

Maternal versus paternal physical and emotional abuse, affect regulation and risk for depression from adolescence to early adulthood

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ABSTRACT

Objective: Current research has established that depression is a common outcome of child abuse. The current study extends previous research by examining the relationship between parental emotional and physical abuse and adolescents' depressive symptoms using a prospective longitudinal design. We anticipated that this relationship would be mediated through problems in affect regulation, consistent with the view that the presence of abuse in the parent–child relationship would derail the development of adaptive affect regulation. Finally, we further examined gender-linked transmission of risk by considering *both* the gender of the parent perpetrating abuse and the gender of the adolescent.

Methods: A sample of high-risk youth ($N = 179$; 46% female) from juvenile justice and clinical settings completed assessments regarding maternal and paternal physical and emotional abuse, affect dysregulation and depressive symptoms during three time points over the course of five years.

Results: The relationship between maternal abuse and depressive symptoms was partially mediated through affect dysregulation at Time 1 and fully mediated at Time 2. In addition, adolescents' reports of maternal abuse at Time 1 predicted their depressive symptoms in early adulthood even after accounting for the partial mediating role of affect dysregulation at each of the three timepoints of the study. It was also found that paternal abuse was related to depressive symptoms through an indirect relationship with affect dysregulation for males, but not females.

Conclusion: These findings suggest that adolescence may be a sensitive developmental period wherein abuse experiences have profound direct and mediated influences on the risk for later depression. Adolescents or young adults who have experienced abuse may benefit from interventions designed to build affect regulation skills.

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There is ample evidence that early childhood abuse is associated with increased risk for depression across the lifespan (e.g., Hankin, 2005; Kim & Cicchetti, 2006, 2010). Retrospective studies show that the effects of abuse on risk for depression persist well into adulthood (Springer, Sheridan, Kuo, & Carnes, 2007) and prospective studies have demonstrated the relationship between abuse and depression in childhood (Kim & Cicchetti, 2006). Less is known, however, about the relationship between abuse experienced in adolescence and the risk for depression. This is surprising given that adolescence is a period of marked increase in risk for multiple types of abuse (Sedlak et al., 2010) and the development of mental health problems, particularly depression (e.g., Hankin, 2006). As a result, there have been calls for prospective longitudinal studies to better understand the impact of abuse on adolescent adjustment (Trickett, Negriff, Ji, & Peckins, 2011).

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We report a prospective study specifically designed to examine how adolescents' experiences of emotional and physical abuse influence their risk for depressive symptoms in adolescence and later into early adulthood. As there is growing interest in the processes that underlie the relationship between abuse and poor adjustment, we discuss affect regulation as an emerging competence in adolescence that protects youth from developing depressive symptoms. We argue that when adolescents experience abuse, the development of affect regulation may be disrupted and result in deficits in this skill. In turn, problems with the regulation of affect may mediate the relationship between abuse and depressive symptoms. We draw on research on the primary role of mothers as attachment figures and examine the differential effects of maternal-versus paternal-perpetrated abuse on risk for depressive symptoms in daughters and sons.

Abuse, Affect Regulation and Depression

Depression is a common outcome of many forms of abuse. Historically, researchers focused on the effects of sexual and physical abuse on risk for adult depression due to the profoundly damaging effects of such experiences (e.g., Boudewyn & Liem, 1995; Gibb et al., 2001; Kaplan, Pelcovitz, & Labruna, 1999). However, recent studies have demonstrated that emotional abuse is also associated with risk for depression (e.g., Liu, Alloy, Abramson, Iacoviello, & Whitehouse, 2009). As a result, more attention has been directed toward understanding the common mechanisms and processes that may mediate risk transmission across development. For example, cognitive vulnerabilities such as cognitive schemas (e.g., Lumley & Harkness, 2009; Wright, Crawford, & Del Castillo, 2009), automatic negative self-associations (e.g., van Harmelen et al., 2010), and rumination (e.g., Raes & Hermans, 2008) have been proposed to arise from abuse experiences and potentially mediate the relationship between abuse and depression over time.

Others have proposed that the disturbance of the development of affect regulation and adaptive functioning (Kim & Cicchetti, 2010), including associated self-regulating strategies such as self-compassion (Vettese, Dyer, Li, & Wekerle, 2011) is implicated in the relationship between abuse and depression. Affect regulation is defined here as the ability to modulate one's emotional states without undue attempts to avoid or suppress difficult emotions. It is also the ability to reflect on or use emotional experiences as an important source of information about oneself and one's experiences in the world (Gratz & Roemer, 2004). Affect regulation can be conceptualized as both a risk and protective factor (Rutter, 2003; Stouthamer-Loeber et al., 1993) since at high levels, adaptive affect regulation can be protective in the context of adversity; however, low affect regulation, or affect dysregulation, is associated with a myriad of mental health problems (Romens & Pollak, 2012; Soenke, Hahn, Tull, & Gratz, 2010).

