A Longitudinal Study of Emotion Regulation, Emotion Lability/ Negativity, and Internalizing Symptomatology in Maltreated and Nonmaltreated Children

Jungmeen Kim-Spoon, **Dante Cicchetti**, and **Fred A. Rogosch**
Jungmeen Kim-Spoon, Department of Psychology, Virginia Polytechnic Institute and State University; Dante Cicchetti, Institute of Child Development, University of Minnesota and Mt. Hope Family Center; Fred A. Rogosch, Mt. Hope Family Center and University of Rochester

Abstract

The longitudinal contributions of emotion regulation and emotion lability/negativity to internalizing symptomatology were examined in a low-income sample (171 maltreated and 151 nonmaltreated children, from age 7 to 10 years). Latent difference score models indicated that, for both maltreated and nonmaltreated children, emotion regulation was a mediator between emotion lability/negativity and internalizing symptomatology, whereas emotion lability/negativity was not a mediator between emotion regulation and internalizing symptomatology. Early maltreatment was associated with high emotion lability/negativity (age 7) that contributed to poor emotion regulation (age 8), which in turn was predictive of increases in internalizing symptomatology (from age 8 to 9). The results imply important roles of emotion regulation in the development of internalizing symptomatology, especially for children with high emotion lability/negativity.

Keywords

emotion regulation; emotion lability/negativity; child maltreatment; internalizing symptomatology; latent difference score

INTRODUCTION

The current view of child psychopathology emphasizes that one of the most important components of healthy social-emotional development is the acquisition of skills to regulate negative emotions (Blair & Diamond, 2008). To date, however, we have a limited understanding of how emotion regulation and emotion lability/negativity jointly contribute to the development of child psychopathology and how caregiving experiences in early life, such as child maltreatment, may influence the development of emotion regulation and emotion lability/negativity as well as psychopathology throughout middle childhood. Our goal in this study was to conduct a comprehensive longitudinal analysis to examine developmental processes that link emotion lability/negativity and emotion regulation to changes in internalizing symptomatology among maltreated and nonmaltreated children from age 7 to 10 years.

In this study, emotion regulation is conceptualized as the ability to modulate one's emotional arousal to foster an optimal level of engagement with the environment (Cicchetti, Ganiban, & Barnett, 1991). Emotion regulation may have implications for the etiology of internalizing

symptomatology. Cicchetti, Ackerman, and Izard (1995) argued that emotion regulation is critical both in initiating, motivating, and organizing adaptive behavior, and in preventing stressful levels of negative emotions and maladaptive behavior. Specifically, an impaired ability to regulate negative emotionality in a context-appropriate fashion is viewed as a diathesis of pediatric depression (Kovacs, Joormann, & Gotlib, 2008). Prior research suggests that children with internalizing symptomatology, such as anxiety and depression, show dysregulated emotional expression and impoverished emotional awareness (Eisenberg et al., 2001a).

Emotion lability/negativity may be described as children's rapidity in responding to emotion eliciting stimuli and simultaneous difficulty in recovering from negative emotion reactions (Dunsmore, Booker, & Ollendick, 2011). Emotion lability/negativity may be related to sensitivity to affective environmental cues. Children with higher sensitivity seem to experience greater emotional and physical responses to difficult situations and also respond emotionally to a greater number of cues (Pietromonaco & Barrett, 2009). Research on normally developing children has demonstrated that emotion lability/negativity is associated with internalizing problems. Specifically, children with higher emotion lability/negativity show lower levels of competent social functioning (Eisenberg et al., 1995), and adolescents who are more emotionally labile report higher levels of depressive symptoms (Larson, Raffaelli, Richards, Ham, & Jewell, 1990; Silk, Steinberg, & Morris, 2003). Developmentally, children with high levels of negative emotionality, such as anger, are more prone to develop both internalizing and externalizing problems in childhood and adolescence (Bates, Pettit, Dodge, & Ridge, 1988; Eisenberg et al., 1995, 2004; Kim & Deater-Deckard, 2011).

Theorists have viewed self-regulation and reactivity as related yet separate entities, with self-regulation considered a process that serves to modulate reactivity (e.g., Rothbart & Bates, 2006), and empirical studies have reported that emotion-related regulation and reactivity/negative emotionality make unique additive relations to adjustment (Eisenberg et al., 1995, 2005). Calkins (1994) proposed a developmental pathway to emotion regulation that involves child's behavioral traits (e.g., reactivity/resistance in response to frustration and adaptability/reactivity in response to novelty) and regulatory style. She argued that some biological or neuro-regulatory mechanisms predispose a child to a particular behavioral trait, and that children's reactivity exerts a strong effect on the child's failure to develop appropriate regulatory strategies to deal with barriers and issues of control. Accordingly, the effects of a behavioral trait (e.g., reactivity to frustration) on social outcomes (e.g., peer interactions) are thought to be mediated via individual differences in emotion regulation that represent the child's ability to control extreme states of arousal or reactivity in such a way that mutual, reciprocal social interactions become possible.

The developmental progression of emotion lability/negativity and emotion regulation over the first four or so years of life is heavily influenced by parenting behaviors. From the perspective of attachment theory, securely attached children are able to use parents effectively to help them regulate their emotions (Bowlby, 1969/1982). Calkins (1994) further suggested that the association between internal and external sources of individual differences in emotion regulation may be explained by caregiver behavior affecting children reciprocally. Specifically, the caregiver's behavior affects the child's immediate emotional reactivity in a given situation, and it may exert a direct effect on the regulatory strategies that the child uses in particular situations as well as in response to the caregiver's overtures.

During the school-age years, children continue to develop emotion regulation skills as they encounter increasing socialization demands from peers as well as from family. Children's emotion regulation skills may represent a mechanism by which skills learned in the family

context translate to the peer realm (Parke & O'Neill, 1999). For example, children who are more adept at regulating negative emotions show higher social competence in peer interactions and higher peer acceptance, resulting in lower levels of internalizing symptomatology. In contrast, poor emotion regulation is a significant predictor of both maladaptive social functioning, such as peer rejection and internalizing symptomatology (Kim & Cicchetti, 2010). Thus, extant literature suggests significant roles of emotion lability/negativity and poor emotion regulation in the development of internalizing symptomatology throughout middle childhood. Nevertheless, no known study has systematically examined the longitudinal relations among emotion lability/negativity, emotion regulation, and internalizing symptomatology during this particular developmental period. Studying the contributions of these two facets of emotional systems to internalizing symptomatology during middle childhood is important given that emotional and social difficulties show considerable stability across middle childhood and into adolescence (Lahey et al., 1995), and that the emergence of psychopathology during middle childhood may well continue to evolve into maladjustment in adolescence.

As has been suggested by researchers and theorists, emotional development is contingent on the nature of the input or experiences made available to the child (e.g., Calkins & Fox, 2002). Early child maltreatment presents a significant threat to the optimal development of emotional understanding and regulation, partly due to the absence of sensitive interactions between the caregiver(s) and the child. In maltreating families, parents are less likely to be available to provide support and scaffolding—from which children can learn constructive strategies to regulate their emotional states—when their children are upset. An unpredictable and disorganized environment, such as those found in maltreating homes (Howes, Cicchetti, Toth, & Rogosch, 2000), would make children particularly vulnerable to frequent negative emotional experiences including anger, frustration, reactivity, and irritability (Alessandri, 1991; Erickson, Egeland, & Pianta, 1989; Shields & Cicchetti, 1998). Thus, maltreated children are likely to experience overwhelming emotional arousal that leads to difficulties managing and processing negative emotions (Cummings, Hennessy, Rabideau, & Cicchetti, 1994).

Indeed, existing literature indicates that maltreated children show numerous deficits in the recognition, expression, and understanding of emotions (e.g., see Camras, Sachs-Alter, & Ribordy, 1996 for a review). Compared to nonmaltreated children, maltreated children exhibit higher levels of negative emotionality, particularly anger reactivity (Erickson et al., 1989; Gunnar & Donzella, 2002), and greater difficulties in regulating affective experiences (Kim-Spoon, Haskett, Longo, & Nice, in press; Shipman et al., 2007). In addition, emotion regulation appears to be important for understanding linkages between maltreatment experiences and maladjustment. For example, maltreated children's deficits in their understanding of negative affect mediated the link between earlier physical abuse and later rejection by peers (Rogosch, Cicchetti, & Aber, 1995). Furthermore, a recent longitudinal study demonstrated that children with early experiences of abuse and neglect were more likely to show poor emotion regulation. Consequently, those children with poor emotion regulation were less likely to be accepted by peers and were likely to show higher internalizing symptomatology one year later, even after controlling for initial levels of internalizing symptomatology (Kim & Cicchetti, 2010).

