Contents

Invited Essay

Emotion dysregulation and adolescent psychopathology: A prospective study

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ABSTRACT

Background: Emotion regulation deficits have been consistently linked to psychopathology in cross-sectional studies. However, the direction of the relationship between emotion regulation and psychopathology is unclear. This study examined the longitudinal and reciprocal relationships between emotion regulation deficits and psychopathology in adolescents.
Methods: Emotion dysregulation and symptomatology (depression, anxiety, aggressive behavior, and eating pathology) were assessed in a large, diverse sample of adolescents ($N = 1065$) at two time points separated by seven months. Structural equation modeling was used to examine the longitudinal and reciprocal relationships between emotion dysregulation and symptoms of psychopathology.
Results: The three distinct emotion processes examined here (emotional understanding, dysregulated expression of sadness and anger, and ruminative responses to distress) formed a unitary latent emotion dysregulation factor. Emotion dysregulation predicted increases in anxiety symptoms, aggressive behavior, and eating pathology after controlling for baseline symptoms but did not predict depressive symptoms. In contrast, none of the four types of psychopathology predicted increases in emotion dysregulation after controlling for baseline emotion dysregulation.
Conclusions: Emotion dysregulation appears to be an important transdiagnostic factor that increases risk for a wide range of psychopathology outcomes in adolescence. These results suggest targets for preventive interventions during this developmental period of risk.

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The adaptive management of emotions is critical for social functioning and psychological well-being (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Denham, 1998; Eisenberg, Fabes, Guthrie, & Reiser, 2000; Gross, 1998b). Thompson (1994) defined emotion regulation as: "the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals" (p. 28). Individual differences in the ability to identify and understand one's own emotions, the emotions of others, and the causes and consequences of different emotions, as well as in the types of strategies used to manage and regulate emotions, are evident by early childhood (Cummings, 1987; Denham, 1998; Eisenberg et al., 1993). These differences in emotional understanding and in the acquisition of

adaptive strategies for managing emotions have far-reaching implications for social functioning (Denham, 1986; Eisenberg et al., 1993; Eisenberg, Fabes, et al., 2000; Saarni, 1999). Children and adolescents with better emotion regulation skills are more socially competent, have higher peer status and better quality relationships, and engage in higher levels of prosocial behavior than youth with poor emotion regulation skills (Denham, 1986; Denham et al., 2003; Denham, McKinley, Couchoud, & Holt, 1990; Eisenberg et al., 1993; Eisenberg et al., 1995; Hubbard & Coie, 1994; Rydell, Berlin, & Bohlin, 2003; Spinard et al., 2006). Children's emotion regulation skills also have been found to predict their social competence at later points in development (Denham et al., 2003; Rydell et al., 2003; Spinard et al., 2006).

Emotion regulation skills also have important implications for mental health (Gross & Muñoz, 1995). Accumulating evidence suggests that emotion regulation is related to the development and maintenance of youth psychopathology. Children with anxiety disorders have been found to have poorer emotional understanding and more difficulty managing negative emotions than non-anxious youth (Southam-Gerow & Kendall, 2000; Suveg & Zeman, 2004).

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Adolescent depression also has been linked to reductions in the use and quality of strategies to manage negative emotions (Garber, Braafladt, & Weiss, 1995; Silk, Steinberg, & Morris, 2003). Engagement in rumination, an emotion regulation strategy involving passive self-focus on feelings of distress and on their causes and consequences (Nolen-Hoeksema & Morrow, 1991), has been associated consistently with adolescent depression both concurrently (Kuyken, Watkins, Holden, & Cook, 2006; Ziegert & Kistner, 2002) and prospectively (Abela, Brozina, & Haigh, 2002; Broderick & Korteland, 2004; Burwell & Shirk, 2007). Poor emotional understanding and difficulty regulating negative emotions also have been documented among children who engage in high levels of aggressive behavior (Bohnert, Crnic, & Lim, 2003; Cole, Zahn-Waxler, & Smith, 1994; Dearing et al., 2002; Eisenberg et al., 2000; Shields & Cicchetti, 1998) and among adolescent females with eating pathology (Sim & Zeman, 2005, 2006).

Adolescence represents a crucial developmental period in which to study the relationship between emotion regulation skills and psychopathology. A central task of adolescence is to learn to regulate affect in adaptive ways, increasingly without the aid of the adults who provide guidance in childhood (Steinberg & Avenevoli, 2000; Steinberg et al., 2006). Changes in biological, cognitive, and social systems during adolescence present innumerable affectively-laden situations in which emotions must be successfully managed to ensure adaptive functioning (Larson & Richards, 1991; Steinberg, 1987). For instance, increasing independent contact with peers introduces a myriad of challenges that require effective emotional response management (e.g., engagement in romantic relationships and sexual behavior, exposure to substance use). These numerous changes lead to increased perceptions of stress and daily hassles (Larson & Ham, 1993; Seidman, Allen, Aber, Mitchell, & Feiman, 1994; Simmons & Blythe, 1987) as well as increases in the experience of negative affect and emotional instability, and a closer linking of stressful events to the emergence of negative affect (Larson & Ham, 1993; Larson & Lampman-Petraitis, 1989; Larson, Moneta, Richards, & Wilson, 2002). Adolescence also is characterized by high risk for the development of psychopathology (Andersen & Teicher, 2008; Hankin et al., 1998; Lewinsohn, Striegel-Moore, & Seeley, 2000; Twenge & Nolen-Hoeksema, 2002). Individuals who have not developed strategies to adaptively manage negative emotions arising from the many challenges of adolescence may be particularly at risk for adverse mental health outcomes (Silk et al., 2007; Steinberg et al., 2006).