There is a strong theoretical and empirical case for proposing affect regulation as a core process that accounts for the relationship between abuse and depression. From a developmental perspective, it is clear that all forms of abuse place the child in an untenable and extremely vulnerable position: When the person whom they rely on to support them to cope with overwhelming distress is the perpetrator of abuse, the child is abandoned and left to cope with extreme distress. As a result, the child's opportunities for learning to regulate difficult emotions are stunted both by the intrusion of overwhelming distress and the abandonment of an effective parent who both soothes the child and helps them to internalize effective self-regulation strategies. Such ideas have been posited in developmental models of disorders such as borderline personality disorder (BPD) which has at its core a fundamental incapacity to regulate affect (Linehan, 1993). Similar notions appear in attachment theory in which parental abuse interferes with the child's access to a safe haven, soothing of distress, and internalization of emotion regulating strategies (Moretti & Obsuth, 2011). Consistent with these views, numerous studies have shown that childhood abuse has been associated with emotion dysregulation (e.g., Camras, Sachs-Alter, & Ribordy, 1996; Cloitre, Stovall-McClough, Zorbas, & Charuvastra, 2008; Kim & Cicchetti, 2010; Riggs, 2010; Romens & Pollak, 2012).

The current study extends previous research by examining the mediating role of affect regulation on the relationship between parental emotional and physical abuse and adolescents' depressive symptoms using a prospective longitudinal design. Consistent with attachment theory and developmental models of personality disorder, we predicted that parental abuse would be associated with problems with affect regulation which in turn would mediate depressive symptoms concurrently and prospectively as teens transitioned to adulthood. These predictions were tested in a clinical sample of adolescents.

Maternal versus Paternal Abuse

Relatively few attempts have been made to determine whether abuse by different perpetrators has differential effects on children. Among studies examining the impact of exposure to interparental violence (IPV), several have found that exposure to maternal- rather than paternal-perpetrated IPV is a stronger predictor of children's aggression (Verlaan & Schwartzman, 2002), particularly in relationships (Hendy et al., 2003; Moretti, Bartolo, Slaney, Odgers, & Craig, under review; Moretti, Penney, Obsuth, & Odgers, 2006). Maternal-perpetrated abuse may play an important role in placing children at risk for several reasons. Hendy et al. (2003) have argued that the primacy of maternal versus paternal maltreatment is due to the primary role of mothers as caregivers. Consequently children may be more frequently exposed to maternal rather than paternal maltreatment. Additionally, we have argued that the effects of maternal maltreatment may be a function of mothers' role as primary attachment figures, as evidenced by research showing that mothers are identified as primary sources of emotional support in childhood and extending well into early adulthood (Rosenthal & Kobak, 2010). As primary

attachment figures, mothers shape the development of their children's capacity for self-regulation, especially regulation of negative affect.

Current Study

The current study examined whether deficits in affect regulation, or 'affect dysregulation', partially or fully mediated the relationship between experiences of child abuse and depressive symptoms in a clinical sample of adolescents. To test these predictions, we examined the concurrent and prospective relationships between maternal- and paternal-perpetrated physical and emotional abuse, depressive symptoms and affect dysregulation over a five-year period. Adolescents were recruited from a provincial assessment center for serious behavioral and emotional problems and from a juvenile justice center, populations known to experience abuse at higher than typical levels (Cicchetti & Toth, 2005).

Based on prior research we expected that depressive symptoms would be elevated among teens who reported exposure to physical and emotional abuse when they entered the study and at the second time point of data collection two years later. We expected reports of abuse at these two time points to predict depressive symptoms at the third time point of data collection five years after the start of the study. We also anticipated that teens exposed to abuse would experience more problems regulating affect at each of the three time points of data collection. Finally, and of most theoretical and clinical importance, we predicted increased risk for depressive symptoms as a result of exposure to abuse would be partially or fully mediated through affect dysregulation, and we expected that mediation would be evident both concurrently and prospectively over the five year study period.

We assessed physical and emotional abuse as reported within six-month periods at each time point in the study for two reasons. First, prior research has raised concerns regarding the reliability of autobiographical memory and recall of lifetime abuse, especially among individuals with a history of depression (Brewin, Reynolds, & Tata, 1999; Mackinger, Pachinger, Leibetseder, & Fartacek, 2000). Second, the cumulative nature of lifetime assessments can potentially confound the interpretation of data from longitudinal studies that include multiple assessments as it can be difficult to determine whether results represent exposure from past versus recent events. The effects of maternal- versus paternal-perpetrated abuse were also examined. We predicted that, compared to paternal abuse, maternal abuse may be a more robust predictor of depressive symptoms and affect dysregulation in daughters and sons at each assessment. Gender-specific risk transmission was also tested by evaluating whether daughters were more influenced than sons by maternal than paternal abuse, and also whether sons were more influenced than daughters by paternal abuse.

