

# Longitudinal growth of post-traumatic stress and depressive symptoms following a child maltreatment allegation: An examination of violence exposure, family risk and placement type

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## ABSTRACT

Few longitudinal studies have analyzed how traumatic experiences (e.g. home removal, violence exposure) influence both depressive and Post-Traumatic Stress (PTS) symptoms in children involved with Child Protective Services (CPS). This study investigated the change trajectories of both depressive and PTS symptomatology as well as their associations over time, focusing on the effect of complex trauma. Data were obtained from the National Survey of Child and Adolescent Well-Being (1999–2007), a nationally representative study of children and adolescents who were referred to child protective services for alleged maltreatment. The Children's Depression Inventory (CDI) scale measured depressive symptoms and the Post Traumatic Stress Disorder section of a version of the Trauma Symptom Checklist for Children (TSCC) provided the measure of current trauma-related symptoms or distress. Analyses were conducted using a parallel process growth curve model. The participants' initial levels of depressive and PTS symptomatology were significantly and positively related; furthermore, any changes in these two outcomes were also correlated longitudinally. The initial assessment of PTS symptoms significantly contributed to the advancement of more severe depressive symptoms over time. No significant differences were found between youth who remained in the home and those removed from the home. However, violence exposure, sexual abuse, gender and age were significant predictors of level and rate of change in both PTS and depressive symptoms. PTS growth factors mediated the longitudinal relationship between witnessing severe violence and depressive symptoms. The findings suggest a complex developmental association between depressive and PTS symptomatology among CPS-involved youth that is rooted in early childhood experiences with complex trauma.

## 1. Introduction

The estimated prevalence of mental health disorders among CPS-involved youth ranges from 11%–80% depending on the nature of the sample. These estimates are much higher in comparison to the general youth population (0.5–25%) (Dovran, Winje, Arefjord, & Haugland, 2012; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Pecora, White, Jackson, & Wiggins, 2009; Shin, 2005). The relatively higher prevalence has been attributed to heightened exposure to complex and ongoing traumas among youth involved with child protective services. Indeed, the bulk of evidence suggests that CPS-involved youth have an increased risk of developing a variety of psychological, emotional and behavior problems (Clausen, Landsverk, Ganger, Chadwick, & Litrownik, 1998; Jee et al., 2010; Jonson-Reid, 1998; Minnis, Everett, Pelosi, Dunn, & Knapp, 2006; Ryan, Herz, Hernandez, & Marshall, 2007; S. Horwitz, M. Hurlburt, & J.

Zhang, 2010a). Across studies using diverse samples (e.g. foster care youth, young urban adults, individuals exposed to a natural disaster) and research methods, a cross-sectional association, or co-morbidity, between major depressive disorder and post-traumatic stress disorder (PTSD) has been well documented (Lawrence, Carlson, & Egeland, 2006; O'Donnell, Creamer, & Pattison, 2004). In a nationally representative study of adolescents, for example, 29% of adolescents diagnosed with a major depressive disorder (MDD) also met criteria for PTSD and 62% of PTSD cases also met criteria for MDD (Kilpatrick et al., 2003a, 2003b). The wide variation in rates of depressive and PTS symptoms among CPS-involved children may be attributed to different samples and varying methodologies that frame assessments and definitions of mental health problems. Co-morbidity between symptoms of PTS and depression has important implications for the etiology, treatment and prognosis of mental health disorders in vulnerable populations. Despite a large body of research

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establishing a cross-sectional relationship between PTSD and MDD, we know little about how these symptoms co-develop over time, particularly among at-risk populations. Consequently, the present study investigated level and change in PTS and depressive symptoms in a nationally representative sample of children and adolescents (ages 8–15) following a referral to CPS for an investigation of abuse or neglect.

Many scholars have noted that existing diagnoses do not capture the real life experiences of youth exposed to complex trauma, nor do they fully account for the severity of their behavior problems (Cook et al., 2005; Van der Kolk & d Andrea, 2010). Empirical research supports this assertion. Most studies have shown that only a small subset of trauma victims develop PTSD (Copeland, Keeler, Angold, & Costello, 2007) (Denton, Frogley, Jackson, John, & Querstret, 2016). As well, PTSD is not the most common diagnosis among youth exposed to complex trauma (Rayburn, McWey, & Cui, 2016). On this basis, Rayburn et al. (2016), have argued that co-morbid diagnoses “may unintentionally convey that different symptoms are independent from one another rather than interrelated (p. 332) (Rayburn et al., 2016).” Developmental Trauma Disorder (DTD) has been proposed as a diagnosis that takes into consideration symptom progression and co-development as causal mechanisms undergirding a single traumatic stress diagnosis for youth experiencing complex trauma. The evidentiary basis for DTD is that preexisting major depression renders individuals more vulnerable to PTSD in the aftermath of trauma (Breslau, Davis, Peterson, & Schultz, 1997; Bromet, Sonnega, & Kessler, 1998) and, conversely, the presence of PTSD increases the risk for first onset of major depression (Breslau et al., 1997; Kessler et al., 1995). The DTD perspective has support from research showing longitudinal interrelationships between growth in PTS and internalizing symptoms over time. O'Donnell et al. (2004), for example, found that while PTSD often occurs independently of depression in the short term, PTSD and depression co-occur and co-develop over the long term (O'Donnell et al., 2004). A more recent study conducted by Rayburn et al. (2016) found that the longitudinal trajectories of PTS and internalizing symptoms generally demonstrate significant sub-symptom covariation (Rayburn et al., 2016). Developmental Trauma theory posits that the co-occurrence of PTSD and MDD implies a shared vulnerability indicative of a general traumatic stress concept.

Research demonstrating the presence of similar risk factors that set in motion both depression and PTSD further supports the DTD model. For example, research has shown that among youth involved with Child Protective Services (CPS), risk factors for co-morbid PTSD and depression include a history of depression, violence exposure, lower levels of social support and gender (Campbell et al., 2007). In this context, DTD theory suggests that youth involved with child protective services are more likely than other groups to exhibit heightened PTS and depressive symptoms because they are more likely to experience a range of adversities throughout their lives. Empirical work has linked complex trauma (i.e. violence exposure and child maltreatment) to an increase in both PTS and depressive symptoms; furthermore, previous studies have demonstrated the cumulative nature of complex trauma that exacerbate PTS symptoms and, in turn, intensify depressed mood. Thus, children exposed to maltreatment, violence and/or loss of caregivers are particularly vulnerable to co-morbid diagnoses. In a nationally representative study of adolescent trauma and mental health, Kilpatrick et al. (2003a, 2003b) found that the odds of co-morbid PTSD and MDD were almost three times higher among adolescents with a history of witnessing violence, and more than twice as high among youth who were sexually or physically victimized compared to adolescents without such a history (Kilpatrick et al., 2003a, 2003b).

Several studies have documented high rates of emotional and behavioral problems among children removed from their homes (Burns et al., 2004a, 2004b; Clausen et al., 1998; Pilowsky, 1995) or who remain with their families with active child welfare cases (Berrick, Barth, & Needell, 1994; Clausen et al., 1998; Farmer et al., 2001). Although the bulk of the public policy and research attention has been

focused on children in OHC, the majority of children with substantiated or highly likely maltreatment in fact remain IHC (S. M. Horwitz, M. S. Hurlburt, & J. Zhang, 2010b). Few examinations, however, have focused on differences between children placed in alternative care settings and who remain in the home. Studies that do exist have yielded inconsistent findings. Some studies have shown that OHC is associated with better psychological and physical outcomes (Davidson-Arad, Englechin-Segal, & Wozner, 2003); others have shown that youth in OHC have worse psychological outcomes (i.e. higher levels of depression and PTSD); and others still have found no relationship between compromised mental health and type of care after controlling for differentials in family risk and violence exposure (Kolko et al., 2010). The conflicting nature of previous research suggests additional examinations of heightened PTS and depressive symptoms by placement type are warranted (Kolko et al., 2010; Shlonsky, Haskins, Wulczyn, & Webb, 2007).

