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Emotion Regulation Mediates the Association Between ADHD and Depressive Symptoms in a Community Sample of Youth

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Abstract The purpose of this study was to examine the longitudinal relationship between attention-deficit/hyperactivity disorder (ADHD) symptoms, emotion regulation (ER) ability, and depressive symptoms within a diverse community sample of 277 youth, ages 9–12 (56 % male). Participants were drawn from a larger study examining adolescent risk behaviors, and completed annual assessments over 3 years. Youth ADHD symptoms were assessed at Time 1 (T1) using the parent-reported Disruptive Behavior Disorders Rating Scale, ER was assessed with the parent-reported Emotion Regulation Checklist at Time 2 (T2), and youth depressive symptoms were assessed using the self-reported Revised Child Anxiety and Depression Scales at Time 3 (T3). Analyses examined T2 ER as a mediator between T1 ADHD symptoms (including the unique contributions of inattentive [IA] versus hyperactive/impulsive [HI] symptoms) and T3 depressive symptoms. Structural equation modeling (SEM) indicated the path model specified provided an excellent fit to the data. Tests of indirect effects suggested that T2 ER appears to be a significant mechanism that underlies the relationship between T1 ADHD and T3 depression, even when accounting for T1 oppositional defiant and depressive symptoms. Furthermore, while both T1 IA and HI symptoms had significant indirect effects on T3 depression through the mechanism T2 ER, HI proved a more robust predictor of T2 ER than IA. Results of this prospective study support cross-sectional findings pointing to ER as a potential mechanism linking ADHD and depressive symptoms in youth. Clinical implications and future directions are discussed.

Keywords Attention-deficit/hyperactivity disorder · Emotion regulation · Depression · Emotion

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Attention-deficit/hyperactivity disorder (ADHD) — a neurodevelopmental disorder affecting 6 % to 9 % percent of children and adolescents in the United States (Polanczyk and Jensen 2008) — is characterized by developmentally-inappropriate levels of inattention, hyperactivity and impulsivity (American Psychiatric 2000). Approximately 10–40 % of youth with ADHD also meet diagnostic criteria for an affective disorder, primarily major depressive disorder (MDD) (Daviss et al. 2009; Elia et al. 2008). Prospective longitudinal studies show that in comparison to their non-disordered peers, youth with ADHD demonstrate higher levels of depressive symptomatology (Lahey et al. 2007; Lee et al. 2008), and a greater likelihood of developing mood disorders in late childhood and adolescence (Biederman et al. 2008; Chronis-Tuscano et al. 2010; Monuteaux et al. 2007). Moreover, compared to healthy controls, children diagnosed with ADHD in early childhood are four times more likely to be diagnosed with MDD or dysthymia prior to age 18 (Hazard Ratio: 4.32, $p<0.001$), and are at greater risk for recurrent episodes of depression throughout childhood and adolescence (adjusted odds ratio [OR], 12.15; 95 % CI, 2.62–56.32; Chronis-Tuscano et al. 2010). Youth diagnosed with ADHD in childhood are also at greater risk for engaging in suicide attempts or self-injurious behavior as young adults than are healthy controls (Hinshaw et al. 2012). Such results underscore the need for a more comprehensive understanding of the relationship between ADHD and depressive symptoms in youth.

In combination, ADHD and depression result in more significant impairments than for either disorder alone (Daviss 2008). For example, compared to youth with ADHD alone, youth with comorbid ADHD and MDD are at greater risk for developing bipolar disorder and oppositional defiant disorder (ODD), require significantly more intensive interventions, and report having more psychosocial and familial problems and experiencing higher levels of stress (Biederman et al. 1996; Jensen et al. 1993). Further, results from a 5-year prospective study of females first diagnosed with ADHD between the ages of 6–18 indicated that girls with comorbid ADHD and MDD demonstrated an earlier onset of depression, higher rate of suicidality and increased rate of hospitalization in comparison to females diagnosed with MDD alone (Biederman et al. 2008). Perhaps most alarming, youth with comorbid ADHD and mood disorders are three times more likely to complete suicide than youth diagnosed with either disorder alone (James et al. 2004). These startling statistics indicate the substantial public health concern associated with comorbid ADHD and depression in youth.

Typically, research on ADHD and/or depression in youth has focused on disorder-level analysis (i.e., youth presenting with one or both disorders). However, research suggests that even youth with elevated, but not diagnostic levels, of either ADHD or depression demonstrate considerable functional impairment and are at significant risk for developing a psychiatric disorder (e.g., Bussing et al. 2010; Keenan et al. 2004, 2008). Children ages 5 to 11 who were identified as at "high risk" for ADHD at baseline (i.e., 1.5 SD above the norm on parent or teacher ADHD ratings) were 10 times more likely than those in the "low risk" ADHD group to demonstrate difficulties with depression 8 years later (Bussing et al. 2010). Examination of sub-threshold symptoms of ADHD may be particularly relevant as recent evidence argues for a dimensional rather than a categorical view of ADHD (Barkley 2006; Marcus and Barry 2011; Nikolas and Burt 2010; Sonuga-Barke 2005). Therefore, research utilizing community samples who present with a broader range of ADHD symptoms may offer a more comprehensive picture of the relationship between ADHD and depressive symptoms than sampling from clinical populations which likely represent more extreme cases of the disorder (Levy and Hay 2001).