Methods

Participants

Participants ($N = 179$; 46% female) were part of a larger longitudinal project examining gender and psychopathology among high-risk youth that was conducted in Vancouver and the Lower Mainland in British Columbia, Canada. Approximately half (55%) of the participants were drawn from centers servicing youth involved in the justice system and the remainder were from a provincial assessment center which receives referrals for youth with serious behavior problems and associated comorbid conditions. Exclusionary criteria included IQ below 70 and the presence of Axis I psychotic symptomatology based on file review and interview. Measures used in the current study were administered at enrollment in the study and at two- and five-years' follow-up. All research protocols and procedures received approval from the Office of Research Ethics at the university of the corresponding author. Informed and voluntary parental consent and youth assent were secured at each time point of data collection until youth reached age 19, at which point they provided consent themselves. Youth received a modest honorarium for their participation.

The average age at enrollment was 15.34 years ($SD_{age} = 1.53$); 17.86 ($SD_{age} = 1.48$) at two-year follow-up; and 19.93 ($SD_{age} = 1.59$) at five-year follow-up. The sample was primarily Caucasian (65.4%), with 22.2% self-identifying as Aboriginal and 10.7% self-identifying as African/Caribbean, Asian, Hispanic or other ethnicity.

Measures

Conflicts Tactics Scale (CTS; Straus, 1979)

The Conflict Tactics Scale is a widely used questionnaire that assesses violence and aggression within relationships. In the current study, we utilized a modified version of the CTS that assesses victimization within family, peer and romantic relationships. Teens' experiences of emotional and physical abuse in their relationships with mothers and fathers in the past six months were assessed using 7 items, including one item that assessed emotional abuse (e.g., "Insulted, put down or called names [for example, name calling such as stupid, lazy, or worthless]") and six items that assessed physical abuse (e.g., "Mother/Father slapped you"). Participants rated each item twice, once regarding experiences of maternal abuse and once regarding paternal abuse. Each item was rated on a 4-point scale (1 = *never* to 4 = *always*). Unsurprisingly, adolescents' reports of exposure to emotional and physical abuse were highly correlated for maternal ($r = .63$; $p < .001$) and paternal abuse ($r = .58$, $p < .001$); thus items were combined to form an index of emotional and physical abuse experienced within teens'

relationships with mothers and fathers, respectively. Maternal and paternal abuse was measured during the first and second time points of data collection. Scores represent the mean of all 7 items for each scale. Coefficient alphas were satisfactory for both maternal abuse ($\alpha = .91$) and paternal abuse ($\alpha = .89$). While accounting for missing data, 55.3% of the sample reported any maternal physical or emotional abuse and 48% reported any paternal physical or emotional abuse.

Affect Regulation Checklist (ARC; Moretti, 2003)

The Affect Regulation Checklist is a 12-item measure adapted from published scales of emotion regulation (e.g., Gross & John, 1998) and augmented with supplementary items to tap three aspects of affect regulation in adolescents. In keeping with contemporary models of regulation of affect, the ARC was developed to tap the multidimensional structure of emotion regulation and assesses two maladaptive factors of affect regulation (i.e., lack of control and suppression of affect) and one adaptive factor (i.e., reflection). Importantly, test items were designed to assess regulatory processes independent of affect. Hence, items do not refer to specific emotions and avoid confounding regulatory processes with emotional states (e.g., “I have a hard time controlling my feelings”). Based on the measure’s construction, scale means are derived from responses on a three-point scale (0 = *not like me* to 2 = *a lot like me*).

The three-factor structure of the ARC and its relationships with emotional and behavioral problems has been confirmed in previous research (see Penney & Moretti, 2010). In the current study, we focused on the affect dysregulation subscale given its direct relevance to the deleterious impact of exposure to abuse (e.g., “It’s very hard for me to calm down when I get upset”). Affect dysregulation was measured during all three time points and demonstrated good reliability at Time 1 ($\alpha = .80$), Time 2 ($\alpha = .79$) and Time 3 ($\alpha = .86$).

Ontario Child Health Study-Youth Self-report (OCHS; Boyle, Offord, Racine, & Sanford, 1993)

The Ontario Child Health Study was used to assess depressive symptoms at the first and second time points of the study when participants were adolescents. This scale was developed based on the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV; American Psychiatric Association, 2000)* descriptions of childhood disorders and items contained in the widely used and well-validated Youth Self-report (Achenbach & Rescorla, 2001). All items are scored on a 3-point scale (0 = *never or not true of me* to 2 = *often or very true of me*), and ask about symptoms occurring in the present or in the past six months. The 15-item depressive symptoms scale (e.g., “I am unhappy, sad, or depressed” and “I get no pleasure from my usual activities”) was included in the analyses. Scores on the depressive symptoms scale range from 0 to 45 with higher scores indicating higher levels of depressive symptoms. The internal reliability in the current sample was good at Time 1 ($\alpha = .86$) and Time 2 ($\alpha = .82$). Mean levels from the OCHS depressive symptoms scores were in the subclinical level (1 SD above the mean) compared to a normative sample (Boyle et al., 1993). More specifically, 47% of the youth were subclinical (1 SD) or above at Time 1 and 43% of the youth were subclinical (1 SD) or above at Time 2.