In the present study, we addressed the following hypotheses and questions. First, we expected that poor emotion regulation and high emotion lability/negativity would be independently predictive of increases in internalizing symptomatology over time. Second, we examined the developmental processes through which emotion regulation and emotion lability/negativity are related to internalizing symptomatology. Specifically, we tested mediation models to examine effects that progress from emotional systems to

psychopathology. Within emotional systems, we tested whether emotion regulation serves as a mediator between emotion lability/negativity and changes in internalizing symptomatology, or alternatively, whether emotion lability/negativity serves as a mediator between emotion regulation and changes in internalizing symptomatology. Furthermore, we tested how an external risk factor, such as maltreatment, and child internal factors, such as emotion lability/negativity and regulation, may transact to influence the development of internalizing symptomatology. We expected stronger contributions of emotion lability/ negativity and dysregulation among maltreated children compared to nonmaltreated children based on the vulnerable-reactive model (Luthar, Cicchetti, & Becker, 2000). The vulnerable-reactive model suggests that negative effects of vulnerable attributes (i.e., high emotional negativity/lability and/or poor emotion regulation) may be heightened with increasing levels of stress. For maltreated children, compared to nonmaltreated children, high emotional negativity/lability is likely to occur without contextual support for the regulation of negativity/lability, and thus to lead to poorer developmental outcomes (Blair, 2010). Finally, we investigated direct and indirect effects of early maltreatment on the development of emotion lability/negativity, emotion regulation, and internalizing symptomatology. We expected that the detrimental effects of early maltreatment on the development of internalizing symptomatology may be mediated by higher levels of emotion lability/ negativity which reduces subsequent ability to regulate emotions thus resulting in increased levels of internalizing symptomatology over time.

METHOD

Participants

The participants included 322 children (171 maltreated and 151 nonmaltreated) who attended a week-long day camp program for inner city children. The average age for the first camp attendance was 7.5 years ($SD = 1.1$) and the current data were drawn from four consecutive years covering age 7 to 10 years. About 61% were boys. Children were from diverse ethnic backgrounds: 63% African American, 21% European American, 14% Latino, and 2% other ethnic groups. Parents of all children provided informed consent for their child's participation and for examination of Department of Human Services (DHS) records pertaining to the family. Maltreated children had been identified by the County DHS as having experienced child abuse or neglect. A recruitment liaison from DHS contacted eligible maltreating families, explained the study, and, if parents were interested, released their names to the study team for recruitment. Comprehensive searches for DHS records were completed and maltreatment information was coded utilizing operational criteria from the maltreatment nosology specified in the Maltreatment Classification System (MCS; Barnett, Manly, & Cicchetti, 1993).

Because the maltreated children were predominantly from low-socioeconomic-status families, demographically comparable nonmaltreated children were recruited from families receiving Temporary Assistance to Need Families (TANF). A DHS recruitment liaison contacted eligible nonmaltreating families and described the study, and, if interested, parents signed a release for their names to be given to the study team for recruitment. DHS record searches were completed for these families to verify the absence of any record of child maltreatment. Trained research assistants also interviewed mothers of children recruited for the nonmaltreatment group to confirm a lack of DHS involvement and prior maltreatment experiences utilizing the Maternal Maltreatment Classification Interview (Cicchetti, Toth, & Manly, 2003). Subsequently, record searches were conducted in the year following camp attendance to verify that all available information had been accessed. Only children from families without any history of documented maltreatment were retained in the nonmaltreatment group. The demographic characteristics of nonmaltreating families were

highly similar to those of the maltreating families, enabling us to assess the independent effects of maltreatment beyond the influences of socio-economic adversity.

Both maltreated and nonmaltreated groups included children from families living in economically disadvantaged neighborhoods. At least 80 % of the families were from the lowest socioeconomic strata (Hollingshead, 1975, Levels 1 or 2). Most of the families were headed by single parents (typically mothers), relied heavily on public assistance, and had a very low income level. The maltreated group and the nonmaltreated group were comparable with respect to family characteristics, including parental marital status (66% of families in the maltreated group and 58% in the nonmaltreated group were headed by single parents), χ^2 (322) = 2.08, p = .17, history of reliance on public assistance (91% of families in the maltreated group and 95% in the nonmaltreated group had received or were receiving TANF), χ^2 (322) = 1.92, p = .12) and income level (a total family income $20,267 for the maltreated group and $21,051 for the nonmaltreated group), t (315) = .59, p = .55. There was a higher percentage of boys in the maltreated group (67% in the maltreated group and 55% in the nonmaltreated group), χ^2 (322) = 5.11, p < .05. The majority of the children were from ethnic minority backgrounds regardless of maltreatment status; however, there were lower numbers of ethnic minority children in the maltreated group (71%) compared to the nonmaltreated group (87%), χ^2 (322) = 13.22, p < .05.

Procedure

After obtaining parental consent and child assent, children participated in a variety of recreational activities in groups of six to eight, with same-age and same-sex peers. Half of the children in each of the groups were maltreated and the other half were nonmaltreated. Each camp group was led by three camp counselors who were unaware of the children's maltreatment status. The counselors were trained on completing assessments based on their observations and interactions with the children in their respective groups. The counselors completed a number of assessment instruments at the end of each week. Each year children encountered a different group of peers in their camp groups and different camp counselors, providing diverse contexts for assessment of their functioning.

Measures

Maltreatment Classification System (MCS)—The MCS provided operational definitions and specific criteria to designate different subtypes of maltreatment. Severity of each subtype was rated along a 5-point scale, with 1 indicating mild maltreatment to 5 indicating severe maltreatment of the specified subtype. *Emotional Maltreatment* involved extreme thwarting of children's basic emotional needs, such as the need for psychological security and for age-appropriate autonomy. *Neglect* was coded when a responsible adult failed to meet a child's needs for food, clothing, shelter, medical-, dental-, or mental-health care, adequate hygiene, physical safety, or education. *Physical Abuse* involved injuries that were inflicted upon a child by non-accidental means. Finally, *Sexual Abuse* was coded when any sexual contact or attempted sexual contact occurred between a child and caregiver for the caregiver's satisfaction or financial benefit. In the present sample, 79% of maltreated children experienced emotional maltreatment, 78% were neglected, 39% had been physically abused, and 15% had been sexually abused. Consistent with the high co-occurrence of subtypes that are found in the literature, 77% of the maltreated children in this sample experienced two or more forms of maltreatment. Adequate reliability was obtained for each maltreatment subtype with kappas ranging from .78 to 1.00 for these subtypes.

Among 133 maltreated children whose information regarding the timing of maltreatment experience was available, the age of onset occurred during infancy (0 to < 18 months) for 63% (n = 84), during toddlerhood (18 months to < 36 months) for 22% (n = 29), and during

preschool (3–5 years) for 15% ($n = 20$). Maltreatment in multiple developmental periods can be considered chronic. Approximately 50% ($n = 66$) of the maltreated children had maltreatment experiences during all three developmental periods (i.e., infancy, toddlerhood, and preschool periods), 26% ($n = 35$) during two developmental periods, and 24% ($n = 32$) during only one developmental period. For 97% of the maltreated children, the child's biological mother was identified as a perpetrator for some form of maltreatment.