Despite the impact of comorbid ADHD and depressive symptoms on youth, few studies have examined mechanisms underlying this relationship. One promising mechanism is emotion regulation (ER) ability, given that deficits in ER ability have been associated with both ADHD and depressive symptoms in youth (Barkley 1997; Compas et al. 2009; Durbin and Shafir 2008; Martel and Nigg 2009; Martel et al. 2009). ER has been conceptualized as a multitude of processes (e.g., attentional control, reward-processing, motivational states, behavioral inhibition) involved in the initiation, modulation and expression of feeling states in order to achieve a goal or adapt to external circumstances (Eisenberg and Spinrad 2004). This definition is principally relevant to youth with symptoms of ADHD, as it emphasizes the role of effortful control — the deliberate modulation of emotional states and subsequent behaviors, which involves the ability to deliberately focus and shift attention, as well as to inhibit or activate behavior as appropriate (Martel 2009; Martel and Nigg 2009; Rothbart and Posner 2006). Deficits in effortful control are implicit in the symptoms of ADHD (Martel 2009) and have been suggested to underlie ER difficulties in theoretical models of ADHD (Barkley 1997).

Significant evidence reveals that youth with ADHD demonstrate ER deficits in comparison to their non-disordered peers. Several studies suggest that youth with ADHD demonstrate an inability to continue a task when frustrated or seek help from their parents, demonstrate extreme levels of negative affect and poorer problem-solving abilities, and focus more on the negative aspects of a task compared to healthy controls (Maedgen and Carlson 2000; Melnick and Hinshaw 2000; Walcott and Landau 2004). Youth with ADHD also display difficulties identifying and processing negative emotions (Norvilitis et al. 2000; Singh et al. 1998). Longitudinal research has shown that youth whose ADHD persists into adulthood demonstrate more emotional impulsiveness (defined by symptoms such as low frustration tolerance, impatience, irritability) than both youth whose ADHD remits in adulthood and those without ADHD, and that this "emotional impulsivity" contributes to family, peer, relationship, financial and driving impairments (Barkley and Fischer 2010).

Comorbid symptoms of ODD have been hypothesized to play a role in emotion dysregulation among youth with ADHD (e.g., Nigg et al. 2004). For example, one study demonstrated that youth with ADHD and high levels of aggression display greater difficulties with ER than youth with ADHD and low aggression, who did not differ from controls (Melnick and Hinshaw 2000). At present, the role of ODD symptoms in the relationship between ADHD and ER remains unclear, but existing studies do suggest that ODD should be considered in research examining ADHD and ER ability.

Difficulties with ER have also been associated with depressive symptoms in youth (Chaplin et al. 2005; Cole et al. 2003; Durbin and Shafir 2008). Specifically, dysfunctional ER ability has been related to both depressive symptoms and disorders in cross-sectional, community samples of youth (Larson et al. 1990; Nolen-Hoeksema et al. 1993; Reijntjes et al. 2007; Silk et al. 2003). While the majority of studies of ER and depressive symptoms have been cross-sectional, one longitudinal study of girls found that difficulties with ER evaluated between the ages of 5–8 years predicted depressive symptoms at age 10, suggesting that difficulties with ER predate depressive symptoms (Feng et al. 2009).

Despite evidence for the association of ER deficits with both ADHD and depression, only two cross-sectional studies

have examined ER as a mechanism underlying the relationship between ADHD and depressive symptoms in youth. First, Seymour and colleagues (2011) reported that ER fully mediated the relationship between ADHD diagnosis and concurrent depressive symptoms in youth ages 11–14, even after accounting for ODD and conduct disorder (CD) diagnoses. Further, Anastopoulos and colleagues (2011) found that youth with ADHD were at significantly elevated risk (OR=5.703, CI=2.991–10.878, $p<0.001$) for demonstrating high levels of emotional lability relative to their unaffected siblings, and that emotional lability partially mediated the relationship between ADHD and depressive symptoms.

While these studies provide the first examination of ER as a mechanism explaining the relationship between ADHD and depression, both of these studies were cross-sectional. Given that mediation analysis assumes a temporal relationship between variables that unfolds over time, the use of cross-sectional data may result in biased estimates of mediation effects (Maxwell and Cole 2007). In contrast, longitudinal data allows for more precise inferences about the temporal and potentially causal relations between variables and within the model (Cole and Maxwell 2003). A second limitation of the prior work is that ER ability was only examined in relation to diagnosable levels of ADHD and depressive symptoms. However, the literature suggests that even children with elevated levels of ADHD symptoms demonstrate a greater likelihood of experiencing difficulties with depression (Bussing et al. 2010), indicating the need for a more comprehensive understanding of the relationship between a broader range of ADHD symptomatology and depressive symptoms in youth. Finally, neither study thoroughly examined the specific contributions of inattentive (IA) versus hyperactivity/impulsive (HI) symptoms to ER ability or depressive symptoms. However, some extant literature suggests that IA and HI may be differentially related to ER ability (e.g., Martel 2009; Martel et al. 2009) and depressive symptoms (e.g., Chronis-Tuscano et al. 2010; Martel et al. 2011) in youth. Therefore, the goal of the current study is to examine the temporal relationship between ADHD symptoms (including the specific contributions of IA versus HI symptoms), ER and depressive symptoms using multiple informants in a large, community sample of youth.