Despite accumulating evidence suggesting that emotion dysregulation is associated with youth psychopathology, a number of important questions remain unanswered regarding the specific nature of this relationship. First, although poor emotion regulation has been documented among children and adolescents with a variety of diagnoses and associations between emotion regulation and symptoms of psychopathology have been observed in a number of studies, the vast majority of this research has been cross-sectional in nature, with some notable exceptions examining rumination specifically (Abela et al., 2002; Broderick & Korteland, 2004; Burwell & Shirk, 2007) and problem behaviors in young children (Eisenberg, Guthrie et al., 2000). Thus, it remains unclear whether emotion dysregulation primarily represents a risk factor for the development of adolescent psychopathology or whether the onset of mental health problems renders adolescents less able to effectively manage their emotions. This distinction has important implications for targeting emotion regulation in treatment versus preventive interventions. If emotion regulation is a risk factor for future psychopathology, techniques targeting emotion regulation skills should be incorporated into both treatment and preventive interventions. Alternatively, if emotion regulation is primarily a consequence of psychopathology or a factor underlying disorder

persistence, such techniques would most usefully be included into treatment rather than prevention.

Second, there is a paucity of research on the extent to which emotion dysregulation represents a global risk factor for poor mental health outcomes versus a vulnerability to specific types of psychopathology. Poor emotion regulation has recently been posited to represent a transdiagnostic factor related to multiple types of psychopathology (Ehring & Watkins, 2008; Gross & John, 2003; Harvey, Watkins, Mansell, & Shafran, 2004; Kring & Sloan, 2010; Moses & Barlow, 2006; Watkins, 2008). Significant cross-sectional associations have been documented between emotion dysregulation and symptoms of depression, anxiety, eating pathology, and substance abuse in transdiagnostic studies of adults and in a recent meta-analysis (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010). Prospective data are needed, however, to determine both the direction of effect in the relationship between emotion regulation deficits and psychopathology, and whether emotion dysregulation represent a vulnerability factor for the development of certain types of psychopathology, but not others. Finally, it is unknown whether different aspects of emotion functioning are best represented by a latent factor of emotion regulation or are better characterized as distinct processes in adolescence. Emotion regulation involves a number of component processes, each of which emerge at different points in development (Denham, 1998). Recent evidence from an adult sample indicated that poor understanding of emotions, negative cognitive reactivity to emotional experiences, and maladaptive emotion management (but not emotional intensity) loaded onto a higher-order factor of emotion dysregulation (Mennin, Holaway, Fresco, Moore, & Heimberg, 2007), but it is unknown whether these processes comprise a broad latent factor in adolescents.

The current investigation aimed to address these gaps in the literature by examining the reciprocal relationships among poor emotion regulation skills and various forms of psychopathology in a sample of adolescents followed prospectively. We expected that adolescents who were better able to identify and understand their emotional experiences would also employ more effective strategies for managing their emotions. Specifically, we predicted that emotional understanding, adaptive expression of negative emotions, and cognitive emotion management strategies would best be represented by a higher-order construct of emotion regulation. We selected these specific aspects of emotion regulation in order to capture different stages in the emotion regulation process, each of which has relevance for psychopathology. Awareness and understanding of emotional experience represents an early stage of the emotion regulation process that requires attending to and differentiating interoceptive emotional cues. Emotional awareness has neural correlates that are distinct from emotion expression and regulation (Lane et al., 1998). It has been argued that emotional awareness is necessary to facilitate adaptive emotion regulation (Saarni, 1999), and poor awareness has been linked to a variety of poor mental health outcomes (Mennin, McLaughlin, & Flanagan, 2009; Novick-Kline, Turk, Mennin, Hoyt, & Gallagher, 2005; Southam-Gerow & Kendall, 2000; Suveg & Zeman, 2004). Emotion expression conveys important information about one's emotional experiences to others, and regulation of expression can directly influence the intensity of emotional experience (Gross, 1998a; Gross & John, 2003). Maladaptive or heightened expression of specific emotions—including anger, fear, and sadness—have been linked to internalizing and externalizing problems in adolescents (Keltner, Moffit, & Stouthamer-Loeber, 1995; Zeman, Shipman, & Suveg, 2002). Cognitive emotion regulation strategies—such as rumination—represent a relatively late stage in the emotion regulation process in which an individual engages in specific thoughts or behaviors in response to emotional experiences, often

as an attempt to maintain or diminish those experiences. Rumination has been linked to adolescent psychopathology both concurrently and prospectively (Abela et al., 2002; Burwell & Shirk, 2007). We anticipated that poor emotion regulation skills in each of these areas would predict subsequent increases in adolescent symptoms of depression, anxiety, aggressive behavior, and eating pathology but that baseline symptomatology would not predict changes in emotion regulation.

Methods

Participants

The sample for this study was recruited from the total enrollment of two middle schools (Grades 6–8) from a small urban community (population 71,538) in Connecticut. The parents of all eligible children ($N = 1567$) in the middle schools were asked to provide active consent for their children to participate. 22% of parents did not return consent forms and could not be reached to obtain consent, and 6% of parents declined to provide consent for their child to participate. The overall cooperation rate in the study at baseline was 72% (see Fig. 1). Of the participants who were present at the baseline assessment, 217 (20.4%) did not participate at the Time 2 assessment. This school district population was highly transient; over the four-year period from 2000 to 2004, 22.7% of students had left the district (Connecticut Department of Education, 2006). We conducted analyses using the sample of 1065 participants who were present at the baseline assessment, excluding participants who were present at Time 2 but not at Time 1.