A central limitation of the existing research centers around the failure to control for the potentially confounding effects of comorbidity (Saigh, Yasik, Oberfield, Halamandaris, & McHugh, 2002) and/or viewing co-morbidity as a cross-sectional phenomenon. Therefore, despite the increasing awareness of the cumulative effects of trauma among at-risk populations, the longitudinal mechanisms underlying the association between violence exposure and depressive symptoms remains unclear. Regarding PTS, a study of children aged 2–14 involved with the child welfare system found trauma symptoms decreased over a 3-year period following a child welfare system investigation (McCrae, 2008). Specific to internalizing problems, findings from a study of adolescents aged 13–16 in out-of-home care found that internalizing symptoms demonstrated a downward trend; however specific covariates influenced the trajectories (McWey, Cui, & Pazdera, 2010). The authors know of only one study to date that has focused on longitudinal interrelationships between symptom growth among CPS-involved youth (Rayburn et al., 2016). This study, however, explored the interrelationships between PTS and a broader class of broadband symptoms that includes not only depressive symptoms but other types of internalizing responses as well. One limitation of this study was that it neither explored differences across placement types nor did it distinguish between witnessing violence and being a victim of violence. Regarding the impact of violence exposure, several studies using different samples and different measures of violence (e.g. home, community, witnessing or victimization) have found that PTS symptoms mediate the relationship between violence exposure and internalizing behavior problems in children (Ruchkin, Henrich, Jones, Vermeiren, & Schwab-Stone, 2007; Yoon, Steigerwald, Holmes, & Perzynski, 2016). Given the positive association between PTS and depressive symptoms and the higher likelihood of developing PTSD following violence exposure, exploring PTS symptoms as potential underlying mechanisms linking violence exposure to both *level* and *growth* of depressive symptoms represents a significant omission in the current literature. This is particularly important given previous research showing that major depressive disorder is relatively uncommon among prepubertal children but increases in frequency in adolescence (Finch, Saylor, & Edwards, 1985).

Due to knowledge gaps in previous studies, the present study aims to explore the longitudinal association between an increase in PTS symptomatology and an increase in depressive symptomatology among CPS-involved youth, as well as studying the influence of maltreatment type, placement type, and violence exposure on the outcome trajectories of both mental health measures. Based on prior studies, we first hypothesize that a poorer initial level of PTS symptoms contributes to the advancement of more severe depressive symptoms over time. We further hypothesize that the progression of PTS symptomatology and heightened depressive symptoms is affected by the youth's level of exposure, both as victims and witnesses, to severe violence at home. In this regard, although children in foster care have been found to be at high risk for future developmental, behavioral and emotional problems, OHC children may fare better clinically than IHC children who are

recommended for placement but who remain with their biological parents (Burns et al., 2004a, 2004b; Davidson-Arad et al., 2003). Therefore, we held no a priori expectation about the nature of these differences. We also examined whether lower levels of violence exposure prior to a maltreatment allegation results in a lower level of PTS and depressive symptoms compared to counterparts with higher levels of violence exposure at the same initial assessment. In addition, we explored whether violence exposure results in a greater increase in PTS and depressive symptomatology longitudinally. Importantly, we examined these relationships while holding constant the impact of child maltreatment type, family risk and adverse childhood experiences (ACEs), gender, race and whether the child has been removed from the home. We used a parallel latent trajectory model for this investigation which is a powerful method that allows for a simultaneous modeling over time of the differences between and within individuals in both PTS and depressive symptom changes.

## 2. Method

### 2.1. Data and participants

Nationally representative data from the National Survey of Children and Adolescent Well-Being (NSCAW) (Dowd, Kinsey, Wheless, & Suresh, 2002) were used for this study. The NSCAW data comprises two groups of children who were subjects of a child welfare system (CWS) abuse or neglect investigation. The total NSCAW cohort includes 6228 children, ages birth to 14 at the time of the sampling, who had contact with the child welfare system within a 15-month period beginning in October 1999. Our analysis focuses on the baseline cohort of 5501 interviewed from those who were subjects of child abuse or neglect investigations conducted by CPS and whose cases were followed beginning at 2 months after the initial investigation up through 36 months (Wave 1: 2–6 months; Wave 3: 18 months; and Wave 4: 36 months). Data from wave 2 were not used in our analyses data because key information (e.g. PTS symptoms) was not collected. The analytical sample is restricted to youth between the ages of 8 and 15 with complete self-report data on PTS and depressive symptoms, yielding a final sample of 2030.

### 2.2. Predictor variables

#### 2.2.1. Exposure to violence

Children were administered the Violence Exposure Scale VEX-R (Fox & Leavitt, 1995), comprised of 23 questions measuring children's exposure to mild (e.g., pushing, yelling, spanking, shoving) to severe (e.g., stabbing, shooting, threatening with a gun or knife) violence in the home. Children younger than 11 years were also shown a cartoon depiction of each act, and all children were asked how often they had witnessed each act at home (never, once, a few times, or lots of times). Two subscales were created – a severe violence witness score and a severe violence victim score. Chronbach's alpha for the two subscales was deemed acceptable (severe violence as witness = 0.83; severe violence as victim = 0.78). The variable was recoded to equal 1 if the child had witness or been the victim of at least one act of severe violence, and 0 otherwise.

#### 2.2.2. Family risk factors

CPS caseworkers identified parent and family risk factors present at the time of the maltreatment investigation based on information obtained in their assessment of the family. The risk factors were selected from several established state risk assessment forms from child welfare systems across five states (Michigan, New York, Washington, Illinois, and Colorado) deemed to be factors most likely to be related to decisions to substantiate cases or make placements into foster care. Each risk factor was marked as either present or absent by caseworkers. Risk factors included active alcohol and drug abuse, serious mental health or emotional problems, intellectual or cognitive impairments, physical

impairments, poor parenting skills, history of domestic violence, caregiver history of maltreatment, recent history of arrest, high family stress, and difficulty meeting basic needs. A cumulative risk score was created using these 11 items with higher scores indicating increased risk (Chronbach's  $\alpha = 0.71$ , mean = 2.04).

#### 2.2.3. Demographics

Adolescent gender, age, and race were included in the model as control variables. Gender was coded as 1 = male and 0 = female; age ranged from 8 to 16; race was coded as white = 1, 0 = non-white; age was a continuous variable coded in years.

