Reactive and regulative temperament dimensions, emotion regulation, and concurrent internalizing and externalizing pathology among youth with ADHD.

Kirsten D. Leaberry

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REACTIVE AND REGULATIVE TEMPERAMENT DIMENSIONS, EMOTION REGULATION, AND CONCURRENT INTERNALIZING AND EXTERNALIZING PATHOLOGY AMONG YOUTH WITH ADHD

By

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ABSTRACT

REACTIVE AND REGULATIVE TEMPERAMENT DIMENSIONS, EMOTION REGULATION, AND CONCURRENT INTERNALIZING AND EXTERNALIZING PATHOLOGY AMONG YOUTH WITH ADHD

Kirsten Leaberry

June 17, 2019

Emerging research has increasingly identified the detrimental effect of internalizing and externalizing comorbidity on the functioning of youth with Attention-Deficit/Hyperactivity Disorder (ADHD). Research in the broad child psychopathology literature has identified a variety of dispositional and developmental risk factors for psychopathology development in youth. However, a conceptual model of psychopathology development has yet to be developed and empirically evaluated in an ADHD sample. Children with ADHD may be particularly vulnerable to exhibiting high rates of psychopathology, given deficits in self-regulation prevalent in this population.

The current study proposed and evaluated a theoretical model of distal and proximal risk factors for internalizing and externalizing pathology development in youth with ADHD. Specifically, this study investigated the influence of reactive and regulative temperament dimensions and emotion regulation on concurrent internalizing and externalizing pathology through utilization of hierarchical regression and path analyses. It was hypothesized that emotion regulation would emerge as a transdiagnostic mechanism to explain the relationship between temperament and psychopathology in youth with
ADHD. Participants were 46 children ages 9-13 with ADHD and their parents, recruited from the community. Both children and parents completed measures to assess temperament, emotion regulation, and psychopathology. Children completed two tasks; a Stroop task and an emotional go/no go, to assess attentional control and inhibitory control regulation dimensions. Hypotheses were partially supported. Results of path analyses indicated emotion regulation explained the relationship between temperamental inhibitory control and broad psychopathology. Additionally, emotion regulation also explained the link between temperamental negative affect and externalizing pathology, but not internalizing pathology. Instead, temperamental negative affect directly estimated internalizing pathology among youth with ADHD. Children who self-reported higher emotion dysregulation performed worse on the laboratory inhibitory control task, but not the laboratory attentional control task. Findings are discussed in terms of theoretical and clinical implications for future research investigating psychopathology development among youth with ADHD.
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CHAPTER I
INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD), one of the most prevalent neurodevelopmental disorders of childhood, (e.g., affecting 5% of youth; APA, 2013) is associated with high comorbidity rates. Approximately 40-70% of children with ADHD are diagnosed with at least one comorbid internalizing (e.g., generalized anxiety disorder, depression) or externalizing disorder, (e.g., oppositional defiant disorder) and a significant portion of these children have diagnoses of both comorbid internalizing and externalizing disorders (Cuffe et al., 2017; Larson, Russ, Kahn, & Halfon, 2011).

Comorbidity among children with ADHD is associated with poorer overall functioning, increased utilization of health and education services, increased family conflict, and greater academic and social impairment (Larson et al., 2011). Youth with ADHD are at great risk for the development of comorbid disorders and associated negative outcomes that significantly impair functioning throughout the lifespan.