The baseline sample included 51.2% ($N = 545$) boys and 48.8% ($N = 520$) girls, resulting in 1065 participants, all of whom provided consent before participating in the study (68% of all eligible students in the schools). Participants (ages 11–14) were evenly distributed across grade level with 31.8% ($N = 337$) of participants in the sixth, 33.9% ($N = 360$) in the seventh, and 34.3% ($N = 364$) in the eighth grade. The race/ethnicity composition of the sample was as follows: 13.2% ($N = 141$) White, 11.8% ($N = 126$) Black, 56.9% ($N = 610$) Hispanic/Latino, 2.2% ($N = 24$) Asian/Pacific Islander, 0.2% ($N = 2$) Native American, 0.8% ($N = 9$) Middle Eastern, 9.3% ($N = 100$) Biracial/Multiracial, and 4.2% ($N = 45$) reported being members of other racial/ethnic groups. 1.3% ($N = 14$) declined to provide this information. 27% ($N = 293$) of participants reported living in single-parent households. The community in which the participating schools reside is a uniformly lower-SES community, with a per capita income of $18,404 (Connecticut Department of Education, 2006). School records indicated that 62.3% of students qualified for free or reduced lunch in the 2004–2005 school year.
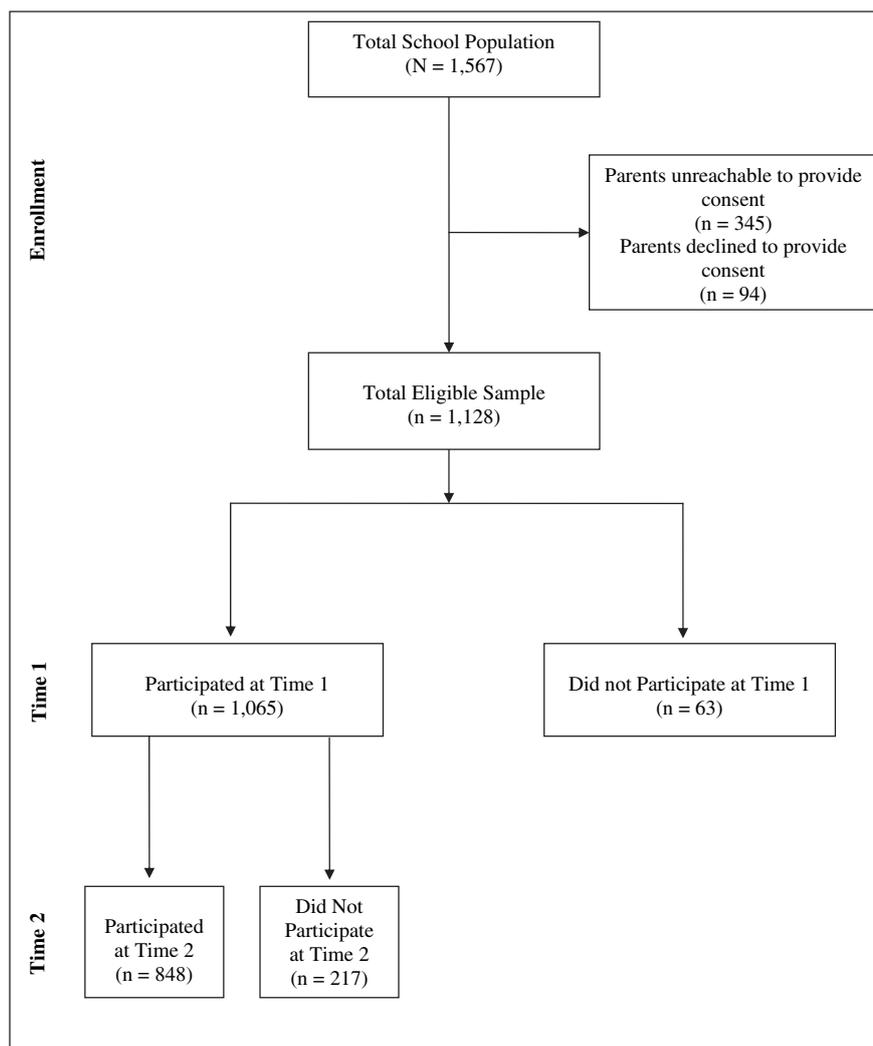

Fig. 1. Study flow of participants in the study.

Psychopathology measures

The Children's Depression Inventory (CDI; Kovacs, 1992) is a widely used self-report measure of depressive symptoms in children and adolescents. The CDI includes 27 items consisting of three statements (e.g., *I am sad once in a while, I am sad many times, I am sad all the time*) representing different levels of severity of a specific symptom of depression. The CDI has sound psychometric properties, including internal consistency, test–retest reliability, and discriminant validity (Kovacs, 1992; Reynolds, 1994). The item pertaining to suicidal ideation was removed from the measure at the request of school officials and the human subjects committee. The 26 remaining items were summed to create a total score ranging from 0 to 52. The CDI demonstrated good reliability in this sample (Time 1 $\alpha = .82$, Time 2 $\alpha = .89$).

Anxiety symptoms

The Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997) is a 39-item widely used measure of anxiety in children. The MASC assesses physical symptoms of anxiety, harm avoidance, social anxiety, and separation anxiety and is appropriate for children ages 8–19. Each item presents a symptom of anxiety, and participants indicate how true each item is for them on a four-point Likert scale ranging from never true (0) to very true (3). A total score, ranging from 0 to 117, is generated by summing all items. The MASC has high internal consistency and test–retest reliability across 3-month intervals, and established convergent and divergent validity (Muris, Merckelbach, Ollendick, King, & Bogie, 2002). The MASC demonstrated good reliability in this sample (Time 1 $\alpha = 0.88$, Time 2 $\alpha = .93$).

Aggressive behavior

A revised version of The Revised Peer Experiences Questionnaire (RPEQ; Prinstein, Boergers, & Vernberg, 2001) was used to assess participants' engagement in aggression toward peers. The RPEQ includes 18 items that assess overt, relational, and reputational aggression toward peers. Participants rate how often they engaged in a specific behavior toward others in the past year (e.g., "I threatened to hurt or beat up another kid") on a 5-point Likert scale ranging from never (1) to a few times a week (5). The RPEQ has also demonstrated good internal consistency for both versions of the measure (aggressor and victim) as well as for the four subscales, and convergent validity (Prinstein et al., 2001). The RPEQ demonstrated good internal consistency in this sample (Time 1 $\alpha = 0.90$, Time 2 $\alpha = .91$).