Emotion Regulation Checklist (ERC)—Counselors' ratings on the Emotion Regulation subscale (8 items) and the Lability/Negativity subscale (15 items) of the ERC (Shields & Cicchetti, 1998) were used to capture processes central to emotion regulation and emotion lability/negativity. The emotion regulation subscale consisted of items assessing adaptive regulation, including socially appropriate emotional displays, empathy, equanimity, and emotional understanding. Higher scores indicated a superior capacity to modulate one's emotional arousal such that an optimal level of engagement with one's environment is fostered (e.g., "Is empathic toward others"; "responds positively to neutral or friendly overtures by peers"). The emotion lability/negativity subscale was composed of items assessing arousal, reactivity, emotional intensity, expression of negative emotions, and mood lability. (e.g., "is prone to angry outbursts"; "exhibits wide mood swings"). Both construct validity and discriminant validity have been demonstrated for the ERC (Shields & Cicchetti, 1998). To evaluate the factor structure of the ERC, we conducted a principal-components factor analysis using the current sample (N = 322). We first analyzed Time 1 data (age 7 or 8 data) and replicated using Time 2 data (1-year follow-up data). This analysis yielded two separate factors of emotion regulation (with factor loadings ranging .42~.84 at Time 1 and .47~.85 at Time 2) and emotion lability/negativity (with factor loadings ranging .29~.86 at Time 1 and .35~.86 at Time 2). The two factors were correlated at −.47 at both times. For the current sample, Cronbach's alphas (averaged for age 7–9) were .81 for the emotion regulation subscale and .95 for the emotion lability/negativity subscale. At the end of each week, each child was rated by two of the child's camp counselors, and the subscale scores were computed by averaging across counselors' ratings. Inter-rater reliabilities were assessed using averaged intraclass correlation coefficients (ICC; absolute agreement between raters) across ages, and they were .72 for the emotion regulation subscale and .85 for the emotion lability/negativity subscale.

Teacher's Report Form (TRF)—Children's internalizing behavior was assessed at the end of each week through completion of the TRF (Achenbach, 1991). The TRF is an extensively used and well-validated assessment instrument designed to evaluate a wide range of child symptomatology. On the TRF, camp counselors rated the frequency of occurrence of a list of 118 problem behaviors that form two broad-band factors of internalizing (e.g., withdrawal, somatic complaints, anxiety-depression) and externalizing (e.g., aggressive behaviors, delinquent behaviors) symptoms. Children were rated by two camp counselors, and the total raw scores for internalizing symptomatology were computed by averaging across counselors' ratings. For the current sample, inter-rater reliability (an averaged ICC over time) was .68. Because our longitudinal analyses focused on change scores (consistent with previous studies of latent different score models), we used raw scores. When we examined the T-scores above the clinical cutoff points ($T > 63$), approximately 9% of the children in the present sample showed internalizing symptomatology above the clinical range at age 7, 6% at age 8, 6% at age 9, and 5% at age 10.

Data Analytic Plan

We tested latent difference score (LDS) models (McArdle, 2009), in which annual change in internalizing symptomatology was estimated and statistically predicted from repeatedly

measured emotion regulation and emotion lability/negativity. We used the Mplus Version 6.0 statistical software package (Muthén & Muthén, 2010) that estimated parameters incorporating full information maximum likelihood (FIML) methods. The FIML estimation procedure allows data from all individuals to be included regardless of their pattern of missing data and is more appropriate than other commonly used methods such as mean substitution (Arbuckle, 1996). In the LDS models, there is a latent intercept, and latent changes are modeled as a function of two components: a linear slope that represents the constant change, or natural change, component (i.e., the mean of the slope factor), and the scores on the same variable at the previous occasion referred to as proportional change.

A notable strength of the LDS analysis lies in its provision of a statistical framework for evaluating dynamic longitudinal changes within time series data while considering interrelations between multivariate change processes (McArdle, 2009). Other analytic approaches for studying changes, such as bivariate latent growth curve modeling, cannot represent time-based dynamic relations, where the effect on change in one variable depends on the state of another variable and any prior change in the system over time. Additionally, compared to the use of a manifest difference score, the LDS model offers an advantage of modeling change in perfectly reliable scores over a time series (by partitioning true scores from measurement errors), thus reducing the likelihood of bias in the estimates of parameters describing that change and enhancing power.

Our preliminary LDS analyses indicated that the means and variances of latent difference score factors were not significantly different from zero for emotion regulation and emotion lability/negativity. Therefore, the time series data of predictors (emotion regulation and emotion lability/negativity) were constructed as a Markov simplex model based on manifest variables instead of a bivariate LDS model. The baseline LDS model for internalizing symptomatology showed significant and negative path estimates for proportional change for internalizing symptomatology indicating annual decreases in internalizing symptomatology from age 7 to 10 (b = −1.08, SE = .17, $p <$ .05). The model-estimated mean levels of internalizing symptomatology decreased from 6.89 at age 7 to 5.83 at age 8, to 4.78 at age 9, and to 3.69 at age 10. From age 7 to age 8, 45% of the children showed decreases, 2% no change, and 53% increases. From age 8 to age 9, 50% of the children showed decreases, 5% no change, and 45% increases. From age 9 to age 10, 52% of the children showed decreases, 3% no change, and 45% increases.

We were primarily interested in developmental processes by which emotion regulation and emotion lability/negativity conjointly contributed to change in internalizing symptomatology. We first tested two separate models estimating only direct effects of emotion regulation or emotion lability/negativity on the latent difference scores of internalizing symptomatology. Next, we tested time-lagged mediation models estimating whether the prospective prediction of emotion lability/negativity was mediated by emotion regulation or vice versa. Finally, we introduced maltreatment as a predictor in the meditational LDS model to test whether the risk factor in the child-rearing environment may directly influence the emotional systems of emotion lability/negativity and emotion regulation and further contribute to the development of internalizing symptomatology.

RESULTS

In the present data, the number of participants varied depending on age and measures because not every child participated in the camp across all years from age 7 to 10. Since we were mainly interested in developmental change in the links between emotional systems and internalizing symptomatology, we included 322 children who had available data for at least two time points over the four assessments. Out of 322 children, 208 children had age 7 data,

238 children had age 8 data, 266 children had age 9 data, and 211 children had age 10 data. About 35% of children had two data points, 44% three data points, and 21% all four data points. To investigate the impact of incomplete data (i.e., missing at random), we performed a series of regression analyses testing if missingness (i.e., number of missing time points) was related to any of the study variables. Missingness was not related to any demographic characteristics—gender, race, family income ($p = .26$~$.98$), or maltreatment status ($p = .27$). The critical value (C.V.) of the Bonferroni multiple test was used to test the significance of the effect of missingness for repeated measures data. The C.V. ($\alpha = .05$ with N > 100) was t 2.73 for internalizing symptomatology (i.e., 4 tests, age 7~10 scores) and t 2.39 for emotion lability/negativity and emotion regulation (i.e., 3 tests, age 7~9 scores). The results indicated that the outcome of internalizing symptomatology and the predictors of emotion lability/negativity and emotion regulation were not affected by the number of missing assessments ($t = .06$~1.94 for internalizing symptomatology, $t = .41$~1.59 for emotion lability/negativity, and $t = .21$~-1.43 for emotion regulation).

Table 1 shows means and standard deviations for study variables separately for maltreated and nonmaltreated children. The differences between maltreated and nonmaltreated children and possible gender differences were investigated by multiple analysis of covariance (MANCOVA) for emotion regulation, emotion lability/negativity, and internalizing symptomatology. The main effects of maltreatment status (maltreated vs. nonmaltreated), gender, and the interaction effect between maltreatment and gender were tested. The significant main effects of maltreatment indicated that maltreated children showed higher levels of internalizing symptomatology than nonmaltreated children from age 7 to 9 ($p < .05$), but not at age 10 ($p = .17$). In addition, maltreated children exhibited significantly higher levels of emotion lability/negativity and lower levels of emotion regulation than nonmaltreated children from age 7 to 9 (p .05). The main effects of gender were not significant for internalizing symptomatology ($p = .24$~$.87$) but were significant for emotion regulation and emotion lability/negativity ($p < .05$) suggesting that girls showed higher emotion regulation and lower emotion lability/negativity from age 7 to 9. There was no significant maltreatment by gender interaction effects ($p = .06$~$.88$). Zero-order correlations among study variables are presented in Table 1. The concurrent correlations between emotion regulation and emotion lability/negativity were moderate, ranging from $r = -.45$ to $-.58$, $p < .05$.