In a study examining the developmental sequence and continuity of childhood disorders, no internalizing or externalizing disorders predicted ADHD; however, ADHD predicted the development of oppositional defiant disorder (ODD) which then predicted subsequent anxiety, depression, and conduct problems (Burke, Loeber, Lahey, & Rathouz, 2005). ADHD has been conceptualized as a neurodevelopmental disorder present early in life. Indeed, studies suggest ADHD symptoms are present before age
seven in most children with ADHD (Kieling et al., 2010). Hyperactive/impulsive ADHD symptoms have been found to predict externalizing symptoms (e.g., oppositionality, argumentativeness, and defiance) in early childhood (Burke et al., 2005.) However, internalizing problems such as anxiety and depression typically present in later childhood or early adolescence (APA, 2013). Researchers have hypothesized that affective aspects of ODD such as anger, irritability, and blame may be predictive of internalizing disorders (e.g., depression, anxiety) while behavioral aspects of ODD such as spitefulness and vindictiveness may predict conduct problems (Burke et al., 2005). Thus, according to this model, affective and/or behavioral symptoms may present secondary to ADHD and predict the development of internalizing and/or externalizing disorders later in childhood. Given ADHD often presents earlier than other disorders of childhood, deficits inherent in ADHD may increase risk for the development of internalizing and externalizing comorbid pathology. Although the diagnostic criteria for ADHD (APA, 2013) primarily capture deficits in core symptoms of inattention, hyperactivity, and impulsivity, there is limited evidence core symptoms are directly associated with increased internalizing and externalizing disorder pathology. Rather, other deficits associated with ADHD, such as poor executive functioning, emotional and/or behavioral dysregulation have been proposed to relate to high comorbidity rates (Jonsdottir et al., 2006). Exploring these deficits may shed light on risk factors related to increased comorbidity rates in ADHD that lead to greater levels of impairment and poorer overall functioning. Identification of risk factors that may serve as the target of treatment interventions is critically needed for this population of children.
Although a range of genetic, biological, environmental, and social factors have been proposed as risk factors for psychopathology, temperament has been one of the most widely explored early biological indicators of childhood psychopathology. Temperament has been defined as a trait-like dimension that captures individual differences in reactivity (e.g., negative affect) and regulation (e.g., effortful control; Rothbart, 2007). Temperamental traits are present in infancy, relatively stable throughout childhood and adolescence, and are predictive of personality and psychopathology across the lifespan (Merviedle, De Clercq, De Fruyt, & Van Leeuwen, 2005). There has been minimal research exploring temperament as a predictor of psychopathology within an ADHD sample; however, a robust relationship exists between temperament and psychopathology among broad samples of children. Indeed, temperamental negative affect has emerged as one of the most salient predictors of both internalizing and externalizing pathology in youth (Kelvin, Goodyer, & Altham, 1996; Mikolajewski, Allan, Hart, Lonigan, & Taylor, 2013; Ormel et al., 2005). Additionally, research suggests that low temperamental effortful control, is also predictive of psychopathology in youth (Rettew & McKee, 2005). Effortful control is a regulatory temperament dimension that has been defined as the ability to inhibit a dominant, prepotent response and activate a subdominant response in order to achieve a goal (e.g., regulate attention or behavior.) It may be particularly relevant to explore mechanisms that may explain the link between temperamental traits and psychopathology among children with ADHD, given ADHD is a neurodevelopmental disorder associated with deficits in both attentional and inhibitory control regulatory processes (Nigg, 2006).
An abundance of research has indicated children with ADHD experience comorbid internalizing and externalizing pathology at disproportionate rates compared to typically developing children (Cuffe et al., 2017); thus, it is surprising that few models exist to account for the development of comorbid psychopathology in youth with ADHD. Emotion regulation has been proposed as one mechanism that may account for the link between temperamental traits and internalizing and externalizing pathology. Emotion regulation (ER) is a multidimensional construct defined as the ability to alter or modify the occurrence, intensity, valence, or duration of emotion through intrinsic and extrinsic multisystemic neurobiological, physiological, cognitive, and/or behavioral processes in order to adapt to environmental demands or to generate goal directed behavior (Eisenberg & Sprinrad, 2004; Gross, 1998; Thompson, 1991). Research has indicated that emotion regulation accounts for the relationship between temperamental negative affect and depression symptoms in children (Yap, Allen, & Sheeber, 2007). Additionally, high temperamental negative affect and low regulatory abilities are related to increased risk for externalizing behavior (Eisenberg et al., 2004). This research implicates both temperamental traits and emotion regulation as potential predictors of internalizing and externalizing pathology development; however, this has yet to be explored in an ADHD sample. The current study explored how temperamental reactivity and regulatory traits increase risk for emotion dysregulation and concurrent internalizing and externalizing pathology among children with ADHD.