### 2.3. Outcome variables

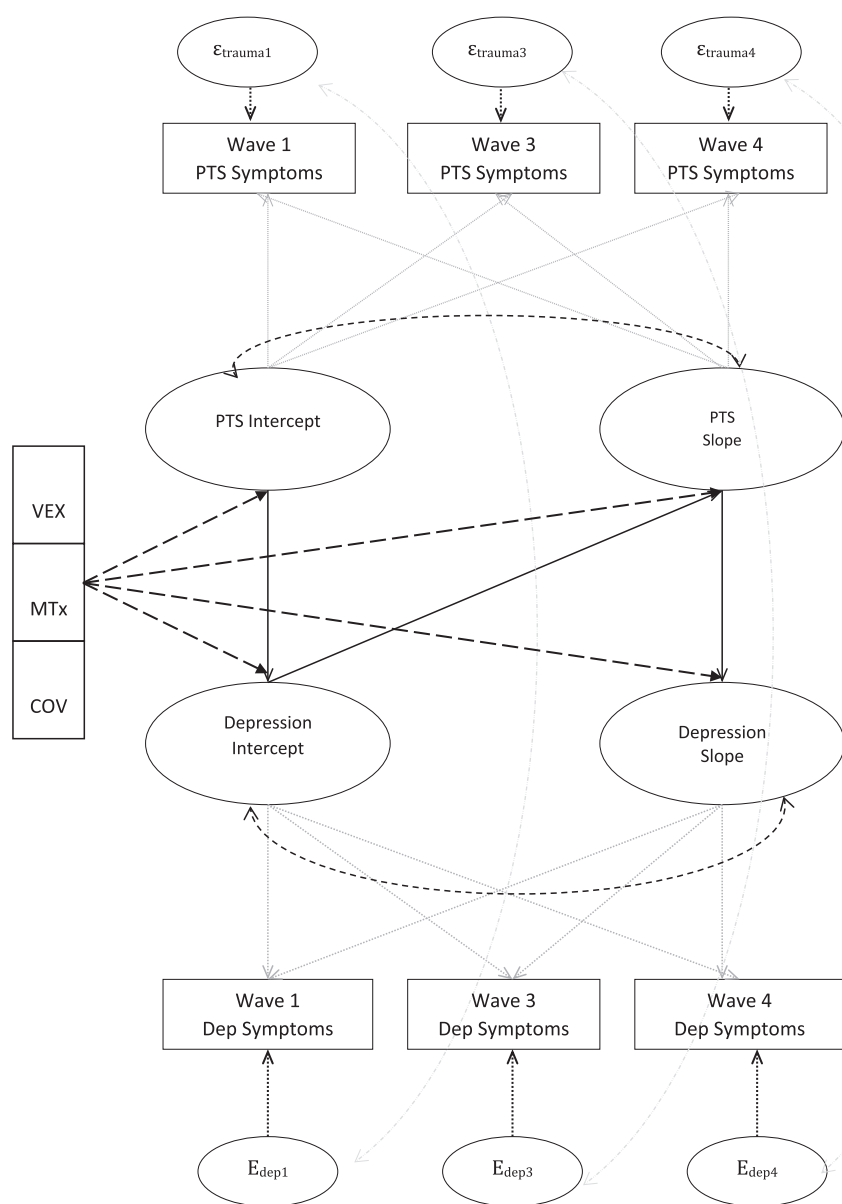
The two outcome measures in this study are depressive symptomatology and PTS symptomatology. PTS symptoms were measured using the Trauma Symptom Checklist for Children (TSCC) for NSCAW (Briere, 2002) which includes 10 items reflecting PTS symptoms (e.g., intrusive recollections of traumatic events, sensory reexperiencing and nightmares, dissociative avoidance, fears) in children and adolescents (ages 8–16) who have been exposed to unspecified traumatic events. Each item uses a 4-point Likert-type scale to indicate the frequency that each symptom has occurred (0 = never; 1 = sometimes; 2 = lots of times; 3 = almost all the time). For example, youth were asked how often they have “bad dreams or nightmares,” and how often “scary ideas or pictures just pop into [their] head.” The TSCC was standardized on a large sample of racially and economically diverse children from urban and suburban settings. T scores at or above 65 are considered clinically significant (“probable PTSD”), and consistent with a diagnosis of PTSD (Briere, 2002). The TSCC has demonstrated good psychometric properties (Briere & Lanktree, 1995; Crouch, Smith, Ezzell, & Saunders, 1999; Fricker & Smith, 2002; Lanktree et al., 2008; Sadowski & Friedrich, 2000). In the present study, internal consistency for the PTS symptoms scale ranged from 0.78 to 0.85 over the three waves.

Depression was measured by 27 items from The Children's Depression Inventory (CDI) (Kovacs, 1992), which is administered to children ages 7 and older, in order to assess the level of depressive symptoms. Each item is measured on a 3-point Likert type scale. Youth were classified as clinically depressed (“probable depression”) if they scored  $\geq 66$ , a threshold that has been used in other epidemiological studies of youth depression (Hyphantis et al., 2012; Smucker, Craighead, Craighead, & Green, 1986). In the present study, internal consistency for the depressive symptoms scale ranged from 0.75 to 0.86 over the three waves. CDI scores greater or equal to 66 indicated clinically relevant depression or “probable” MDD.

### 2.4. Research Questions

This prospective study examined CPS-involved youth at three time points. We set to evaluate the prevalence and course of symptoms of PTSD and depression, as well as their longitudinal interrelationships. Our research aims were (1) To assess the prevalence of symptoms of PTSD and depression at three points in time and the changes in prevalence over time. (2) To assess the interrelationship of symptoms of PTSD and depression, at each assessment and across time and (3) To assess the bi-directional associations between symptoms of PTSD and depression across time. Specifically, we examined the following questions:

- Is there evidence of a co-developmental process between PTS and Depressive symptoms over time (Hypothesis 1)?
- Does initial level and growth in PTS symptoms predict initial level and growth in depressive symptoms (Hypothesis 2)?
- What is the impact of complex trauma on PTS severity and depressive symptom severity and change over time (Hypothesis 3)?



**Fig. 1.** Parallel Process Model (PPM) of PTS and Depressive Symptom Trajectories.

- Does initial level and growth in PTS symptoms mediate the relationship between violence exposure (as witness or victim) and initial level and growth in depressive symptomatology over time (Hypothesis 4)?

## 2.5. Data analysis

Prior to multivariate analyses, descriptive statistics were run on all study variables. Building a parallel process model is a complex procedure, therefore modeling must be conducted in a step-sequential approach (Muthén & Muthén). A series of latent growth curve models (LCGM) were estimated to test and explore the relationships between PTS and depressive symptom growth factors independently. First, unconditional models were examined separately for PTS and depressive symptoms. Model fit was determined with generally accepted fit indices and thresholds (Hu & Bentler, 1999). A parallel process latent growth curve model (PPLGCM) was estimated that included both outcome measures across three waves with maximum likelihood estimation using a Structural Equation Modeling (SEM) framework. Next, we explored subgroup differences in trajectories by placement type. We next estimated a PPM to explore symptom co-development. Following the

estimation of non-directional influences, we incorporated directional effects to explore symptom co-development. In our last step, we incorporated variables as covariates first and then as potential mediating effects. Analyses were conducted using MPlus 7.4 (Muthén & Muthén). The overall weighted response rate was 64.2% at baseline (Biemer, 2010; Dolan et al., 2011) and follow-up data collections at 18- and 36-months were maintained above 80%. Survey weights were developed to account for the complex survey design of the data and adjust for survey non-response and frame non-coverage. Missing data were handled using the full-information maximum likelihood (FIML) procedure, which utilizes all available information from the observed data.

Given the flexibility of the modeling approach, time loadings were set at 0, 1.5, 3 years to correspond to each measurement wave. First, the growth trajectory shape of PTS and externalizing symptoms was investigated separately to test how well the hypothesized trajectory shape fits the data. In the second step, several hypothesized relations among PTS and externalizing symptoms were modeled. We examined the covariance structure between initial level and rate of change in both PTS and externalizing symptoms factors to understand the co-development of different attributes, capturing communality in primary growth factors. Our model incorporated correlations between primary



growth factors of PTS and depressive symptoms *within* each subdomain (e.g. a correlation between the initial level and slope of PTS symptoms) and *between* different subdomains (e.g. a correlation between the slope of PTS symptoms and the slope of depressive symptoms). These correlations enabled us to capture unique variance components, methodological biases or time specific patterns of reporting biases, (Wickrama, Lee, O'Neal, & Lorenz, 2016) respectively. We explored co-developmental processes across subdomains by including directional influences between slope parameters of PTS and depressive symptoms.