Adult Self-report (ASR; Achenbach & Rescorla, 2003)

The Adult Self-report is the adult version of the Achenbach Youth Self-report (Achenbach & Rescorla, 2001) and was used to assess depressive symptoms during the third time point of data collection when the participants entered young adulthood. The ASR is a 123-item measure that assesses general psychopathology and behavioral difficulties within the past six months in individuals aged 18–59. Responses are measured on a three-point scale (0 = *never or not true* to 2 = *often or very true*) and summed. The DSM-based affective subscale was utilized. It consists of 15 items (e.g., “I cry a lot”, and “I think about killing myself”) that are consistent with the OCHS depression scale (Boyle et al., 1993). This is a widely used instrument with demonstrated validity and reliability (Achenbach & Rescorla, 2001). Internal reliability in the present sample was satisfactory ($\alpha = .87$).

Missing Data and Imputation

Of the 179 youth involved at Time 1, 55% were retained at Time 2 and 47% of the original sample were retained at Time 3. This attrition rate is consistent with other research with similar high-risk populations (e.g., Kim & Cicchetti, 2006). Before completing the analyses, the impact of missingness was assessed by comparing participants who completed Times 2 and 3 with those who did not on demographic variables and baseline study measures. No significant mean differences were found on demographic variables, maternal or paternal abuse, affect dysregulation or depressive symptoms. In addition, no differences were found in the correlations between all Time 1 variables for those who completed all three time points and those who only completed one or two time points of the study.

Missing data were handled through the use of Full Information Maximum Likelihood Estimation (FIML). This method assumes data is missing at random and uses all available data points to produce a vector of means and a covariance matrix that is model-dependent and is considered to be one of the most robust and advanced methods of dealing with missing data (e.g., Arbuckle, 2005). It has also been shown to be appropriate for use with longitudinal data and with data missing due to

Table 1

Means, standard deviations, and range for paternal abuse, maternal abuse, affect dysregulation and depressive symptoms across the 3 waves.

	Mean (FIML estimation)	Mean (original data)	SD	Range
W1 Maternal abuse	1.38	1.39	.57	1.00–3.71
W1 Paternal abuse	1.32	1.32	.51	1.00–3.71
W1 Depressive symptoms	8.84	8.86	5.91	0–27.00
W1 Affect dysregulation	.97	.96	.61	0–2.00
W2 Maternal abuse	1.21	1.21	.35	1.00–2.75
W2 Paternal abuse	1.22	1.21	.39	1.00–3.50
W2 Depressive symptoms	8.84	8.32	4.88	0–20.00
W2 Affect dysregulation	.86	.86	.58	0–2.00
W3 Depressive symptoms	8.10	8.10	3.22	0–24.00
W3 Affect dysregulation	.93	.93	.61	0–2.00

attrition (Graham, 2009). Comparison of means, variances and correlations in our original data and imputed data revealed consistent estimates and relationships among variables (see Table 1).

Statistical Analysis

We first examined zero-order correlations to evaluate predictions regarding the concurrent relationship between exposure to maternal and paternal abuse, depressive symptoms and affect dysregulation at each time point of data collection. The differential relationship of maternal versus paternal abuse was also assessed in this manner.

Prospective and mediated relationships were tested within a *path analysis* framework. Path analysis provides a confirmatory approach to data analysis in which the expected set of relationships among variables can be specified *a priori*

and modeled simultaneously. Path analysis also allows for a direct and empirical comparison of model parameters across groups (e.g., across males and females) through multiple group modeling. All models were fit using AMOS Version 19.0 (Arbuckle, 2005). Models were evaluated according to the most commonly used critical values for the fit indices. Chi-square is a discrepancy function and represents the differences between the observed covariance matrix and the predicted covariance matrix. A non-significant chi-square is regarded as an acceptable model, such that the observed covariance matrix is similar to the predicted matrix. The root mean square error of approximation (RMSEA) is a parsimony-adjusted index which favors a parsimonious model. In addition, RMSEA allows for confidence intervals to be calculated in order to test for both a perfect fit and a close fit. A RMSEA is considered close if it is below .05, and acceptable if it is between .05 and .08 (Hu & Bentler, 1999). The comparative fit index (CFI) is one of the most commonly used incremental fit indices, which attempts to assess the improvement of the proposed model to the null model, where all relationships are assumed to be 0 (Hu & Bentler, 1999). The CFI is also less affected by sample size. A cutoff of .90 to .95 is suggested for an acceptable fit for the CFI (Hu & Bentler, 1999).