Methods

Participants and Procedures

Participants were drawn from a larger study of 277 children who were between the ages of 9–12 at initial enrollment and were followed prospectively over a period of 3 years. Children and their parents were recruited though media outreach and mailings to schools, libraries and youth organizations within the Washington DC metropolitan area. Inclusion criteria for the larger study allowed the participation of any adolescent within the specified age range who spoke English and had a consenting parent/guardian.

Families who met inclusion criteria based on a telephone screen were invited to a baseline assessment at the University of Maryland. At this time, a more detailed description of study procedures was provided, and parent and child participants signed informed consent and assent which were approved by the university Institutional Review Board (IRB). Subsequently, parents and children were separated, so that each could be given standardized, specific instructions for the completion of rating forms. Although both mothers and fathers were eligible to participate, due to limited paternal responses (e.g., at Time 1 only 24 of 277 [9 %] fathers completed forms), we rely on maternal report for these analyses. Follow-up data for all participants was collected at yearly intervals (Time 1[T1], Time 2[T2], Time 3[T3]) and consisted of the same data collection procedures outlined above. At T2 and T3, follow-up rates were 85.2 % and 82.3 % respectively.

Sample demographics are presented in Table 1. Average child age (in years) within the sample was 11.0 ($SD=0.81$) at

Table 1 Participant demographic and clinical characteristics

Variable	*M* (*SD*)	% (*n*)
Time 1		
Child Characteristics		
Age, years	11.00 (0.81)	
Sex, % male		56.3 (156)
Ethnicity[a]		
Caucasian		49.3 (136)
African American		35.5 (98)
Hispanic/Latino		2.9 (8)
Asian		1.4 (4)
Other		10.9 (30)
ADHD symptoms	0.37 (0.38)	
ODD symptoms	0.12 (0.21)	
Depressive symptoms	1.67 (0.10)	
Mother age, years[a]	41.86 (6.21)	
Median Family Income[b]	$85,000	
Time 2		
ER[c]	24.60 (6.98)	
Time 3		
Depressive symptoms[d]	1.66 (0.10)	

For ADHD, ODD and depressive symptoms, M (SD) are presented for log transformed variables due to non-normal distributions

ADHD attention-deficit/hyperactivity disorder, *ER* Emotion Regulation, *ODD* Oppositional Defiant Disorder

[a] $n=267$

[b] $n=264$

[c] $n=234$

[d] $n=236$

T1, 12.1 (*SD* = 0.91) at T2, and 13.1 (*SD* = 0.89) at T3. Fifty-six percent of the sample was male, 49.3 % Caucasian, 35.5 % African-American, 2.9 % Latino, 1.4 % Asian and 10.9 % of mixed or other ethnicity. Maternal age at T1 ranged from 26 to 55-years old (*M* = 41.9, *SD* = 6.21), and median family income was $85,000 (<$1,000–$850,000 year).

Measures

Demographics At each assessment point, parents/guardians completed a basic demographics form which included personal information about both the parent/guardian and child including: age, gender, ethnicity/race, and annual family income.

Youth ADHD and ODD Symptoms Youth DSM-IV ADHD symptoms were assessed by maternal-report on the Disruptive Behavior Disorders (DBD) symptom checklist (Pelham et al. 1992), which assesses ADHD, ODD and conduct disorder symptoms on a 4-point Likert scale from *not at all* to *very much*. Symptoms rated as occurring *pretty much* or *very much* were considered present to a clinically significant degree. Given that prior research has suggested that ODD symptoms may account for ER deficits in youth with ADHD (Melnick and Hinshaw 2000; Nigg et al. 2004), and that oppositional behavior and irritability are significantly related to the development of depression in adulthood (Stringaris et al. 2009), ODD symptoms were accounted for in the analyses. The version of the DBD symptom checklist used in this study included items from both DSM-III-R and DSM-IV, but for the current study, symptom counts only included DSM-IV symptoms. Items occurring at a clinically significant level were summed to create total symptom counts for each disorder (ADHD and ODD). Additionally, for ADHD, IA symptoms occurring at clinically significant levels were summed to create a total IA score, and HI items occurring at clinically significant levels were summed to create a total HI score.

The DBD has been used with adolescents up to age 18 and gender- and age-based norms are available (Pelham et al. 1992). Internal consistency for the DBD in this sample was moderate to high on the IA symptom (α=0.78), HI symptom (α=0.89), total ADHD symptom (α=0.91) and total ODD symptom (α=0.75) scales.