Temperament and Self-Regulation in ADHD

Rothbart’s theory of temperament. Several theories of temperament exist. Rothbart’s tripartite model of temperament has received considerable attention in the
general child psychopathology literature (Rothbart, 2007). According to this model, temperment in childhood and adolescence is composed of three dimensions: *effortful control, surgency, and negative affect*. As defined above, effortful control involves the ability to modulate or inhibit a dominant attentional response (i.e., attentional control) and/or a dominant behavioral response (inhibitory control) and activate subdominant attentional and/or behavioral response in order to modulate reactivity (Capaldi & Rothbart, 1992; Rothbart, 2007). Surgency is a reactive temperamental dimension that is characterized by impulsivity, high novelty seeking behavior, and low fear (Dollar & Stifter, 2012). Negative affect, a broad reactivity temperament dimension, encompasses frustration, depressive mood, and fear reactivity traits. Both reactivity and regulatory temperamental dimensions have been proposed to predict psychopathology in children (Bradley 2000; Steinberg & Drabick, 2015); however, children with ADHD may be particularly vulnerable to experiencing poor temperamental effortful control due to regulatory deficits inherent in ADHD (Nigg, 2006).

**Temperamental regulation deficits in children.** Temperamental self-regulation has been defined in the literature as control and orienting processes necessary to modulate reactivity (Eisenberg, Eggum, Sallquist, & Edwards, 2010). The ability to self-regulate is critical to prevent maladaptive responding to emotional or negative stimuli encountered in daily life. The most consistent finding in the ADHD temperament literature has been that children with ADHD exhibit temperamental, biologically based deficiencies in effortful control, indicating they experience difficulties regulating attention and inhibiting behavior (De Pauw & Merviedle, 2011; Nigg, 2006). Indeed, trait theories of ADHD have recently emerged to theorize temperamental dimensions related
to the development of inattention versus hyperactive/impulsive ADHD symptoms. In a review of trait models, Martel (2009) theorizes effortful control is related to the development of ADHD inattentive symptoms while negative emotionality, or negative affect, is related to hyperactive/impulsive symptoms and the development of comorbid externalizing problems. Difficulties in effortful control are theorized to arise as a result of executive dysfunction inherent in ADHD (Martel, 2009). Given evidence of the strong relationship between ADHD symptoms and effortful control, it may prove useful to examine attentional and inhibitory control facets of effortful control separately to determine if specific temperamental regulation deficits are related to specific emotion regulation deficits and concurrent psychopathology symptoms among children with ADHD. Attentional and inhibitory control processes will be described in detail, as difficulties regulating attention and inhibiting behavioral responses are theorized to relate to emotion dysregulation (Gross, 1998).

Attention Regulation

Attentional network theory of attentional control. Attentional regulation, also termed attentional control (AC) is a complex, multidimensional construct that has been defined as the ability to selectively attend to and coordinate incoming stimuli in order to “maintain a calm state of mind, delay gratification, tolerate change, and create the cognitive and behavioral response to selected stimuli exclusively” (Luszczynska, Diehl, Gutierrez-Dona, Kuusinen, & Schwarzer, 2003). Thus, AC permits individuals to regulate attention in an adaptive manner to achieve a goal (e.g., ignore distracting stimuli in order to concentrate on a specific stimulus; allocate attention away from negative stimuli to reduce distress.) The construct AC captures several attentional processes that
will be described to provide a more thorough understanding of AC. The processes include attentional *alerting, orienting, and conflict resolution* (Johnson et al., 2008). The attentional alerting system allows one to maintain an alert state in order to attend to incoming stimuli, while the attentional orienting system is responsible for selecting incoming information to be further processed (Johnson et al., 2008). The conflict resolution system resolves conflict between competing stimuli (Johnson, 2008).

The attentional network theory provides a framework for understanding the role of neural executive attention networks in the regulation of attention (Rueda, Posner, & Rothbart, 2005). The anterior cingulate cortex (ACC) has been strongly implicated as the “main node” of the executive attention network, responsible for detecting and monitoring conflicting information (Rueda et al., 2005). The ACC is connected to limbic system structures responsible for processing of emotions; thus, the attentional network system becomes critical in the detection (i.e., alerting system) and modulation (i.e., orienting and conflict resolution systems) of emotional or potentially threatening stimuli (Rueda et al., 2005). The ACC is responsible for detecting conflicts in information processing and triggering top down control of attention and emotion. Research suggests deficient conflict monitoring of threat-related information is associated with high rates of anxiety (Bishop, Duncan, Brett, & Lawrence, 2004). Research examining AC broadly has indicated poor AC is prevalent in both children with ADHD and in children with internalizing problems (Muris, Mayer, van Lint, & Hofman, 2008).