Eating pathology

Eating attitudes and behavior were assessed using the Children's Eating Attitudes Test (ChEAT; Maloney, McGuire, & Daniels, 1988). The ChEAT is a 26-item measure that assesses attitudes and behaviors associated with anorexia nervosa and bulimia nervosa. Participants rate how often they engage in specific behaviors or have specific beliefs about food on a 6-point Likert scale ranging from never (1) to always (6). The ChEAT was scored continuously for the current study, yielding a potential range of scores of 0–156. This method has been used in other studies (Barr, Petit, Vigna, & Prior, 2001) yielding sound psychometric properties (McVey, Davis, Tweed, & Shaw, 2004). The ChEAT has demonstrated good test–retest reliability and convergent validity (Maloney et al., 1988; Smolak & Levine, 1994). The continuously scored ChEAT was internally consistent in this sample (Time 1 $\alpha = 0.94$, Time 2 $\alpha = .96$).

Emotion dysregulation measures

Poor emotional understanding

Emotional understanding was assessed using an 8-item subscale from the Emotion Expression Scale for Children (EESC; Penza-Clyve & Zeman, 2002) that provides statements involving lack of emotional awareness and understanding. Children respond to items on a 5-point Likert scale ranging from not at all true (1) to extremely true (5). The 8 items are summed to generate a total score ranging from 8 to 40. Higher scores on this subscale reflect *lack* of emotional understanding. Representative items from this scale are, "*I have feelings that I can't figure out*" and "*I often do not know how I am feeling*". The EESC has high internal consistency and moderate test–retest reliability, and the construct validity of the measure has been established (Penza-Clyve & Zeman, 2002). This scale has been used with early adolescents (Sim & Zeman, 2005, 2006). This subscale demonstrated good reliability in this sample (Time 1 $\alpha = .82$, Time 2 $\alpha = .92$).

Dysregulated emotion expression

The Children's Sadness Management Scale (CSMS) and Anger Management Scale (CAMS) assess both adaptive and maladaptive aspects of emotion expression and regulation for the specific emotions of sadness and anger (Zeman, Shipman, & Penza-Clyve, 2001). We used the Dysregulation subscale of each of these measures, which assesses the extent to which children engage in maladaptive or inappropriate expressions of emotion, such as excessive crying. Higher scores on this scale reflect higher levels of emotion dysregulation. The CSMS contains 12 items, and the CAMS contains 11 items. Children respond on a 3-point Likert scale ranging from hardly ever (1) to often (3). The Dysregulation scale for each measure contains 3 items that are summed to create scores ranging from 3 to 9. The scales have demonstrated adequate reliability, and their construct validity has been established (Zeman et al., 2001). These scales have been used in prior research with early adolescents (Sim & Zeman, 2005, 2006). Representative items from the dysregulation scale are, "*I attack whatever it is that is making me angry*", (CAMS) and "*I cry and carry on when I'm sad*" (CSMS). The Dysregulation subscale of the CSMS (Time 1 $\alpha = 0.61$, Time 2 $\alpha = .60$) and CAMS (Time 1 $\alpha = 0.56$, Time 2 $\alpha = .66$) each demonstrated adequate reliability.

Rumination

The Children's Response Styles Questionnaire (CRSQ; Abela et al., 2002) is a 25-item scale that assesses the extent to which children respond to sad feelings with rumination, defined as self-focused thought concerning the causes and consequences of depressed mood, distraction, or problem-solving. The measure is modeled after the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991) that was developed for adults. For each item, youth are asked to rate how often they respond in that way when they feel sad on a 4-point Likert scale ranging from almost never (1) to almost always (4). The rumination subscale includes 13 items that are summed to generate a score ranging from 13 to 42. Sample items include: "*Think about a recent situation wishing it had gone better*" and "*Think why can't I handle things better?*" The reliability and validity of the CRSQ have been demonstrated in samples of early adolescents (Abela et al., 2002). The CRSQ rumination scale demonstrated good reliability in this study (Time 1 $\alpha = 0.86$, Time 2 $\alpha = .90$).

Procedure

Participants completed study questionnaires during their homeroom period. Symptom and emotion regulation measures were each assessed initially at Time 1 and again seven months later at Time 2. This time frame was chosen to allow the maximum time

between assessments while ensuring that all assessments occurred within the same academic year to avoid high attrition. Participants were assured of the confidentiality of their responses and the voluntary nature of their participation. The study was conducted in compliance with the Yale Institutional Review Board.

Data analysis

Structural equation modeling was used to examine study hypotheses using AMOS 6.0 software (Arbuckle, 2005). Analyses were conducted using the full information maximum likelihood estimation method, which estimates means and intercepts to handle missing data. Both emotion dysregulation and symptoms were modeled as latent variables. We tested two measurement models for emotion dysregulation. The first included four observed variables representing distinct emotion regulation processes: emotional understanding, dysregulated expression of sadness and of anger, and rumination. The second measurement model included three inter-related latent factors representing each of these emotion regulation processes separately (i.e., emotional understanding, dysregulated expression, and rumination). Each of the four psychopathology outcomes was measured with one scale; as such, latent factors were created using parcels of items from each scale. Parcels were created using the domain representative approach, which accounts for the multidimensionality of each of the psychopathology outcomes (Little, Cunningham, Shahar, & Widaman, 2002). Four parcels were created for each symptom outcome, and each parcel included items from the various subscales of that measure. In structural equation modeling, the use of parcels to model constructs as latent factors, as opposed to using an observed variable representing a total scale score, confers a number of psychometric advantages including greater reliability, reduction of error variance, and increased efficiency (Little et al., 2002).

