:10.1186/s12888-016-1110-4 [PubMed: 27825325]

Norris FH, Stevens SP, Pfefferbaum B, Wyche KF, & Pfefferbaum RL (2008). Community resilience as a metaphor, theory, set of capacities, and strategy for disaster readiness. Am J Community Psychol, 41(1–2), 127–150. doi:10.1007/s10464-007-9156-6 [PubMed: 18157631]

Obradovic J, Burt KB, & Masten AS (2010). Testing a dual cascade model linking competence and symptoms over 20 years from childhood to adulthood. J Clin Child Adolesc Psychol, 39(1), 90–102. doi:10.1080/15374410903401120 [PubMed: 20390801]

Paul L, Felton JW, Adams ZW, Welsh K, Miller S, & Ruggiero KJ (2015). Mental Health Among Adolescents Exposed to a Tornado: The Influence of Social Support and Its Interactions With Sociodemographic Characteristics and Disaster Exposure. Journal of Traumatic Stress 28(3), 232–239. doi:doi: 10.1002/jts.22012 [PubMed: 26031997]

Prinz RJ, Foster S, Kent RN, & O'Leary KD (1979). Multivariate assessment of conflict in distressed and nondistressed mother-adolescent dyads. J Appl Behav Anal, 12(4), 691–700. doi:10.1901/jaba.1979.12-691 [PubMed: 541311]

Raker EJ, Lowe SR, Arcaya MC, Johnson ST, Rhodes J, & Waters MC (2019). Twelve years later: The long-term mental health consequences of Hurricane Katrina. Social Science & Medicine, 242.

Raposa EB, Hammen CL, & Brennan PA (2011). Effects of child psychopathology on maternal depression: the mediating role of child-related acute and chronic stressors. J Abnorm Child Psychol, 39(8), 1177–1186. doi:10.1007/s10802-011-9536-0 [PubMed: 21735051]

Ruggiero K, Price M, Adams Z, Stauffacher K, McCauley J, Danielson CK, Knapp R, Hanson RF, Davidson T, Amstadter AB, Carpenter MJ, Saunders BE, Kilpatrick DG, & Resnick HS (2015).

Web intervention for adolescents affected by disaster: Population-based randomized controlled trial. Journal of the American Academy of Child & Adolescent Psychiatry., 54(9), 709–717. [PubMed: 26299292]

Ruggiero KJ, Davidson TM, McCauley J, Gros KS, Welsh K, Price M, … Amstadter AB (2015). Bounce Back Now! Protocol of a population-based randomized controlled trial to examine the efficacy of a Web-based intervention with disaster-affected families. Contemp Clin Trials, 40, 138–149. doi:10.1016/j.cct.2014.11.018 [PubMed: 25478956]

Schafer JL, & Graham JW (2002). Missing data: our view of the state of the art. Psychol Methods, 7(2), 147–177. [PubMed: 12090408]

Statista. (2018). The 10 most significant natural disasters worldwide by death toll from 1980 to 2017. Retrieved from https://www.statista.com/statistics/268029/natural-disasters-by-death-toll-since-1980/

Thabet AA, & Vostanis P. (1999). Post-traumatic stress reactions in children of war. J Child Psychol Psychiatry, 40(3), 385–391. [PubMed: 10190340]

Trickey D, Siddaway AP, Meiser-Stedman R, Serpell L, & Field AP (2012). A meta-analysis of risk factors for post-traumatic stress disorder in children and adolescents. Clin Psychol Rev, 32(2), 122–138. doi:10.1016/j.cpr.2011.12.001 [PubMed: 22245560]

Uttervall M, Hultman CM, Ekerwald H, Lindam A, & Lundin T. (2014). After the flood: resilience among tsunami-afflicted adolescents. Nord J Psychiatry, 68(1), 38–43. doi:10.3109/08039488.2013.767373 [PubMed: 23445215]

Varda DM, Forgette R, Banks D, & Contractor N. (2009). Social network methodology in the study of disasters: Issues and insights prompted by post-Katrina research. Population Research and Policy Review., 28(1), 11–29.