**The measurement of AC in children with ADHD.** An abundance of research suggests children with ADHD demonstrate deficits in sustained attention, defined as the ability to pay attention for a prolonged period of time (APA, 2013). Deficits in sustained
attention are most often assessed through use of behavioral inhibition tasks (e.g., go/no go tasks, continuous performance tasks; Ridderinkhof, Wery, Wildenberg, Seglowitz, & Carter, 2004). These tasks require individuals to attend to a computer screen, selectively respond to relevant stimuli, and inhibit a response to irrelevant stimuli. The number of omission errors on these tasks (i.e., failures to respond to relevant stimuli) is often indexed as a measure of sustained attention (i.e., more errors represents poorer attention; Trommer, Hoeppner, & Armstrong, 1998). Children who exhibit deficits in sustained attention on executive functioning tasks demonstrate significantly greater rates of grade retention and poorer academic performance (Biederman et al., 2004). Sustained attention has likely been studied widely in the ADHD literature due to the association with academic impairment.

There has been minimal research applying the attentional network theory of AC to the child ADHD literature. Indeed, several researchers have commented on the “general failure” to define inattention within the ADHD literature (Huang-Pollock & Nigg, 2003; Mullane et al., 2010). Several cognitive tasks have been utilized to assess AC performance in the child temperament literature. In a review on temperament, development, and personality, Rothbart (2007) notes that laboratory based attentional tasks can be utilized to assess individual differences in effortful control. Particularly, tasks that require children to monitor and resolve conflict between interfering responses are linked to attention networks (Rothbart, 2007). Several tasks have been designed to assess conflict monitoring including the Stroop task, attention network test, and the flanker task (Rothbart, 2007). Neuroimaging studies have revealed activation in brain regions associated with top down control (e.g., dorsal lateral PFC, posterior inferior PFC).
and conflict monitoring (e.g., anterior cingulate cortex) during the Stroop task (Milham, Banich, & Barad, 2003). Research utilizing the Stroop task has indicated both children with ADHD and children with internalizing pathology (i.e., depressed mood, anxiety) exhibit task-based deficits in AC (Doost, Taghavi, Moradi, Yule, & Dalgleish, 1997; Homack & Riccio, 2004; Killgore, Gruber, Yurgelon-Todd, 2007). Despite the fact that research has identified deficits in AC span across both attentional and internalizing disorders of childhood, studies exploring the relationship between ADHD, AC, and internalizing pathology do not yet exist.

**Distal risk factors for poor AC in children with ADHD.** Conflict monitoring requires activation of the dorsolateral prefrontal cortex (PFC) and the ACC to facilitate top down control of attention and emotion (Rueda et al., 2005). An abundance of evidence has indicated that children with ADHD demonstrate deficiencies in frontal-striatal brain regions implicated in executive functioning, such as the PFC, orbitofrontal cortex, striatum, and ACC (Tripp & Wickens, 2009). Children with ADHD who exhibit deficits in executive functioning experience difficulties maintaining attention, task-switching, *modulating attention and arousal, and resolving conflict* (Castellanos et al., 2006). Interestingly, research has indicated that children with ADHD demonstrate significant weaknesses in broad executive functioning domains, yet “Executive functioning weaknesses are neither necessary or sufficient to cause all cases of ADHD” (Willcutt et al., 2005). For instance, in a meta-analysis of 83 studies assessing executive functioning utilizing 13 neurocognitive measures (e.g., stop signal reaction time task, continuous performance task, trailmaking test, tower of Hanoi, working memory span, etc.) in children with ADHD compared to controls ($n=3734$ with ADHD, $n=2969$
without ADHD), the weighted mean effect size across all comparisons was $d = .54$, while the weighted mean effect size for all measures ranged from $d = .43$ to $.69$ (Willcutt et al., 2005). This meta-analysis indicated a moderate effect size for EF measures in children with ADHD. Additionally, this research has indicated there is no “universal” executive functioning deficit in ADHD. Thus, although executive functioning deficits may contribute some variance in ADHD symptomatology, research suggests “one” executive functioning deficit cannot fully account for the heterogeneity in ADHD presentations.