We first tested each of the measurement models for emotion dysregulation to determine which model provided the best fit to the data. To test the longitudinal hypotheses, emotion dysregulation at Time 1 was examined as a predictor of Time 2 symptoms, controlling for symptoms at Time 1. This hypothesis was tested by estimating a single model that included all four outcomes of interest (depression, anxiety, aggression, and eating pathology) and allowed us to model the inter-correlations among the four outcomes. Next, we estimated a second model examining each of the four types of symptoms at Time 1 as predictors of emotion dysregulation at Time 2, controlling for emotion dysregulation at Time 1. All analyses controlled for gender and race/ethnicity.

Results

Attrition

Analyses comparing participants who completed both assessments to those who did not revealed that participants who completed the baseline but not the follow-up assessment were more likely to be female, $\chi^2 = 6.85$, $p < .01$, from a single-parent household, $\chi^2 = 8.93$, $p < .01$, and to have lower levels of eating pathology, $F(1, 761) = 6.43$, $p < .05$, $\eta^2 = 0.01$, but did not differ in grade level, race/ethnicity, baseline anxiety, depression, or aggressive behavior, or emotion regulation characteristics (p-values > 0.10). Participants who were present at Time 2 but not Time 1 were not included in analyses.

Descriptive statistics

Table 1 displays the mean and standard deviation of all measures at each time point and the zero-order correlations among

all study measures. The means in this sample are similar to values obtained in other samples of early adolescents (i.e., within 1 standard deviation) (Muris et al., 2002; Penza-Clyve & Zeman, 2002; Twenge & Nolen-Hoeksema, 2002; Zeman et al., 2001). Each of the emotion processes were positively inter-correlated, indicating that dysregulation in one of these processes was associated with dysregulation in the others. As expected, emotion dysregulation was positively associated with all four types of symptomatology, which were themselves positively inter-related.

Measurement model of emotion dysregulation

The first measurement model of emotion dysregulation was constructed using four indicator variables reflecting distinct types of emotion dysregulation: poor emotional understanding, dysregulated expression of anger and of sadness, and ruminative responses to distress. Hu and Bentler (1999) recommend that values of the comparative fit index (CFI) exceeding .95 indicate adequate fit and that the root-mean-square error of approximation (RMSEA) should be less than 0.06. For the hypothesized model, $\chi^2(2) = 1.21$, $p = .299$, CFI = .99, and RMSEA = .01 (90% CI: .00−.06). Thus, all fit indices indicated that the single-factor measurement model of emotion dysregulation fit the data very well. Because adding more parameters to a model typically improve the fit, the Akaike Information Criteria (AIC) was also used to compare the two measurement models; AIC takes into account both model fit and the number of parameters included in the model (Burnham & Anderson, 2002). Smaller values indicate better fit. For the single-factor model, AIC = 26.4.

The second measurement model of emotion dysregulation was constructed using three inter-correlated latent factors representing poor emotional understanding, dysregulated expression (including anger and sadness), and rumination. Latent variables were constructed from parcels of items from each of these scales using the domain representative approach. Although this model also demonstrated adequate fit, it did not fit the data as well as the single-factor model: $\chi^2(32) = 83.0$, $p < .001$, CFI = .98, and RMSEA = .04 (90% CI: .03−.04). The AIC for the three-factor model was considerably higher than for the single-factor model (AIC = 149.0). Therefore, we used the single-factor model of emotion dysregulation in all subsequent analyses.

Measurement models of symptomatology

We next examined the fit of our measurement models for each of the four types of psychopathology. Fit indices indicated that each of these models fit the data well: depression, $\chi^2(2) = 1.83$, $p = .400$, CFI = .99, and RMSEA = .01 (90% CI: .00−.05); anxiety, $\chi^2(2) = 9.95$, $p = .007$, CFI = .99, and RMSEA = .06 (90% CI: .02−.09); aggressive behavior, $\chi^2(2) = 2.25$, $p = .324$, CFI = .99, and RMSEA = .01 (90% CI: .00−.06); and eating pathology, $\chi^2(2) = 9.86$, $p = .007$, CFI = .99, and RMSEA = .06 (90% CI: .02−.09).

Emotion dysregulation predicting symptoms

We next estimated a model examining the longitudinal association between emotion dysregulation at Time 1 and each of the four types of psychopathology at Time 2, controlling for Time 1 psychopathology. This model was estimated including all four types of psychopathology simultaneously to adjust for the inter-relations between each of the outcomes. The covariance between each type of psychopathology at Time 1 and between emotion dysregulation and each type of psychopathology at Time 1 were also estimated in the model (see Fig. 2). In this model, Time 1 emotion dysregulation was significantly associated with Time 2

Table 1
Correlations, means, and standard deviations of Time 1 symptoms and emotion dysregulation.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. CDI T1	—															
2. MASC T1	.28**	—														
3. RPEQ T1	.31**	.11**	—													
4. ChEAT T1	.32**	.17**	.35**	—												
5. EESC T1	.40**	.42**	.23**	.19**	—											
6. CSMS T1	.21**	.31**	.20**	.22**	.30**	—										
7. CAMS T1	.25**	.01	.26**	.15**	.19**	.20**	—									
8. CRSQ T1	.42**	.55**	.16**	.18**	.56**	.35**	.16**	—								
9. CDI T2	.54**	.13**	.28**	.24**	.23**	.17**	.24**	.23**	—							
10. MASC T2	.24**	.53**	.14**	.30**	.31**	.25**	.02	.35**	.33**	—						
11. RPEQ T2	.23**	.12**	.38**	.15**	.16**	.08*	.17**	.17**	.27**	.20**	—					
12. ChEAT T2	.21**	.18**	.23**	.18**	.15**	.11**	.06	.19**	.27**	.22**	.32**	—				
13. EESC T2	.25**	.34**	.15**	.29**	.40**	.20**	.13**	.36**	.33**	.50**	.33**	.18**	—			
14. CSMS T2	.11**	.21**	.13**	.12**	.15**	.21**	.05	.24**	.15**	.34**	.23**	.12**	.33**	—		
15. CAMS T2	.18**	.10**	.19**	.13**	.22**	.15**	.30**	.20**	.18**	.17**	.24**	.05	.32**	.32**	—	
16. CRSQ T2	.35**	.41**	.17**	.11**	.19**	.27**	.09*	.48**	.44**	.69**	.28**	.23**	.59**	.40**	.24**	—
Mean and (SD)	9.67 (6.44)	40.20 (15.39)	5.73 (6.18)	36.75 (26.26)	18.67 (7.02)	4.71 (1.50)	5.43 (1.63)	10.84 (7.65)	10.63 (8.15)	34.80 (18.05)	6.69 (6.80)	38.52 (29.63)	18.40 (7.47)	4.79 (1.53)	5.52 (1.73)	10.18 (8.07)