Mediation was assessed concurrently by examining the indirect effects as calculated by AMOS 19.0 (Arbuckle, 2005) and using a Sobel test in order to confirm the significance of the indirect effect. To evaluate mediation over time, we assessed the fit of our model predicting that abuse experiences increase risk for depression through affect dysregulation at three time points across adolescence and early adulthood. Mediation was tested in an exploratory fashion by examining the indirect effects as calculated in AMOS 19.0 when only examining the effect of affect dysregulation. In addition, a nested model in which affect dysregulation did not mediate parental abuse was compared to the full mediation model. The combination of these techniques allows for investigative statistical inferences to be made regarding mediation over time.

Results

1. Concurrent and Prospective Relationships between Maternal and Paternal Child Abuse, Affect Dysregulation and Depressive Symptoms.

Table 2 presents zero-order correlations for the complete sample. To assess the mediating role of affect dysregulation over time, an exploratory longitudinal model was created for maternal abuse as depicted in Fig. 1. In order to determine pathways independent of previous levels of functioning, maternal abuse, affect dysregulation and depressive symptoms at Times 2 and 3 were all covaried from their previous time points. In addition, maternal abuse at Time 1 was used as a covariate for depressive symptoms and affect dysregulation at Times 2 and 3.

Fig. 1 depicts only the significant paths between maternal abuse, affect dysregulation and depressive symptoms across time. With data from all time points considered, maternal abuse was not directly related to adolescent and young adult affect dysregulation at Times 2 or 3 or to adolescent depressive symptoms at Time 2. The model showed an acceptable chi-square ($\chi^2(21) = 27.62, p = .15$), root mean square error of approximation (RMSEA = .04) and CFI fit (CFI = .96), and accounted for 14% of the variance in depressive symptoms at Time 1, 32% at Time 2 and 43% at Time 3.

As predicted, maternal abuse at Time 1 was significantly related concurrently with Time 1 depressive symptoms and affect dysregulation independently ($\beta = .23, p < .01$ and $\beta = .23, p < .01$ respectively). Once affect regulation was added as a mediator to the model, the effects of maternal abuse were attenuated but remained significantly related to depressive symptoms ($\beta = .19, p < .05$), suggesting partial mediation. Additionally, an indirect effect of maternal abuse on depressive

Table 2
Correlations between maternal and paternal abuse, affect dysregulation and depressive symptoms across the 3 waves.

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1.	W1 Maternal abuse	1									
2.	W1 Paternal abuse	.36***	1								
3.	W1 Depressive symptoms	.24**	.09	1							
4.	W1 Affect dysregulation	.18*	.15	.34***	1						
5.	W2 Maternal abuse	.56***	.15	.17	.25*	1					
6.	W2 Paternal abuse	-.04	.10	.03	-.03	.14	1				
7.	W2 Depressive symptoms	.32**	-.06	.18	.45***	.19 ^a	-.01	1			
8.	W2 Affect dysregulation	.13	-.03	.41***	.21*	.27**	.11	.42***	1		
9.	W3 Depressive symptoms	.35**	-.10	.20	.37**	.24	.06	.42**	.38**	1	
10.	W3 Affect dysregulation	.17	-.06	.45***	.17	.13	.09	.32*	.60***	.53***	1

^a $p \leq .06$.

* $p < .05$.

** $p < .01$.

*** $p \leq .001$.

symptoms through affect dysregulation ($\beta = .05$) was confirmed by a Sobel test ($z_{\text{sobel}} = 1.78, p = .07$). Given that the Sobel test is a conservative test of indirect effects and has low power (Kenny, 2012), a trend can be considered supportive of partial mediation. Thus, the total effect of maternal abuse on depressive symptoms is a sum of its direct and indirect effects ($\beta = .24$).

Evidence of a full mediation emerged when we examined Time 2 concurrent relationships between maternal abuse, affect regulation and depressive symptoms. Consistent with predictions, a significant relationship emerged between maternal abuse, depressive symptoms and affect regulation independently ($\beta = .19, p = .05$, and $\beta = .27, p < .01$, respectively). Once affect dysregulation was included as a mediator in the model, the relationship between maternal abuse and depressive symptoms was no longer significant ($\beta = .09, p = .37$). In addition, an indirect effect of maternal abuse on depressive symptoms at Time 2 through affect dysregulation ($\beta = .08$) was confirmed with a Sobel test ($z_{\text{sobel}} = 2.16, p < .05$), supporting a fully mediated relationship.