Next, we investigated whether level of PTS symptoms influenced rate of change in depressive symptoms over time. Previous research exploring the risk factors for heightened PTS and depressive symptoms have focused on three broad domains including individual (age/developmental level), relational (e.g., caregiver, social support) and sociocultural risks (e.g., poverty, presence of stressful life events including violence exposure). Therefore, once the best fitting model was identified, indicators of complex trauma (e.g., violence exposure, maltreatment type, adverse child experiences) and sociodemographic variables (gender, race, age at time of allegation) were incorporated as covariates. Finally, we examined the mediating influences of violence exposure, as witness and victim, on the interrelationship between PTS and developmental symptoms. We expected to find decreases in both symptoms over time. More importantly, we expected to find substantial individual differences in level and change in both PTS and depressive symptoms. Given the conflicting findings of previous literature, we had no a priori expectations about differences in trajectories between foster youth and youth who remained in the home. Next, we estimated a parallel process model using both domains simultaneously to examine symptom co-development. Based on prior work (Rayburn et al., 2016) we expected to find correlations between growth parameters indicating highly interrelated symptoms across time. We expected that both initial level and change in PTS symptoms would have strong correlations with initial level and change in depressive symptoms. Regarding the direction of covariates, we expected to find age and gender differences in PTS symptoms with females and younger children exhibiting higher levels of PTS and depressive symptoms. We also expected that youth exposed to complex trauma (violence exposure, physical and sexual abuse) would be more likely to have higher initial levels of both symptoms that worsen with time (Fig. 1).

### 3. Results

#### 3.1. Descriptive statistics of sample

Table 1 provides descriptive statistics for the sample. The sample was evenly split between females (50.1%) and males (49.9%). The average age of children in the sample at Wave 1 was 11.58 years (*s.d.* = 2.26). Forty-seven percent of children in the study sample were White (non-Hispanic), 27.9% were Black (non-Hispanic), 18.2% were Hispanic, and 6.9% were of other races or ethnicities. Caseworkers reported neglect (failure to provide or lack of supervision) as the most severe type of maltreatment in 46.5% of the cases, followed other “physical abuse” (27.2%), “other” types of maltreatment (15.3%), and sexual abuse (11.0%). Slightly over 50% of the sample had a prior maltreatment report. The average number of family risk factors was 2.04.

Table 1 also provides the bivariate associations between study variable and placement type. As shown, significant associations were found across all variables. Youth in OHC were also more likely to be black, have more previous reports of maltreatment (IHC = 49%; OHC = 66%), more adverse family risk factors (IHC = 1.83; OHC = 4.23) and any severe violence exposure as either a witness (IHC = 53%; OHC = 59%) or victim (IHC = 5.6%; OHC = 11.2%). Notably, compared to youth IHC, youth in OHC were significantly more likely to demonstrate probable depression (IHC = 10.4%; OHC = 16.0%) and PTSD (IHC = 11.2%; OHC = 18.8%) immediately

**Table 1**  
Baseline characteristics of sample.

| Variable  | Total | OHC   | IHC   | Chi <sup>2</sup> |
|---|-------|-------|-------|------------------|
| <b>Predictor</b>  |       |       |       |                  |
| <i>Type of abuse</i>                                      |       |       |       |                  |
| Physical  | 27.2  | 21    | 27    | < 0.001          |
| Sexual  | 11    | 9.5   | 11.2  |                  |
| Supervisory neglect                                       | 27.1  | 28.5  | 26.9  |                  |
| Physical neglect  | 19.4  | 21.3  | 19.4  |                  |
| Other   | 15.3  | 19.7  | 14.7  |                  |
| <i>Exposure to violence</i>                               |       |       |       |                  |
| Severe violence victimization                             | 6     | 11.2  | 5.6   | 0.003            |
| Severe violence exposure                                  | 54    | 59    | 53    | < 0.001          |
| Prior investigation of maltreatment                       | 6.2   | 5     | 6.3   | 0.123            |
| Prior reports of maltreatment                             | 50.8  | 66    | 49    | < 0.001          |
| <i>Child gender</i>                                       |       |       |       |                  |
| Male  | 49.9  | 47.1  | 50.3  | 0.045            |
| Female  | 50.1  | 52.9  | 49.7  |                  |
| <i>Child age</i>  |       |       |       |                  |
| 0–6   | 46.3  | 45.2  | 46.4  | 0.045            |
| 7–10  | 29.2  | 25.9  | 29.6  |                  |
| 11–15   | 24.6  | 29    | 24    |                  |
| <i>Child race/ethnicity</i>                               |       |       |       |                  |
| Non-Hispanic Black  | 27.9  | 33.8  | 27.1  | < 0.001          |
| Non-Hispanic White  | 47    | 45.2  | 47.2  |                  |
| Hispanic  | 18.2  | 14.3  | 18.7  |                  |
| Other   | 6.9   | 6.7   | 6.9   |                  |
| Adverse child experiences                                 | 2.04  | 4.23  | 1.83  | < 0.001          |
| <b>Outcome variable</b>                                   |       |       |       |                  |
| <i>Clinically significant symptoms</i>                    |       |       |       |                  |
| Child trauma symptom $\geq$ 65 (clinic. sig.), wave 1     | 12.1  | 18.8  | 11.2  | < 0.001          |
| Child trauma symptom $\geq$ 65 (clinic. sig.), wave 3     | 7.1   | 14.6  | 6.1   | < 0.001          |
| Child trauma symptom $\geq$ 65 (clinic. sig.), wave 4     | 5.1   | 6.9   | 4.8   | < 0.001          |
| Child depression symptom $\geq$ 66 (clinic. sig.), wave 1 | 11.2  | 16.0  | 10.4  | < 0.001          |
| Child depression symptom $\geq$ 66 (clinic. sig.), wave 3 | 6.9   | 9.0   | 6.7   | < 0.001          |
| Child depression symptom $\geq$ 66 (clinic. sig.), wave 4 | 3.9   | 6.3   | 3.6   | < 0.001          |
| <i>Symptoms</i>   |       |       |       |                  |
| Post-traumatic stress total T score, wave 1               | 49.62 | 52.44 | 49.30 | < 0.001          |
| Post-traumatic stress total T score, wave 3               | 48.27 | 50.52 | 48.07 | < 0.001          |
| Post-traumatic stress total T score, wave 4               | 47.11 | 49.24 | 46.82 | < 0.001          |
| Depression total CIDI std. score, wave 1                  | 50.07 | 51.29 | 49.97 | < 0.001          |
| Depression total CIDI std. score, wave 3                  | 47.37 | 47.58 | 47.19 | < 0.001          |
| Depression total CIDI std. score, wave 4                  | 45.90 | 47.82 | 45.64 | < 0.001          |

following the maltreatment allegation at wave 1 and during waves 3 and 4. Independent sample *t*-tests of PTS and depressive symptoms revealed significant differences between IHC and OHC at all three waves (see Table 1). We examined symptom change between W1 and W3, and between W3 and W4 separately for youth IHC and OHC. Looking at differences within cohorts, both PTS ( $t = 3.22$ ,  $p < 0.001$ ) and depressive symptoms ( $t = 4.41$ ,  $p < 0.001$ ) declined significantly between W1 and W3 but not between W3 and W4 for youth in OHC. Among youth IHC, PTS ( $t = 3.3$ ,  $p = 0.001$ ) and depressive symptoms ( $t = 5.2$ ,  $p < 0.001$ ) significantly declined between W1 and W3. As well, both PTS ( $t = 2.5$ ,  $p < 0.014$ ) and depressive symptoms declined significantly between W3 and W4 ( $t = 2.2$ ,  $p = 0.03$ ) (results not shown). Across the period, 56.3% of the sample had PTS symptoms that were above average but less than the clinical cutoff score required for a PTSD diagnosis ( $\geq 65$ ).