We examined time-lagged mediation effects testing whether the prospective prediction of emotion lability/negativity was mediated by emotion regulation or vice versa. These models were tested using two-group structural equation modeling (SEM) approaches to explore any significant differences between maltreated and nonmaltreated groups. We first tested the Direct Effect models in which the time series data of emotion regulation or emotion lability/negativity predicted the latent difference scores of internalizing symptomatology. As shown in Table 2, the non-significant difference between the configural invariance model (where all parameters were freed to be estimated) and the Equal Direct Effect model (where direct effects were fixed to be equal between the two groups) indicated that there was no difference in the direct effects of emotion regulation and emotion lability/negativity between maltreated and nonmaltreated children. Higher emotion regulation predicted decreases in internalizing symptomatology at all ages ($p < .05$). In contrast, higher emotion lability/negativity predicted increases in internalizing symptomatology from age 7 to 8 and from age 8 to 9 ($p < .05$), but its effect became weaker between ages 9 and 10 ($p = .08$). We also tested the reciprocal effects of internalizing symptomatology on emotion regulation and emotion lability/negativity (e.g., age 7 internalizing age 8 emotion regulation, diff1 age 9 emotion regulation, diff2 age 9 emotion regulation; `diff' indicates the latent difference score factor as shown in Figures 1 and 2), but these reciprocal regressions were not significant and thus were not retained in further analyses.

Next, two sets of longitudinal mediation models were tested: first, with emotion regulation as a mediating variable in the prospective relations between emotion lability/negativity and change in internalizing symptomatology, and second, with emotion lability/negativity as a mediating variable in the prospective relations between emotion regulation and change in internalizing symptomatology. As shown in Figure 1, we examined time-lagged mediation effects, for example, age 7 emotion lability/negativity   age 8 emotion regulation   change in internalizing symptomatology between ages 8 and 9. Within each set, we performed two-group SEM analyses and compared two nested models to evaluate any differences between maltreated and nonmaltreated children. In the first model, the Configural Invariance Model, all the parameters were freed to be estimated for maltreated and nonmaltreated children. In the second model, Equal Direct and Mediated Effect model, both direct and indirect effects were fixed to be equal between maltreated and nonmaltreated groups. Table 2 presents the summary results for the comparisons between the two nested models within the two sets of mediation analyses.

In the first set of models in which emotion regulation was tested as a mediator, the model fit of the Equal Direct and Mediated Effect model was not significantly worse than that of the Configural Invariance Model, indicating no difference in direct and indirect effects of emotion lability/negativity between maltreated and nonmaltreated groups. As shown in Figure 1, the results of the Equal Direct and Mediated Effect model indicated that higher levels of emotion lability/negativity at age 7 and at age 8 predicted lower levels of emotion regulation at age 8 and at age 9 respectively, which in turn predicted a subsequent increase in internalizing symptomatology from age 8 to 9 and from age 9 to 10 respectively. These influences were above and beyond, or controlling for, autoregressive effects and natural change (i.e., decreasing slope) in internalizing symptomatology. However, previously significant direct effects of emotion lability/negativity on internalizing symptomatology became non-significant. Sobel's approximate significance tests (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002) for the significance levels of the indirect effects revealed that both of the paths involved in the mediation (emotion lability/negativity   emotion regulation   diff2/3) were significant: $z = 2.21$, $p < .05$ for prediction of diff2; and $z = 2.15$, $p < .05$ for diff3. The significance test for the equality of the two mediated effects indicated consistent (of similar magnitude) mediated effects of emotion regulation for the link between emotion lability/negativity and internalizing symptomatology across ages ($z = 0.03$, $p = .98$).

In the second set of models in which emotion lability/negativity was tested as a mediator of the link between emotion regulation and change in internalizing symptomatology, the Equal Direct and Mediated Effect Model was selected as the best model indicating no significant difference in direct and indirect effects of emotion regulation between maltreated and nonmaltreated groups (see Table 2). Specifically, higher levels of emotion regulation directly predicted a subsequent decrease in internalizing symptomatology at all ages (b = −2.52, SE = .56, $p < .05$ for age 7 emotion regulation   diff1; b = −2.62, SE = .58, $p < .05$ for age 8 emotion regulation   diff2; b = −2.43, SE = .58, $p < .05$ for age 9 emotion regulation   diff3). However, neither the cross-lagged effects of emotion regulation on emotion lability/negativity ($p = .38$ ~.50) nor the direct effects of emotion lability/negativity on internalizing symptomatology difference scores ($p = .18$ ~.49) were significant. Therefore, the results of our mediation models clearly indicated that emotion regulation is a mediating process for the longitudinal association between emotion lability/negativity and internalizing symptomatology, whereas emotion lability/negativity is not a mediating process for the longitudinal association between emotion regulation and internalizing symptomatology.

The results of mediation models suggested that emotion regulation may be a proximal process through which the effects of emotion lability/negativity on internalizing symptomatology are actualized for both maltreated and nonmaltreated children. We further investigated whether child maltreatment, as a risk factor in the rearing environment, may directly influence the processes through which emotion lability/negativity and emotion regulation are related to developmental changes in internalizing symptomatology. Given the significant differences in gender and ethnic composition between maltreated and nonmaltreated groups, we included gender and ethnicity as covariates. Significant effects of these covariates indicated that girls, compared to boys, showed higher emotion regulation and lower emotion lability/negativity at age 7 but greater increases in internalizing symptomatology between ages 7 and 8. White children, compared to ethnic minority children, exhibited greater increases in internalizing symptomatology between ages 7 and 8 and between ages 9 and 10.

As shown in Figure 2, maltreatment predicted low initial levels of emotion regulation and high initial levels of emotion lability/negativity and internalizing symptomatology. In addition, beyond the predictions of initial levels, maltreatment predicted lower levels of emotion regulation at age 8 and higher levels of emotion lability/negativity at age 9, after controlling for autoregressive effects. Consistent with the previous mediation model without the maltreatment predictor, higher levels of emotion lability/negativity at age 7 and at age 8 predicted lower levels of emotion regulation at age 8 and at age 9 respectively, which in turn predicted a subsequent increase in internalizing symptomatology from age 8 to 9 and from age 9 to 10 respectively. In contrast, the direct effects of emotion lability/negativity on difference scores of internalizing symptomatology were not significant.

The significance levels of the indirect effects of maltreatment involving multiple mediators were evaluated by product-of-coefficients tests using Delta method standard errors (Taylor, MacKinnon, & Tein, 2008). For the two-path mediated effects that involved a single-mediator, there were significant mediated effects of maltreatment on the age 7–8 difference score of internalizing symptomatology through age 7 emotion regulation (b = .49, SE = .19, $p < .05$) and on the age 8–9 difference score of internalizing symptomatology through age 8 emotion regulation (b = .32, SE = .15, $p < .05$). The mediated effect of maltreatment on the age 9–10 difference score of internalizing symptomatology through age 9 emotion regulation was not significant (b = .03, SE = .04, $p = .50$). In addition, there was a significant mediated effect of maltreatment on the age 8–9 difference score of internalizing symptomatology through age 7 and age 8 emotion regulation scores (two mediators; b = .22, SE = .10, $p < .05$). The mediated effect of maltreatment on the age 9–10 difference score of internalizing symptomatology through age 7, age 8, and age 9 emotion regulation scores (three mediators) approached significance (b = .07, SE = .04, $p = .06$). Most importantly, evidence was found for a significant three-path mediated effect involving two mediators, emotion lability/negativity and emotion regulation, in series (i.e., maltreatment   emotion lability/negativity   emotion regulation   internalizing symptomatology). The mediated effect of maltreatment on age 8–9 difference score of internalizing symptomatology via two mediators of age 7 emotion lability/negativity and age 8 emotion regulation was significant (b = .10, SE = .05, $p < .05$).

DISCUSSION

We investigated longitudinal processes by which emotion lability/negativity and emotion regulation contribute to the development of internalizing symptomatology in an effort to better understand the nature of the link between the developing emotional systems and child psychopathology. We used latent difference score models to fill the gap in the literature by examining cross-lagged associations among emotion lability/negativity, emotion regulation,

and child internalizing symptomatology and determining directionality in the prediction of emotion lability/negativity and emotion regulation for change in internalizing symptomatology.

In the current sample of maltreated and nonmaltreated children, emotion regulation and emotion lability/negativity both were significant independent predictors of internalizing latent change scores, with lower levels of emotion regulation and higher levels of emotion lability/negativity being associated with increases in internalizing symptomatology from one year to the next. The findings dovetail with others demonstrating the significant roles of emotion regulation and emotion lability/negativity in the development of child psychopathology in normative samples (e.g., P. M. Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996; Eisenberg et al., 2001a). Prior research has shown that negative emotionality, such as anger, was related to elevated levels of internalizing symptomatology among children and adolescents (Oldehinkel, Hartman, Ferdinand, Verhulst, & Ormel, 2007; Rydell, Berlin, & Bohlin, 2003). Furthermore, dysregulation of anger predicted higher levels of internalizing symptomatology, whereas constructive coping with anger was related to lower levels of internalizing symptomatology among children (Zeman, Shipman, & Suveg, 2002).