Weaver TL, & Clum GA (1995). Psychological distress associated with interpersonal violence: A meta-analysis. Clinical Psychology Review, 15(2), 115–140.

Weiss JA, Cappadocia MC, MacMullin JA, Viecili M, & Lunsky Y. (2012). The impact of child problem behaviors of children with ASD on parent mental health: the mediating role of acceptance and empowerment. Autism, 16(3), 261–274. doi:10.1177/1362361311422708 [PubMed: 22297202]

Wickrama KA, & Kaspar V. (2007). Family context of mental health risk in Tsunami-exposed adolescents: Findings from a pilot study in Sri Lanka. Soc Sci Med, 64(3), 713–723. doi:10.1016/j.socscimed.2006.09.031 [PubMed: 17084953]

Wind TR, & Komproe IH (2012). The mechanisms that associate community social capital with post-disaster mental health: A multilevel model. Soc Sci Med, 75(9), 1715–1720. doi:10.1016/j.socscimed.2012.06.032 [PubMed: 22883254]

Highlights

- Families enduring more severe disasters had family members with more distress/PTSD.
- Following a disaster, there was an association between parental distress and adolescent PTSD.
- There was an association between family conflict/communication and adolescent PTSD.
- Family conflict/communication and parent distress were unrelated.

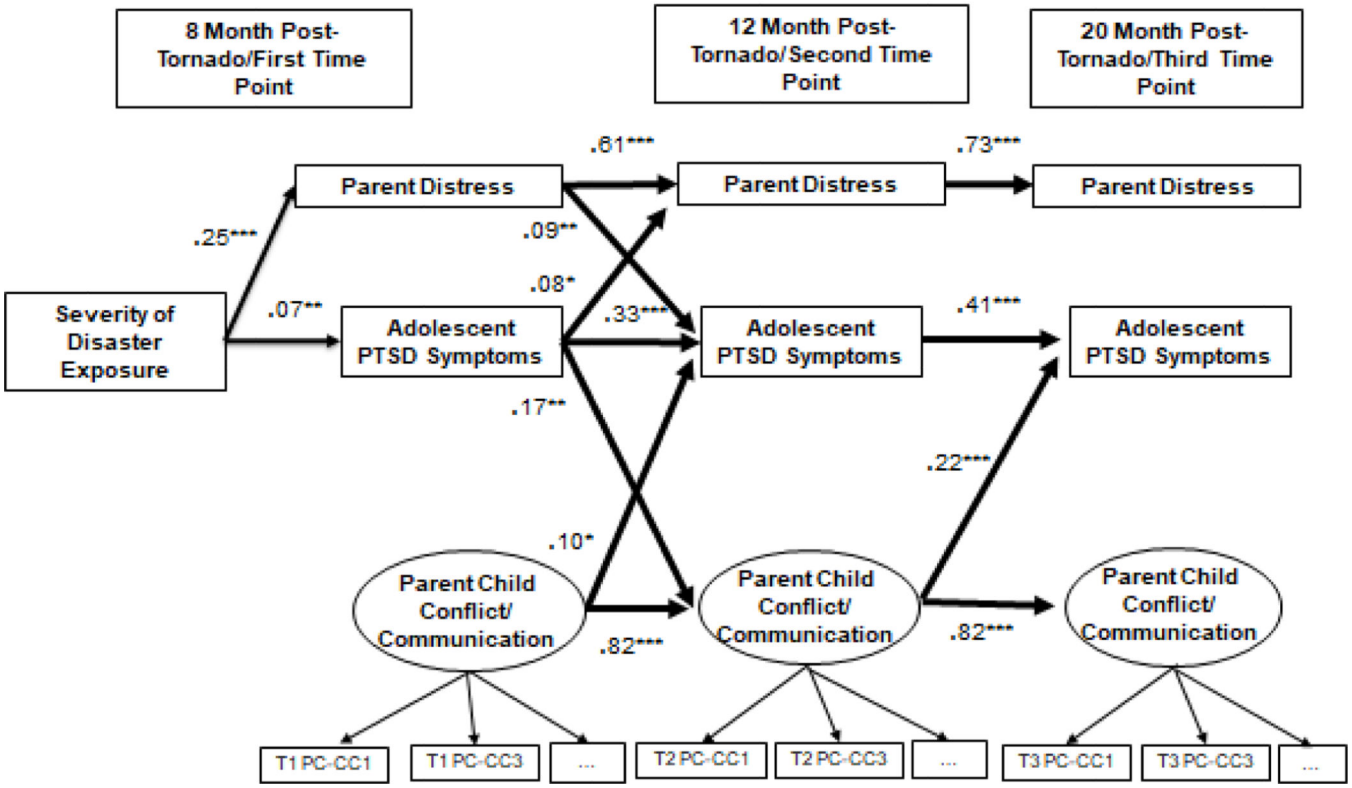

Fig. 1.

Final study model.

Note. Solid black lines indicate significant effects (*p<.05, **p<.01, ***p<.001); non-significant effects among primary study variables are omitted for ease of presentation. Additionally, effects of covariates (i.e., adolescent age, race, gender, treatment, number of traumatic events, household income, adolescent alcohol use and depressive symptoms) and inter-correlations of main constructs within time were included, though not displayed here.

Table 1

Exploring differences between those who were included in (*N*=1271) and those who were excluded from (*N*=729) analyses.