#### 3.2. Trajectories of PTSD and depressive symptoms and placement type

To examine changes in PTS and depressive symptoms over time, we conducted a series of Latent Growth Curve Models (LGCM) (Bollen & Curran, 2006). Two latent factors were estimated: the first defines initial levels of PTS and depressive symptoms (i.e. the

**Table 2**  
Model fit statistics.

| Growth model type                           | Chi <sup>2</sup> (df), p | RMSEA (90% CI)       | CFI/TLI     | SRMR |
|---|--------------------------|----------------------|-------------|------|
| PTS symptoms                                |                          |                      |             |      |
| Linear                                      | 42.338(5), < 0.001       | 0.056 (0.040, 0.074) | 0.964/0.956 | 0.06 |
| Depressive symptoms                         |                          |                      |             |      |
| Linear                                      | 39.617(5), < 0.001       | 0.058 (0.042, 0.076) | 0.974/0.969 | 0.09 |
| PPM with non-directional effects            | 7.59(5), 0.181           | 0.016 (0.000, 0.031) | 0.999/0.996 | 0.01 |
| PPM with directional effects and covariates | 68.291(58), 0.17         | 0.013 (0.000, 0.023) | 0.982/0.966 | 0.03 |

Note: PTS = post-traumatic stress; PPM = parallel process model; RMSEA = root mean square error of approximation; CFI/TLI = tucker-lewis fit index, Comparative fit index/SRMR = standardized root mean square error.

intercept), and the second explores the constant of the trajectory of change in PTS and depressive symptoms over time (i.e. linear; time was coded as 0, 1.5 and 3 to accord with survey wave). Furthermore, we assessed whether the LGCMs were different for the youth in IHC and in OHC by using a multi-group SEM procedure.

### 3.2.1. Model selection and model fit for the latent growth curve models (LGCMs)

Table 2 shows the model fit statistics. As can be seen in Table 3, the overall model fit indices of the unconditional models fit the data well (PTS symptoms:  $\chi^2(5) = 42.34$ ,  $p < 0.001$ , CFI = 0.964, TLI = 0.956 RMSEA = 0.056 (0.040, 0.074), SRMR = 0.06; Dep symptoms:  $\chi^2(5) = 39.62$ ,  $p < 0.001$ , CFI = 0.974, TLI = 0.969 RMSEA = 0.058 (0.042, 0.076), SRMR = 0.09). The average intercept and slope was statistically significant for both PTS and depressive symptoms. The mean trajectory for both PTS and depressive symptoms significantly decreased throughout the period. The variances of the intercept and slope were statistically significant as well, indicating substantial variability in initial levels and changes in both PTS and depressive symptoms. The negative covariance between the intercept and slope suggests that on average individuals with higher initial levels of PTS and depressive symptoms are more likely to experience declines over time.

### 3.2.2. Differences in PTS and depressive symptoms among youth IHC and OHC

To test whether youth in IHC and OHC have different growth factors for PTS and depressive symptoms, we used a multi-group latent growth curve modeling approach to compare the intercept and slope of each. Our approach was to first estimate the unconstrained model for each symptom. To test for weak factorial invariance across groups, the chi-square from the unconstrained model was compared to a model with

the growth parameters constrained to be equal across groups. The model with all the parameters freely estimated in the two groups fit the data well for both depressive symptoms (CFI = 1.000, SRMR = 0.004) and PTS symptoms (CFI = 0.985, SRMR = 0.060). The strict invariance model with growth factors constrained to be equal across groups also demonstrated a good fit ( $\chi^2 = 25.62$ ,  $p = 0.001$ , CFI = 0.984, SRMR = 0.062). The difference was small in magnitude and statistically insignificant (change  $\chi^2 = (25.62-21.52)$  change  $df = 3$ ,  $p = 0.749$ ). These results suggested that the growth parameters of the LGCM for both depressive and PTS symptoms are structurally similar for youth IHC and OHC. Therefore, we were justified in treating them as coming from a single population in subsequent analyses.

### 3.2.3. Bidirectional associations between PTSD and depressive symptoms over time (Hypothesis 1)

As shown by Table 2, the parallel process growth model had excellent fit to the data  $\chi^2(5) = 7.59$ ,  $p = 0.181$ , CFI = 0.999, TLI = 0.996, RMSEA = 0.016, SMR = 0.009. Results indicated that both level and change in PTS and depressive symptoms were significantly associated. The correlations among primary growth factors were statistically significant which is evidence for the existence of parallel growth between symptoms. Higher levels of PTS symptoms (i.e. intercept) were associated with higher levels of depressive symptoms ( $r = 0.637$ ,  $p < 0.001$ ). Change in PTS symptoms were positively associated with change in depressive symptoms ( $r = 0.688$ ,  $p < 0.001$ ), that is increases (decreases) in depressive symptoms are significantly associated with increases (decreases) in PTS symptoms over time. In addition, time specific indicators were significantly associated within each wave and were all highly significant ranging from 0.280 to 0.472. The correlation coefficients between PTS and depressive symptom intercepts and slopes are 0.603,  $p < 0.001$ , and 0.689 ( $p < 0.001$ ), respectively. These non-directional associations depict the co-occurrence of PTS and depressive symptoms and their co-development over time. The R-squared for the observed variables revealed that between 38.7%–86.8% of the observed individual differences are accounted for by the latent growth factors for depressive symptoms and between 55.9%–67.9% of the observed individual differences are accounted for by the latent growth factors for PTS symptoms. The remaining variance is due to measurement error and/or systematic occasion-specific variance in this model.

Table 3 shows the unstandardized parameter estimates, standard errors and statistical significance of the unconditional PPM. Statistically significant mean levels existed for intercept and slope of both PTS (int = 50.61,  $p < 0.001$  and slope =  $-1.35$ ,  $p < 0.001$ ) and depressive symptoms (int = 50.42,  $p < 0.001$  and slope =  $-2.69$ ,  $p < 0.001$ ). The mean trajectory of both symptoms decreased significantly over time. The variances of the intercepts for both symptoms were statistically significant suggesting that some youth have higher initial levels of PTS and depressive symptomatology while others have lower levels, and others still have levels closer to the average values. The negative correlation between intercepts and slopes indicates that individuals with heightened PTS and depressive symptoms initially were more likely to experience declines in each symptom over time.

**Table 3**  
Unstandardized parameter estimates, standard errors, and statistical significance of unconditional Parallel Process model.

|                    | PTS symptoms |        | Depressive symptoms |       |
|--------------------|--------------|--------|---------------------|-------|
|                    | Estimate     | SE     | Estimate            | SE    |
| Initial level      | 50.61***     | 0.672  | 50.42***            | 0.714 |
| Linear change      | $-1.35$ ***  | 0.478  | $-2.69$ ***         | 0.534 |
| Quadratic change   | 0.110        | 0.120  | 0.447***            | 0.146 |
| Variances          |              |        |                     |       |
| Var (intercept)    | 84.90***     | 29.90  | 123.03***           | 47.89 |
| Var (slope)        | 7.490        | 15.70  | 30.45               | 21.03 |
| Var (quadratic)    | 0.573        | 0.575  | 0.433               | 0.889 |
| Residual variances |              |        |                     |       |
| 3–6 months         | 50.12*       | 26.744 | 18.76               | 40.56 |
| 18 months          | 48.74***     | 9.152  | 52.80***            | 14.77 |
| 36 months          | 49.28***     | 6.759  | 29.44***            | 8.15  |
| 48 months          | 22.76**      | 10.213 | 68.91*              | 35.52 |

\*  $p < 0.10$ .