We further tested longitudinal mediation models and found that emotion regulation served as a mediator for the longitudinal link between emotion lability/negativity and change in internalizing symptomatology among maltreated and nonmaltreated children. Specifically, those with high emotion lability/negativity showed poor emotion regulation in the following year. In turn, poor emotion regulation predicted a subsequent increase in internalizing symptomatology. However, there was no evidence of emotion lability/negativity serving as a mediator between emotion regulation and change in internalizing symptomatology. In addition, the reciprocal effects of internalizing symptomatology on emotion regulation and emotion lability/negativity were not significant. According to the vulnerable-reactive model (Luthar et al., 2000), we expected that the contribution of vulnerability factors (i.e., high emotion lability/negativity and poor emotion regulation) would be stronger for maltreated children than for nonmaltreated children. However, the patterns of mediational processes were similar between maltreated and nonmaltreated children. In these longitudinal mediation analyses, the weight of evidence favored emotion regulation as a mediator, thereby supporting the proposition that regards emotionality as a primary precursor of individual differences in emotion regulation (Calkins, 1994) and suggesting the critical role of emotion regulation in linking emotion lability/negativity to internalizing symptomatology. Although caution is required when interpreting statistical mediation from correlational (albeit longitudinal) data, the results may implicate emotion lability/negativity as a vulnerability factor that may impair the development of appropriate regulatory strategies to manage emotion, which in turn may contribute to the emergence and maintenance of internalizing symptomatology (Calkins & Fox, 2002; Cicchetti & Toth, 1998).

Therefore, consistent with the perspective that views impairment in self-regulatory responses to negative affect as predictive of childhood depression (e.g., Kovacs et al., 2008), our findings underscore the important role of emotional systems in the development of internalizing symptomatology in childhood. Prior studies have tested and demonstrated unique additive effects for emotion lability/negativity and regulation on child adjustment. For example, negative emotionality was a significant predictor of internalizing problems, whereas regulation was not (Eisenberg et al., 2005). Using latent difference score modeling, we examined the temporally lagged prediction of changes in internalizing symptomatology from prior levels of emotion regulation and emotion lability/negativity, and found that the detrimental effects of high emotion lability/negativity on changes in internalizing symptomatology were fully mediated by poor emotion regulation among maltreated and

nonmaltreated children. Such findings imply that emotion regulation skills can be a significant protective factor for child maladjustment problems (e.g., Gottman, Katz, & Hooven, 1996; Thompson & Calkins, 1996). Adaptive emotion regulation is likely to be developed among children with low emotion lability/negativity and may facilitate school-age children's abilities to establish positive peer relationships by promoting prosocial attributes (such as empathy) and further enhance their social competence. Children with higher social competence, in turn, are less likely to become vulnerable to developing internalizing symptomatology (Burt, Obradovic, Long, & Masten, 2008; Kim & Cicchetti, 2010). Thus, our findings provide preliminary evidence suggesting that children's vulnerability to emotion lability/negativity can interfere with the development of adaptive emotion regulation necessary for healthy socioemotional adjustment.

The current results have important implications for prevention and intervention processes with at-risk children with high emotion lability/negativity. As represented in our meditational model, interventions that enhance children's ability to develop effective skills in regulating negative emotional experiences (the mediating process) may in turn prevent the development of internalizing symptomatology. In particular, policies and programs that focus on parenting behaviors are likely to be beneficial. Extant literature suggests that parents can provide support to children to promote effective emotion regulation by directing positive emotion and behaviors toward children, helping children to label and discuss emotions, soothing their children's reactions through appropriate physical contact, encouraging activities such as reading or drawing that reduce arousal, and redirecting attention and cognitively reframing emotions with children (e.g., Calkins & Hill, 2007; Eisenberg et al., 2001b; Morris et al., 2010). Furthermore, school-based preventive intervention that focuses on promoting social emotional competence among school-aged children seems to be effective in fostering emotion regulation development. For example, the PATHS (Promoting Alternative Thinking Strategies) curriculum has been shown to be successful in reducing in internalizing problems among elementary school students by teaching them to identify, understand, and discuss their emotions (Greenberg, Kusche, Cook, & Quamma, 1995).

We tested whether the longitudinal effects of emotion lability/negativity and emotion regulation are influenced by early maltreatment experiences. Our findings suggested that the developmental outcome of internalizing symptomatology is a function of emotional lability/negativity and maturation of regulation as well as the extent to which the non-optimal caregiving behavior, such as maltreatment, fails to support the development of self-regulation (Calkins & Fox, 2002). The significant mediated effects of earlier maltreatment on later internalizing symptomatology illustrate the cumulative progressive consequences of developing emotion systems resulting in changes in child adjustment. In particular, our results indicated two different ways by which earlier maltreatment experiences were related to later internalizing symptomatology. First, harmful effects of maltreatment on changes in internalizing symptomatology were mediated through heightened levels of emotion lability/negativity that contributed to poor emotion regulation over time. The findings suggest that early stress may present risks for disturbances in emotional systems (Blair, 2010; Shields & Cicchetti, 1998) and ultimately for increases in internalizing symptomatology.

Second, our results emphasize the important role of emotion regulation in the development of internalizing symptomatology in childhood. That is, emotion regulation had relatively consistent mediating effects between early maltreatment and changes in internalizing symptomatology from age 7 to 10 years. The mediating effects of emotion regulation between maltreatment and changes in internalizing symptomatology were stronger for the time between age 7 to 9 years compared to the time between age 9 to 10 years. The finding may be explained by decreases in the effects of emotion regulation in changes in

internalizing symptomatology as shown in Figure 2 as well as by decreases in the magnitude of the concurrent association between emotion regulation and internalizing symptomatology as shown in Table 1. The finding is consistent with a prior study that used LDS models and reported that the effects of negative emotion, such as anger, on changes in internalizing symptomatology gradually declined from age 4.5 to 11 years among children from the Study of Early Child Care and Youth Development (Kim & Deater-Deckard, 2011). In addition, the weakening mediated effects of early maltreatment on changes in internalizing symptomatology may be due to decreases in the effects of early maltreatment on emotion regulation as shown in Figure 2. Taken together, these findings imply that emotion regulation may be more important for the development of internalizing symptomatology in the earlier school years than in the later school years because younger children lack the cognitive and social resources that help older children cope with stress (P. M. Cole, Luby, & Sullivan, 2008). For the same reason, younger children's ability to regulate emotion and alter emotional responses may be more vulnerable to environmental stress such as maltreatment.

Our findings illustrate that early caregiving experiences have important implications for the development of self-regulatory processes. Not surprisingly, maltreating families are characterized by affect dysregulation, disorganized roles, chaotic interactions, and rigid relationship skills when compared to nonmaltreating families (Howes et al., 2000). For maltreated children whose environments may be unpredictable and frightening, their caregivers often fail to provide the much-needed structure and regulation. For example, physically abusive parents often lack impulse control, especially when aroused perhaps because they are biologically predisposed to overreact to stressful stimuli (Milner, 2000). Therefore, when living in an environment laden with distress and conflict, many abused children may experience high levels of arousal and vigilance (Rogosch et al., 1995). Such sustained exposure to stress may alter biological stress responses (Gunnar & Quevedo, 2007) and resulting extreme irritability may impede the development of adaptive regulation (Calkins, 1994). One caveat in interpreting the effects of child maltreatment on development of emotional systems is that maltreatment history is one indication of stressors in the family ecology, likely representing possible presence of broader conflict and violence in family relationships. Thus, future research should benefit from considering diverse factors of family emotional climate (e.g., parenting styles, marital relationships, positive and negative emotion displayed among family members).

