2018

DOES EMOTION REGULATION MODERATE THE ASSOCIATION BETWEEN IMPAIRMENT AND DEPRESSION IN ADOLESCENTS WITH ADHD?

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DOES EMOTION REGULATION MODERATE THE ASSOCIATION BETWEEN IMPAIRMENT AND DEPRESSION IN ADOLESCENTS WITH ADHD?

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

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Master of Arts, Clinical Psychology
Appalachian State University, 2013

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Virginia Commonwealth University
Richmond, Virginia
September, 2016
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Abstract

DOES EMOTION REGULATION MODERATE THE ASSOCIATION BETWEEN IMPAIRMENT AND DEPRESSION IN ADOLESCENTS WITH ADHD?

By Laura D. Eddy, M.A.

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at Virginia Commonwealth University

Virginia Commonwealth University, 2018.

Major Directors: Joshua M. Langberg, Ph.D., Associate Professor of Psychology, Department of Psychology and Heather A. Jones, Ph.D., Assistant Professor of Psychology, Department of Psychology

In comparison with their peers, adolescents with ADHD are at increased risk for developing depression, with prevalence estimates for comorbid depression ranging from 14% to 20%. Youth with comorbid ADHD and depression are at greater risk for multiple negative outcomes compared to youth with ADHD alone, including suspension from school, failing a grade, or difficulties in peer relationships. Identifying risk factors for depression among adolescents with ADHD is important for facilitating early identification and treatment efforts. Research completed to date indicates that interpersonal impairment and emotion regulation may mediate the relationship between ADHD and depression. However, a comprehensive longitudinal model including both interpersonal impairment and emotion regulation has not been tested. The aim of the current study was to evaluate whether the indirect pathway from ADHD to depression through interpersonal impairment is moderated by emotion regulation in a longitudinal study of young adolescents with ADHD (N = 239; M age at follow-up = 12.30, SD = .92). Data were collected at three time points over 18 months. Parents completed ratings of
externalizing symptoms (ADHD and Oppositional Defiant Disorder, [ODD]), parent-child relationship problems, and peer relationship problems. Adolescents rated their depressive symptoms and emotion regulation problems. Moderated mediation models as outlined by Preacher, Rucker, and Hayes (2007) were used to test whether the indirect effects of externalizing symptoms on depression through parent-child relationship stress and peer relationship problems are moderated by emotion regulation. Controlling for baseline levels of depression, results suggest that these pathways vary as a function of emotion regulation, such that the paths from externalizing symptoms to depression are significantly stronger among adolescents with high levels of emotion regulation problems. In addition, an exploratory analysis of the associations between multiple aspects of emotion regulation problems and depression revealed that access to emotion regulation strategies was the only unique predictor of later depression among adolescents with ADHD.
MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD

Does Emotion Regulation Moderate the Association between Impairment and Depression in Adolescents with ADHD?

Attention-deficit/hyperactivity disorder (ADHD) is a neurobiological condition characterized by symptoms of inattention and/or hyperactivity and impulsivity (American Psychiatric Association [APA], 2013). Children who meet criteria for a diagnosis of ADHD demonstrate the presence of six or more symptoms of inattention (e.g., difficulty sustaining attention, careless mistakes, difficulty organizing tasks and activities) and/or six or more symptoms of hyperactivity/impulsivity (e.g., often fidgets, talks excessively, has trouble playing quietly). Moreover, these symptoms must be present before age 12, appear in two or more settings, and have a negative impact on functioning (APA, 2013).

Children with ADHD typically demonstrate impairment in multiple domains of functioning, including academics and interpersonal relationships (Caci et al., 2014). At school, children with ADHD score lower on standardized tests (Frazier et al., 2007; McConaughy, Volpe, Antshel, Gordon, and Eiraldi, 2011) and are more likely to be required to repeat a grade (Fried et al., 2016; Loe and Feldman, 2007) in comparison to their peers. With respect to peer relationships, children with ADHD are rated by their parents and teachers as less socially competent and more socially impaired than their peers (McConaughy et al., 2011; Solanto, Pope-Boyd, Tryon, & Stepak, 2009). Moreover, they tend to have fewer friends overall (Bagwell, Molina, Pelham, & Hoza, 2001), are less likely to have reciprocal friendships (Hoza et al., 2005), and experience higher rates of victimization and peer rejection (Wiener & Mak, 2009).

Impairment in interpersonal relationships also extends to the family. For example, lower levels of parent-child attachment are observed in families of children with ADHD (Bauermeister, 2007) and parents of children with ADHD report more parenting stress and lower levels of parenting.
satisfaction relative to parents of undiagnosed children (Lange et al., 2005; Theule, Wiener, Tannock and Jenkins, 2013). Families of children with ADHD also report more overall stress and conflict (Biederman et al. 1999; DuPaul et al 2001), as well as higher perceived family burden relative to families of children without ADHD (Bauermeister, 2007).

Importantly, symptoms and impairment associated with ADHD persist over time, and approximately 70% of children diagnosed with ADHD continue to meet diagnostic criteria in adolescence (August, Braswell, & Thuras, 1998; Barkley, Fischer, Edelbrock & Smallish, 1990; Hinshaw, Owens, Sami & Fargeon, 2006). Adolescents with ADHD continue to perform lower on standardized tests of achievement (Frazier et al., 2007; Molina et al., 2009), have fewer friends, and experience higher levels of peer rejection (Bagwell, 2001). Moreover, youth diagnosed with ADHD in childhood are less likely to be rated as socially well-adjusted in adolescence, even when their ADHD symptoms have improved (Lee, Lahey, Owens, & Hinshaw, 2008).

**Comorbid Depression**

In addition to the range of impairments described above, youth with ADHD are also at a higher risk of developing depression relative to their peers, with prevalence estimates for comorbid depression with ADHD ranging from 14-20% (Biederman et al., 1992; Chen et al., 2013; Elia et al., 2008; Larson, Russ, Kahn, & Halfon, 2011). Importantly, this association is not epiphenomenal, meaning the high rates of depression observed among youth with ADHD cannot be fully attributed to the general effects of a chronic psychiatric condition (Blackman, Ostrander, & Herman, 2005) or to overlapping symptom criteria (e.g., concentration difficulties; Biederman et al., 1995). In addition, ADHD symptom severity does not directly predict the occurrence of depression in adolescents (Daviss, Diler, & Birmaher, 2009), suggesting that the relationship
between ADHD and depression is indirect, or may be due to a combination of genetic and environmental risk factors. The limited research completed to date points to a common genetic vulnerability underlying both major depressive disorder and ADHD which may or may not be expressed, depending on environmental factors (Biederman et al., 1992; Pliska, 1998).

Although the exact causes of elevated rates of depression in this population are still unknown, it is clear that youth with comorbid ADHD and depression experience significantly more impairment than youth with ADHD alone (Blackman et al., 2005). For example, youth with ADHD and comorbid depression have an even higher likelihood of experiencing significant negative life events, such as suspension from school and failing a grade (Daviss & Diler, 2012). In addition, youth with ADHD experience an earlier onset of depression, longer depressive episodes, and more severe impairment associated with depression compared to peers without ADHD who are diagnosed with depression (Biederman et al., 2008). Perhaps of greatest concern, adolescents with ADHD report more suicidal ideation and self-harm behaviors compared to adolescents without ADHD (Chronis-Tuscano et al., 2010) even when controlling for the effects of comorbid depression (Hurtig, Taanila, Moilanen, Nordström, & Ebeling, 2012). Interestingly, rates of depression among young adolescents with ADHD do not significantly differ from rates of depression among young adolescents without ADHD (Bagwell, Molina, Kashdan, Pelham & Hoza, 2006; Ruchkin, Lorberg, Kozosov, Schwab-Stone, Sukhodolsky, 2008). However, by late adolescence and early adulthood, significantly higher levels of depressive symptoms are present among individuals with ADHD (Rabiner, Anastopoulos, Costello, Hoyle, & Swartzwelder, 2008). This increase in depressive symptoms suggests certain factors may be present during the early adolescent period that contribute to the development of depressive symptoms.
Predictors of depression in ADHD. The identification of youth with ADHD who are at greatest risk for developing symptoms of depression is important, as early identification could aid in prevention and treatment efforts. However, there is still much debate about what factors increase risk for depression among youth with ADHD. A review of the limited research on co-occurring ADHD and depression concluded that a history of impairment in multiple domains of functioning may be an important factor in understanding the etiology of depression in the context of ADHD (Daviss, 2008). This parallels the cognitive-behavioral model of ADHD proposed by Safren, Sprich, Chulvick and Otto (2004) which links the deficits characteristic of ADHD to symptoms of depression and anxiety (see Figure 1).

![Figure 1. A Cognitive-Behavioral Model of ADHD.](image)

According to this model, the neurobiological impairments inherent to ADHD (e.g., inattention, inhibition and self-regulation) lead to functional impairment and difficulties in multiple life domains. As a result, individuals with ADHD develop negative cognitions about
their own competence, thereby increasing their risk for comorbid internalizing symptoms and disorders. In fact, there is some evidence that adults with a diagnosis of ADHD and depression display more negative cognitions than adults with a diagnosis of ADHD only (Mitchell, Benson, Knouse, Kimbrel, & Anastopoulos, 2013). While this model provides a framework for understanding why rates of comorbid depression may increase over time, it does not emphasize any particular area of impairment.

**Interpersonal Impairment**

One area of impairment which may be particularly relevant to the development of depression is interpersonal impairment. Indeed, the interpersonal theory of depression suggests that difficulties in interpersonal processes (interpersonal impairment) are central to the development of depression (for a review, see Hames, Hagan & Joiner, 2013). Interpersonal impairment, including relationships with peers, parents, and other family members, plays an important role in the development of depression among adolescents (Katz et al., 2011; Hammen et al., 2008). For example, Katz and colleagues (2011) demonstrated that impairment in peer relationships mediated the relationship between social withdrawal in childhood and depression in adolescence in a longitudinal sample followed from birth to age 20 (N = 702). Family relationship impairment is also highly associated with depression in adolescents (O' Shea, Spence, & Donovan, 2014). In particular, conflict in the parent-child relationship seems to have a reciprocal relationship with depressive symptoms in adolescence, such that conflict predicts later depressive symptoms, and these symptoms in turn predict future conflict (Brière, Archambault, & Janosz, 2012). As noted above, families of children with ADHD report higher levels of parent-child conflict and parent-child relationship problems (Pressman et al., 2006), and children with ADHD often demonstrate peer relationship problems, such as fewer close friendships and
MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD

higher levels of peer rejection (Bagwell et al., 2001). Moreover, parent-child relationship problems, conflict and peer relationship problems occur at particularly high rates among children with ADHD who also display comorbid symptoms of Oppositional Defiant Disorder (ODD; Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001; Li et al., 2014). ODD is a common comorbid condition in children with ADHD, with prevalence rates as high as 50 to 70% (Biederman et al., 2008; Costello et al., 2003).