\*\*  $p < 0.05$ .

\*\*\*  $p < 0.01$ .

**Table 4**

Direct effect and correlation between growth factors.

|               | Direct effects |            | Correlations between growth factors |            |               |            |
|---------------|----------------|------------|-------------------------------------|------------|---------------|------------|
|               | Dep intercept  | Dep slope  | PTS intercept                       | PTS slope  | Dep intercept | Dep slope  |
| PTS intercept | 0.498***       | – 0.098*** | –                                   | – 0.789*** | 0.603***      | – 0.494*** |
| PTS slope     |                | 0.530***   |                                     | –          | – 0.393***    | 0.689***   |
| Dep intercept |                |            |                                     |            | –             | – 0.593*** |
| Dep slope     |                |            |                                     |            |               | –          |

\*\*\*  $p < .001$ .

### 3.2.4. Directional effects of initial level and change in PTS symptoms on initial level and change in depressive symptoms (Hypothesis 2)

The model including directional effects and covariates provided an excellent fit:  $\chi^2(58) = 68.29$ ,  $p = 0.170$ , CFI = 0.982, TLI = 0.966, RMSEA = 0.013, SMR = 0.03 (Table 2). Table 4 shows the directional influences of PTS intercept and slope on rate of change in depressive symptomatology over time. The results showed a significant association between the level or initial value of heightened PTS symptoms at W1 and rate of change in depressive symptoms over time ( $b = -0.098$ ,  $p < 0.001$ ). Statistically, this is an interaction effect between time and the initial level of PTS symptoms influencing depressive symptom growth. Therefore, the influence of PTS symptoms on depressive symptoms gains strength over time. As well, results suggested that changes in PTS symptoms were significantly associated with changes in depressive symptoms ( $b = 0.530$ ,  $p < 0.001$ ). Finally, initial level of PTS symptoms was related to initial level of depressive symptoms ( $b = 0.498$ ,  $p < 0.001$ ). A significant amount of variability remained in the initial values of both PTS and depressive symptoms ( $\text{var}(\text{dep}) = 69.05$ ,  $p < 0.001$ ;  $\text{var}(\text{PTS}) = 42.33$ ,  $p < 0.001$ ). The correlation between the depressive symptom intercept and PTS symptom slope is  $-0.393$  ( $p < 0.001$ ), the depressive symptom intercept and depressive symptom slope is  $-0.593$  ( $p < 0.001$ ); the PTS symptom intercept and PTS symptom slope is  $-0.789$  ( $p < 0.001$ ); and the depressive symptom slope and PTS symptom intercept  $-0.494$  ( $p < 0.001$ ) (see Table 4).

### 3.2.5. Predictors of both PTS and depressive symptom trajectories (Hypothesis 3)

Accounting for the influence of contextual risk factors reduced the correlations between PTS symptoms and depressive symptoms from 0.637 to 0.576 for initial level and between from 0.688 to 0.499 for change. All associations between PTS and depressive symptoms remained significant even when contextual effects were considered. The final model, adding contextual factors, explained 10.3% and 14.9% of the variance in initial level of PTS and depressive symptoms, and 3.9% and 7.6% of PTS and depressive change. The standardized parameter estimates for the final PPM model are shown in Table 5. Note that a negative coefficient is interpreted as a slower decrease in symptomatology since the slopes of both PTS and depressive symptoms are negative. Witnessing severe violence at home had a significant effect on the intercept of both PTS ( $b = 0.278$ ,  $p < 0.001$ ) and depressive symptoms ( $b = 0.232$ ,  $p < 0.001$ ). Child age was significantly associated with heightened initial PTS ( $b = -0.125$ ,  $p < 0.03$ ) and growth in PTS over time ( $b = 0.144$ ,  $p < 0.08$ ). Sexual abuse was related to higher initial PTS levels at baseline ( $b = 0.212$ ,  $p < 0.022$ ). Among females, depressive symptom severity was significantly higher compared to males ( $b = 0.267$ ,  $p < 0.001$ ) and females had slower decreases in depressive symptoms over time ( $b = 0.210$ ,  $p < 0.015$ ). Severe violence victimization ( $b = -0.265$ ,  $p < 0.001$ ) and family risk factors ( $b = -0.187$ ,  $p < 0.076$ ) were both associated with slower decreases in depressive symptoms.

**Table 5**

PTS and depressive symptoms scores parallel process growth model standardized parameter estimates.

| Covariates                               | Intercept | SE    | Linear slope | SE    |
|--|-----------|-------|--------------|-------|
| <i>PTS symptoms</i>                      |           |       |              |       |
| Female                                   | – 0.032   | 0.069 | – 0.103      | 0.087 |
| Child age (years)                        | – 0.125** | 0.059 | 0.144**      | 0.076 |
| Maltreatment type                        |           |       |              |       |
| Physical abuse                           | 0.050     | 0.075 | – 0.005      | 0.075 |
| Sexual abuse                             | 0.212**   | 0.098 | 0.008        | 0.119 |
| Other                                    | – 0.038   | 0.061 | 0.040        | 0.077 |
| Adverse childhood experiences            | 0.062     | 0.070 | – 0.122      | 0.078 |
| Violence exposure (severe witness)       | 0.278***  | 0.075 | – 0.097      | 0.089 |
| Violence exposure (severe victimization) | 0.070     | 0.089 | – 0.142      | 0.105 |
| IHC vs. OHC                              | – 0.045   | 0.066 | 0.048        | 0.089 |
| Race/ethnicity                           |           |       |              |       |
| NH White v non-White                     | 0.052     | 0.066 | – 0.068      | 0.081 |
| <i>Depressive symptoms</i>               |           |       |              |       |
| Female                                   | 0.267***  | 0.057 | – 0.210**    | 0.101 |
| Child age (years)                        | – 0.035   | 0.067 | 0.074        | 0.098 |
| Maltreatment type                        |           |       |              |       |
| Physical abuse                           | 0.121     | 0.082 | – 0.091      | 0.116 |
| Sexual abuse                             | 0.112     | 0.084 | – 0.023      | 0.108 |
| Other                                    | – 0.012   | 0.066 | – 0.028      | 0.101 |
| Adverse childhood experiences            | 0.031     | 0.074 | – 0.187*     | 0.113 |
| Violence exposure (severe witness)       | 0.232***  | 0.062 | – 0.077      | 0.095 |
| Violence exposure (severe victimization) | 0.081     | 0.079 | – 0.265**    | 0.139 |
| IHC vs. OHC                              | – 0.021   | 0.055 | 0.033        | 0.094 |
| Race/ethnicity                           |           |       |              |       |
| NH White v non-White                     | 0.058     | 0.070 | – 0.019      | 0.101 |

Note: IHC = in home care; OHC = out of home care.