Some limitations of this study should be noted. First, even though this study involved multiple informants (e.g., DHS records and different counselors across the occasions), the use of additional informants (e.g., self-reports, peer and parent ratings) in future work is recommended to preclude possible biases resulting from shared method variance. In addition, future studies should integrate multiple levels of assessment (e.g., observations in laboratory paradigms and in naturalistic contexts, interviews, and self reports). Second, we focused on the effects of earlier maltreatment experiences and did not consider the quality of caregiving during the longitudinal period between ages 7 and 10. Therefore, we could not examine possible effects of the on-going caregiving environment, as well as cumulative effects of caregiving above and beyond early maltreatment. Third, in our longitudinal analyses there was a relatively large percentage of missing data. We were able to demonstrate that our data did not appear to violate the assumption of missing at random, and thus we used the direct maximum likelihood approach (i.e., FIML) that is predominantly recommended for the latent difference score analyses. Nevertheless, it is not entirely clear how the degree and the patterns of missingness might have affected our ability to accurately depict within-person developmental changes. Finally, an important avenue for future research is to examine how underlying genetic and neurobiological mechanisms interact with experiences to contribute to the links among emotion lability/negativity, emotion regulation, and child psychopathology.

Despite these limitations, this investigation addressed several shortcomings present within the developmental psychopathology literature. In the low-income sample that was assessed for an extended period in middle childhood, we examined whether prospective effects of emotion lability/negativity and emotion regulation on internalizing symptomatology differed between maltreated and nonmaltreated children. Methodologically, the current study is the first study that examines mediation effects of emotion regulation and emotion lability/negativity using longitudinal autoregressive models (i.e., emotion lability/negativity at time 1 → emotion regulation at time 2 → change in internalizing symptomatology between time 2 and time 3) involving optimally reliable latent change scores using more than two measurement occasions. In particular, mediational processes unfold over time, and our latent difference score modeling approach based on multivariate repeated measures data enhances the ability to present evidence toward the plausibility of the causal nature of relations within a mediational chain (D. A. Cole & Maxwell, 2003), avoiding limitations of prior cross-sectional research or longitudinal research using two-wave panel models which allow predictions of only a single time lag.

Findings from this study indicate that emotion regulation and emotion lability/negativity may be important factors in identifying distinct pathways to child psychopathology. In particular, low emotion lability/negativity and adaptive emotion regulation play protective roles in the development of internalizing symptomatology. Furthermore, our results may offer helpful insight toward enhancing prevention and intervention efforts for internalizing symptomatology in childhood. The findings suggest that emotion regulation is an important mediational process between emotion lability/negativity and internalizing symptomatology. Therefore, to alter processes that emotional difficulties are related to subsequent development of internalizing symptomatology in middle childhood, a focus on improving emotion regulation skills in children is likely to be an effective strategy that can impede the progression of internalizing problems. The results also imply that emotion regulation can be targeted in order to reduce the deleterious effects of emotion lability/negativity heightened by early maltreatment experiences, thus preventing internalizing symptomatology.

Acknowledgments

References

Achenbach, T. Manual for the Teacher's Report form and 1991 Profile. University of Vermont, Department of Psychiatry; Burlington, VT: 1991.

Alessandri SM. Play and social behavior in maltreated preschoolers. Development and Psychopathology. 1991; 3:191–205. doi: 10.1017/S0954579400000079.

Arbuckle, JL. Full information estimation in the presence of incomplete data. In: Marcoulides, GA.; Schumacker, RE., editors. Advanced structural equation modeling: Issues and techniques. Erlbaum; Mahwah, NJ: 1996. p. 243-277.

Barnett, D.; Manly, JT.; Cicchetti, D. Defining child maltreatment: The interface between policy and research. In: Cicchetti, D.; Toth, SL., editors. Child abuse, child development, and social policy. Ablex; Norwood, NJ: 1993. p. 7-73.

Bates JE, Pettit GS, Dodge KA, Ridge B. Interaction of temperamental resistance to control and restrictive parenting in the development of externalizing problems. Developmental Psychology. 1998; 34:982–995. doi: 10.1037/0012-1649.34.5.982. [PubMed: 9779744]

Blair C. Stress and the development of self-regulation in context. Child Development Perspectives. 2010; 4:181–188. doi: 10.1111/j.1750-8606.2010.00145.x. [PubMed: 21779305]

Blair C, Diamond A. Biological processes in prevention and intervention: The promotion of self-regulation as a means of preventing school failure. Development and Psychopathology. 2008; 20:899–911. doi: 10.1017/S0954579408000436. [PubMed: 18606037]

Bowlby, J. Attachment and loss: Vol. 1. Attachment. Basic Books; New York: 1969/1982.

Burt KB, Obradovic J, Long JD, Masten AS. The interplay of social competence and psychopathology over 20 years: Testing transactional and cascade models. Child Development. 2008; 79:359–374. doi: 10.1111/j.1467-8624.2007.01130.x. [PubMed: 18366428]

Calkins S. Origins and outcomes of individual differences in emotion regulation. In N. A. Fox (Ed.), The development of emotion regulation: Biological and behavioral considerations. Monographs of the Society for Research in Child Development. 1994; 59(2–3):53–72. Serial No. 240. [PubMed: 7984167]

Calkins SD, Fox NA. Self-regulatory processes in early personality development: A multilevel approach to the study of childhood social withdrawal and aggression. Development and Psychopathology. 2002; 14:477–498. doi: 10.1017/S095457940200305X. [PubMed: 12349870]

Calkins, SD.; Hill, AL. Caregiver influences on emerging emotion regulation: Biological and environmental transactions in early development. In: Gross, J., editor. Handbook of Emotion Regulation. Guildford; New York: 2007. p. 229-248.

Camras, LA.; Sachs-Alter, E.; Ribordy, SC. Emotion understanding in maltreated children: Recognition of facial expressions and integration with other emotion cues. In: Lewis, M.; Sullivan, MW., editors. Emotional development in atypical children. Lawrence Erlbaum Associates, Inc.; Hillsdale, NJ, England: 1996. p. 203-225.

Cicchetti D, Ackerman BP, Izard CE. Emotions and emotion regulation in developmental psychopathology. Development and Psychopathology. 1995; 7:1–10. doi: 10.1017/S0954579400006301.

Cicchetti, D.; Ganiban, J.; Barnett, D. Contributions from the study of high risk populations to understanding the development of emotion regulation. In: Garber, J.; Dodge, K., editors. The development of emotion regulation and dysregulation. Cambridge University Press; New York: 1991. p. 15-49.

Cicchetti D, Toth S. The development of depression in children and adolescents. American Psychologist. 1998; 53:221–241. doi: 10.1037/0003-066X.53.2.221. [PubMed: 9491749]

Cicchetti, D.; Toth, S.; Manly, JT. Maternal Maltreatment Classification Interview. Mt. Hope Family Center; Rochester, NY: 2003. Unpublished manuscript

Cole DA, Maxwell SE. Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. Journal of Abnormal Psychology. 2003; 112:558–577. doi: 10.1037/0021-843X.112.4.558. [PubMed: 14674869]

Cole PM, Luby J, Sullivan MW. Emotions and the development of childhood depression: Bridging the gap. Child Development Perspectives. 2008; 2:141–148. doi: 10.1111/j.1750-8606.2008.00056.x. [PubMed: 19956783]

Cole PM, Zahn-Waxler C, Fox NA, Usher BA, Welsh JD. Individual differences in emotion regulation and behavior problems in preschool children. Journal of Abnormal Psychology. 1996; 105:518–529. doi: 10.1037/0021-843X.105.4.518. [PubMed: 8952185]

Cummings EM, Hennessy K, Rabideau G, Cicchetti D. Responses of physically abused boys to interadult anger involving their mothers. Development and Psychopathology. 1994; 6:31–42. doi: 10.1017/S0954579400005861.

Dunsmore, JC.; Booker, JA.; Ollendick, TH. Parental emotion coaching and child emotion regulation as protective factors for children with oppositional defiant disorder. 2011. Manuscript submitted for publication

Eisenberg N, Cumberland A, Spinrad TL, Fabes RA, Shepard SA, Reiser M, Guthrie IK. The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. Child Development. 2001a; 72:1112–1134. doi: 10.1111/1467-8624.00337. [PubMed: 11480937]

Eisenberg N, Fabes RA, Murphy B, Maszk P, Smith M, Karbon M. The role of emotionality and regulation in children's social functioning: A longitudinal study. Child Development. 1995; 66:1360–1384. doi: 10.2307/1131652. [PubMed: 7555221]

Eisenberg N, Losoya S, Fabes RA, Guthrie IK, Reiser M, Murphy B, Padgett SJ. Parental socialization of children's dysregulated expression of emotion and externalizing problems. Journal of Family Psychology. 2001b; 15:183–205. doi: 10.1037/0893-3200.15.2.183. [PubMed: 11458628]

Eisenberg N, Sadovsky A, Spinrad TL, Fabes RA, Losoya SH, Valiente C, Shepard SA. The relations of problem behavior status to children's negative emotionality, effortful control, and impulsivity: Concurrent relations and prediction of change. Developmental Psychology. 2005; 41:193–211. doi: 10.1037/0012-1649.41.1.193. [PubMed: 15656749]

Eisenberg N, Spinrad TL, Fabes RA, Reiser M, Cumberland A, Shepard SA, Thompson M. The relations of effortful control and impulsivity to children's resiliency and adjustment. Child Development. 2004; 75:25–46. doi: 10.1111/j.1467-8624.2004.00652.x. [PubMed: 15015673]

Erickson, MR.; Egeland, B.; Pianta, R. The effects of maltreatment on the development of young children. In: Cicchetti, D.; Carlson, V., editors. Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect. Cambridge University Press; New York: 1989. p. 647-684.