Interpersonal impairment in ADHD. Given that interpersonal impairment is common among adolescents with ADHD (Sibley, Evans, & Serpell, 2010), it seems likely that this may serve as a mechanism which increases risk for the development of comorbid depression. Currently, there is some evidence which supports this hypothesis. The strongest evidence is provided by Humphreys and colleagues (2013), who utilized two samples (one cross-sectional and one longitudinal) to investigate pathways from symptoms of ADHD to depression. The authors used structural equation modeling to examine the paths from ADHD symptom domains (inattention and hyperactivity) to depression with a cross-sectional sample of children (including children diagnosed with ADHD and non-diagnosed children) between the ages of 5 and 10 years old ($N = 230, M_{age} = 7.39$). Multiple mediation pathways were tested, including parent-child relationship problems, peer relationship problems, and academic achievement. Results indicated that pathways between inattention and depression were significantly mediated by parent-child relationship problems and peer problems but not academic impairment. Paths from hyperactivity to depression were not significant alone or mediated by any of the domains of impairment, suggesting that inattention is a more important predictor of depression. Next, Humphreys and colleagues used a longitudinal community sample ($N = 472$) selected from a larger birth cohort study. Data were collected from participants at ages 5, 15, and 20. Structural equation modeling
was used to assess paths from childhood attention problems (inattention) to depression at age 20, through peer problems, academic problems, and parent-child relationship problems at age 15. In this sample, the indirect path to depression through parent-child relationship problems was significant (point estimate = .07, SE = .03, p < .05) and the indirect path through peer problems approached significance (point estimate = .03, SE = .01, p = .08). The path through academic problems was not significant. These findings suggest difficulties with peer and parent-child relationships are important in understanding the association between ADHD and depression.

The findings reported by Humphreys et al. (2013) represent an important first step in understanding the relationship between ADHD and depression, but an important question remains unanswered. The prevalence of interpersonal impairment is quite common in youth with ADHD, with 80% of children with ADHD displaying peer rejection scores one standard deviation or more above the mean (MTA Cooperative Group, 1999; Pelham & Bender, 1982) and between 56% and 76% having no mutual friendships (see Hoza, 2007 for a review). When family functioning and parent-child relationship problems are considered, the degree of interpersonal impairment is even more striking. For example, in a study of family interactions in adolescents with ADHD and their parents (n = 87 families), the families of children with ADHD displayed two times or more the level of conflict reported by families in the control group (n = 32) according to the adolescent, mother and father reports on the Conflict Behavior Questionnaire (CBQ; Prinz, Foster, Kent & O’Leary, 1979; Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001). In addition, parents of children with ADHD report higher levels of parenting stress (Theule et al., 2013), with some studies finding that parents report parenting stress up to two standard deviations higher on the Parenting Stress Inventory (PSI; Loyd &Abidin, 1985) compared to control families (Mash & Johnston, 1983). These findings imply the vast majority
of adolescents with ADHD experience significant interpersonal impairment with peers and families, yet rates of depression in this population are far more limited (14-20%; Biederman et al., 1992; Chen et al., 2013; Elia et al., 2008; Larson et al., 2011). The heterogeneity in the development of depression among adolescents with ADHD begs the question of whether other variables may play a role in determining under what conditions interpersonal impairment leads to depression. One potentially important moderating factor is emotion regulation.

**Emotion Regulation**

Multiple definitions of emotion regulation have been presented (e.g. Cole et al., 2004; Gottman & Katz, 1989; Thompson, 1994), and there is no single agreed-upon definition. However, consensus within the field suggests that emotion regulation involves both intrinsic and extrinsic processes which serve the function of monitoring and modifying emotions. The definition provided by Thompson (1994) is frequently cited, and states, “emotion regulation consists of the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one’s goals” (Thompson, 1994, p. 27-28). More recent reviews of the emotion regulation literature have refined and expanded upon this definition in three ways (e.g., Cole et al., 2004; Southam-Gerow & Kendall, 2002). First, it is important to note that emotion can be viewed as both a regulator of behavior and something which is itself subject to regulation (Southam-Gerow & Kendall, 2002). In other words, emotion regulation must be understood as more than the process of managing one’s emotions, since it may also involve the mobilization of emotion to organize behavior. For instance, a student who feels increased feelings of anxiety at the prospect of an upcoming exam may choose to begin studying earlier because of their emotional experience. Second, emotion regulation involves the monitoring, evaluation, and modification of
one’s own emotions in a goal-oriented process. This emphasis on goal accomplishment underscores the functionality of emotion regulation and is important for understanding how deficits in emotion regulation lead to impairment. Finally, emotion regulation is not tantamount to emotional restraint, as the process of increasing emotions may be appropriate and adaptive in some circumstances (e.g., displaying excitement in response to a spouse’s promotion at work; Cole et al., 2004; Southam-Gerow & Kendall, 2004). Hence, emotion regulation consists of several processes and is therefore best understood as a multidimensional construct.

Reflecting this understanding of emotion regulation, many self and parent report measures of emotion regulation include subscales tapping several distinct processes. For example, the Deficits in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) includes six subscales (e.g., lack of emotional awareness, limited access to emotion regulation strategies) and the Emotion Regulation Index for Children and Adolescents (ERICA, MacDermott et al., 2010) includes three (Emotional Control, Emotional Self-Awareness and Situational Responsiveness). The multiple aspects or processes involved in emotion regulation may increase risk for psychopathology through different mechanisms. For example, increased use of strategies such as rumination, avoidance (either behavioral or experiential) and suppression (either suppression of thoughts or suppression of emotions) is considered less adaptive and a risk factor for increased psychopathology. Increased use of thought suppression results in increased emotional arousal (Wegner, Broome & Blumberg, 1997), and higher levels of rumination appear to interfere with problem solving (Ward, Lyubomirsky, Sousa, & Nolen-Hoeksema, 2003). In contrast, other emotion regulation processes are considered more adaptive, such as problem solving, reappraisal and acceptance. Problem solving in this context refers to modulating emotions by taking steps to change a problematic situation or manage its consequences. Reappraisal refers to the use of
reframing or generating positive cognitions to manage painful situations, and acceptance within the process of emotion regulation has been defined as nonjudgmental acceptance of one’s emotions. In fact, these processes are commonly emphasized in cognitive-behavioral and mindfulness-based approaches to treating psychopathology. These various aspects of emotion regulation appear to be differentially related to various types of psychopathology, as indicated by a recent meta-analysis (Aldao, Nolen-Hoeksema, & Schweizer, 2010) which found strong positive relationships between depression and rumination and suppression and avoidance as well as a strong negative relationship between problem solving and depression. Interestingly, reappraisal and acceptance, which are theorized to be negatively related to depressive symptoms and are typically targeted in cognitive-behavioral treatments, were not significantly associated with depression (Aldao et al., 2010).

**Emotion regulation and ADHD.** Although difficulties with emotion regulation are a transdiagnostic phenomenon, they have long been associated with ADHD. Early conceptualizations of ADHD included struggles with emotion regulation as a key feature of the disorder; however, this focus on emotion regulation was omitted from later definitions starting in the late 1960’s (see Barkley, 2015 for a review of the history of emotion regulation in the conceptualization of ADHD). Recently there has been a renewed interest in emotion regulation difficulties among children with ADHD (see Bunford, Evans & Wymbs, 2015). Given that theories of ADHD conceptualize the disorder as primarily a disorder of inhibition (Barkley, 1997), it follows that children with ADHD may also have difficulties with the regulation of emotion, since regulation often begins with the inhibition of a response. However, youth with ADHD may struggle with other aspects of emotion regulation aside from inhibition-based processes. For instance, there is some evidence that children with ADHD display difficulties
with accurate emotional recognition (Kats-Gold et al., 2007; Singh et al. 1998) and display problems with empathetic responding to both positive and negative emotions displayed by others (Braaten & Rosen, 2000). Importantly, emotion regulation difficulties are particularly likely to occur in children with ADHD who are also diagnosed with a comorbid externalizing disorder such as ODD (Factor, Rosen, & Reyes); however, there is compelling evidence that emotion regulation difficulties are not attributable solely to the presence of ODD (Sobanski et al, 2010).

The strongest evidence for emotion regulation difficulties in the context of ADHD is provided by a recent meta-analysis, which investigated associations between ADHD and several different dimensions of emotional functioning in children and adolescents (Graziano & Garcia, 2016), including emotion recognition/understanding, emotional reactivity/lability, emotion regulation, and empathy/callous and unemotional traits. Graziano and Garcia (2016) found that the strongest association across studies was the association between ADHD and emotional reactivity/lability ($d = .95$), followed by ADHD and emotion regulation ($d = .80$; Graziano & Garcia, 2016). Importantly, these findings held when controlling for conduct problems as a moderator, suggesting that these struggles cannot be attributed solely to the overlap between ADHD and co-occurring oppositional or conduct symptoms. Emotional reactivity in this study was conceptualized as the threshold, intensity and duration of affective arousal, which could occur in the context of either negative or positive emotions. Graziano and Garcia (2016) note that the terms “emotional lability” and “emotional impulsivity” are often used to refer to the same construct. These findings suggest that youth with ADHD are more likely than their peers to experience strong positive or negative emotional reactions and are more “reactive” in the context of frustrating situations. Importantly, some definitions of emotion regulation include these processes within the definition of emotion regulation (e.g., the recognition of emotion as a
regulated phenomenon; Southam-Gerow & Kendall, 2002). Despite the evidence for associations between emotion regulation problems and ADHD, prevalence estimates suggest that only 35% of young adolescents with ADHD demonstrate emotion regulation difficulties markedly higher (one to two standard deviations) than mean scores reported in other populations (Bunford et al., 2014). Clearly, not all adolescents with ADHD demonstrate notable difficulties with emotion regulation. This raises the possibility that the presence or absence of emotion regulation problems may be important in understanding the development of comorbid internalizing symptoms in adolescents with ADHD.