Note: CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children; RPEQ = Revised Peer Experiences Scale for Children; ChEAT = Children's Eating Attitudes Test; EESC = Emotion Expression Scale for Children; CSMS = Children's Sadness Management Scale; CAMS = Children's Anger Management Scale (aggressive behavior); CRSQ = Children's Response Styles Questionnaire; *$p < 0.05$, **$p < 0.01$.
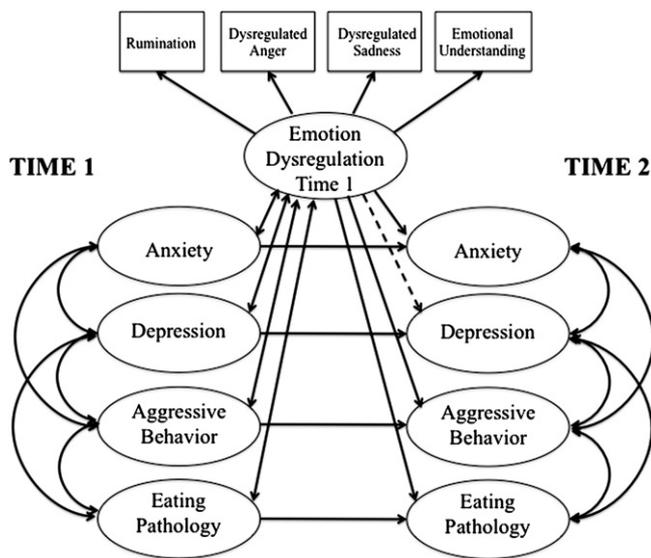
Fig. 2. Note. Figure represents final structural equation model of Time 1 emotion dysregulation predicting Time 2 psychopathology, controlling for psychopathology at Time 1. See Results section for standardized path coefficients (lines drawn with a single-headed arrow). All paths shown are significant ($p < .05$), except those drawn with broken lines. All constructs were modeled as latent variables. Due to space constraints, indicator variables for psychopathology variables and covariates (sex and race/ethnicity) are not displayed.

symptoms of anxiety ($\beta = .19$, $p < .001$), aggression ($\beta = .18$, $p < .001$), and eating pathology ($\beta = .20$, $p < .001$), controlling for Time 1 symptomatology. In contrast, Time 1 emotion dysregulation was not associated with depressive symptoms at Time 2 after adjustment for Time 1 psychopathology, ($\beta = .01$, $p = .879$). The model fit the data well, $\chi^2(641) = 1615.1$, $p < .001$, CFI = .96, RMSEA = .03 (90% CI: .03–.04).

Psychopathology predicting emotion dysregulation

To evaluate whether the presence of psychopathology at Time 1 was associated prospectively with emotion dysregulation at Time 2, we estimated a model with each of the four types of psychopathology at Time 1 predicting emotion dysregulation at Time 2, controlling for Time 1 emotion dysregulation. The covariance among the four types of psychopathology was also modeled. Emotion dysregulation at Time 2 was not associated with Time 1 anxiety symptoms ($\beta = .06$, $p = .351$), depressive symptoms ($\beta = .00$, $p = .973$), aggressive behavior ($\beta = .07$, $p = .131$), or eating pathology ($\beta = .03$, $p = .517$), after controlling for Time 1 emotion dysregulation. The model demonstrated good fit to the data, $\chi^2(284) = 832.0$, $p < .001$, CFI = .96, RMSEA = .04 (90% CI: .03–.04).

Discussion

Accumulating evidence suggests that emotion regulation deficits are associated with a wide range of psychopathological outcomes in children and adults (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Bohnert et al., 2003; Silk et al., 2003; Sim & Zeman, 2006; Zeman et al., 2002). Because nearly all of this research has been cross-sectional, however, it is unknown whether emotion dysregulation is a risk factor for the development of psychopathology or whether emotion regulation deficits are simply a consequence of having a mental health problem. Clarifying the direction of association between emotion regulation and psychopathology has implications both for theoretical conceptualizations

of emotion regulation (Campos, Frankel, & Camras, 2004; Cole, Martin, & Dennis, 2004) and for interventions aimed at preventing and treating adolescent psychopathology. The current study utilized prospective data from a large sample of adolescents that included multiple measures of emotion regulation and symptoms of psychopathology to illuminate this issue. We found that emotion regulation deficits predicted subsequent changes in symptoms of anxiety, aggressive behavior, and eating pathology but not depression. In contrast, psychopathology did not predict subsequent changes in emotion dysregulation. These findings provide perhaps the strongest evidence to date for the role of emotion dysregulation as a risk factor for adolescent psychopathology. Moreover, our results indicate that psychopathology is not associated with increases in emotion dysregulation over time, suggesting that emotion regulation deficits are a predictor and not a consequence of adolescent psychopathology.