Gottman JM, Katz LF, Hooven C. Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. Journal of Family Psychology. 1996; 10:243–268. doi: 10.1037/0893-3200.10.3.243.

Greenberg MT, Kusche CA, Cook ET, Quamma JP. Promoting emotional competence in school-aged children: The effects of the PATHS curriculum. Development and Psychopathology. 1995; 7:117–136. doi: 10.1017/S0954579400006374.

Gunnar MR, Donzella B. Social regulation of the cortisol levels in early human development. Psychoneuroendocrinology. 2002; 27:199–220. doi: 10.1016/S0306-4530(01)00045-2. [PubMed: 11750779]

Gunnar M, Quevedo K. The neurobiology of stress and development. Annual Review of Psychology. 2007; 58:145–173. doi:10.1146/annurev.psych.58.110405.085605.

Hollingshead, AF. Four Factor Index of Social Status. Yale University; 1975. Unpublished manuscript

Howes PW, Cicchetti D, Toth SL, Rogosch F. Affective, organizational, and relational characteristics of maltreating families: A systems perspective. Journal of Family Psychology. 2000; 14:95–110. doi: 10.1037/0893-3200.14.1.95. [PubMed: 10740685]

Kim J, Cicchetti D. Longitudinal pathways linking child abuse and neglect, emotion regulation, peer rejection, and psychopathology. Journal of Child Psychology and Psychiatry. 2010; 51:706–716. doi: 10.1111/j.1469-7610.2009.02202.x. [PubMed: 20050965]

Kim J, Deater-Deckard K. Dynamic changes in anger linking to developmental trajectories of internalizing and externalizing problems: The moderating role of attention. Journal of Child Psychology and Psychiatry. 2011; 52:156–166. doi: 10.1111/j.1469-7610.2010.02301.x. [PubMed: 20825522]

Kim-Spoon J, Haskett ME, Longo GS, Nice R. Longitudinal study of self-regulation, positive parenting, and adjustment problems among physically abused children. Child Abuse and Neglect. in press.

Kovacs M, Joormann J, Gotlib IH. Emotion (dys)regulation and links to depressive disorders. Child Development Perspectives. 2008; 2:149–155. doi: 10.1111/j.1750-8606.2008.00057.x. [PubMed: 20721304]

Lahey BB, Loeber R, Hart EL, Frick PJ, Applegate B, Zhaing Q, Russo MF. Four-year longitudinal study of conduct disorder in boys: Patterns and predictors of persistence. Journal of Abnormal Psychology. 1995; 104:83–93. doi: 10.1037/0021-843X.104.1.83. [PubMed: 7897057]

Larson R, Raffaelli M, Richards MH, Ham M, Jewell L. Ecology of depression in late childhood and early adolescence: A profile of daily states and activities. Journal of Abnormal Psychology. 1990; 99:92–102. doi: 10.1037/0021-843X.99.1.92. [PubMed: 2307772]

Luthar SS, Cicchetti D, Becker B. The construct of resilience: A critical evaluation and guidelines for future work. Child Development. 2000; 71:543–562. doi: 10.1111/1467-8624.00164. [PubMed: 10953923]

MacKinnon DP, Lockwood CM, Hoffman JM, West SG, Sheets V. A comparison of methods to test mediation and other intervening variable effects. Psychological Methods. 2002; 7:83–104. doi: 10.1037/1082-989X.7.1.83. [PubMed: 11928892]

McArdle JJ. Latent variable modeling of differences and changes with longitudinal data. Annual Review of Psychology. 2009; 60:577–605. doi: 10.1146/annurev.psych.60.110707.163612.

Milner, JS. Social information processing and child physical abuse: Theory and research. In: Hansen, DJ., editor. Nebraska Symposium on Motivation. Vol. Vol. 45. University of Nebraska Press; Lincoln: 2000. p. 39-84.

Morris AS, Silk JS, Morris MDS, Steinberg L, Aucoin KJ, Keyes AW. The influence of mother-child emotion regulation strategies on children's expression of anger and sadness. Developmental Psychology. 2010; 47:213–225. doi: 10.1037/a0021021. [PubMed: 21244160]

Muthén, LK.; Muthén, B. Mplus user's guide [Computer software and manual]. 6th Ed.. Muthén & Muthén; Los Angeles, CA: 2010.

Oldehinkel AJ, Hartman CA, Ferdinand RF, Verhulst FC, Ormel J. Effortful control as modifier of the association between negative emotionality and adolescents' mental health problems. Development and Psychopathology. 2007; 19:523–539. doi: 10.1017/S0954579407070253. [PubMed: 17459182]

Parke, R.; O'Neill, R. Social relationships across contexts: Family-peer linkages. In: Collins, A.; Larson, B., editors. The Minnesota symposia on child psychology: Vol. 30. Relationships as developmental contexts. Erlbaum; Mahwah, NJ: 1999. p. 211-239.

Pietromonaco PR, Barrett LF. Valence focus and self-esteem lability: Reacting to hedonic cues in the social environment. Emotion. 2009; 9:406–418. doi: 10.1037/a0015691. [PubMed: 19485618]

Rogosch FA, Cicchetti D, Aber JL. The role of child maltreatment in early deviations in cognitive and affective processing abilities and later peer relationship problems. Development and Psychopathology. 1995; 7:591–609. doi: 10.1017/S0954579400006738.

Rothbart, MK.; Bates, JE. Temperament. In: Damon, W.; Eisenberg, N., editors. Handbook of Child Psychology: Vol. 3. Social, emotional, and personality development. 6th ed.. Wiley; New York: 2006. p. 99-166.

Rydell A, Berlin L, Bohlin G. Emotionality, emotion regulation, and adaptation among 5- to 8-year-old children. Emotion. 2003; 3:30–47. doi: 10.1037/1528-3542.3.1.30. [PubMed: 12899315]

Shields A, Cicchetti D. Reactive aggression among maltreated children: The contribution of attention and emotion regulation. Journal of Clinical Child Psychology. 1998; 27:381–395. doi: 10.1207/s15374424jccp2704_2. [PubMed: 9866075]

Shipman KL, Schneider R, Fitzgerald MM, Sims C, Swisher L, Edwards A. Maternal emotion socialization in maltreating and non-maltreating families: Implications for children's emotion regulation. Social Development. 2007; 16:268–285. doi: 10.1111/j.1467-9507.2007.00384.x.

Silk JS, Steinberg L, Morris AS. Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. Child Development. 2003; 74:1869–1880. doi: 10.1046/j.1467-8624.2003.00643.x. [PubMed: 14669901]

Taylor AB, MacKinnon DP, Tein J. Tests of the three-path mediated effect. Organizational Research Methods. 2008; 11:241–269. doi: 10.1177/1094428107300344.

Thompson RA, Calkins SD. The double-edged sword: Emotional regulation for children at risk. Development and Psychopathology. 1996; 8:163–182. doi: 10.1017/S0954579400007021.