Youth ER Ability Mothers completed the Emotion Regulation Checklist (ERC; Shields and Cicchetti 1997) at T2. The ERC is a 24-item measure which assesses the frequency with which youth exhibit a variety of positive and negative ER-related behaviors. Items are rated on a 4-point Likert scale (1 = *rarely/never*; 4 = *almost always*). For the present study, the Lability/Negativity (L/N) subscale was used, as it most closely reflects ER deficits hypothesized to be present in youth with ADHD (Martel 2009; Nigg et al. 2004). The ERC-L/N subscale is composed of 15 items (11 items positively scored; 4 items reverse scored) focusing on the inappropriate regulation of negative emotions, variability of mood and inflexibility of emotional response, emotional intensity, and dysregulation of positive emotion. Sample items include: "Is easily frustrated", "Can bounce back and recover quickly when upset or frustrated (e.g., doesn't pout or stay sullen, anxious or sad after emotionally distressing events)", and "Transitions well from one activity to another; doesn't become angry, anxious, distressed or overly excited when moving from one activity to another". Raw scores from the 15 items that compose the ERC- L/N subscale are summed, and higher scores are reflective of poorer emotion regulation (i.e., greater dysregulation). Previous research with the ERC has demonstrated good construct validity and has been shown to discriminate between well-adjusted and maltreated youth (Shields and Cicchetti 1997; Shields et al. 2001). For the current study, internal consistency was good (α=0.85).

Youth Depression Symptoms Youth depressive symptoms at T1 and T3 were assessed using the self-report Revised Child Anxiety and Depression Scales (RCADS; Chorpita et al. 2000; Spence 1997). The RCADS is a 47-item instrument that assesses DSM-IV symptoms of anxiety and depression in youth between the ages of 8 to 18. Items are rated on a 4 point Likert scale from 0 (*never*) to 3 (*always*). In the current study, the depression subscale, consisting of 10 items assessing common symptoms of depression including sadness, anhedonia, neurovegetative symptoms, and feelings of worthlessness, was used. Raw scores on the depression subscale can be converted to grade and gender specific T-scores providing more clinically-relevant data; therefore, T-scores were utilized in data analysis (Weiss and Chorpita 2011). Research examining 1,641 children and adolescents demonstrated that the factor structure of the RCADS is consistent with DSM-IV depression and that internal consistency is good (Chorpita et al. 2000). Internal consistency for the present sample was good (α=0.84).

To ensure the independence of the ER, ODD and depression constructs in the current study the ERC-L/N subscale, DBD ODD subscale and RCADS depression subscale items were examined for overlap. While no items overlapped on the ERC-L/N and RCADS; one item from the DBD ODD subscale ("Often loses temper") overlapped with an ERC-L/N subscale item ("Has angry outbursts/tantrums easily"). Therefore, it was removed from the ERC-L/N subscale calculation (changing α=0.85 to α=0.81). This revised ER variable was used for all analyses.

Data Analytic Plan

Prior to conducting analyses, distributions of the variables were inspected. Consistent with previous studies (Arnett

et al. 2012; Piehler et al. 2012), distributions that were non-normal were log transformed which included: T1 ADHD (total, IA and HI), ODD, depressive symptoms and T3 depressive symptoms (skew ranged from: 1.13 to 3.81, kurtosis: 2.23–18.92). The resulting transformations improved the kurtosis and skewness values for all the non-normal variables.

Additionally, missing data patterns were examined which revealed that missing data for the variables in the model ranged from 0.0 % to 15.5 %. Only three variables had missing data: T1 depression (4.3 %), T3 depression (14.8 %) and T2 ER (15.5 %). Accordingly, Little's (1988) missing completely at random (MCAR) test using SPSS missing values program was then conducted. The findings suggested that the missing data appeared to be missing at random, $\chi^2(44)=34.45$, $p=0.85$. Given that the data appeared to be MCAR, we used full information maximum likelihood estimation (FIML) procedures to estimate the missing parameters in the model (Enders 2001). FIML employs an algorithm that utilizes all of the observed data to estimate and infer probable values for the individuals' missing data. This procedure yields less biased and superior parameter estimates from samples with moderate missing data (Enders 2001).

Bivariate correlations were examined between hypothesized covariates (e.g., child gender, ODD symptoms) and the variables within the specified model (i.e., T1 ADHD and depressive symptoms; T2 ER and T3 depressive symptoms). Child gender and T1 ODD symptoms were included given that previous studies have found these two factors significantly associated with ADHD, ER ability, and depression (e.g., Naninck et al. 2011; Stringaris and Goodman 2009; Stringaris et al. 2012; Zahn-Waxler et al. 2008).

Structural equation modeling was performed using M*plus* 6 (Muthen and Muthen 2008). Three waves of data were used: T1, T2, and T3 and the model incorporated cross-lagged and auto-regressive paths. The cross-lagged paths tested the indirect effect of T1 ADHD on the distal outcome T3 depressive symptoms through the T2 mediator ER, while controlling for the covariates of depression, gender and ODD, in the model. The auto-regressive path represents stability of the construct over time (T1 depressive symptoms on T3 depressive symptoms). To evaluate model fit the conventional fit indices, the comparative fit index (CFI), root mean square error of approximation (RMSEA) and standard root mean square residual values (SRMR) were used. Values great than 0.95 for CFI, and RMSEA and SRMR values of 0.08 or less suggest good model fit (Garson 2009).