**Emotion regulation and internalizing symptoms.** Chronic struggles with emotion regulation are associated with the development of internalizing symptoms (Garnefski, Kraaij, & van Etten, 2005; Silk & Steinberg, 2003). For example, emotion regulation mediates the pathway between stressful life events and internalizing symptoms (both depression and anxiety) among adolescents (McLaughlin, & Hatzenbuehler, 2009). Furthermore, some studies have investigated emotion regulation as a potential mediator of the relationship between ADHD and depression (Anastopoulos et al., 2011; Seymour et al., 2012; Seymour et al., 2014). The most rigorous evidence is provided by Seymour and colleagues (2014), who found that emotion regulation mediated the association between ADHD symptoms and depression, using a longitudinal sample of children between the ages of 9 and 12 ($N = 277; M$ age = 11). In addition, two cross-sectional studies have found evidence that emotion regulation mediates the link between ADHD and depression (Anastopoulos et al., 2011; Seymour et al., 2012). These findings suggest that emotion regulation may play a role in the relationship between ADHD and depression in addition to interpersonal impairment. Adolescents who have difficulty modulating their emotional response may struggle to cope with stressful or adverse events, such as difficulties in their
relationships with peers or family. Thus, it may be that difficulties with emotion regulation interact with interpersonal impairment to increase risk for depression in adolescents with ADHD. Furthermore, this process may be bidirectional, such that emotional regulation in turn will contribute to higher levels of conflict and stress within the parent-child relationship. For example, for adolescents who experience frequent conflict with parents, an inability to regulate their reactions and behavior when upset will likely lead to even more conflict and interpersonal impairment. This may also explain why not all adolescents with ADHD develop depression. Specifically, it may be that only adolescents with ADHD who struggle to monitor, manage, and process the negative emotions that stem from interpersonal impairment develop depressive symptoms. By examining whether the interaction between interpersonal impairment and difficulties with emotion regulation predicts increased depressive symptoms among children with ADHD, it may be possible to identify children with the highest risk of developing a depressive disorder.

To date, the influence of interpersonal impairment and emotion regulation on the development of depression has been studied separately in this population, and there has been no investigation of whether these variables interact in meaningful ways to increase risk for depression. Emotion regulation may act as a moderator, determining under what conditions the indirect pathway from ADHD to depression through interpersonal impairment is strongest. To address these questions statistically, it is necessary to assess conditional indirect effects, also referred to as moderated mediation effects (Preacher, Rucker & Hayes, 2007; see Figure 2). Furthermore, although emotion regulation is a multidimensional construct, most prior research testing pathways between emotion regulation and depression in youth with ADHD has treated emotion regulation as a unitary construct and has not explored whether specific aspects of
emotion regulation are particularly important in understanding the risk for developing depression (e.g., Seymour et al., 2014). Additionally, past studies assessing mediation relationships between ADHD and depression have primarily used childhood samples (Anastopoulos et al., 2011; Humphreys et al., 2013; Ostrander et al. 2006; Seymour et al., 2014). Since the prevalence of depression increases during adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003), it is important to build on the current literature by evaluating these associations in young adolescent samples. Adolescence is also a time associated with greater disruptions in the parent-child relationship (Pasley and Gecas, 1984), which Humphreys et al. (2013) identified as the single most important mediating variable in the path from ADHD to depression. For example, adolescence is the most difficult stage in terms of parenting (Pasley and Gecas, 1984) and is associated with decreases in parent-child closeness (Steinberg, 1988) and increases in parent-child conflict (Steinberg, 1988).

**Present Study**

The primary aim of the current study was to test a longitudinal moderated mediation model of the association between externalizing symptoms (ADHD and ODD) and depression through interpersonal impairment, with emotion regulation serving as a moderator of this indirect pathway. Given the findings from Humphreys et al. (2013), pathways to depression through impairment in parent-child relationships and peer relationships were evaluated. This model was tested using a sample of young adolescents comprehensively diagnosed with ADHD, and multiple informants were used to minimize within-source bias (Spector & Brannick, 2009). Specifically, consistent with best-practice recommendations, depressive symptoms were measured through adolescent self-report (Moretti, Fine, Haley, & Marriage, 1985) and ADHD symptoms, ODD symptoms, and interpersonal impairment were measured by parent-report
MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD

(Sibley et al., 2012). Emotion regulation symptoms were also measured by adolescent self-report.

I predicted that the indirect pathways from externalizing symptoms to depression through parent-child relationship problems and peer relationship problems would be moderated by emotion regulation, such that this pathway would be significantly stronger for youth with high levels of emotion regulation difficulties compared to youth with low levels of emotion regulation difficulties. Finally, the relationships between specific facets of emotion regulation difficulties and depression among youth with ADHD were examined to assess whether certain facets were more strongly related to the development of depression in children with ADHD. In line with past findings linking three aspects of emotion regulation to internalizing symptoms in adolescents (Neumann, Lier, Gratz, & Koot, 2009), I hypothesized that lack of emotional clarity, non-acceptance of emotional responses, and limited access to emotion regulation strategies would most strongly relate to depression.

Method

Participants

Participants included 239 adolescents between the ages of 10 and 14 years old, who participated in a larger randomized controlled trial ($N = 326$). The sample for this study included participants from two cohorts of the trial, who completed a measure of emotion regulation. This sample is 73.2% Male and 91.7% Non-Hispanic. The average income level for the sample was $58,735 ($SD = 49,313$). Participants identified as White (79.9%), Black (10%), Biracial (7.9%) and Other (2.2%). A summary of this information as well as additional demographic information including intelligence, achievement scores, and parental education level is provided in Table 1.
Participants for the trial were recruited from seven middle schools in the Midwestern United States. See Table 1 for additional participant demographics.

Table 1.

**Participant Characteristics and Correlations with Model Variables**

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
<th>Mean (SD)</th>
<th>Depression</th>
<th>Parent-Child Relationship Problems</th>
<th>Peer Relationship Problems</th>
<th>Emotion Regulation Difficulties</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>175 (73.2%)</td>
<td>.04</td>
<td>.06</td>
<td>.004</td>
<td>.11</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>64 (26.8%)</td>
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<td></td>
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</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>191 (79.9%)</td>
<td>.01</td>
<td>.08</td>
<td>.09</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>24 (10%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biracial</td>
<td>19 (7.9%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>4 (2.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Hispanic</td>
<td>217 (91.7%)</td>
<td>-.034</td>
<td>-.005</td>
<td>-.096</td>
<td>-.061</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>8 (3.1%)</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>13 (5.2%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Medication</strong></td>
<td>170 (71.1%)</td>
<td>-.034</td>
<td>-.005</td>
<td>-.096</td>
<td>-.061</td>
<td></td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td>12.30 (.92)</td>
<td>-.047</td>
<td>-.009</td>
<td>-.04</td>
<td>-.06</td>
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</tr>
<tr>
<td><strong>IQ</strong></td>
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<td>-.02</td>
<td>-.06</td>
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<td>.02</td>
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<td><strong>Reading</strong></td>
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<td>-.03</td>
<td>-.002</td>
<td>.003</td>
<td>-.02</td>
<td></td>
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<tr>
<td><strong>Math</strong></td>
<td>92.38 (16.67)</td>
<td>-.16</td>
<td>-.06</td>
<td>-.01</td>
<td>-.02</td>
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</tr>
<tr>
<td><strong>Maternal Education</strong></td>
<td>14.05 (2.28)</td>
<td>-.04</td>
<td>-.12</td>
<td>-.01</td>
<td>-.01</td>
<td></td>
</tr>
<tr>
<td><strong>Paternal Education</strong></td>
<td>13.66(2.5)</td>
<td>.03</td>
<td>-.04</td>
<td>-.04</td>
<td>-.04</td>
<td></td>
</tr>
</tbody>
</table>

Note. *p < .05, **p < .01, ***p < .001. Medication refers to use of medication to treat ADHD at Time One. Reading was assessed by the Wechsler Individual Achievement Test, Third Edition (WIAT-III) Basic Reading composite score, Math achievement was assessed by the WIAT-III Basic Math composite score. Full Scale IQ scores (IQ) were measured using four subtests of the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV). Mother Education and Father Education are reported in years. Age is reported in years.
Procedures

Study procedures were approved by the Institutional Review Boards (IRB) of the universities where data were collected. The goal of the parent study was to evaluate the efficacy of two types of school-based intervention for young adolescents with ADHD as compared to a community control condition. Participants were recruited by way of study announcement letters, flyers, and direct referrals from school staff during the spring semester prior to receiving the intervention the following semester. Eligible participants were randomly assigned to one of three treatment conditions and received the intervention over the course of one academic year. The first treatment condition (Challenging Horizons Program, After School; CHP-AS) was a school-based intervention (delivered as an afterschool program) which targeted organization, planning, study skills as well as personal social goals, delivered using a mix of group and individual meetings. This program was delivered by trained study staff (graduate and undergraduate student counselors) two days per week over the course of one academic year (September to May). The second treatment condition (Challenging Horizons Program, Mentoring; CHP-M) involved the delivery of the same treatment via weekly individual meetings with a teacher or other staff member at the participants’ school (the “mentor”). The assigned mentors were trained to deliver the intervention and were supervised by study staff. The third condition (community control) received no intervention but were provided with a list of potential resources in the community at the beginning of the academic year. The percentage of participants in the present study who received the CHP-AS and CHP-M was 33.1% and 36%, respectively; and the percentage of participants in the community control condition was 30.9%. Participants completed the measures described below at the baseline study eligibility visit, at the end of the school year (9 months later) and 18-months post-baseline.
Potential participants underwent a phone eligibility screening and a diagnostic evaluation to determine eligibility. Interested parents who contacted study personnel were provided with a comprehensive description of the study and asked to complete an eligibility screen regarding their children; those who endorsed the presence of at least four of nine DSM-IV-TR symptoms of inattention and/or a prior diagnosis of ADHD were scheduled for an evaluation. Children were eligible to participate in the study if they (a) attended a participating school; (b) met full DSM-IV-TR diagnostic criteria for ADHD-Predominantly Inattentive Type or ADHD-Combined Type on the Children’s Interview for Psychiatric Syndromes, Parent Version (P-ChIPS; Weller, Weller, Fristad, Rooney, & Schecter, 2000), or by supplemental teacher ratings on the Disruptive Behavior Disorders Rating Scale (DBD; Pelham, Gnagy, Greenslade, & Milich, 1992) in conjunction with P-ChIPS report; (c) showed impairment on the Impairment Rating Scale (IRS; scores ≥ 3 demonstrate impairment; Fabiano et al., 2006); and (d) had an estimated IQ of at least 80 as assessed using the Wechsler Intelligence Scale for Children – Fourth Edition (WISC-IV; Wechsler, 2003). To meet full diagnostic criteria for ADHD, at least six inattentive or hyperactive/impulsive symptoms had to be endorsed at clinically significant levels by caregivers on the P-ChIPS. These symptoms had to be associated with impairment in multiple domains and age of onset had to be during elementary school. If a parent endorsed fewer than six symptoms, non-overlapping teacher endorsed ADHD symptoms were used to supplement parent ratings as long as the parent endorsed at least 4 symptoms within a domain. Two doctoral-level psychologists reviewed assessment data to determine eligibility status and diagnoses. Children were ineligible if they met diagnostic criteria for a pervasive developmental disorder, bipolar disorder, psychosis, or obsessive-compulsive disorder on the P-ChIPS. The Parent Children’s Interview for Psychiatric Syndromes, Parent Version (P-ChIPS; Weller, et al., 2000), is a

Measures

Measures in this study included assessments of externalizing symptoms (ADHD and ODD) at Time 1, depression at Time 1 and Time 3, emotion regulation at Time 2, and interpersonal impairment at Time 2 (impairment in parent-child relationship and impairment in peer relationships).