\*\*\*  $p < .001$ 

### 3.2.6. The mediating effect of initial level and change in PTS on the relationship between violence exposure and initial level and change in depressive symptoms (Hypothesis 4)

Our last step was to test the mediating effect of violence exposure on the relationship between initial level and change in PTS symptoms and change in depressive symptoms over time. Possible mediation paths extend from the variable codifying violence exposure as victim or witness to the intercept or slope of PTS symptoms and then to the slope for depression (i.e., change in depression). Specifically, we examined whether violence exposure influences an initial level or change in PTSD, and whether the initial level or change, in turn, predicts depressive symptom growth. The bias-corrected bootstrap confidence interval for the mediating effect of initial PTS and PTS slope on the relationship between witnessing at least one act of extreme violence and progression of depressive symptomatology revealed a statistically significant result. We found that both initial level of PTS ( $b = 0.164$ ; BootCI = [0.104, 0.415]) and rate of change in PTS ( $b = -0.975$ ; BootCI = [– 1.04, – 0.705]) mediate the relationship between violence exposure as witness and rate of change in depressive symptoms. This means that witnessing extreme violence is related to 1) heightened initial PTS

symptoms which, in turn, are related to changes in depressive symptoms over time; and 2) slower decreases in PTS symptom change which, in turn, are related to slower decreases in depressive symptom change.

#### 4. Discussion

This study set out to examine the long-term prevalence, course, and interrelations between symptoms of PTSD and depression among CPS-involved youth and to examine subgroup differences between youth IHC and OHC. This study offers the most precise longitudinal estimate of the prevalence of heightened PTS and depressive symptoms, and the more stringent psychiatric diagnosis of PTSD and MDD, in a random sample of children reported for suspected child maltreatment (Kolko et al., 2010). At baseline, the overall prevalence for probable PTSD and depression was 12.1 and 11.2, and the corresponding mean scores were 49.62 and 50.07, respectively. Our wave 1 estimates are the same as those reported by Kolko et al. (2010) using the same data (Kolko et al., 2010). After examining multiple methodological and diagnostic explanations, these authors suggested the lower than expected estimates may be explained by the uncanny ability of at risk youth to develop hardiness and coping skills that help them withstand extreme levels of adversity (Kolko et al., 2010). Because their study was cross-sectional, they could not draw any definitive conclusions regarding longer term mental health outcomes. Therefore, to expand the longitudinal formulations in this area, we examined how trajectories of PTS and depressive symptoms unfold over time following the initial maltreatment allegation. Using prospective data over a 3-year period following an allegation of maltreatment, several important findings emerged. We found that the estimates of probable PTSD and depression prevalence declined significantly from baseline. Compared to youth placed in foster care following a maltreatment allegation, the prevalence of probable PTSD and depression, as well as their co-occurrence, was lower among youth who remained IHC. Results also indicate that estimates of probable PTSD and depression prevalence were similar to each other at each time point for both youth IHC and OHC. As well, symptoms of both PTS and depression were statistically different among youth IHC and OHC but both cohorts showed significant decreases over time.

Although the prevalence of both PTS and depressive symptoms decreased over time in both groups, the LGCMs revealed the presence of significant inter- and intra-individual variability in symptom trajectories. As a result, during at least one period, symptoms remained elevated for over half of the children referred for CWS whose symptoms did not rise to the level of clinical significance (Kolko et al., 2010). Our examination of the co-development between PTS and depressive symptomatology revealed that lower initial PTS symptoms were related to lower initial depressive symptoms and to change in depressive symptoms over time. The finding that symptoms of depression and PTSD are highly inter-related across time is consistent with DTD theory's unified traumatic stress construct. Our effort to explain the processes undergirding symptom co-development focused on the effects of placement type, maltreatment type, violence exposure, adverse living arrangements and sociodemographic characteristics. Methodologically, substantial residual variance in the PTS and depressive symptom baseline was related to level and type of violence exposure, age, gender and family risk, and through this to negative changes in mental health over time.

The present investigation suggests that the point in time closest to the maltreatment allegation represents a critical transition point for average declines in PTS and depressive symptoms and not home removal per se (Rayburn et al., 2016). In explaining this finding, we revisit an issue that has been discussed and debated extensively, namely how and whether foster care is related to poor mental health outcomes. Previous research has shown that children in foster care are often placed in high-risk home environments characterized by adversity and risk (Ehrle, Geen, & Clark, 2001, 2002; Pilowsky, 1995) and therefore may experience similar types/amounts of traumatic stress during their

transition to out-of-home care and thereafter. The present findings support this contention, in part. More specifically, the PPM revealed that children who are removed from the home have worse mental health trajectories; however, irrespective of placement type, symptoms significantly improved in both groups over time. Consequently, despite different initial prevalence estimates of probable PTSD and depression, the overall trajectory or symptom course was similar for youth IHC and OHC. We further found that despite a low prevalence of probable PTSD, more than half the sample demonstrated higher than average symptoms during the period under investigation. Finally, once the effects of complex trauma and family risk were included in the model, no differences between youth IHC and OHC were found. There are several possible explanations for our findings. First, it is possible that foster care youth would have had worse trajectories had they remained in the home and/or had no CPS intervention. Previous research suggests that youth who were not removed from an abusive home, but should have been, have worse outcomes compared to youth who remain in the home or were placed in OHC. It is also possible that existing diagnostic criteria are masking important subgroup differences. While sorting out these competing explanations is beyond the scope of the present research, our prospective longitudinal analysis leads us to question the assumption that foster care youth have worse mental health outcomes than youth who are not removed after controlling for the effects of maltreatment type, violence exposure and family risk. Future research should continue to explore the role of complex trauma in differentially shaping the long-term outcomes of youth who remain in the home and those who do not.

The analysis showed important age and gender differences in symptoms of depression and PTS and their co-development over time. In this regard, the results align with previous studies showing that older children involved with CPS tend to have different mental health needs compared to younger children (Cook et al., 2005). For example, we found that older children have lower initial trauma symptoms but they also experience faster declines in PTS symptoms over time. These findings are consistent with previous research demonstrating that younger children may be more vulnerable because they have fewer protective mechanisms to manage and interpret their traumatic experiences. Research has also shown that the development of both cognitive affective regulation and presence of peer group support that buffers unpleasant life experiences may not be achieved until middle childhood or adolescence (Salmon & Bryant, 2002). Compared to males, females were more likely to suffer from higher levels of depression that worsen over time. This gender difference may be attributed to differential exposure to complex trauma as well as to differences in gender role socialization and acceptable patterns of behavior for men and women. Generally, girls tend to exhibit internalizing behaviors when coping with stress, whereas boys are more likely to externalize or act out (Maschi, Morgen, Bradley, & Hatcher, 2008). From the standpoint of intervention and prevention, gender-specific approaches focusing on different responses to trauma, as well as differences in its causes and consequences for males and females, would be beneficial in minimizing symptom progression and co-development over time.

Childhood exposure to ongoing domestic and community violence as victims, witnesses, or both, has been previously associated with heightened levels of externalizing and PTS symptoms and corresponding psychopathology (i.e. PTSD) (Cicchetti & Toth, 2000; Clark, De Bellis, Lynch, Cornelius, & Martin, 2003; Livingston, Lawson, & Jones, 1993; Turner, Finkelhor, & Ormrod, 2006). Considering maltreatment first, studies have shown that most CPS involved children have experienced some form of maltreatment such as neglect, physical abuse or sexual abuse (Health & Human, 2006). Previous research suggests that abused and neglected children have a comparable risk for heightened PTS symptoms. As well, in the current investigation, abused and neglected children were found to have a comparable risk for heightened PTS, even after controlling for co-morbid depressive symptoms and violence exposure. This is important since studies have



shown that neglected children show high rates of exposure to domestic violence and externalizing behavior (Kolko et al., 2010). There was one exception, however. Even after controlling for sociodemographic characteristics, childhood adversity, and violence exposure at home, we found that sexual abuse was significantly related to the initial level of PTS symptoms (but not depressive symptoms) compared to neglect (both physical and supervisory). This is consistent with a large body of evidence that PTSD has relatively stronger associations with sexual abuse (Pine & Cohen, 2002). Taken together, these findings suggest that sexual abuse may be a key factor in explaining the onset of PTS symptoms above and beyond the effects of co-morbidity and other types of adversities, including violence exposure but that children with heightened PTS and depressive symptoms show similar rates of growth over time once these same factors are considered.