We tested for the indirect effect of T1 ADHD on T3 depressive symptoms through T2 ER using the RMediation program (Tofighi and MacKinnon 2011). RMediation builds on the widely cited and used PRODCLIN program (MacKinnon et al. 2007) by producing more accurate confidence intervals using three methods: distribution of products, Monte Carlo simulation and asymptotic normal distribution. Simulation studies suggest that the distribution of products yields more accurate confidence intervals and more optimal coverage (i.e., more power) than resampling methods including the percentile and bias-corrected bootstrap methods (MacKinnon et al. 2007; Tofighi and MacKinnon 2011).

We also conducted secondary analyses exploring the specificity of the ADHD dimensions of IA and HI symptoms in relation to T2 ER and T3 depression. Structural equation modeling and tests of indirect effects were conducted as outlined above.

Results

Clinical Characteristics

Child participants in the study demonstrated a wide range of parent-reported ADHD symptoms at T1 ranging from 0 to 17 (of a possible 18). Twenty-seven (9.7 %) children met DSM-IV symptom criteria for ADHD (i.e., 6 out of 9 symptoms of either IA or HI) according to maternal report. Similarly, the range of ODD symptoms at T1 ranged from 0 to 7 (of a possible 8), and 11 (4 %) participants met DSM-IV symptom criteria for ODD per maternal report. Of the 27 participants who met DSM-IV symptom criteria for ADHD, three (11.1 %) also met symptom criteria for ODD at Time 1. In terms of depressive symptoms, at T1 the average T-score reported was 47.59 (SD=11.94), with 14 (5.1 %) participants reporting T-scores in the clinically significant range (i.e., $T\geq 70$). At T3, the average depression T-score was 45.83 (11.58), and 9 (3.2 %) participants reported T-scores in the clinically significant range.

Correlational Analyses

Pearson-product moment correlations revealed that T1 ADHD was positively related to T1 ODD and depressive symptoms, T2 ER, and T3 depressive symptoms (Table 2). Additionally, T1 ODD symptoms were significantly positively correlated with T2 ER and T3 depressive symptoms. Depressive symptoms at T1 were positively related to T2 ER and T3 depressive symptoms. T2 ER was significantly positively correlated with T3 depressive symptoms. Child gender was not significantly related to T1 ADHD, ODD or depressive symptoms, T2 ER or T3 depressive symptoms (Table 2).

Structural Equation Modeling

Prior to testing the full model, we investigated the total effects of ADHD, T1 depression and the covariates ODD and gender without T2 ER in the model. The results suggested that T1 depression and ODD ($\beta=0.14$, $SE=0.062$, $p<0.05$) were both positively related to T3 depression, while T1 ADHD

Table 2 Bivariate correlations among study variables

Variables	1	2	3	4	5	6
Time 1						
1. ADHD symptom	–					
2. ODD symptoms	0.544***	–				
3. Depressive symptoms	0.259***	0.102	–			
4. Child Gender	0.038	0.074	−0.015	–		
Time 2						
5. ER	0.616***	0.519***	0.180**	0.076	–	
Time 3						
6. Depressive symptoms	0.184**	0.163**	0.570***	−0.003	0.233***	–

ADHD Attention-Deficit/Hyperactivity Disorder, *ER* Emotion Regulation, *ODD* Oppositional Defiant Disorder, *SES* Socio-economic Status

*$p<0.05$

**$p<0.01$

***$p<0.001$

($\beta=-0.01$, $SE=0.062$, $p=0.91$) and gender ($\beta=0.02$, $SE=0.05$, $p=0.74$) did not appear to have a significant direct effect on the outcome.

Next, the full SEM model (Fig. 1) was tested and it provided an excellent fit to the data, χ^2 (3)=18.94, $p=0.01$, CFI=0.95, and SRMR=0.039. The autoregressive path of T1 depressive symptoms to T3 depressive symptoms was significant suggesting levels of depression were moderately stable over time. In terms of the cross-lagged path, T1 ADHD was positively and significantly associated to T2 ER, and the variable explained 37 % of the variance in ER. Controlling for T1 depressive symptoms and the covariates (i.e., ODD and gender) in the model, T2 ER predicted T3 depressive symptoms, and the variables in the model explained 37 % of the variance in T3 depression. These findings suggest the direct effects of ODD ($\beta=0.11$, $SE=0.064$, $p=0.08$) on T3 depression diminished when T2 ER was added to the model.

Tests of the indirect effects suggest that T1 ADHD (indirect effect=0.085, $SE=0.043$; 95 % CI [0.002, 0.171], $p<0.05$) appears to have a significant indirect influence on T3 depressive symptoms through the mechanismT2 ER. In other words, T1 ADHD appeared to contribute to T2 ER, which in turn heightened the risk of T3 depression.