Construct	Mean (*SD*): Included, Excluded	*t* or χ^2 value (*p* value); effect size if significant
Age	14.55 (1.75), 14.61 (1.75)	0.653 (*ns*)
Sex	**48.1% Female, 52.9% Female**	**3.513 (*p*<.05); Cramer's *V*: 0.04**
Race	71.6% Caucasian, 73.6% Caucasian	0.324 (*ns*)
Income	**3.18 (1.91), 2.90 (1.91)**	**−2.481 (*p*<.05); Cohen's *d*: 0.14**
Alcohol Use	0.15 (0.87), 0.10 (0.58)	−1.376 (*ns*)
Depression	0.45 (0.92), 0.39 (0.87)	−1.439 (*ns*)
Prior Traumas	1.06 (1.16), 1.07 (1.12)	0.104 (*ns*)
Severity of Exposure	**2.15 (1.77), 2.54 (1.16)**	**3.391 (*p*<.001); Cohen's *d*: 0.26**
First -Parent Distress	5.14 (5.08), 5.30 (5.29)	0.623 (*ns*)
First - A. PTSD	1.27 (2.27), 1.27 (2.21)	0.011 (*ns*)
First – PC-CC	0.13 (0.78), 0.15 (0.78)	0.242 (*ns*)
Sec- Parent Distress	4.77 (4.42), 4.85 (4.58)	0.267 (*ns*)
Sec-A. PTSD	1.22 (2.49), 1.42 (2.83)	1.055 (*ns*)
Sec-PC-CC	**0.21 (0.75), 0.05 (0.78)**	**−2.305 (*p*<.05); Cohen's *d*: 0.21**
Third- Parent Distress	4.64 (4.13), 4.50 (4.50)	−0.452 (*ns*)
Third-A. PTSD	1.19 (2.46), 1.29 (2.75)	0.462 (*ns*)
Third-PC-CC	0.17 (0.75), 0.13 (0.80)	−0.448 (*ns*)

Note. First, Sec, Third=First, Second, Third Time-Point; A.PTSD=Adolescent PTSD, PC-CC=Parent-Child Conflict-Communication.

Table 2

Correlations among Primary Study Variables (*N*=1271).

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Severity	--								
2.First -P. Dist.	.34***	--							
3. First - A. PTSD	.07**	.20***	--						
4. First – PC-CC	.08	.16***	.39***	--					
5. Sec-P. Dist.	.16***	.65***	.20***	.16***	--				
6. Sec-A. PTSD	.11*	.22***	.46***	.30***	.23***	--			
7. Sec-PC-CC	.11*	.23***	.31***	.67***	.19***	.28***	--		
8. Third-P. Dist.	.15**	.61***	.23***	.22***	.70***	.20***	.19***	--	
9. Third-A. PTSD	.05	.23***	.51***	.37***	.24***	.53***	.34***	.18***	--
10. Third-PC-CC	.11*	.20***	.37***	.62***	.14**	.30***	.61***	.13**	.39***

Note.