Previous research has shown that childhood exposure to violence as a victim or bystander can lead to depression, lowered self-esteem, and social isolation (Avery, Massat, & Lundy, 2000; Carlson, Furby, Armstrong, & Shlaes, 1997; Staudt, 2001). The present study as well pointed to the existence of a direct relationship between violence exposure and both initial level and growth in both PTS and depressive symptoms. Like others, our findings highlight the different patterns of associations between different types of violence exposure and mental health issues (Yoon et al., 2016). Witnessing severe violence was related to heightened PTS and depressive symptoms whereas victimization was only found to be related to depressive symptom growth over time. This conflicts with previous research that has failed to uncover a relationship between witnessing violence in the home and the development of internalizing problems (Cooley-Quille, Boyd, Frantz, & Walsh, 2001; Farrell & Bruce, 1997; Fitzpatrick, 1993; Yoon et al., 2016). A possible explanation for the differences across studies may be attributed to the uniqueness of our methodological approach; unlike the present study, previous research has generally not considered the impact of symptom comorbidity on the development of internalizing psychopathology. Second, internalizing symptoms represent a broader class of mental health problems of which depression is only one. In this regard, our findings are consistent with previous research showing that more severe acts of violence (e.g. being bitten, kicked, punched, beaten, or threatened with a weapon) have strong relationships with depression (Naar-King, Silvern, Ryan, & Sebring, 2002; Wind & Silvern, 1992). Other research has shown that exposure to severe violence is associated with PTS symptoms such as hypervigilance, which help adolescents recognize and respond to threatening situations (Gaylord-Harden, Cunningham, & Zelencik, 2011).

In the present study, witnessing violence was a robust finding which points to its role as a significant risk factor for co-occurring mental health issues above and beyond the presence of other risk factors, including adverse child experiences and maltreatment type. After controlling for child maltreatment, maternal mental health and substance use, violence exposure in the home was significantly related to co-morbid mental health. A clear implication for the prevention and intervention of psychological disorders among potential child maltreatment victims exposed to violence is that a much wider range of treatments need to be assessed, monitored and streamlined to reflect symptom profiles and patterns of change. Such knowledge will assist CPS workers particularly since previous studies have shown that violence against children is common in families experiencing domestic violence (Hughes, Chau, & Poff, 2011). In addition, studies have shown that although CPS workers recognize domestic violence exposure as a potential risk factor for child maltreatment, few families are referred for services absent evidence of another co-occurring issue, such as maternal mental health issues or substance use. For these children, interventions that address concerns about mothers' parental capacity that are devoid of specific interventions that focus on addressing the effects of witnessing severe violence will not address consequential co-morbid mental health issues over the long term.

The results of our study supported initial level and PTS symptom

growth as a potential mechanism that explains the relationship between witnessing severe violence and depressive symptom rate of change. We found that violence exposure mediates the longitudinal association between the PTS symptomatology and the development of depressive symptoms. This means that some of the effect of violence exposure on symptoms of depression operates through heightened PTS symptomatology. Lower levels of violence exposure resulted in significant reduction of PTS symptoms and produced a slower decline in depressive symptoms later in life. In contrast, high levels of violence exposure exacerbated PTS symptoms three years after the initial maltreatment allegation. This finding has two important implications. First, it suggests that exposure to violence is a key factor for explaining why the influence of PTS symptoms on depressive symptoms gains strength over time. Second, it points to the possibility that exposure to violence is a key developmental precursor of the interrelationship between PTS and depressive symptom co-development. Taken together, our results show that witnessing violence in the home is a critical component undergirding a unified traumatic stress construct that explains the magnitude of the interrelationship between depression and PTSD during a key developmental transition.

#### 4.1. Limitations

This study used parallel latent growth curve modeling to capture the development of two central mental health conditions over time in an at-risk population of children. The analytical strategy also provides new information regarding the effects of age, gender and complex trauma on depressive and PTS symptom trajectories following an allegation of maltreatment. Nevertheless, this work is not without limitations. Firstly, attrition and non-response bias are issues in any longitudinal study of high risk populations. Fortunately, Mplus accounts for missing data using Full Information Maximum Likelihood (FIML) estimation and limits potential biases by employing an expectation maximization (EM) algorithm (Duncan et al., 2006). In addition, we were unable to distinguish between types of OHC in this study due to small sample sizes. Placement experiences may vary according to placement type and therefore future research should incorporate other forms of OHC into similar analyses. As well, our study is limited by different definitions of child maltreatment (Coulton, Crampton, Irwin, Spilsbury, & Korbin, 2007) both substantively (i.e. between self-report and agency data) and methodologically (i.e. prospective vs. retrospective data). Therefore, our results are limited to youth who are involved in the CPS system due to alleged maltreatment via a substantiated allegation. Certainly, different measures or samples may yield different findings. The NSCAW data has been used extensively in studies of at risk children and is useful for enhancing our knowledge about the role of child welfare intervention in the lives of children who have experienced maltreatment. Nevertheless, the data set itself is a possible limitation given that various individuals collected data over a wide range of time and the data were collected over a decade ago. It is also important to acknowledge that our analysis does not directly address diagnostic comorbidity but rather symptom overlap and symptoms indicative of a probable diagnosis. This means that even though heightened symptoms are reflective of more the more stringent psychiatric diagnosis of PTSD and MDD, it is not absolute. Finally, while conventional LGCM is well-suited for studying change in attributes when the growth function for all members of the population is the same form, it is possible that the heterogeneous rate of change across individuals may produce qualitatively distinct patterns resulting in different patterns of growth. We leave this as an endeavor for future research.

#### 4.2. Implications for practice

The results of this study point to the importance of implementing a trauma-informed perspective as a first step towards identifying potential mental health problems among subsets of CPS involved youth with

domestic violence exposure, a history of child maltreatment and other trauma histories. The implementation of a trauma informed perspective will require that all systems encountering children understand how to best address their needs. Creating trauma-informed systems requires routine screenings for trauma exposure, that child welfare workers use evidence-informed practices, making resources on trauma widely available, and ensuring continuity of care across service providers (Ko et al., 2008). Attending to the mental health challenges faced by victims of child abuse and domestic violence is critical to prevent the co-development of trauma and depressive symptoms and adult onset PTSD and MDD. Further, long-term mental health requires not only understanding the most effective treatments that minimize symptomatology; it also necessitates understanding the processes and pathways undergirding the etiology of developmental trauma to develop streamlined treatment modalities. Given the fact that symptom co-development is associated with complex trauma exposure, it is important for child welfare workers to be able to assess the extent and impact of violence exposure, either as witness or victim, on the child. This necessitates that professionals have expertise in assessing trauma exposure, evaluating trauma-related distress, and implementing appropriate trauma-focused treatments and that they understand how violence exposure and victimization effects family dynamics and child adjustment across a variety of settings. In this regard, the development of a trauma informed perspective in CPS may provide a more comprehensive understanding of the trauma exposure histories of youth involved with their care. Given high rates of trauma exposure and co-occurring adverse childhood experiences, a more nuanced understanding of these issues and their long-term impact will help child welfare professionals more efficiently focus their obligations and activities to address service gaps (Greeson et al., 2011).

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