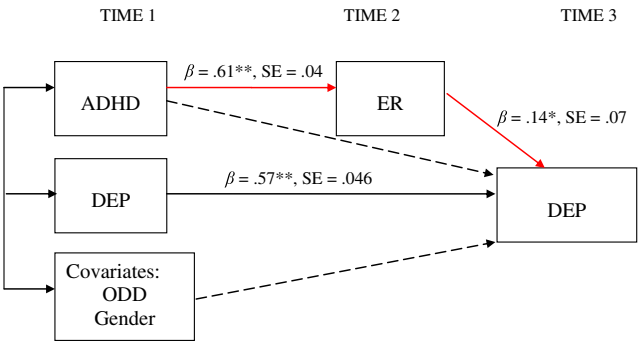

Fig. 1 Temporal relationship between ADHD, ER and Depression. Path coefficients presented are standardized. *Red paths* denote indirect effects of T1 ADHD and ODD on T3 depressive symptoms through T2 ER ability. *ADHD* attention-deficit/hyperactivity disorder, *DEP* depressive symptoms, *ER* emotion regulation ability, *ODD* oppositional defiant disorder. *Dashed lines* indicates non-significant path. *$p<0.05$, **$p<0.001$

Alternative Model Examining IA vs. HI

Within the extant literature, the specific contributions of IA versus HI symptoms to ER ability or depressive symptoms have not been comprehensively examined. Therefore, we sought to further examine the specificity of T1 IA and HI symptoms in relation to T2 ER and T3 depression. The path model (Fig. 2) provided an excellent fit to the data, χ^2 (4)=18.24, $p<0.01$, CFI=0.97, SRMR=0.037. Results demonstrated that T1 IA and HI symptoms were not significantly associated with T3 depression. However, IA and HI were both positively associated with T2 ER, and together explained 40 % of the variance in T2 ER.

Further, in order to determine whether the magnitude of the estimate between IA and T2 ER was significantly different from the magnitude of the estimate between HI and T2 ER, we conducted a Steiger's Z (Steiger 1980; Uitenbroek 1997) test to examine which ADHD variable (i.e., HI and IA) was a better predictor of T2 ER. The results revealed that HI

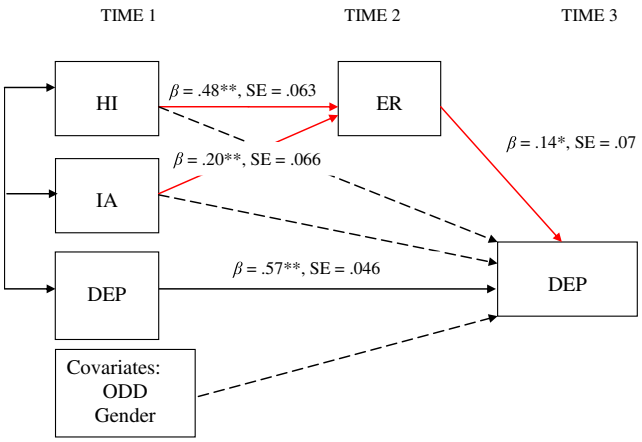

Fig. 2 Temporal relationship between Inattentive Symptoms, Hyperactive/Impulsive symptoms, ER and Depression. Path coefficients presented are standardized. *HI* Hyperactivity/Impulsivity. *IA* Inattention. *DEP* depressive symptoms. *ER* emotion regulation ability. *ODD* oppositional defiant disorder. *Red paths* denote indirect effects of T1 IA, and HI on T3 depressive symptoms through T2 ER ability. *Dashed lines* indicates non-significant path. *$p<0.05$, **$p<0.001$

appeared to have a statistically stronger relationship on T2 ER compared to IA, $Z=3.39$, $p<0.05$.

Finally, given that the missing data ranged from 0 % to 15.5 % on the various variables in our model, we tested our primary model without using FIML procedures. T1 ADHD to T2 ER was $\beta=0.63$ (compared 0.61 with FIML) while the association between T2 ER to T3 DEP was $\beta=0.12$ (compared to 0.14 with FIML). These results suggest that the patterns and magnitude of the relationships were nearly identical to the results using FIML procedures thus strongly supporting the robustness of our original model.

Discussion

The goal of the present study was to examine the longitudinal relationship between ADHD symptoms, ER, and depressive symptoms within a community sample of children. Recent cross-sectional research has suggested that ER ability in youth with ADHD may be an important mechanism underlying the development of subsequent depression or depressive symptoms (Anastopoulos et al. 2011; Seymour et al. 2011). However, the present study is, to our knowledge, the first prospective investigation of the relationship between ADHD symptoms, ER ability and depressive symptoms in youth. Furthermore, we extended existing literature by examining the specific contributions of IA versus HI symptoms on later ER ability and depressive symptoms.