Zeman J, Shipman K, Suveg C. Anger and sadness regulation: Predictions to internalizing and externalizing symptoms in children. Journal of Clinical Child and Adolescent Psychology. 2002; 31:393–398. doi: 10.1207/153744202760082658. [PubMed: 12149977]
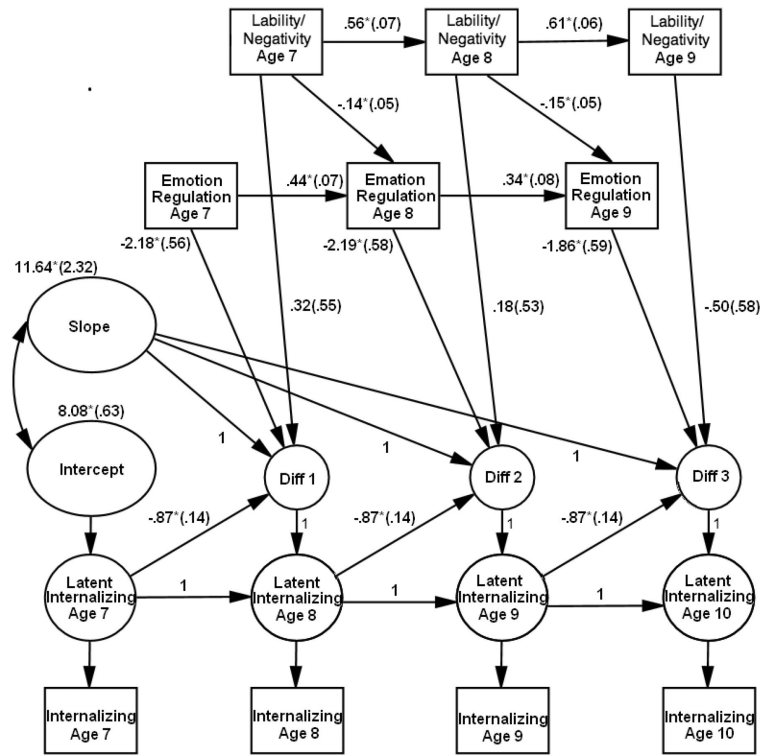
Figure 1.
Latent Difference Score Model of Emotion Lability/Negativity Predicting Internalizing Symptomatology, Mediated by Emotion Regulation.

Note. Unstandardized parameter estimates (SE) are presented. For clarify of presentation, residual variances and concurrent correlations among emotion lability/negativity, emotion regulation, and internalizing symptomatology are not shown. Diff = latent difference score factor; e = measurement error. Model fit: χ^2 = 92.10, *df* = 58, *p* = .004, RMSEA = .04, CFI = .96. * *p* < .05.
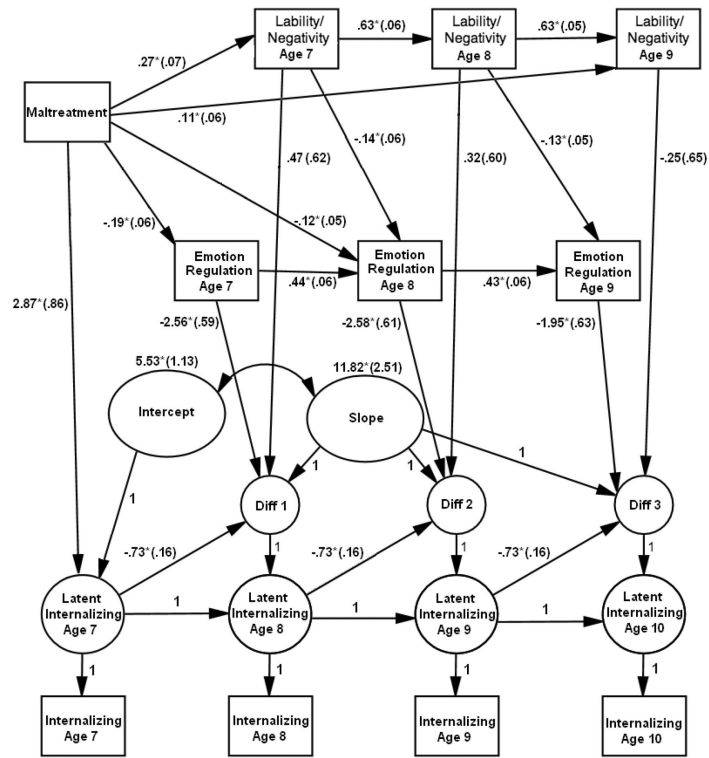
Figure 2.
Latent Difference Score Model of Maltreatment Effects on Emotion Lability/Negativity, Emotion Regulation, and Internalizing Symptomatology.

Note. Unstandardized parameter estimates (SE) are presented for high emotion lability/ negativity/low emotion lability/negativity groups. For clarity of presentation, residual variances, the effects of gender and ethnicity covariates, and correlations among emotion lability/negativity, emotion regulation, and internalizing symptomatology are not shown. For the effects of maltreatment subtypes on age 8 and age 9 emotion lability/negativity and emotion regulation and on diff1/2/3 were not significant, only significant paths are presented. Diff = latent difference score factor; e = measurement error. Model fit: χ^2 = 87.20, df = 27, p = .000, RMSEA = .08, CFI = .94.
† p = .05, * p < .05.

Table 1

Descriptive Statistics and Bivariate Correlations of Emotion Regulation, Emotion Lability/Negativity, and Internalizing Symptomatology

	1	2	3	4	5	6	7	8	9	Maltreated M (SD)	Nonmaltreated M (SD)	Univariate F
1. ER (age 7)	—									2.86 (.50)	3.04 (.43)	3.75†
2. ER (age 8)	.54*	—								2.80 (.44)	3.03 (.42)	16.31*
3. ER (age 9)	.49*	.54*	—							2.86 (.44)	3.04 (.44)	12.17*
4. EL (age 7)	−.58*	−.48*	−.43*	—						1.97 (.61)	1.67 (.52)	8.73*
5. EL (age 8)	−.36*	−.57*	−.57*	.67*	—					1.98 (.56)	1.69 (.56)	13.46*
6. EL (age 9)	−.34*	−.45*	−.45*	.67*	.69*	—				1.96 (.54)	1.68 (.51)	16.18*
7. INT (age 7)	−.60*	−.32*	−.32*	.45*	.13	.26*	—			7.98 (6.98)	5.20 (5.11)	6.09*
8. INT (age 8)	−.17*	−.48*	−.48*	.23*	.37*	.31*	.25*	—		6.97 (5.10)	5.24 (4.35)	8.80*
9. INT (age 9)	−.22*	−.27*	−.27*	.18*	.18*	.30*	.25*	.27*	—	6.76 (5.97)	5.12 (4.57)	8.10*
10. INT (age 10)	−.28*	−.20*	−.20*	.28*	.14	.04	.32*	.24*	.44*	6.31 (5.45)	5.37 (4.98)	1.92

Note. ER = emotion regulation; EL = emotion lability/negativity; INT = internalizing symptomatology. N = 171 for Maltreated group and N = 151 for nonmaltreated group. Bivariate N = 107 to 266.

† $p = .05$,

* $p < .05$

Table 2

Comparisons of Latent Difference Score Models for Differences in Direct and Mediated Effects of Emotion Regulation and Emotion Lability/Negativity on Internalizing Symptomatology between Maltreated and Nonmaltreated Children

Model Label		x^2	df	p	CFI	RMSEA	Comparison	x^2	df	p(d)
Direct effects of emotion regulation										
a. Configural Invariance		39.78	28	.07	.97	.04				
b. Equal Direct Effects		**40.85**	**31**	**.11**	**.97**	**.03**	**a vs. b**	**1.07**	**3**	**.79**
Direct effects of emotion lability/negativity										
a. Configural Invariance		50.14	28	.01	.94	.05				
b. Equal Direct Effects		**51.13**	**31**	**.01**	**.95**	**.05**	**a vs. b**	**.99**	**3**	**.80**
Emotion regulation as a mediator for emotion lability/negativity	*Internalizing Symptomatology*									
a. Configural Invariance		89.22	50	.00	.95	.05				
b. Equal Direct and Mediated Effects		**92.10**	**58**	**.00**	**.96**	**.04**	**a vs. b**	**2.88**	**8**	**.94**
Emotion lability/negativity as a mediator for emotion regulation	*Internalizing Symptomatology*									
a. Configural Invariance		101.54	50	.00	.94	.06				
b. Equal Direct and Mediated Effects		**105.88**	**58**	**.00**	**.94**	**.05**	**a vs. b**	**4.33**	**8**	**.83**

Note. CFI = comparative-fit index; RMSEA = root mean square error of approximation; x^2 = difference in likelihood ratio tests; *df* = difference in *df*; *p(d)* = probability of the difference tests. Best-fitting models are in bold face.