* $p<.05$,

** $p<.01$,

*** $p<.001$;

First, Sec, Third=First, Second, Third Time-Point; P. Dist.=Parent Distress, A.PTSD=Adolescent PTSD, PC-CC=Parent-Child Conflict-Communication.

Table 3

Standardized Betas (and SE) for Final Model Results: Part 1 of 2 (*N*=1271).

Predictor	First-P. Distr.	First-A. PTSD	First-PC-CC	Second-P. Distr.	Second-A. PTSD	Second-PC-CC
Severity of Exposure	.25*** (.02)	.07** (.02)	.07 (.05)	--	--	--
Adolescent Age	.03 (.02)	.01 (.02)	.12* (.04)	.01 (.03)	.00 (.04)	−.03 (.06)
Adolescent Gender	.00 (.03)	−.05 (.02)	−.16** (.04)	.01 (.03)	.01 (.04)	.08 (.05)
Adolescent Race	.10*** (.02)	.02 (.02)	.05 (.04)	−.03 (.03)	.02 (.04)	.05 (.05)
Treatment D. Code 1	.02 (.03)	.00 (.02)	−.04 (.05)	−.02 (.03)	−.02 (.04)	.09 (.06)
Treatment D. Code 2	.03 (.03)	.00 (.03)	.04 (.05)	−.02 (.03)	−.06 (.04)	.10 (.06)
Number of Traumas	.05 (.03)	.13*** (.02)	.22*** (.05)	.05 (.03)	.05 (.05)	−.06 (.06)
Household Income	−.34*** (.03)	−.01 (.02)	.00 (.05)	−.17*** (.03)	−.01 (.05)	−.02 (.06)
Alcohol Use	.02 (.03)	.04 (.02)	.03* (.04)	.05 (.03)	.04 (.02)	.04 (.03)
Depression	.10*** (.02)	.62*** (.01)	.22*** (.04)	−.06 (.04)	.09** (.03)	−.09 (.06)
First- P. Distr.	--	--	--	.61*** (.02)	.10** (.03)	.06 (.05)
First - A. PTSD	--	--	--	.08* (.04)	.33*** (.02)	.17* (.06)
First - PC-CC	--	--	--	.03 (.05)	.10* (.04)	.82*** (.04)

Note.

* *p*<.05,

** *p*<.01,

*** *p*<.001;

First, Sec=First, Second Time-Point; P. Distr.=Parent Distress, A.PTSD=Adolescent PTSD, PC-CC=Parent-Child Conflict-Communication.

Table 4

Standardized Betas (and SEs) for Final Model Results: Part 2 of 2 (*N*=1271).

Predictor	Third - P. Distr.	Third - A. PTSD	Third - PC-CC
Adolescent Age	.01 (.03)	−.08 (.05)	−.04 (.07)
Adolescent Gender	.01 (.03)	−.09 (.05)	−.06 (.06)
Adolescent Race	.02 (.04)	.04 (.04)	.04 (.06)
Treatment D. Code 1	.05 (.04)	−.07 (.05)	−.06 (.07)
Treatment D. Code 2	.10 (.04)	−.04 (.05)	.01 (.07)
Number of Traumas	−.01 (.07)	.12 (.04)	.00 (.07)
Household Income	−.02 (.04)	.10 (.05)	.05 (.07)
Alcohol Use	−.02 (.03)	.04 (.02)	−.07 (.04)
Depression	.08 * (.03)	.11 *** (.03)	.08 (.07)
Sec. - P. Distr.	.73 *** (.03)	.06 (.04)	.05 (.07)
Sec. - A. PTSD	−.04 (.04)	.41 *** (.03)	.05 (.06)
Sec. - PC-CC	.03 (.06)	.22 *** (.05)	.82 *** (.05)

Note.

* $p<.05$,

** $p<.01$,

*** $p<.001$;

Sec, Third=Second, Third Time-Point; P. Distr.=Parent Distress, A.PTSD=Adolescent PTSD, PC-CC=Parent-Child Conflict-Communication.