Overall, our results suggested that T2 ER appears to be an important mechanism in the relationship between T1 ADHD symptoms and T3 depressive symptoms. That is, ADHD symptomatology at an earlier developmental time point may heighten future ER difficulties, which in turn may lead to higher levels of later depression symptoms. These results extend cross-sectional research suggesting that ER mediates the relationship between ADHD diagnosis and concurrent depressive symptoms in youth (Anastopoulos et al. 2011; Seymour et al. 2011) by providing strong support of mediation in a longitudinal design and within a diverse community sample. As ineffective utilization of ER strategies (e.g., using fewer problem-focused and active distraction strategies and more avoidant, passive, and aggressive strategies) has been associated with depression in youth (Garber et al. 1995; Reijntjes et al. 2007; Silk et al. 2003), it may be that youth with ADHD, due to deficits with inattention, hyperactivity and impulsivity, have difficulty effectively coping with their negative emotions (i.e., poor ER) leading to increased feelings of depression. In fact, a growing literature demonstrates an inverse relationship between effortful control, the ability to deliberately shift attention and inhibit behavior, and symptoms of depression in children and adolescents (Muris 2006; Muris et al. 2008; Oldehinkel et al. 2007; Verstraeten et al. 2009). Unfortunately, in our sample we do not have a measure of T1 ER, making it impossible to confirm that ADHD symptoms predate ER difficulties. Further longitudinal research is therefore needed to elucidate the temporal relationship between ER and ADHD in youth.

Our study also sought to address a notable gap in the literature by examining the longitudinal relationship between continuous ADHD and depressive symptoms. Within both the research and clinical communities, there has been increasing emphasis placed on the dimensional rather than categorical nature of mental disorders in which symptoms fall on a continuum ranging from normative to atypical levels (Cuthbert and Insel 2010; NIMH Strategic Plan 1.4). Recent empirical evidence argues for a dimensional rather than a categorical view of ADHD (Barkley 2006; Marcus and Barry 2011; Nikolas and Burt 2010; Shaw et al. 2011; Sonuga-Barke 2005). Our results emphasize the importance of such a dimensional examination of symptoms, as the majority of youth in our study demonstrated ADHD symptom counts below diagnostic threshold, yet there was a strong positive correlation between T1 ADHD symptoms, T2 ER and T3 depressive symptoms. These findings support the work of Bussing and colleagues (2010) who also found that within a community sample, children with elevated ADHD symptoms demonstrate a greater likelihood of experiencing difficulties with depression (Bussing et al. 2010). Even at sub-clinical levels, symptoms of psychopathology have been found to increase a child's odds of developing a diagnosable disorder, and are associated with significant levels of impairment (Keenan et al. 2008). For instance, Keenan and colleagues (2004) found that while less than 1 % of 5-to-8-year-old girls met diagnostic criteria for major depression, girls with sub-clinical depression reported significant impairment. Therefore, children demonstrating sub-clinical levels of ADHD and depression may experience impairments similar to those with full-blown diagnoses, potentially related to more overarching difficulties with ER. Our results support the need for the global assessment of inattention, hyperactivity/impulsivity and ER ability in all children as these problems may predate issues later issues with depression.

Prior research examining the relationship between ER and ADHD has suggested the potentially confounding nature of ODD symptoms in this relationship (e.g., Nigg et al. 2004). For example, one prior study found that youth with ADHD and high levels of aggression displayed greater difficulties with ER than youth with ADHD and low aggression, who did not differ from controls (Melnick and Hinshaw 2000). Additionally, a significant body of research has demonstrated that oppositionality in youth is a predictor of depression in young adulthood (Copeland et al. 2009; Stringaris et al. 2009), and that oppositionality and depression are linked by irritability (Stringaris et al. 2012; Stringaris and Goodman 2009). Therefore, in our models we accounted for ODD symptoms as a covariate. Our results suggested that even when

accounting for T1 ODD, T1 ADHD continued to have a significant indirect influence on T3 depressive symptoms through the mechanism T2 ER. Furthermore, when T1 IA and HI symptoms were examined in the model, they both demonstrated a stronger relationship to T2 ER than T1 ODD symptoms. Taken together, our results suggest the unique relationship between ADHD symptoms, ER ability, and the development of depressive symptoms in youth.

Surprisingly, our sample did not demonstrate a significant relationship between gender and ADHD symptoms, depression symptoms, or ER ability despite well-documented evidence of such associations within the literature (e.g., Hinshaw et al. 2012; Naninck et al. 2011; Nolen-Hoeksema 2012; Rucklidge 2010; Zahn-Waxler et al. 2008). Our findings may be related to our overall low base rates of ADHD and depressive symptoms. However, of the 27 participants who met ADHD symptom criteria for ADHD at T1, 63 % were male. Similarly, of the 14 participants reporting clinically significant depression at T1, 78 % were female. Therefore, while in the overall sample gender was not significantly associated with ADHD symptoms, depressive symptoms or ER ability, for youth demonstrating clinically significant levels of ADHD or depression, the gender ratios more closely approximated the prior literature (Nolen-Hoeksema 2012). Future longitudinal research should explore these relationships in youth with diagnosable levels of ADHD and/or depression.