A two-pathway model of ADHD has been posited to explain the heterogeneity in executive dysfunction that occurs in children with ADHD (Castellanos et al., 2006). According to this theory, executive functioning deficits can be distinguished as either “cool” or “hot.” The “cool” and “hot” systems are theorized as two neurobiological systems. The “cool” system, composed of the dorsolateral PFC, governs suppression of prepotent responses and maintenance of information in memory. This system is responsible for executive control and cognitive regulation. On the other hand, the “hot” system, composed of the orbital and medial prefrontal cortices, controls flexible appraisal of affect and modulation of arousal in situations with high affective involvement. Thus, this system is theorized to control the top down regulation of emotion (Castellanos et al., 2006). This research suggests that a subset of children with ADHD may experience deficits in cognitive control. Cognitive control has been defined as broad executive processes (e.g., goal and context representation, attention allocation, stimulus-response mapping) that permits for moment to moment (i.e., flexible) goal-directed behavior (Botvinick, Braver, Barch, Carter, & Cohen, 2000). Thus, cognitive control encompasses a wide range of cognitive processes rather than one specific cognitive process or domain.
AC is a more specific attentional process that captures the ability to modulate attention and resolve conflict. Although there has been extensive research exploring cognitive control and subsequent executive functioning deficits in children with ADHD, there has been substantially less research exploring more specific AC processes among children with ADHD. Inherent deficits in frontal striatal circuitry and brain structures implicated in conflict monitoring (e.g., ACC) may result in deficits in AC among children with ADHD. Deficits in AC appear to underlie both internalizing and attentional disorders of childhood (Muris et al., 2008). Research exploring the specificity of executive functioning deficits by examining AC among children with ADHD may shed light on the relationship between ADHD, AC, and internalizing pathology.

Inhibitory Control

Inhibitory control defined. Inhibitory control (IC) is a multisystematic and multidimensional construct that has been defined in the literature as the ability to suppress responses that interfere with task demands (Carlson & Wang, 2007; Rothbart & Posner, 1985). Nigg (2001) has theorized a two-process model of inhibition that conceptualizes two discrete IC processes 1) executive inhibition and 2) motivational inhibition (Nigg, 2001). Executive inhibition involves the inhibition of impulsive action by postponing, refraining from, or cancelling an action (e.g., inhibit an impulse to raise hand before teacher finishes asking question). Failures in executive inhibition result in an inappropriate motor response (Bari & Robbins, 2013; Nigg, 2001). Executive IC has been assessed through utilization of several neurocognitive tasks of response inhibition (e.g., go/ no go task, continuous performance task) in which children have to respond exclusively to target stimuli and inhibit a motor response (e.g., do no click computer
mouse) to irrelevant stimuli (Berlin, Bohlin, Nyberg, & Janlos, 2004). Failure to inhibit a
response (i.e., commission error) is often indexed as a measure of response inhibition.
Response inhibition requires cognitive control, broadly, to resist interference from
alternative responses within a timely manner (Ridderinkhof et al., 2004). Response
disinhibition is theorized to arise as a result of deficiencies in executive neurobiological
systems, such as the PFC and basal ganglia. Neuroimaging studies have revealed
activation of the dorsomedial PFC and ventrolateral PFC during response inhibition tasks,
indicating these tasks require cognitive control (Ridderinkhof et al., 2004).

Motivational IC refers to the ability to reduce or delay a behavioral response that
is driven by fear/anxiety or by cues for punishment (Nigg, 2001). For instance, a child
bullying a sibling may refrain from further provocation if the child receives a cue for
punishment (e.g., hears parents approaching.) A child with poor motivational IC may fail
to inhibit an impulse to further provoke a sibling even in the face of punishment. Nigg
(2001) highlights that motivational IC is highly influenced by negative emotion or
reward/punishment. Motivational IC is mediated by neurobiological structures implicated
in affective and reward processing such as subcortical and limbic structures (Nigg, 2001).
Failure of motivational IC results in inflexibility in response, impulsivity, poor decision-
making, and poor performance monitoring (Bari & Robbins, 2013). Research suggests
executive and motivational IC processes are impaired in both children with ADHD
(Nigg, 2001) and children with externalizing disorders (Hobson et al., 2011; Matthys et
al., 2012).