Externalizing symptoms. The Disruptive Behavior Disorders Rating Scale (DBD; Pelham et al., 1992) is a 45-item parent-completed symptom scale which assesses for symptoms of ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD). The wording of the items closely corresponds to the symptom criteria for ADHD, ODD, and CD, as included in the DSM-III-R (APA, 1987). Respondents indicate the frequency with which a child or adolescent displays a certain behavior using a 4-point scale, with 0 indicating “not at all” and 3 indicating “very much”. Higher scores indicate greater symptomatology. This scale has demonstrated adequate to strong internal consistency (α = .96; α = .95, and α = .75 for ADHD, ODD and CD scales, respectively). The DBD rating scale has been validated using an adolescent sample (Van Eck et al., 2010). For the current study, a composite score of externalizing symptoms was created by summing the scores from the ADHD symptoms scale and the ODD symptoms scale of the DBD. Scores for the ADHD scale may range from 0 to 54 and scores for the ODD scale may range from 0 to 24; accordingly, scores for the composite score could range from 0 to 78.
Internal consistency for the current sample was strong ($\alpha = .93$ and $\alpha = .94$ for ADHD and ODD scales, respectively, and $\alpha = .93$ for the combined externalizing symptom score scale).

**Emotion regulation.** The Deficits in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item self-report measure of emotion regulation difficulties. This measure allows for the calculation of a total overall score as well as six subscale scores (Lack of Emotional Awareness, Nonacceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Limited Access to Emotion Regulation Strategies, and Lack of Emotional Clarity). The total score for the DERS ranges from 36 to 180. The DERS total score has demonstrated strong evidence for test-retest reliability ($\rho_1 = .88$; with re-administration time ranging from four to eight weeks; Gratz & Roemer, 2004). Test-retest reliability for the DERS subscales was adequate, with Spearman’s rho coefficients ranging from .57 to .89 (Gratz & Roemer, 2004). The DERS total score has demonstrated evidence for internal consistency ($\alpha = .93$) and Cronbach’s alphas for the subscales scores are all above .80 (Gratz & Roemer, 2004). Internal consistency for the current sample was also strong ($\alpha = .91$ for the total score).

**Depression.** The Reynolds Adolescent Depression Scale, Second Edition (RADS-2; Reynolds, 1987) is a 30-item, standardized, self-report measure of the severity of depressive symptoms. The RADS-2 is appropriate for individuals ages 11 to 20 years old. Adolescents are prompted to rate how often a given depressive symptom occurs from 1 (almost never) to 4 (most of the time). Higher scores indicate higher levels of depressive symptoms. The RADS-2 has demonstrated evidence for internal consistency ($\alpha = .91$) and test-retest reliability (.87) in adolescent samples (Reynolds & Mazza, 1998). The RADS-2 generates a total scale, as well as four subscales scores (Dysphoric Mood, Anhedonia/Negative Affect, Negative Self-Evaluation,
and Somatic Complaints). Higher scores indicate more severe levels of depressive symptoms, and raw scores may range from 30 to 120. Scores are reported as $t$-scores. A clinical cut-off score of 77 ($t = 60$) is used. This cutoff score has correctly classified adolescents diagnosed with major depression (Reynolds, 1987). Internal consistency for the current sample was strong ($\alpha = .93$).

**Parent-Child Relationship Problems.** The Stress Index for Parents of Adolescents (SIPA; Sheras, Abidin, & Konold, 1998) is a 90-item parent-completed measure, which assesses stress in three domains: adolescent-related, parent-related and the adolescent-parent relationship. Parents respond to items using a 5-point Likert scale, ranging from “strongly disagree” to “strongly agree”. In the current study, only the Adolescent-Parent Relationship Domain (APRD) was used. This scale included 16 items and measured the perceived quality of the relationship parents have with their children, including items which assess degree of communication and amount of affection. Higher scores indicate more problems in the parent-child relationship. The APRD has demonstrated evidence for internal consistency across multiple studies ($\alpha$’s ranging from .81 to .91; Sheras et al., 1998; Weiner, Biondic, Grimbus, & Herbert, 2016; Wheatley & Whille, 2009) and evidence for test-retest reliability (ICC = .91; Sheras et al., 1998). Internal consistency was also strong in the current study ($\alpha = .91$).

**Peer-Relationship Problems.** The Weiss Functional Impairment Rating Scale – Parent Report (WFIRS-P; Weiss, Wasdell, & Bomben, 2004) is a fifty-item, parent-completed measure of ADHD-related impairment in six domains: Family, Learning and School, Life Skills, Child’s Self-Concept, Social Activities, and Risky Activities. Respondents are asked to use a 4-point scale (0=never, 3=very often) to rate the impact of their child’s ADHD and related symptoms on behaviors and activities in each of the domains listed above. The current study used the Social
Activities subscale, which contained seven items. Examples of items on the WFIRS-P Social Activities scale include “problems getting along with other children,” “problems keeping friends,” and “problems participating in afterschool activities.” A mean score was calculated for the Social Activities scale. The minimum score on this scale is 0 and the maximum score is 3. Although the original instructions for this scale suggest that a mean domain score of 1.5 or higher indicates clinically elevated impairment (Weiss et al., 2004), more recent work suggests that a mean score of .71 identifies the presence of clinically significant impairment (Thompson, Llyod, Joseph, & Weiss, 2017). The WFIRS-P has demonstrated adequate internal consistency, with α’s exceeding .70 in all domains (Gajria et al., 2015). The Social Activities subscale demonstrated evidence for strong internal consistency in previous studies (α = .86; Gajria et al., 2015) and the current study (α = .82).

Analytic Plan

Data Preparation

Examination of the data across all time points revealed that the amount of missing data across the variables ranged from 0 to 27%. The measures with the most missing data were the RADS-2 (Time 3) and the DERS (18% and 27%, respectively). The remaining variables only displayed 0 to 5% missing data. To assess the evidence for the assumption that data were missing at random, Little’s Missing Completely At Random (MCAR) test (Little, 1988) was performed. Little’s MCAR is a chi-square statistic used to assess whether the pattern of missing values is systematic, by assessing whether mean scores on variables of interest significantly differ when cases with missing data are included or excluded. If Little’s MCAR is significant, there is evidence to suggest that data are missing not at random (MNAR). The Little's MCAR test conducted for this data resulted in a chi-square = 57.508 (df = 53; p = .312), providing
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evidence for the assumption that data were missing at random. To address the missing data, multiple imputation was used to estimate values for these data points through SPSS. Multiple imputation combined with bootstrapping procedures (described below) is recommended to address missing data in the context of mediation analyses (Wu & Jia, 2013). This procedure has been found to produce comparable results to full information maximum likelihood when data is missing at random (Wu & Jia, 2013). According to the guidelines proposed by Graham, Olchoski, and Gilreath (2007), 20 imputations is an appropriate number for analyses using data in which 10 to 30% of cases are missing.

The data used in this study were collected during a randomized controlled trial evaluating the efficacy of a school-based intervention. Previous investigations using these data found no significant intervention effects with regard to depression (Bunford et al., 2015) and no significant intervention effects for interpersonal impairment (Evans et al., 2016). Furthermore, the three treatment groups did not differ on emotion regulation problems as assessed by the subscales or the total score (Bunford et al., 2015). To further corroborate these findings, three mixed two-way analysis of variance (ANOVA’s) were conducted, to test whether group assignment had a significant effect across time with regard to mediating or outcome variables (parent-child relationship problems, peer relationship problems, or depression). Results suggested that there was no significant effect of time by condition on peer relationship problems, \(F[2, 316] = 1.074, p = .343\). Mauchly's Test of Sphericity indicated that the assumption of sphericity had been violated for the analyses testing the effects of condition by time on depression \(\chi^2[2] = 70.27, p < .001\) and parent child relationship problems, \(\chi^2[2] = 7.763, p = .02\), and therefore, a Greenhouse-Geisser correction was applied. There was no significant effect of condition by time on depression, \(F[1.76, 282.75] = .179, p = .808\) or parent-child relationship problems, \(F[2, 316] = 1.074, p = .343\).
[1.907, 299.46] = .162, p = 850). As a result, it was deemed unnecessary to enter intervention condition as a covariate in the model.

Data were checked for violations of assumptions of normality. The assumptions of normality were checked by examining Shapiro-Wilk and Kolmogorov-Smirnov tests and p-values, skewness, and kurtosis values for each variable. Significant values of the Shapiro-Wilk and Kolmogorov-Smirnov tests provide evidence that variables may not demonstrate a normal distribution, and skewness and kurtosis values are indicative of the degree of non-normality for each variable. Skewness and kurtosis values that exceed ±2 are generally considered to be problematic (Field, 2009). An examination of the Shapiro-Wilk and Kolmogorov-Smirnov tests as well as skewness and kurtosis values for each variable indicated that only two variables displayed problematic levels of non-normality, as evidenced by kurtosis values that exceeded ±2 (DERS total score, kurtosis = 2.27; DERS Non-Acceptance of Emotional Responses, kurtosis = 2.16). Considering this level of non-normality, bootstrapping procedures were used to estimate the mediation and moderated mediation models. Furthermore, to assess the relationships between aspects of emotion regulation and depression, bootstrapping was used in conjunction with path analysis. Bootstrapping methods are preferred to other tests of mediation such as Baron and Kenny (1986), since bias-corrected bootstrapped estimates of the confidence intervals for indirect effects do not assume normality of the distribution of sampled indirect effects (Preacher et al., 2007). As a result, bootstrapping is robust to violations of normality (Preacher et al., 2007).

To assess for multicollinearity, variance inflation factors and tolerance statistics were examined. Tolerance statistics less than .10 and VIF of greater than 10 are indicative of multicollinearity (Tabachnick & Fiddell, 2007). Accordingly, the variance inflation factors
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(VIF) and tolerance statistics for all variables were examined. Results suggested that the data met the assumptions of collinearity, with VIF ranging from 1.12 to 3.92 and tolerance statistics ranging from .255 to .878.

Finally, bivariate correlations between demographic factors and parent child relationship problems, peer relationship problems and depression were examined (see Table 1). Depression was not related to sex, race, ethnicity, age, parental education level, IQ, medication use or achievement scores. As a result, it was deemed unnecessary to control for these variables.

Table 2.

<table>
<thead>
<tr>
<th>Variable Descriptives and Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>1. Externalizing Symptoms</td>
</tr>
<tr>
<td>2. Baseline Depression</td>
</tr>
<tr>
<td>3. Parent-Child Relationship Problems</td>
</tr>
<tr>
<td>4. Peer Relationship Problems</td>
</tr>
<tr>
<td>5. Emotion Regulation Problems</td>
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<tr>
<td>6. Depression</td>
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<td>.55</td>
</tr>
<tr>
<td>20.50</td>
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</table>

* p < .05. **p < .01. ***p < .001.