Conceptual models of emotion regulation posit that regulation involves processes related to the evaluation and interpretation of emotional information, the modulation of emotional expression, and the management of emotional experiences, responses to emotions, and related thoughts and behaviors (Cole et al., 2004; Mennin, Heimberg, Turk, & Fresco, 2005; Thompson, 1994). We examined the inter-relations of three of these distinct emotional processes and their associations with adolescent psychopathology. Consistent with prior studies documenting that diverse emotion processes related to emotional awareness and the management of emotional experiences loaded onto a unitary dimension of emotion regulation (Aldao & Nolen-Hoeksema, 2010; Mennin et al., 2007), we found that emotional understanding, expression of sadness and of anger, and ruminative responses to distress loaded onto a single latent factor of emotion dysregulation. This model provided a better fit to the data than a more differentiated three-factor model. Our finding of a single emotion dysregulation factor suggests that adolescents who are better able to understand their emotional experiences also are more likely to express their emotions and respond to negative affect in adaptive ways. This is not particularly surprising, given that an understanding of one's own emotions is critical for the adaptive expression and management of emotional experiences (Saarni, 1999). Moreover, both emotional understanding and management have been found to be mediated by common neural mechanisms (Lieberman et al., 2007). These findings contrast, however, with some prior work suggesting greater differentiation of emotion regulation processes (Garnefski, Kraaij, & Spinhoven, 2001; Gratz & Roemer, 2004). One explanation for this discrepancy is that these previous studies examined a wider range of emotion regulation facets than we examined in the present study, including acceptance of one's emotions, reactivity to emotional experiences, and ability to engage in goal-directed behavior and refrain from impulsivity (Gratz & Roemer, 2004). Alternatively, it may be that emotion regulation skills become increasingly differentiated across development. The prefrontal cortex undergoes considerable remodeling during adolescence, particularly in regions known to be associated with emotion regulation (e.g., the anterior cingulate cortex) (Cohen, 2006; Giedd, 2004). These developmental changes may be accompanied by greater differentiation of emotion regulation strategies that require cognitive control (e.g., engaging in goal-directed behavior) or higher-order reflective processing (e.g., acceptance of emotional experiences). Because prior studies finding greater differentiation of emotion regulation processes have focused on older samples, the different pattern of findings may relate to changes in the structure of emotion regulation across development. Examination of developmental changes in the structure and inter-relatedness of different facets of emotion regulation represents an important area for future research.

Emotion dysregulation predicted the subsequent development of multiple forms of psychopathology, including anxiety, aggression, and eating pathology, and the strength of the prospective association was similar across outcomes. These results suggest that emotion regulation deficits are a fairly global risk factor for adolescent psychopathology, consistent with recent evidence from a cross-sectional study of young adults (Aldao & Nolen-Hoeksema, 2010) and a meta-analysis examining the associations of emotion regulation with depression, anxiety, substance abuse, and disordered eating (Aldao et al., 2010). The current findings thus bolster recent conceptualizations of emotion dysregulation as a transdiagnostic factor relevant to multiple types of psychopathology (Ehring & Watkins, 2008; Kring & Sloan, 2010; Moses & Barlow, 2006; Watkins, 2008). The significant associations between emotion dysregulation and psychopathology reported here mirror findings from a number of cross-sectional studies documenting emotion regulation deficits in youths with anxiety disorders (Southam-Gerow & Kendall, 2000; Suveg & Zeman, 2004), problematic aggressive behavior (Bohnert et al., 2003; Cole et al., 1994; Dearing et al., 2002; Shields & Cicchetti, 1998), and disordered eating (Sim & Zeman, 2005, 2006). Our findings regarding the prospective association of emotion dysregulation with subsequent symptom development extend this previous work, supporting theoretical conceptualizations of emotion regulation deficits as an important determinant of psychopathology (Gross & Muñoz, 1995; John & Gross, 2004; Mennin, 2004; Mennin et al., 2005; Moses & Barlow, 2006).

However, emotion dysregulation was not associated with later increases in depressive symptoms. These results contradict somewhat the findings of previous cross-sectional and experience sampling studies documenting associations between emotion regulation deficits and adolescent depressive symptoms (Garber et al., 1995; Sheeber, Allen, Davis, & Sorensen, 2000; Silk et al., 2003). These findings were surprising because rumination was included as one indicator of emotion dysregulation, and a robust prospective association between rumination and depressive symptoms has been reported in a wide range of studies (Nolen-Hoeksema & Morrow, 1991), including among children and adolescents (Abela et al., 2002; Broderick & Korteland, 2004; Burwell & Shirk, 2007). Here, all of the individual emotion regulation measures, including rumination, demonstrated significant cross-sectional relationships with depressive symptoms (see Table 1). Post-hoc linear regression analyses indicated that rumination and dysregulated anger and sadness expression predicted changes in depressive symptoms, but emotional understanding did not. The lack of association between emotional understanding and depression likely contributed to our finding of no prospective relationship between composite emotion dysregulation and depressive symptoms. It may be that adolescents who develop symptoms of depression are not *unaware* of their emotional experiences; rather, they may be all too aware of their negative emotions. Indeed, the self-focus on one's feelings that defines rumination suggests that adolescents who are at risk for depression may attend to their emotions—particularly negative emotions—too frequently, which contributes to the development of depressive symptoms over time (Nolen-Hoeksema & Morrow, 1991). Given the novelty of this finding, however, the lack of a prospective relationship between emotion dysregulation and depressive symptoms should be interpreted with caution. Future longitudinal studies are needed to determine whether other aspects of emotion dysregulation that were not measured in the current study—such as acceptance of emotions—are associated specifically with risk for adolescent depression.

One of the central unresolved questions in the field of emotion regulation is whether dysregulated emotions are risk factors for, versus sequelae of, psychopathology and negative affect (Campos et al., 2004). The current study provides novel evidence indicating that the presence of psychopathology does not predict subsequent changes in emotion dysregulation over time. Changes in emotion dysregulation were unrelated to baseline symptoms of anxiety, depression, aggressive behavior, or eating pathology. Although these findings suggest that emotion dysregulation is not a consequence of psychopathology, additional research is needed to determine whether the onset of psychiatric disorders predicts changes in emotion regulation and whether these results hold for different periods of developmental risk transitions (e.g., the transition from adolescence to young adulthood).