**Distal risk factors for poor IC in children with ADHD.** There has been a
movement in the ADHD field to explore a motivational pathway of ADHD that results
from altered reinforcement and reward processing (Sonuga-Barke, 2002).

Neurobiological theories highlight the role of limbic structures, such as the ventral-striatum, in dysfunctional reward processing (Plichta et al., 2009). The ventral striatum is a structure implicated in decision-making, risk, and, reward. Research suggests a subset of children with ADHD experience both 1) reward sensitivity and 2) hyporesponsiveness of the ventral striatum (i.e., reduced activity) during reward anticipation (Scheres, Milham, Knutson, & Castellanos, 2006; van Hulst et al., 2017). Both reduced activation of the ventral striatum and reward sensitivity are hypothesized to lead children with ADHD to prefer immediate rewards, discount the value of future rewards, and find delay aversive. As a result, these children often display poor IC in the face of reward/punishment. Studies have also highlighted the role of dopaminergic systems in contributing to deficient motivation and reward processing (Sagvolden, 2005). According to dopamine reward theories, dysfunction in the mesolimbic dopamine branch leads to altered reinforcement of novel behavior, which results in delay aversion, impulsiveness, and disinhibition (Sagvolden, 2005). In sum, hypofunctioning of limbic structures (e.g., ventral striatum) and dysfunction in mesolimbic dopamine systems have been theorized as distal risk factors that lead to altered reinforcement of behavior, and subsequent deficits in IC among children with ADHD. Additionally, as described previously, deficits in executive IC among children with ADHD may also arise as a result of deficits in cognitive control, inherent in a subset of children with ADHD (Castellanos et al., 2006).

**The measurement of IC in ADHD.** Temperamental IC has been assessed utilizing parent and child self-report measures. The IC temperament dimension assesses “the capacity to plan, and suppress inappropriate responses” (Capaldi & Rothbart, 2002).
In a validity study assessing the factor structure of the Early Adolescent Temperament Questionnaire (EATQ) in a large sample \((n = 1,055)\) of children and adolescents, IC was most highly correlated with aggression symptoms \((r = .49)\) followed by inattentive/hyperactive symptoms \((r = .44)\) indicating children with low temperamental IC demonstrate both greater externalizing and ADHD symptoms (Muris & Meesters, 2008).

Several task-based assessments have been utilized to measure IC in children. Go/no go tasks are a commonly used task paradigm to assess IC. Participants are continuously presented with frequent (i.e., occur for 75% of trials) “go” stimuli and less frequent (i.e., occur for 25% of trials) “no go,” stimuli (Schulz et al., 2007) and are required to respond to “go” stimuli exclusively. Commission errors (i.e., responding to “no go” stimuli”) are indexed as a measure of IC (Schultz et al., 2007). Classic go/no go paradigms utilize letters or picture stimuli. More recently, emotional go/no go paradigms have emerged to permit for the assessment of IC to affective stimuli. Emotional go/no go paradigms assess IC of affective stimuli by assessing participants’ responses to a variety of emotional valences (i.e., happy, sad, etc.; Schultz et al., 2007). Recently, an emotional go/no-go paradigm was adapted to assess response inhibition toward several facial cues in children with and without ADHD (Kochel, Leutgeb, & Schienle, 2014). Children with ADHD compared to controls made more commission errors on all emotional valences. The authors concluded that response inhibition to emotion signals is altered in children with ADHD (Kochel et al., 2014). Emotional go/no go tasks may prove useful to assess executive and motivational aspects of IC; yet, it is unclear if task-based assessments of IC correlate with temperamental measures of IC. However, it is evident that temperamental
IC is impaired in both children with ADHD and children with externalizing pathology (Muris & Meesters, 2008).