Statistical Analyses

The primary aims of this study were to test whether the indirect and direct pathways from ADHD to depression through parent-child relationship problems and peer relationship problems were moderated by emotion regulation. Accordingly, mediational analyses were first used to assess for the presence of mediation. When evidence for mediation was found, moderated mediation analyses were conducted, as these analyses test whether the indirect effect of a predictor variable (X) on an outcome variable (Y) through a mediating variable (M) is moderated by another variable (V). As Hayes (2013) describes, “the mechanism represented by the X \rightarrow M \rightarrow Y chain of events operates to varying degrees (or not at all) for certain people or in certain contexts” (Hayes, 2013, p. 358). All mediation and moderated mediation analyses included
baseline levels of depression as a covariate. Controlling for baseline levels of outcome variables leads to an increase in statistical power, by accounting for some of the variance in outcome scores, thereby decreasing standard errors for the effects of interest (Kahan, Jairath, Doré, & Morris, 2014).

Process modeling strategies, as recommended by Preacher et al. (2007) were used to assess for mediation and moderated mediation. The PROCESS macro 2.15 (Hayes, 2016) used in this study allows for testing of mediation and moderation models and provides a framework to test both direct and indirect effects. For simple mediation models, model 4 was used to assess evidence for mediation. For moderated mediation models, model 15 was used to assess evidence for moderated mediation. Multiple criteria for assessing moderated mediation have been proposed in the literature (e.g., Edwards & Lambert, 2007, Muller et al., 2005). Most recently, Hayes (2015) developed a procedure to test for moderated mediation which allows the calculation of an index of moderated mediation which can be tested using bias-corrected bootstrap estimates. If the test is significant, evidence for moderated mediation exists. Subsequently, this effect can be probed by examining confidence intervals at different values of the moderator to understand how the indirect relationship varies as a function of the moderator (Hayes, 2015). For these analyses, 95% confidence intervals (CIs) were considered significant if they did not include zero and 10,000 replications (bootstrapping tests) were used.

Finally, path analysis was used to examine the relationships between the scales of DERS and depression at Time 3. Path analysis was conducted using structural equation modeling (SEM) software (AMOS 5.0). SEM is useful for testing causal processes and is robust to violations of multicollinearity (Byrne, 2001).

**Results**
All analyses were run in SPSS 24 (SPSS, IBM, version 24.0) and AMOS 5.0 (AMOS, IBM, Version 5.0).

**Aim One**

A mediation model was constructed to assess the indirect relationship between externalizing symptoms and depression through parent-child relationship problems. Adolescent report of depressive symptoms at Time 3 was entered as the outcome variable, parent report of externalizing symptoms was entered as the predictor variable, and parent report of parent-child relationship problems was entered as the mediator. Depression at Time 1 was entered as a covariate. Results indicated that the indirect effect was significant \( ab = .069, 95\% CI [.036, .115] \). In addition, depression at Time 1 predicted depression at Time 3 \( B = .222, p < .001, 95\% CI [.149, .296] \), but the direct path from externalizing symptoms to depression was not significant \( c = -.069, 95\% CI [-.144, .006] \). See Figure 2 for a visual depiction of this model.

Next, to test the hypothesis that emotion regulation difficulties moderated the indirect relationship between externalizing symptoms and depression through parent-child relationship problems, a moderated mediation model was constructed. Adolescent report of depressive symptoms (Time 3) was entered as the outcome variable, externalizing symptoms (Time 1) was entered as the predictor variable, parent-child relationship problems was entered as a mediator, emotion regulation difficulties was entered as a moderator and baseline depressive symptoms (Time 1) was entered as a covariate. Results indicated that emotion regulation difficulties significantly moderated the indirect relationship between externalizing symptoms and depression through parent-child relationship problems when controlling for depression at Time 1 (Index of Moderated Mediation = .001, 95% CI = .0001, .0033]). Depression at Time 1 was also a
significant predictor of depression at Time 3 in the final model, \((B = .102, p = .004, 95\% CI [.031, .173])\). See Figure 3 for a visual representation of this model.

The Index of Moderated Mediation indicated the indirect relationship from externalizing symptoms to depression through parent child relationship problems varied significantly as a function of emotion regulation difficulties. To better understand this moderated mediation effect, the data were graphed, displaying the difference in depression among groups with either very high (90\textsuperscript{th} percentile) or very low (10\textsuperscript{th}) levels of emotion regulation difficulties relative to this sample (see Figure 4). These thresholds are sometimes applied when there is not a widely accepted clinical cutoff (see Barkley, 2013 for an example of this approach used in the context of examining group differences associated with ‘high’ levels of sluggish cognitive tempo symptoms). The direct relationship between externalizing symptoms and depression was not significantly moderated by emotion regulation difficulties.

Post-hoc power analyses were conducted using the software package GPower (Faul and Erdfelder, 1992). Power was examined for the regression analyses conducted within the simple mediation models and the moderated mediation models. The statistical power for these analyses ranged from .99 to 1.0, \((N = 239; \alpha\text{ set as } p < .05)\) suggesting that the tests were sufficiently powered. Power analyses were not conducted for tests of indirect effects, due to the fact that bootstrapping techniques were used to provide estimates of indirect effects. Bootstrapping approaches are based on the premise that each sample can be treated as a representation of the large population from which the sample is drawn; accordingly, the sampling distribution of any statistic can be produced by calculating the statistic of interest using multiple ‘resamples’ of the data. Most commonly, the number of bootstrapped samples calculated is set to 1000 or higher.
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As a result, bootstrapping techniques are very unlikely to be underpowered (Preacher et al., 2007).

Aim Two

A mediation model was created to test for an indirect relationship between externalizing symptoms and depression through peer relationship problems. Externalizing symptoms were entered as the predictor variable, depression at Time 3 was entered as the outcome variable, peer relationship problems were designated as the mediator, and depression at Time 1 was entered as a covariate. Results indicated that the indirect effect was significant ($ab = .048, 95\% CI [.023, .086])$. In addition, depression at Time 1 predicted depression at Time 3 ($B = .241, p < .001, 95\% CI [.166, .315])$ and the direct path from externalizing symptoms to depression was not significant ($c = -.049, 95\% CI [-.123, .025])$. See Figure 5 for a visual depiction of this model.

Next, to test the hypothesis that emotion regulation difficulties moderated the indirect relationship between externalizing symptoms and depression through peer relationship problems, a moderated mediation model was constructed. In this model, adolescent report of depressive symptoms (Time 3) was entered as the outcome variable, externalizing symptoms (Time 1) were entered as the predictor variable, peer relationship problems (Time 2) were entered as a mediator, emotion regulation difficulties were entered as a moderator and baseline depressive symptoms (Time 1) were entered as a covariate. Results indicated that emotion regulation difficulties significantly moderated the indirect relationship between ADHD, peer relationship problems and depression when controlling for depression at Time 1 (Index of Moderated Mediation $= .001, 95\% CI = .0000, .0023])$. For a figure depicting this model, see Figure 6. In the final model, emotion regulation difficulties ($B = .254, p = .0002, 95\% CI [.123, .384])$ and depression at Time 1 ($B = .116, p < .0001, 95\% CI [.086, .226])$ also significantly predicted depression.
MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD

The Index of Moderated Mediation indicated the indirect relationship from externalizing symptoms to depression through parent child relationship problems varied significantly as a function of emotion regulation problems. To better understand this moderated mediation effect, the data were graphed, displaying the difference in depression among groups with either very high (90th percentile) or very low (10th) levels of emotion regulation difficulties in the presence of low, moderate or high levels of peer relationship problems (See Figure 7).

Post-hoc power analyses were conducted using the software package GPower (Faul and Erdfelder, 1992). Power was examined for the regression analyses conducted within the simple mediation model and the moderated mediation model. The post hoc analyses revealed the statistical power for these analyses ranged from .99 to 1.0, (\(N=239\); alpha set as \(p < .05\)).

**Aim Three**

The third aim of this study was to assess which aspects of emotion regulation were most strongly related to depression among adolescents with ADHD. Bivariate correlations among subscales of the DERS and depression were examined (see Table 3).

Table 3. 

**Correlations between DERS scales and Depression**

<table>
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<th>1</th>
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<th>4</th>
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<th>6</th>
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<td>1. Depression</td>
<td>--</td>
<td>.341**</td>
<td>.298**</td>
<td>.290**</td>
<td>.072</td>
<td>.430**</td>
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<td>.517**</td>
<td>.099</td>
<td>.660**</td>
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<td>.494**</td>
<td>.159*</td>
<td>.546**</td>
<td>.188*</td>
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<td>4. Impulse</td>
<td>--</td>
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<td>.709**</td>
<td>.239**</td>
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<td>5. Awareness</td>
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<td>.206**</td>
<td>.223**</td>
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<td>6. Strategies</td>
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<td></td>
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<td>.345**</td>
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<td>7. Clarity</td>
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**Mean**

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**SD**

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<td>4.50</td>
<td>6.45</td>
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</tbody>
</table>

Note. **indicates \(p<.001\); * indicates \(p < .05\). Depression was assessed by the RADS at Time 3. Acceptance refers to the Nonacceptance of Emotional Responses scale on the DERS; Goals
refers to Difficulties Engaging in Goal-Directed Behavior scale on the DERS, Impulse refers to the Impulse Control Difficulties scale on the DERS, Awareness refers to Lack of Emotional Awareness, Strategies refers to the Limited Access to Emotion Regulation Strategies scale on the DERS, and Clarity refers to the Lack of Emotional Clarity scale on the DERS.