Longitudinally, evidence of partial mediation emerged through the association between maternal abuse at Time 1 and depressive symptoms at Time 2 through affect dysregulation at Time 1 and its relationship with affect dysregulation at Time 2 ($\beta = .05$). Examination of the paths across the three time points of the study revealed that the relationship between maternal abuse at Time 1 and depressive symptoms at Time 3 was partially mediated through the relationship between maternal abuse at Time 1 and affect dysregulation at Time 1, which was subsequently mediated through affect dysregulation at Times 2 and 3. The significant relationship originally observed between maternal abuse at Time 1 and depressive symptoms at Time 3 ($r = .35, p < .01$) remained significant although partially attenuated ($\beta = .24, p < .05$). A modest indirect effect was also found from maternal abuse at Time 1 on depressive symptoms at Time 3 through affect dysregulation at Times 1–3 ($\beta = .04$). This relationship suggests that maternal abuse in adolescence may have a particularly deleterious impact on vulnerability to depressive symptoms as teens enter early adulthood.

Similarly, the relationship between maternal abuse at Time 2 and depressive symptoms at Time 3, which originally showed a trend towards significance ($r = .24, p = .08$), was fully mediated ($\beta = .05, p = .71$) once affect dysregulation at Times 2 and 3 were modeled as mediators. A moderate indirect effect was also found from maternal abuse at Time 2 on depressive symptoms at Time 3 through affect dysregulation at Times 2 and 3 ($\beta = .08$).

In order to confirm longitudinal mediation, we compared the full model as depicted in Fig. 1 with a nested model in which affect dysregulation did not mediate the relationship between maternal abuse and depressive symptoms across

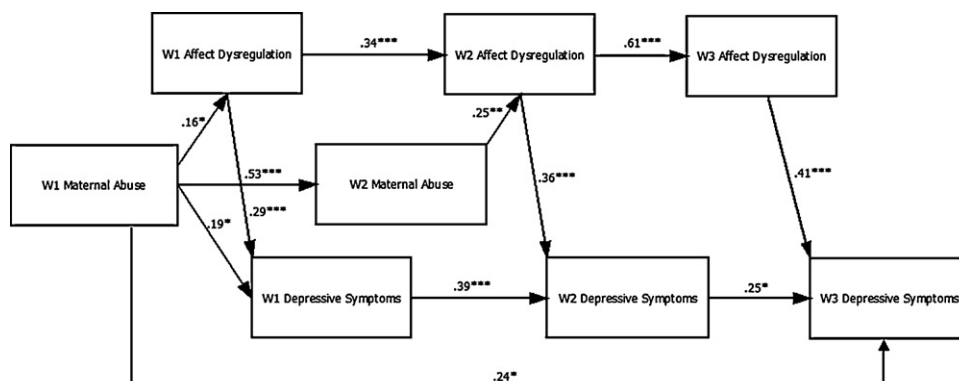


Fig. 1. Path analysis for longitudinal mediation model across all three time points with standard estimates (β). * $p < .05$, ** $p < .01$, *** $p \leq .001$.

the three time points. A chi-square difference test supported the full model ($\Delta\chi^2(3) = 10.65, p < .05$), providing additional support for the longitudinal mediated model.

We were unable to adequately fit the same model (Fig. 1) when examining longitudinal associations between paternal abuse, affect dysregulation and depressive symptoms.

2. Effects of Maternal Versus Paternal Abuse on Affect Dysregulation and Depressive symptoms for Sons and Daughters. (or change the first title to sentence form).

An important question is whether our findings apply similarly across child gender. Zero-order correlations suggested that the relationship between maternal abuse, depressive symptoms, and affect regulation were generally similar for males and females. Paternal abuse was not significantly related to depressive symptoms for males or females at Times 1, 2 or 3; however paternal abuse was significantly related to affect dysregulation concurrently at Times 1 and 2 for males ($r = .37, p < .01$; $r = .33, p < .05$, respectively) but not for females ($r = -.08, p > .05$; $r = -.08, p > .05$, respectively). Fisher's r to z transformation demonstrated these correlations were significantly different at both time points ($z = 2.50, p < .01$ and $z = 1.82, p < .05$, respectively).

Based on these differences, we examined whether our longitudinal mediation model for maternal abuse based on the entire sample fit similarly for girls versus boys using a multi-group comparison model and a chi-square difference test. No gender differences were found ($\Delta\chi^2(12) = 15, p > .05$).