**Temperamental Reactivity and Emotion Regulation in ADHD**

**Temperament as a precursor for emotion dysregulation.** Temperamental theories provide a link between emotion-related traits and psychopathology. As described earlier, temperamental traits can be categorized as either reactive traits or regulative traits (Rothbart, 2007). *Negative affect is a reactive* temperamental trait composed of frustration, depressive mood, and fear traits (Hankin et al., 2017). Research suggests that children high in temperamental negative affect are more reactive, easily aroused, and have a tendency to experience negative moods such as sadness, worry, and irritability/anger (Hankin et al., 2017; Rothbart, 2007). An abundance of research has revealed the strong link between temperamental traits and psychopathology in youth (Hankin et al., 2017; Merviedle et al., 2005). For instance, in a recent study, Hankin et al. (2017) explored negative affect as a risk factor for general psychopathology (i.e., “p” factor composed of both internalizing and externalizing symptoms) in a large sample of youth. Results revealed that higher negative affect was associated with greater psychopathology (i.e., “p” factor.) The authors concluded that negative affect may be a “broad-based, transdiagnostic risk to child psychopathology.” Negative affect is a broad dimension in that it captures reactivity and arousal to both internalizing-driven emotions (e.g., sadness, anxiety) and externalizing-driven emotions (e.g., anger, irritability, frustration). Children who demonstrate reactivity to emotional stimuli may be at increased risk to experience strong and overwhelming emotions. Increased expression of negative emotions and a limited capacity to regulate strong internalizing and
externalizing-driven emotions (i.e., poor emotion regulation) may then, in turn, result in broad psychopathology (i.e., internalizing and externalizing problems.) Thus, emotion regulation may serve as one mechanism linking temperamental negative affect to broad psychopathology. Although the Hankin et al (2017) study and other similar studies have proven useful in identifying the link between temperamental traits and psychopathology, few studies have explored mechanisms that may explain this link. Research investigating mechanisms that may explain the link between temperament and psychopathology are particularly relevant in an ADHD sample, given high rates of comorbid psychopathology in youth with ADHD.

**Emotion Regulation**

Emotion regulation (ER) has been proposed as one mechanism that may explain the link between temperamental traits and psychopathology (Bradley, 2000). ER is multisystemic in that it involves modulation of emotions through neurobiological, physiological, cognitive, behavioral, and social mechanisms (Zeman et al., 2006). Neurobiological theories highlight the role of the amygdala and frontal brain regions in ER. These findings suggest that functional connectivity between the amygdala and frontal regions (i.e., dorsolateral PFC, dorsal medial PFC, orbitofrontal cortex) occurs during regulation of negative affect (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Morawetz, Bode, Baudewig, & Heekren, 2017.) More efficient coupling between the amygdala and frontal structures is hypothesized to relate to more effective and successful emotion regulatory capacities (Banks et al., 2007; Morawetz et al., 2017).

The polyvagal theory (Porges, 2001) has been proposed to explain the physiological basis of ER. Porges’ theory (2001) provides a link between autonomic
nervous system functioning and emotional/affective experience. According to the polyvagal theory, there are several hierarchical autonomic structures critical for regulating cardiac output in response to an environmental challenge to maintain homeostasis. One mechanism in particular, the “vagal brake,” is hypothesized to play a critical role in the modulation of cardiac output by either providing inhibitory or disinhibitory input to the heart to speed or slow heart rate. During an environmental challenge or when encountered with a perceived stressor, the “vagal brake” aids in modulation of heart rate through activation of the parasympathetic nervous system. Thus, the “vagal brake” plays a crucial role in promoting calming and self-soothing regulatory behavior. Dysfunction of the vagal brake triggers activation of a phylogenetically older system, the dorsal vagal complex, which then results in increased sympathetic nervous system activity such as increased heart rate, stimulation of sweat glands, and inhibition of the gastrointestinal tract. This theory highlights the significance of cardiac mechanisms critical for regulating physiological responses. Maladaptive regulation results in greater physiological symptoms of emotion dysregulation (e.g., fight or flight response.)