Four subscales of the DERS (Nonacceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, and Limited Access to Emotion Regulation Strategies) were significantly correlated with depression at Time 3. Lack of Emotional Clarity and Lack of Emotional Awareness subscales of the DERS were not significantly correlated with depression. To simultaneously evaluate the association between the DERS subscales and depression a path analysis was conducted using structural equation modeling (SEM) software (AMOS 5.0). SEM is useful for testing causal processes and is robust to violations of multicollinearity (Byrne, 2001). Depression was entered as the dependent variable, and the six subscales of the DERS were entered simultaneously as predictors. The kurtosis and skewness statistics for all variables entered in the path analysis were within the limits suggested by West, Finch, and Curran as appropriate to proceed with SEM (skewness < 2; kurtosis < 7). Since the model was fully saturated (degrees of freedom = 0), it demonstrated a perfect fit to the data; accordingly, model fit statistics are not reported. An examination of path coefficients provides an assessment of the magnitude of the relationships among the variables in the model. Path analysis provides standardized estimates which may be interpreted as effect sizes, such that values of .10 are classified as small effects, values of .30 are classified as moderate effects and values of .50 are classified as large effects (Cohen, 1988). Out of all the emotion regulation subscales, the only significant unique predictor of depression was Limited Access to Emotion Regulation Strategies ($\beta = .37$, $p < .001$). See Figure 8 for a visual depiction of the path analysis model with standardized regression weights for each path tested in the model.
Discussion

The goal of this study was to evaluate whether two types of interpersonal impairment, parent-child relationship problems and peer relationship problems, mediated the longitudinal association between externalizing symptoms and depression among adolescents with ADHD. In addition, moderated mediation models were tested to assess whether emotion regulation problems moderated the direct and indirect paths from externalizing symptoms to depression through parent-child relationship and peer relationship problems. Finally, the paths between specific aspects of emotion regulation difficulties at Time 2 and depression at Time 3 were assessed simultaneously, to better understand which specific aspects of emotion regulation contribute to depression in this population. Analyses revealed that both parent-child and peer relationship problems mediated the association between externalizing symptoms and depression and that emotional regulation significantly moderated this association. The Limited Access to Emotion Regulation Strategies subscale emerged as the most important predictor of depression. These findings are discussed in more detail in subsequent sections along with clinical implications and directions for future research.

The Role of Interpersonal Impairment

Findings from the present study lend support to a growing body of research highlighting the importance of interpersonal impairment in understanding why up to a third of children with ADHD develop comorbid depression in adolescence or emerging adulthood (32%; Anastopolous et al., 2016). Specifically, the findings are consistent with Humphreys et al. (2013), who found evidence for indirect paths from ADHD symptoms to depression through interpersonal impairment in a cross-sectional clinical sample (M age = 7.39) and a longitudinal community sample followed from childhood to age 20 (Humphreys et al., 2013). The present study builds on
Humphreys et al. (2013) by longitudinally documenting the importance of both parent-child relationships and peer relationships in determining risk for depression in a clinical sample of young adolescents with ADHD. Importantly, these associations were cross-rater, with parent ratings of interpersonal impairment predicting adolescent ratings of depression. Overall, the present study suggests that the parent-child relationship quality continues to be a relevant factor during the period of adolescence, a developmental stage when relationships with peers are typically stronger predictors of adjustment (Laible, Carlo, & Raffaelli, 2000). Further, these findings are clinically significant given that adolescents with ADHD and depression experience more severe impairment, suicidal ideation, and self-harm behaviors, compared to their peers without ADHD who are diagnosed with depression (Biederman et al., 2008; Chronis-Tuscano et al., 2010).

Importantly, it can be difficult to improve parent-adolescent and adolescent-peer relationships, as the sheer existence of externalizing symptoms can interfere with the development of positive peer relationships and pulls for higher levels of conflict in parent-child relationships (Edwards et al., 2001; Li et al., 2014). It is no coincidence that the overwhelming majority of behavioral parent training programs, which are considered a first-line treatment for young children diagnosed with ADHD along with psychopharmacological treatment (American Academy of Pediatrics, 2011), include a strong focus on improving the parent-child relationship through an emphasis on praise, positive reinforcement and the implementation of special play times during which parents are instructed to refrain from commands, instructions or questions (e.g., Barkley, 2013; see Chacko et al., 2015 for a review of behavioral parent training for children with ADHD). Similarly, some treatments of adolescents with ADHD also include a focus on communication and the parent-adolescent relationship (e.g., Robin, 2015). This reflects
the understanding of the importance of this relationship and an awareness that this relationship is very often strained due to the frustrations associated with parenting a child with high levels of externalizing symptoms.

Similarly, longitudinal research has consistently documented difficulties in peer relationships among children and adolescents with ADHD (Bagwell et al., 2001; Li et al., 2014); moreover, peer rejection (a more severe aspect of peer relationship difficulties) is linked to poorer long-term outcomes such as delinquency, anxiety and global impairment (Mrug et al., 2012). Findings from the present study strongly suggest that both parent-child relationships and peer relationships play an important role in determining risk for depression.

**Moderating Effects of Emotion Regulation Difficulties**

In addition to the importance of interpersonal relationships, the findings of this study also provide valuable insights about the role that adolescent qualities play in strengthening or weakening the relationship between externalizing symptoms, interpersonal impairment and depression. When high levels of emotion regulation difficulties are present, adolescents’ symptoms of depression are more elevated in comparison to adolescents with low levels of emotion regulation difficulties, across all levels of interpersonal impairment (parent-child relationship problems or peer relationship problems). This is consistent with prior cross-sectional research which found associations between externalizing symptoms, emotion regulation problems, and depression (Anastopoulos et al., 2011; Seymour et al., 2012; Seymour et al., 2014). In the present study, adolescents with both high levels of emotion regulation difficulties and high levels of interpersonal impairment also reported the highest levels of depressive symptoms (see Figures 4 and 7). In fact, there was a 17-point difference (1.87 standard deviations) in mean depression scores between adolescents who were high on both emotion
regulation problems and peer relationship problems and adolescents low on both peer relationship problems and emotion regulation problems. Similarly, there was a 14-point difference (1.54 standard deviations) in mean depression scores between adolescents who were high on both emotion regulation problems and parent-adolescent relationship problems and adolescents low on both parent-adolescent relationship and emotion regulation problems.

In sum, heterogeneity in the development of depression among adolescents with ADHD may be at least partly attributed to differences in emotion regulation ability. This may help explain why not all adolescents with ADHD develop clinical levels of depression, despite most struggling with impairments in parent and peer relationships. The results of the current study suggest that adolescents with relatively stronger emotion regulation abilities experience a lower overall risk for depression, even in the context of interpersonal impairment. In contrast, adolescents with both high levels of interpersonal impairment and greater emotion regulation difficulties appear far more likely to develop symptoms of depression. This is consistent with past work in typically developing samples documenting increased risk for negative outcomes in the presence of interpersonal impairment and emotion regulation abilities. For instance, Adrian et al. (2010) reported that family and peer relationship problems indirectly increase risk for nonsuicidal self-injury behaviors (NSSI) through emotion dysregulation in adolescent girls. This suggests that even in the presence of interpersonal impairment, an adolescent’s ability to identify emotions, manage painful emotions and utilize coping strategies to effectively regulate emotion is a key factor which can either increase or decrease risk for developing symptoms of depression. Adolescents with difficulties identifying and regulating their emotional responses may respond to interpersonal conflict with greater distress, or may experience helplessness or low confidence in their ability to regulate or cope with their own distress. Over time, this may lead to an
attributional style in which negative events are perceived as internal, stable, and global as outlined by Seligman’s learned helplessness theory of depression (Seligman, 1975); this style is positively associated with symptoms of depression (Gladstone & Kaslow, 1995). In contrast, adolescents who experience interpersonal impairments but who feel confident and effective regarding their ability to regulate emotion and manage distress may experience less helplessness, thereby preventing the development of depression.

Moreover, it is probable that emotion regulation and interpersonal impairment influence each other. Increased levels of family conflict and relationship problems are very likely to have a negative impact on an adolescent’s developing ability to self-regulate their emotion. Concurrently, an adolescent who struggles to regulate his or her emotions may elicit responses such as frustration, anger, impatience or dismissiveness from those around them. Parental influence on children’s understanding and expression of emotion is not a new concept—this process has been defined as “emotion socialization” (Eisenberg, Cumberland, & Spinrad, 1998), and it is considered a part of normative development. More recently, Morris and colleagues (2007) outlined a theoretical model of adolescents’ emotion regulation in which learning process (i.e., modeling, social referencing), parenting practices specific to emotional behavior, and the emotional climate of the family (e.g., attachment, emotional expressiveness) all influence adolescent emotion regulation abilities. Further, the authors discuss how adolescent characteristics influence these three areas and suggest that in particular, adolescents who are more likely to experience high emotional arousal (what the authors refer to as “emotional reactivity”) may stand to benefit the most from parental support and guidance in the development of emotion regulation abilities and conversely, are the most likely to be negatively affected by critical or dismissive reactions from parents (Morris, Silk, Steinberg, Myers, & Robinson,
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(2007). An adolescent with deficits in self-regulation (i.e., the types of deficits associated with externalizing difficulties broadly and ADHD specifically) may therefore be more likely to experience disruptions or difficulties in the process of developing emotion regulation abilities through these multiple paths.

Although interactions between interpersonal impairment and emotion regulation difficulties are not unique to adolescents with ADHD, they are likely to be particularly salient in this population because of the higher prevalence of both interpersonal impairment and emotion regulation difficulties. For example, in a large cross-sectional study, the mean scores of adolescents with ADHD on measures of emotion lability (defined as a pattern of behavior including low frustration tolerance, irritability, and sudden shifts in mood) were 1.5 standard deviations higher compared to mean scores of adolescents without ADHD (Sobanski et al., 2010). Interpersonal relationship problems are also quite common, occurring within both parent-child relationships (Pressman et al., 2006) and friendships (Bagwell et al., 2001); moreover, interpersonal impairments occur at even higher rates when comorbid ODD symptoms are present (Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001; Li et al., 2014). Therefore, investigating how and when these constructs influence one another is of particular importance for this population.

**Associations Between Facets of Emotion Regulation and Depression**

Bivariate correlations showed that Nonacceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Lack of Emotional Awareness, and Limited Access to Emotion Regulation Strategies all had moderate positive associations with depression. However, when considered simultaneously, Limited Access to Emotion Regulation Strategies emerged as the only significant predictor. To better understand
this association, it is helpful to more closely review the items included on the Limited Access to Emotion Regulation Strategies scale from the DERS. The authors of the measure describe this scale as “reflecting the belief that … little can be done to regulate emotions effectively, once an individual is upset (p. 47, Gratz & Roemer, 2004).” Specific items on this scale all begin with the statement “When I’m upset” and require respondents to indicate how strongly they agree with statements such as: I believe that I will remain that way for a long time; It takes me a long time to feel better; I believe that there is nothing I can do to make myself feel better; and I know that I can find a way to eventually feel better (reverse scored). These items appear to reflect both a lack of effective strategies to cope with negative emotions and a low sense of self-efficacy (e.g., “I believe there is nothing I can do to make myself feel better”) with regard to the ability to regulate negative emotion. It is easy to understand how an adolescent who both experiences high levels of strong negative emotions and feels little to no confidence in his or her ability to manage these emotions could be more susceptible to depression. Moreover, this is consistent with findings that self-efficacy in adolescents shows a strong negative relationship with internalizing symptoms (both anxiety and depression; Muris, 2002; Sharon, Goodness, & Buhrmester, 2002).