As a secondary aim, we also sought to examine the specific contributions of IA versus HI symptoms to ER ability and depressive symptoms. Our results suggested that T1 symptoms of both IA and, to a greater degree, HI were significantly related to T2 ER. Additionally, T1 IA and HI both had significant indirect effects on T3 depression through the mediator T2 ER. Such findings are not surprising in light of the work suggesting a "dual pathway model" of ADHD which involves "top-down" executive control and "bottom-up" affective/reactive processes (Nigg 2006; Martel and Nigg 2006). From this model, symptoms of HI would be related to affective and reactive processes including the propensity to certain affective states and reactive control. On the other hand, top-down processing which includes effortful control, requires greater mental resources and would be reflected in symptoms of inattention. Therefore, both top-down and bottom-up processes are required for appropriate regulation of emotional states. It is likely that the combination of both IA and HI symptom together results in greater emotion dysregulation as research suggests that youth with ADHD-combined type experience greater emotional lability than those with either ADHD –Inattentive subtype or Hyperactive/Impulsive subtype (Anastopoulos et al. 2011).

While promising, our results should be interpreted in light of some limitations. First, our measurement of ER relied solely on parent-report measures. However, the construct of ER can be most completely understood by examining multiple processes related to emotion including: biological responses (central and peripheral nervous system responses), behavioral responses, sociocultural influences and psychosocial factors (Thompson et al. 2008). While we only utilized parent-report of youth ER, other methods of assessment include self-report of ER, behavioral observations, and physiological/biological indicators (e.g., skin conductance, neural activity) (Melnick and Hinshaw 2000; Morris et al. 2006; Zeman et al. 2007). Inherently, each form of ER measurement has its own limitation. For example, our use of parent-report, while often considered more reliable and valid than self-report of ER (Morris et al. 2006), may result in bias due to parental stress, parental psychopathology or parents' own ER abilities (Fergusson et al. 1993; Han and Shaffer 2012). Moving forward, multimodal assessment of ER is necessary to parse apart different experiences of youth ER, but also delineate the processes involved in ER (e.g., monitoring versus evaluation versus modification of emotion). Utilization of multiple forms of ER measurement may provide more comprehensive insight into the relationship between ER and youth psychopathology, such as ADHD and depression.

A second limitation is that the current study included only the assessment of sub-clinical levels of ADHD and depression. As a result, we are unable to generalize our findings to youth with diagnosable levels of either disorder. ADHD symptoms were measured solely on the basis of parent-report; typically, a structured diagnostic interview, teacher reports, and assessment of age on onset and functional impairment would be required for a formal diagnosis of ADHD (Pelham et al. 2005). Examining the longitudinal relationship between ADHD diagnosis, depression and ER will be an important next step. Furthermore, it would be advantageous to begin such a study early in development in order to more concretely establish the temporal relationship between disrupted ER and ADHD. While we hypothesized that ADHD symptoms contributed to poor ER and therefore increased risk of depressive symptoms, it may be that difficulties with ER predated the emergence of both ADHD and depressive symptoms. Finally, given that we did not have a T1 ER measure and a T2 ADHD assessment, we were unable to explore important autoregressive relationships (i.e., T1 ADHD on T2 ADHD) and possible cross-lagged paths (i.e., T1 ER to T2 ADHD). These are alternative scenarios that future studies needs to examine in greater depth to provide an even more accurate estimation of the temporal relationships between these key variables.

Despite these limitations, the findings from this study have important clinical implications. In particular, our results support the importance of assessing ER in youth with even subclinical levels of ADHD, since risk for difficulties with ER and depressive symptoms do not appear to be limited to youth with ADHD diagnoses. Given that depression has an estimated prevalence of 2 % in children and 4 %–8 % in adolescents (Avenevoli et al. 2008; Birmaher et al. 1996; Kessler et al. 2001; Shaffer et al. 1996), and that approximately 15 %–20 %

of youth will experience a depressive episode by mid-adolescence (Birmaher et al. 1996; Lewinsohn et al. 1993), additional efforts targeting the prevention of depression are needed. Providing all children, particularly those with elevated levels of inattention and hyperactivity/impulsivity, and perhaps their parents, with psychoeducation or more intensive treatment focused on the identification and regulation of emotions appears critical in order to reduce the number of youth with clinical levels of depression (Southam-Gerow et al. 2012). One promising intervention is a modified version of Parent–child Interaction Therapy which focuses on Emotion Development (PCIT-ED). In a randomized clinical trial, PCIT-ED was more effective that psychoeduction for improving executive functioning and emotion recognition skills in preschoolers with depression (Luby et al. 2012). Current efforts are underway to modify PCIT-ED for youth with ADHD (Lewis-Morrarty et al. 2013). Furthermore, when clinicians and schools screen for symptoms of psychopathology in youth, it appears important to assess ER abilities as well.

In conclusion, the results of the present study advance the literature implicating deficits in ER as an important factor in the development of mood problems in youth with ADHD symptoms. Investigating the temporal relationship between ADHD, ER and depressive symptoms in youth holds the potential to advance prevention efforts to reduce the development of depression in at-risk youth.

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