To further explore the impact of paternal abuse, we investigated the fit of the longitudinal paternal abuse model for girls versus boys even though none of our models for paternal abuse showed acceptable fit for the overall sample. In contrast to maternal abuse, the paternal longitudinal model was significantly different across gender ($\Delta\chi^2(11) = 20.76, p < .05$). Closer inspection of the standardized coefficients suggested that for males, there was an indirect effect of paternal abuse on concurrent depressive symptoms through affect dysregulation at Times 1 and 2. More specifically, paternal abuse was related to affect dysregulation in sons concurrently at Time 1 ($\beta = .36, p < .001$) and Time 2 ($\beta = .32, p < .05$) and affect dysregulation was significantly related to concurrent depressive symptoms at Time 1 ($\beta = .34, p < .01$) and Time 2 ($\beta = .48, p < .001$). At both time points, paternal abuse was not directly related to depressive symptoms, which indicates that there was an indirect relationship between paternal abuse and depressive symptoms though affect dysregulation concurrently at Time 1 ($\beta = .13, z_{\text{sobel}} = 2.47, p < .05$) and 2 ($\beta = .19, z_{\text{sobel}} = 2.28, p < .05$). In addition, for males, an indirect relationship between paternal abuse at Time 1 appeared to have an indirect relationship with depressive symptoms at Time 2 ($\beta = .06$) and Time 3 ($\beta = .06$) through affect dysregulation. Paternal abuse at Time 2 also appeared to have an indirect relationship with depressive symptoms at Time 3 ($\beta = .19$). These effects were not found for females. Multi-group comparison results across gender should be interpreted with caution due to the low sample size.

Discussion

Parental abuse is a significant risk factor for problems in regulating affect and depressive symptoms in adolescents. We proposed that the effects of parental abuse on adolescents' depressive symptoms would be mediated through deficits in affect regulation. Such deficits place adolescents at risk for depressive symptoms as they transition to adulthood, limiting their competence in managing difficult emotions and interpersonal challenges. We tested this prediction separately for maternal versus paternal abuse, deriving composite scores for emotional and physical abuse given the very high inter-correlations between these indices, which has been frequently observed in other research (Melançon and Gagné, 2011; Springer et al., 2007).

Our results were largely supportive of our predictions when we examined the concurrent and long-term effects of maternal abuse. As predicted, maternal abuse was associated with adolescents' reports of affect dysregulation and depressive symptoms concurrently and prospectively as teens transitioned to early adulthood. The relationship between maternal abuse and depressive symptoms was partially mediated through affect dysregulation at the first time point of the study when participants were adolescents, and fully mediated at the second time point. Maternal abuse at entry to the study also predicted their depressive symptoms in early adulthood five years later even after accounting for the partial mediating role of affect dysregulation at each of the three time points of the study. These longitudinal relationships through affect regulation showed modest to moderate effects.

These findings suggest that adolescence may be a sensitive developmental period wherein abuse experiences have profound direct and indirect effects on the risk for later depression. Such a view is congruent with growing evidence that adolescence is a unique and complex developmental transition involving neurobiological, cognitive and socio-emotional shifts (Ernst & Fudge, 2009; Moretti & Peled, 2004; Spear, 2000). Our results are consistent with other research showing that experiences of abuse increase vulnerability to depressive symptoms in early (Baldry & Winkel, 2004) and mid-adolescence (Seeds, Harkness, & Quilty, 2010). Previous research also confirms that such experiences have lingering effects into early adulthood (Webb, Heisler, Call, Chickering, & Colburn, 2007) and later adulthood (Springer et al., 2007) that may be due to the negative impact of abuse on the ability to regulate emotions (Soenke et al., 2010) and to disturbances in neurobiological processes associated with child abuse (Nanni, Uher, & Danese, 2012).

Our results are also consistent with the growing literature on the importance of attachment in determining adolescent adjustment (Allen et al., 2002; Moretti & Obsuth, 2011; Moretti et al., 2006). Although autonomy is a developmental imperative during adolescence, teens continue to turn to their mothers for secure base and safe haven (Rosenthal & Kobak,

2010). Mothers not only provide emotional soothing but also important lessons in how to cope with difficult emotional experiences, strategies that can be internalized as adolescents move into adulthood. When emotional and physical abuse is part of the mother–adolescent relationship, opportunities for support and learning are profoundly compromised, limiting adolescents' ability to manage emotional distress. It is possible that such deficits lead to compensatory attempts to regulate emotional distress through substance use or other risk-taking behaviors (e.g., Allen, Chango, Szewedo, Schad, & Marston, 2012).

We found that the impact of maternal abuse, which was fully or partially mediated through affect dysregulation, was equally related to daughters' and sons' levels of depressive symptoms. These findings are consistent with our past research and that of others showing that maternal abuse exerts a pervasive influence over daughters' and sons' adjustment (Moretti et al., submitted for publication; Seeds et al., 2010). However, an interesting pattern of gender differences emerged when we explored the differential impact of paternal abuse on affect dysregulation and depressive symptoms in daughters and sons. Although no evidence was found that paternal abuse influenced daughters, an indirect relationship was found between paternal abuse and sons' depressive symptoms at Times 1 and 2. More specifically, sons exposed to paternal abuse reported more problems in regulating affect at Times 1 and 2 and this in turn predicted depressive symptoms at these time points. This is consistent with previous findings suggesting males may be impacted more by paternal abuse (Baldry & Winkel, 2004). Our findings suggest that boys may be sensitive to paternal abuse through its effects on emerging affect regulation competencies, perhaps as a result of gender modeling. However, such an interpretation is preliminary and requires further examination through research examining gender-specific developmental trajectories of the impact of maternal versus paternal abuse.