In addition, these findings are consistent with the results of a study conducted by Silk et al. (2003), which utilized experience sampling methods to assess in-the-moment emotional experiences and responses among adolescents between the ages of 12 and 17 (N = 152; M age = 13.89; SD = 1.65; 48% male). The authors found that adolescents who experienced more intense and labile negative emotions (sadness, anger, and anxiety) scored higher on measures of depressive symptoms and problem behavior. In addition, adolescents who reported more frequent use of ineffective emotion regulation strategies (denial, avoidance, rumination or impulsive/involuntary action) also scored higher on measures of depression and problem
behavior (Silk et al., 2003). Use of ineffective strategies likely overlaps with the Lack of Emotion Regulation Strategies scale as assessed by the DERS, as some of the items on this scale may tap into ineffective strategies such as rumination (e.g., I believe that wallowing in it is all I can do) or denial/avoidance (I believe there is nothing I can do to make myself feel better). Thus, adolescents who score higher on this scale may display not only a lack of effective emotion regulation strategies but also a higher likelihood of using ineffective techniques such as denial or avoidance. Overall, the implication of these findings is that depression is associated with both the experience of negative emotion (intensity and lability) as well as how effectively adolescents are able to respond to and regulate these emotions.

Clinical Implications and Future Directions

The results of this study provide important implications for future research. It is important to consider not only whether adolescents are using emotion regulation strategies but also, the effectiveness of these strategies. For example, avoidance or denial of negative emotion may provide short-term relief from negative emotion, thus functioning as an emotion regulation strategy. However, these strategies are not considered effective as they do not provide long-term relief and are associated with higher levels of depression (Silk et al., 2003) in addition to a higher likelihood of symptom recurrence (Holahan, Moos, Holahan, Brennan, & Schutte, 2005). A related direction for future research is considering how the use of emotion regulation strategies may be affected by the context of the environment that an adolescent lives in. Adolescents may face challenges in making decisions about which strategies to use in which situations, and this may be particularly important when they are not in control over their environment. For example, an adolescent could attempt to distract themselves from feelings of sadness by playing video games. However, if playing video games violates rules set by parents or teachers, this strategy
will be ineffective for the situation and could potentially make the situation worse by increasing conflict with parents or teachers. Similarly, an adolescent may learn and apply an emotion regulation strategy in school, but unless the behavior is reinforced by peers or teachers, strategy implementation is unlikely to be sustained. In sum, measurement of emotion regulation is complex and may need to include the assessment of access to, confidence in, and environmental support for, the use of emotion regulation strategies.

It is important to acknowledge that this study focused on impairment with family and peers and that other facets of emotion regulation may be more strongly linked to different types of impairment. In fact, there is evidence that emotional perception (defined as awareness and accurate identification of the emotions of oneself and others) is positively correlated with social support from peers and with ratings of satisfaction with social support (Ciarrochi, Chan, & Bajgar, 2001). Thus, deficits in emotion perception which correspond closely to constructs measured by Lack of Emotional Awareness and Lack of Emotional Clarity may be linked to impairment in different social domains. Similarly, the constructs assessed by Impulse Control Difficulties (e.g., “When I’m upset I have difficult controlling my behaviors”) may overlap with emotional impulsivity, which has been defined as intense and variable emotional reactivity (Barkley & Fischer, 2010). Emotion impulsivity has been associated with behavioral problems and aggression in children with ADHD (Rosen & Factor, 2015), which can lead to both peer difficulties (Bagwell, Molina, Pelham, & Hoza, 2001) and strained parent-child relationships (Burke et al., 2008). In conclusion, it may be that whereas access to emotion regulation strategies has a direct influence on the development of depression in this population, the most important aspect of emotion regulation varies as a function of impairment domain.

Implications for the Cognitive Behavioral Theory of ADHD
These findings also provide support for the validity of the cognitive-behavioral model of ADHD in adolescents, whereby comorbid depression emerges from impairment and failure experiences. However, one construct which was not explicitly addressed in the current study is the impact of impairments on cognitions about the self, which are theorized to be an important explanatory mechanism linking impairment and depression in the cognitive behavioral theory of ADHD. Past studies have produced mixed results on the question of whether children with ADHD demonstrate more negative thoughts about the self or lower self-esteem, with some studies reporting evidence for lower self-esteem (e.g., Edbom, Lichtenstein, Granlund, & Larsson, 2006) and other studies demonstrating evidence that children with ADHD are more likely to overestimate their competence (e.g., Hoza, Pelham, Dobbs, Owens, & Pillow, 2002). The most recent research indicates that children with ADHD are no more likely to overestimate their competence than their peers are, with only 10% of children overestimating their competence in multiple domains (Bourchtein et al., 2017). However, among adolescents with ADHD, there is evidence that ADHD is associated with significantly lower ratings of self-esteem (Harpin, Mazzone, Raynaud, Kahle, & Hodgkins, 2016), especially when comorbid conduct problems are present (Glass, Flory, Martin & Hankin, 2011). This suggests that consistent with the cognitive behavioral model of ADHD, individuals with ADHD develop negative cognitions and views of themselves over time, which is likely attributable to a history of impairment and underachievement. Moreover, these cognitions likely play a role in increasing risk for depression in this population.

**Implications for Treatment**

Historically, studies investigating the use of cognitive-behavioral therapies to treat ADHD in children have failed to find significant effects on ADHD symptoms and associated

Perhaps for that reason, there were no investigations of CBT for adolescents with ADHD until
the publication of two recent studies testing the use of a CBT protocol in adolescent samples.
Both studies found that adolescents with ADHD demonstrated significant improvements on
ratings of externalizing symptoms and impairment (Antshel, Faraone, & Gordon 2014; Sprich,
Safren, Finkelstein, Remmert, & Hammerstein, 2016). Specifically, Sprich, Safren, Finkelstein,
Remmert, and Hammerstein (2016) demonstrated significant reductions on ratings of ADHD
symptom severity and global impairment in a randomized controlled trial of CBT (N = 36; M
age_{CBT} = 15.17, SD_{CBT} = 1.01; M_{Waitlist age} = 15.09, SD_{Waitlist} = 1.11). Antshel et al. (2014)
demonstrated significant improvements on inattentive symptoms, externalizing behavior, and
peer, family and academic functioning (estimate of Cohen’s d from .85 to 1.51) in an
uncontrolled trial of CBT conducted in a community specialty clinic (N = 68; M age = 16.4, SD
= 1.3). Significant improvements were also demonstrated on adolescent ratings of self-esteem (d
= .86) and, adolescents with ADHD and depression benefitted significantly more than
participants with ADHD alone (Antshel et al., 2014). These results have promising implications
for the use of CBT to treat adolescents with ADHD, particularly when combined with the results
reported herein supporting the cognitive behavioral model of ADHD in adolescents.

Findings from the present study also have important implications for understanding
which adolescents with ADHD are most at risk for developing symptoms of depression. As
discussed above, adolescents with ADHD who experience high levels of parent-child
relationship problems or high levels of peer relationship problems may experience a relatively
elevated risk for symptoms of depression, particularly when they also demonstrate greater
difficulties with emotion regulation. Given evidence children and adolescents with ADHD
demonstrate far more difficulties in interpersonal relationships, particularly in the presence of comorbid ODD symptoms (Bagwell et al., 2001; Edwards et al., 2001; Li et al., 2014), targeting interpersonal impairment using interventions aimed at improving social relationships would seem to be an obvious recommendation. However, it is important to acknowledge that interventions which seek to directly address social skills and interpersonal impairment, particularly in the realm of peer relationships, have not demonstrated reliable improvements in interpersonal functioning (for a review of this area of research, see Mikami, Jia, & Na, 2014). A paradigm shift may be needed with respect to treating the interpersonal impairments of children and adolescents with ADHD.

Currently, interventions for children and adolescents with ADHD are designed to address specific types of impairment. Social skills interventions target impairment in interpersonal domains (Mikami et al., 2014) and parenting interventions address family conflict and relationships (Chronis, Chacko, Fabiano, Wymbs, & Pelham, 2004). Since ADHD is now understood as a chronic condition that persists into adulthood in the majority of cases (Biederman et al., 2010), it may make sense to also consider treatment in terms of symptom management as opposed to solely focusing on symptoms/impairment reduction. In other words, in addition to specifically targeting impairment, treatment could be designed to promote coping. Treatments designed from this perspective would focus on teaching and reinforcing the use of coping skills that can be applied in multiple domains (including, but not limited to, emotion regulation skills). Importantly, this would have the added benefit of helping adolescents learn to deal with frustrations in multiple domains and during later stages of life, when increased demands and changing environments may result in greater challenges. For example, an adolescent with ADHD and/or significant levels of externalizing symptoms may receive an
intervention that helps improve his academic performance and classroom behaviors. While these improvements represent important progress and may even result in reduced parent-child conflicts at home, the adolescent will likely continue to struggle with peer relationships due to symptoms such as forgetfulness, talking too much, or interrupting others—all hallmark symptoms of ADHD. In sum, even when adolescents with ADHD exhibit a significant response to treatment, behavior is rarely normalized, and they are likely to experience negative affect and emotion, such as frustration, anger, and sadness. Thus, it may be helpful to include an explicit focus on increasing broad coping skills in treatments for ADHD, including practice and rehearsal of coping skills. As others have noted, ADHD is characterized by inconsistent performance (e.g., Ramsay & Rostain, 2015), and supporting individuals as they practice and implement skills can help account for this issue.

A focus on teaching and rehearsing coping skills could also help prepare individuals for developmental transitions, which can be challenging for individuals with ADHD (for a detailed discussion of how and why transitional periods may be challenging for individuals with ADHD, see Turgay et al., 2012). The increasing environmental demands associated with transitions (e.g., transition to middle school) can result in symptoms of ADHD becoming more apparent and impairing (Langberg et al., 2008). For example, an adolescent with ADHD whose symptoms are well managed and who is not experiencing significant impairment in academics in high school may begin to struggle again when faced with the academic demands of a college or university. In such situations, coping and problem solving skills would be important tools to help manage these challenges effectively by identifying and recognizing problems/problematic behaviors and accessing additional supports if necessary. Importantly, some adolescents who are functioning well may not be engaged in active treatment; therefore, they may have an even harder time when
they begin to struggle during life transitions as they face the additional challenge of reengaging in treatment (e.g., seeking out pharmacological treatment which generally requires an updated documentation of a diagnosis). In sum, current evidence-based treatments for ADHD represent symptom management, not symptom resolution. As such, we should expect individuals with ADHD will continue to experience challenges and impairment and creating interventions which target coping broadly may be an important future direction for research on treatments for ADHD.