Our findings have important implications for preventive interventions targeting adolescent mental health problems. Empirically-supported treatments and prevention programs for each of the types of adolescent psychopathology examined here rely heavily on cognitive-behavioral techniques (Clarke, DeBar, & Lewinsohn, 2003; Gillham, Reivich, Jaycox, & Seligman, 1995; Kazdin, 2003; Schmidt et al., 2007; Stice, Shaw, Burton, & Wade, 2006). Such interventions may benefit from the inclusion of techniques targeting emotion regulation processes. Inclusion of techniques specifically aimed at increasing emotional awareness and understanding, effectively managing expressions of sadness and of anger, and reducing engagement in rumination could improve the efficacy of preventive and treatment interventions. Several existing interventions provide a template for how to incorporate such techniques into cognitive-behavioral treatments. For example, a recent treatment specifically designed to reduce ruminative self-focus led to significant reductions in depression and associated comorbidity in a small sample of adults (Watkins et al., 2007). This treatment utilized traditional cognitive-behavioral techniques, including functional analysis, to identify and reduce engagement in rumination and to help individuals adopt thinking styles that promote problem-solving and adaptive processing of emotional experiences (Watkins et al. 2007). Mennin (2004, 2006) developed a treatment for anxiety disorders that specifically aims to improve emotional awareness and understanding and decrease engagement in maladaptive emotion regulation strategies. Although both of these interventions warrant further empirical evaluation, the techniques targeting emotion regulation may provide additional clinical benefits if incorporated into existing evidence-based prevention programs for adolescent psychopathology. Kovacs et al. (2006) have developed a particularly promising intervention specifically for children and adolescents targeting self-regulation of distress. The intervention seeks to delineate children's typical responses to distressing situations, identify the contexts that elicit maladaptive management of distress, and replace habitual maladaptive responses to distress with alternative responses from the child's own repertoire of emotion regulation skills to ameliorate negative mood (Kovacs et al., 2006). Several additional treatments that incorporate emotion-focused techniques into cognitive-behavioral therapy for child and adolescent internalizing disorders have been developed and await evaluation in randomized trials (Suveg, Kendall, Comer, & Robin, 2006; Trosper, Buzzella, Bennett, & Ehrenreich, 2009). These treatments each utilize techniques that have direct relevance for preventive interventions targeting emotion dysregulation. Empirical evaluation of preventive interventions that include techniques aimed at promoting adaptive understanding and regulation of emotions represents an important area for future research.

Study findings should be interpreted in light of the following limitations. First, we relied on self-reported symptoms of psychopathology rather than DSM-IV-TR diagnoses based on a structured clinical interview. Although administration of a structured interview to establish diagnoses would represent a methodological improvement, the validity of the self-report measures used in this study is well-established (Timbremont, Braet, & Dreessen, 2004; Wood, Piacentini, Bergman, McCracken, & Barrios, 2002). Nonetheless,

future research is needed to examine the association between emotion regulation processes and the development of DSM-IV-TR disorders. Our use of self-report measures of emotion regulation is another limitation of the study. Utilization of self-report measures in research examining emotion regulation in youth presents challenges because of the difficulties inherent in disentangling initial emotion activation from emotion regulation (Cole et al., 2004) and with reporting on one's *typical* responses to emotional experiences. Biases associated with memories of affective experiences, such as a tendency to remember the most recent experiences or the experiences associated with the most intense emotions (Fredrickson, 2000; Stone et al., 1998), may influence reports of trait emotion regulation. Increasingly, measures that do not rely on self-report, such as psychophysiological and observational measures, are being used to assess emotion and emotion regulation (Chaplin, 2006; Cole, Zahn-Waxler, Fox, Usher, & Welsh, 1996). Such measures are useful in experimental settings to examine state changes in emotion and physiological responses and subsequent regulatory behavior, but they are unfeasible for use with large community samples. Given the size of the sample and the longitudinal nature of the current investigation, utilization of self-report measures presented the only reasonable option for assessing emotion regulation. Moreover, recent evidence suggests good concordance between self-reported and physiologic measures of emotion regulation among youth (Hessler & Katz, 2007), and numerous prior studies examining emotion regulation and youth psychopathology have also relied on self-report measures of emotion regulation (Sim & Zeman, 2005; Southam-Gerow & Kendall, 2000; Zeman et al., 2002). Fourth, our assessment of emotion regulation was not exhaustive. We focused here on emotional understanding, regulation of sadness and of anger, and rumination but did not measure other aspects of emotion regulation likely to be relevant to risk for psychopathology, including negative reactivity to emotional experiences and management of fear (Mennin et al., 2005, 2007). Examination of the associations of these aspects of emotion regulation with adolescent psychopathology is an important area for future research. Finally, although we believe that the racial/ethnic diversity of our sample represents a strength of the study, the preponderance of adolescents of Hispanic ethnicity in our sample may limit the generalizability of our findings. These findings therefore warrant replication in samples with a more representative ethnic distribution.

We extend the literature linking emotion regulation processes to youth psychopathology by providing, to our knowledge, the first longitudinal evidence documenting the role of emotion dysregulation as a risk factor for the development of adolescent psychopathology. Emotion dysregulation predicted subsequent increases in symptoms of anxiety, aggressive behavior, and disordered eating but not depression. Importantly, the results provided no evidence for an association between psychopathology and subsequent increases in emotion dysregulation, indicating that the presence of psychopathology did not render adolescents less able to effectively manage their emotions. Thus, emotion dysregulation appears to serve as a risk factor for, rather than a consequence of, psychopathology in adolescents. These findings have important implications for preventive interventions targeting adolescents and suggest that techniques that promote emotional understanding and the adaptive expression and modulation of negative emotions should be incorporated into existing interventions.

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