A number of clinical implications can be drawn from this study. First, as we have noted elsewhere, there is overwhelming evidence that preventing child abuse is paramount in decreasing vulnerability to the broad array of mental health, physical health, social and emotional problems with which it is associated. The need to prevent child abuse is even more critical given recent findings demonstrating that individuals with a history of child abuse respond more poorly to psychological and psychopharmacological treatments for depression (e.g., Nanni et al., 2012). Our findings extend previous work by highlighting the importance of preventing abuse specifically in adolescence, a period which is frequently associated with exacerbated conflict within families that sometimes leads to family violence and breakdown. This is important as parents, mental health practitioners and government stakeholders often assume that prevention is something that is done only in early childhood. We argue that prevention needs to be developmentally timed, targeting periods of vulnerability with appropriate interventions that target key challenges in such transitions.

Our findings point to three intervention targets. First, parents may benefit from parenting programs preventatively as their children approach adolescence, or as a risk reduction intervention in families with high levels of parent–teen conflict and adolescent conduct problems. There is strong evidence that parenting interventions are effective in preventing and reducing conduct problems, with an excellent return on investment (Roberts & Grimes, 2011). Parents may particularly benefit from parenting programs that help them to regulate their own feelings of frustrations in parenting their teen, and to recognize the importance in providing a secure base and safe haven within the parent–teen relationship as pivotal in their child's development of affect regulation (Moretti, Obsuth, Mayselless, & Scharf, 2012; Moretti & Obsuth, 2009). Such issues may be particularly critical in clinical populations where parents struggle with mental health problems that stem from their own experiences of abuse as children and teens. Second, given the relationship between parents' experiences of abuse and their increased risk of perpetrating abuse on their children (e.g., Haapasalo & Virtanen, 1999), risk may be reduced through interventions to parents themselves. Third, adolescents or young adults who have experienced abuse may also benefit from interventions designed to build affect regulation skills such as cognitive behavior or dialectical behavior therapy (McMain, Korman, & Dimeff, 2001).

Although our results add to the growing literature on the long-term influence of abuse on vulnerability to depression and the importance of affect regulation as a mediator in this relationship, several limitations temper our conclusions. First, although our sample size was sufficient for the analyses we undertook, a larger sample would have allowed us to conduct growth curve modeling, which potentially could provide a better understanding of the diversity of developmental trajectories in the relationship between abuse, affect regulation and depressive symptomatology. Second, a Sobel test was used to confirm mediation in the Time 1 and Time 2 concurrent models. Bootstrapping is currently a more desirable test of mediation, but could not be employed due to missing data (Kenny, 2012). Third, we relied on an exploratory analysis for longitudinal mediation as a Sobel test could not be employed in cases where mediation occurs longitudinally over several time points. Currently, there are no other statistical tests available to test longitudinal associations of this nature. Nonetheless, tentative conclusions can be made. Third, as is common in research with high-risk samples, sample retention was compromised. We were able to employ robust missing data procedures and basic findings were highly similar in original and imputed data. In addition, participants who completed all three time points of the study did not differ on Time 1 variable means and correlations from those who completed one or two time points of the study. However, a higher retention rate would increase confidence in the current findings. We also relied entirely on self-report instruments to assess adolescents' experiences of abuse, affect regulation and depressive symptoms. There are advantages and limitations in relying on self-report data from adolescents; however, collateral reports would have added to our study. Finally, we focused on adolescents' recent experiences of physical and emotional abuse, assessing incidents occurring within a six-month window at each time point in the study. This allowed us to estimate the effects of new experiences of abuse and to avoid confounding these estimates with previous occurrences. However, we did not examine other forms of abuse (e.g., sexual abuse, neglect, and family violence) and we cannot comment on whether some forms of abuse may be particularly salient in predicting affect dysregulation and

depressive symptoms. In addition, we did not examine the effects of multiple and cumulative lifetime abuse which has been shown to be especially deleterious to psychological (Boxer & Terranova, 2008) and physical health (Danese, Pariante, Caspi, Taylor, & Poulton, 2007).

Notwithstanding these limitations, an important strength of our study is the use of a prospective longitudinal design allowing us to model complex relationships over a sensitive developmental period that has previously received little attention. While the clinical sample that we retained may temper generalizations to normative populations, it has important implications for working with high-risk families where the base rate of abuse experiences is elevated. Further research with both typical and atypical populations will add to our understanding of the complex influences of maternal and paternal abuse on adolescents' vulnerability to depression and to the processes and mechanisms that account for such risks.

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