Limitations of the Current Study

Although the findings of this study provide important information, several limitations should be discussed. First, the length of time over which data were collected was 1.5 years, which is a relatively short period of development to assess the paths outlined in the moderated mediation models. Specifically, measuring these constructs over a longer time period would likely provide more information about how they interact and influence one another. Second, another drawback of the current study was the low levels of depression present in the sample. The mean score on adolescent-reported depression on the RADS-2 at Time 3 was 45.26 ($SD = 9.05$), which falls well below the clinical threshold of 70. Overall, 93.3% of the sample scored within the “normal” range on the RADS-2 ($t = 60$ or lower). The remainder of the sample scored in the mild clinical depression range (3.8%; $t = 61$ to 64), moderate clinical depression range (1.3%; $t = 65$ to 69) and severe clinical depression range (1.7%; $t = 70$ or higher). Furthermore, there were minimal differences in mean levels of depression between Time 1 and Time 3, with a mean difference score of 6 points. The low level of depressive symptoms observed in this study suggests that the findings should be interpreted with caution. These low levels may reflect the fact that this sample consisted of younger adolescents ($M$ age = 12.30; $SD = .92$). As such, it is
unclear whether the findings of this study would generalize to older adolescents with ADHD with higher rates of clinical depression (Rabiner et al., 2008).

Moreover, the sample used in this study was predominantly male (73%). This is consistent with gender ratios in other clinical samples of adolescents with ADHD, which are generally primarily male (e.g., 66% male reported in Antshel et al., 2014; 78% male reported in Sprich et al., 2016). However, gender differences in the prevalence of depression during adolescence are well-documented, with females displaying significantly higher rates of depression compared to males (Hankin et al., 1998; Nolen-Hoeksema, 2001). Moreover, gender differences in patterns of emotion regulation difficulties and emotion regulation strategies have also been observed (Silk et al., 2003; Zimmerman & Iwanski, 2014). It is interesting to consider whether different patterns in emotion regulation difficulties may partially explain the lower prevalence of females in clinical samples of adolescents with ADHD. For example, females may be less likely to present to clinical settings due to different patterns of emotion regulation strategies associated with lower levels of interpersonal impairment and comorbid depression. In sum, these findings should be interpreted with caution, as they may not generalize to females, who were underrepresented in this sample.

Another important limitation to note is the amount of missing data on the RADS-2 at Time 3 and the DERS at Time 2. Missing data was addressed by first assessing whether there was evidence for the assumption that data were missing at random; subsequently, multiple imputation was applied to address this missing data. It is important to acknowledge that despite the evidence gathered by Little’s MCAR test, there is still a possibility that data may have been missing in a systematic way, as it is impossible to determine that data are missing at random with complete certainty.
Finally, there were some measurement-related limitations to the current study. The DERS has many strengths, but there are two aspects of its design which limit the interpretations that can be drawn. First, the DERS was designed to measure deficits in emotion regulation skills. Importantly, not demonstrating a deficit does not necessarily equate to demonstrating a skill or strength. For example, an adolescent who scores in the low range on “Lack of Emotional Awareness” scale from the DERS may not necessarily have excellent emotional awareness skills. Accordingly, our interpretations of findings utilizing this measure are limited to understanding the role such deficits play, and it is difficult to draw conclusions about the role of emotion regulation strengths and abilities. Second, the phrasing of the items on the DERS all start with the same stem, “When I am upset…. (Gratz & Roemer, 2004).” As a result, the DERS is best understood as a measure of adolescents’ emotion regulation difficulties in the context of negative emotions. It does not provide information about how effectively adolescents regulate positive emotion.

In addition to the limitations of the DERS discussed above, there are certain limitations to the measure of peer relationship impairment. This measure was a parent-report of adolescents’ peer functioning and, therefore, it has some inherent drawbacks. Parent ratings of adolescents’ peer relationship functioning may not necessarily correspond with the reports provided by adolescents themselves. However, the use of parent ratings of peer relationships does result in a statistical model utilizing the reports of multiple raters (parent report of symptoms and impairment; adolescent report of depression and emotion regulation difficulties). Statistical models using reports from multiple informants are more methodologically more rigorous, since they minimize the likelihood of shared variance due to a single rater (Spector & Brannick, 2009).
Finally, the measure of parent-child relationship problems used in the current study has some limitations as well. Recommendations for the comprehensive assessment of family relationships call for the use of structured or semi-structured interviews, as well as data from multiple raters and, ideally, direct observations of parent-adolescent interactions (see Hawes & Dadds, 2013 for a discussion of this topic). In contrast, the measure used to assess parent-adolescent relationship problems in this study was a single self-report measure completed by the parent. Nevertheless, strong associations observed between externalizing symptoms, depression, and parent-adolescent relationship problems, even with this relatively brief measure of parent-adolescent relationship problems, suggests a more comprehensive assessment would produce similar findings.

**Summary and Conclusions**

The goal of this study was to investigate potential mechanisms which explain why adolescents with ADHD are at increased risk for developing depression compared to their peers. Simple mediation models and moderated mediation models were used to test whether the indirect effects of externalizing symptoms on depression through parent-child relationship stress and peer relationship problems are moderated by emotion regulation. Results of the mediation models and the moderated mediation models suggest impairment in interpersonal relationships (both parent-child relationships and peer relationships) is an important mechanism through which externalizing symptoms increase risk for later depressive symptoms among adolescents with ADHD. Importantly, these pathways varied in strength as a function of emotion regulation, such that the paths from externalizing symptoms to depression are significantly stronger among adolescents with high levels of emotion regulation problems. Finally, an exploratory analysis of the associations between multiple aspects of emotion regulation problems and depression
revealed access to emotion regulation strategies was the most important predictor of later depression among adolescents with ADHD.

The results of this study support existing evidence for viewing interpersonal impairment and emotion regulation problems as explanatory variables in the relationship between externalizing symptoms and depression. These findings extend previous work by suggesting the interaction between interpersonal impairment and emotion regulation problems influences risk for later depressive symptoms in this population, such that the presence of both interpersonal impairments and emotion regulation difficulties are associated with the highest risk for later depression. Future directions for research in this area could include investigations into factors which impact the development of emotion regulation difficulties as well as investigating treatments that target emotion regulation. The results of the study also support the validity of the cognitive behavioral model of ADHD and suggest that additional trials testing the application of CBT in this population are warranted. CBT treatment could include targeting broad-based coping skills, with a focus on emotion regulation skills to address the difficulties which are associated with the chronic nature of ADHD. This may be particularly helpful in times of developmental transition.
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doi:10.1177/1087054705278777


MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD


MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD


Johnston, C. (1996). Parent characteristics and parent-child interactions in families of nonproblem children and ADHD children with higher and lower levels of oppositional-

doi:10.1007/BF01448375


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Appendix A-1

Simple Mediation with Parent-Child Relationship

*Figure 2.* Mediation model depicting the direct and indirect relationships between externalizing symptoms and depression. Standardized coefficients shown outside parentheses; standard errors are shown inside parentheses. Dashed paths are nonsignificant (*p*'s > .05). Analyses controlled for depression at Time 1, which in the final model was significantly positively associated with depression at Time 3 (*b* = .22, *SE* = .037, *t* = 5.95, *p* < .001). *p* < .05. **p** < .01. ***p*** < .001.
Figure 3. Indirect effects model of Externalizing Symptoms predicting Depression at Time 3 via Parent-Child Relationship Problems at Time 2, moderated by Emotion Regulation Problems at Time 2. Standardized coefficients shown outside parentheses; standard errors are shown inside parentheses. Dashed paths are nonsignificant (p’s > .05). Analyses controlled for depression at Time 1, which in the final model was significantly positively associated with depression at Time 3 (b = .15, SE = .04, t = 4.10, p < .001). *p < .05. ** p < .01. ***p < .001.
Figure 4. Relationship between parent-child relationship problems and depression is depicted at very low (10th) and very high (90th percentile) levels of emotion regulation problems. Parent-child relationship problems was measured using the Adolescent-Parent Relationship Domain (APRD) of the Stress Index for Parents of Adolescents (SIPA; Sheras, Abidin, & Konold, 1998). Higher scores on this measure indicate more severe problems in the parent-adolescent relationship. Depression was measured using the Reynolds Adolescent Depression Scale, Second Edition (RADS-2; Reynolds, 1987). Scores are reported as t-scores, and a clinical cut-off score of 60 is used to identify clinically significant levels of depression (Reynolds, 1987).
Appendix A-4

Simple Mediation with Peer Relationships

Figure 5. Mediation model depicting the direct and indirect relationships between externalizing symptoms and depression. Standardized coefficients shown outside parentheses; standard errors are shown inside parentheses. Dashed paths are nonsignificant (p’s > .05). Analyses controlled for depression at Time 1, which in the final model was significantly positively associated with depression at Time 3 (b = .24, SE = .038, t = 6.399, p < .001). *p < .05. **p < .01. ***p < .001.
Appendix A-5

Moderated-Mediation with Peer Relationships

Figure 6. Moderated-Mediation with Peer Relationships. Indirect effects model of Externalizing Symptoms predicting Depression at Time 3 via Peer Relationship Problems at Time 2, moderated by Emotion Regulation Problems at Time 2. Standardized coefficients shown outside parentheses; standard errors are shown inside parentheses. Dashed paths are nonsignificant ($p$'s > .05). Analyses controlled for depression at Time 1, which in the final model was significantly positively associated with depression at Time 3 ($b = .15$, $SE = .04$, $t = 4.10$, $p < .001$). *$p < .05$. **$p < .01$. ***$p < .001$. 

Index of Moderated Mediation = .0011, $SE = .0006$, 95% CI = .0000, .0023
Appendix A-6

Depression Scores at Low and High Levels of Emotion Regulation

Figure 7. Depression Scores at Low and High Levels of Emotion Regulation. Relationship between peer relationship problems and depression is depicted at very low (10th) and very high (90th percentile) levels of emotion regulation problems. Peer relationship problems were assessed using the WFIRS Social Activities Domain. On the WFIRS, a score of .71 identifies the presence of clinically significant impairment (Thompson, Lloyd, Joseph, & Weiss, 2017). Depression was measured using the Reynolds Adolescent Depression Scale, Second Edition (RADS-2; Reynolds, 1987). Scores are reported as t-scores, and a clinical cut-off score of 60 is used to identify clinically significant levels of depression (Reynolds, 1987).
**MODERATED MEDIATION MODEL OF DEPRESSION AND ADHD**

### Appendix A-7

**Relationships Between Emotion Regulation Subscales and Depression**

![Diagram showing relationships between emotion regulation subscales and depression](image)

*Figure 8. Standardized coefficients shown next to paths. Dashed paths are nonsignificant (p’s > .05). *p < .05. **p < .01. Acceptance refers to the Nonacceptance of Emotional Responses scale on the DERS; Goals refers to Difficulties Engaging in Goal-Directed Behavior scale on the DERS, Impulse refers to the Impulse Control Difficulties scale on the DERS, Awareness refers to Lack of Emotional Awareness on the DERS, Strategies refers to the Limited Access to Emotion Regulation Strategies scale on the DERS, and Clarity refers to the Lack of Emotional Clarity scale on the DERS.*

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