Cognitive behavioral theories of ER highlight several cognitive and response modulation processes necessary for efficient ER. One of the most prominent models of ER is Gross’s process model (1988). In his theory, Gross (1988) identifies multidimensional distal and proximal processes of ER that flow from “upstream strategies” (e.g., attentional deployment) to “downstream” responses (behavior change.) In this model, Gross proposes that individuals first have the opportunity to regulate emotions by selecting the situations to which they choose to attend. Attentional deployment is a distal strategy that involves selecting the aspect of the situation to which
one attends through use of strategies such as distraction, concentration, and rumination. These cognitive strategies permit individuals to focus on nonemotional aspects of the situation and move attention to or away from emotional aspects of a situation. Cognitive change can occur secondary, or proximal, to attentional deployment. During the cognitive change process, individuals can alter the meaning (e.g., through cognitive appraisal, cognitive reframing, etc.) they ascribe to the emotional situation. Individuals can then modulate their response to an emotional situation by altering expressive behavior (e.g., changing their facial expression,) or seeking external coping resources (e.g., using relaxation techniques). Thus, this research suggests ER is not only multisystematic, but also involves several multidimensional cognitive and behavioral processes (e.g., attentional control, cognitive change, behavioral response modulation.)

**ER in ADHD.** Recent literature suggests emotion dysregulation is present in a substantial portion of children with ADHD. Emotion dysregulation occurs when there are impairments in one or more of the multisystemic processes (e.g., neurobiological, physiological, cognitive, behavioral, social) that govern the ability to modulate an emotional state (Shaw et al., 2014). In a recent review of ER in children with ADHD, Shaw and colleagues (2014) revealed emotion dysregulation occurs in approximately 25-45% of children with ADHD. This subset of children demonstrates difficulties managing both positive and negative emotions. They often excessively display exuberance and excitement, which may present as emotional and behavioral immaturity (Bunford, Evans, & Wymbs, 2015). Alternatively, they also exhibit difficulties managing negative emotions such as anger, irritability, frustration, sadness, and worry (Bunford et al., 2015). Because children with ADHD often display high rates of both positive and negative
emotions, they often display a pattern of frequent, rapid, and intense shifts in emotions (i.e., emotional lability; Anastopoulos et al., 2011; Leaberry, Rosen, Fogleman, Walerius, & Slaughter, 2017; Sobanski et al., 2010). There has been a shift in the child literature to incorporate ER into models of psychopathology development, given research suggesting negative affect and emotion dysregulation span across multiple disorders of childhood (Zeman et al., 2006).

**Emotion dysregulation and internalizing and externalizing pathology in children with ADHD.** Research has revealed *independent* associations (i.e., ER linked to internalizing and ER linked to externalizing) between emotion dysregulation and comorbid internalizing versus externalizing pathology among children with ADHD. In one large study of 1186 children with ADHD (ages 6-18), the relationship between emotion dysregulation (measured utilizing a parent report measure) and comorbidity was assessed (Sobanski et al., 2010). Children with ADHD were classified as exhibiting either 1) low 2) moderate or 3) severe levels of emotion dysregulation. Children with ADHD exhibiting severe emotion dysregulation were significantly more likely to have a comorbid disorder. Seventy-nine percent of children with ADHD and severe emotion dysregulation exhibited comorbid ODD, 45% of children exhibited a comorbid anxiety disorder, and 22% of children exhibited comorbid depression. These results presented evidence that comorbidity among children with ADHD is associated with high rates of parent-reported emotion dysregulation (Sobanski et al., 2010).

Several other studies have indicated children with ADHD and either a comorbid internalizing or externalizing disorder display higher rates of emotion dysregulation (Anastopoulous et al., 2011; Leaberry et al., 2017; Spencer et al., 2011). In a study
utilizing ecological momentary assessment to provide an ecologically valid assessment of emotion dysregulation, children with ADHD and an internalizing disorder displayed high levels of emotional lability over a 28-day period (Leaberry et al., 2017). Children with ADHD and comorbid ODD also displayed high rates of emotional lability over time (Leaberry et al., 2017). Longitudinal research exploring the relationship between ADHD symptoms (time 1), emotion dysregulation (time 2), and depressive symptoms (time 3) have indicated that emotion dysregulation fully mediates the relationship between ADHD symptoms and later depressive symptomatology (Seymour, Chronis-Tuscano, Iwamoto, Kurdziel, & MacPherson, 2014). Emotion dysregulation may also serve as a mechanism to account for the relationship between ADHD symptoms and the development of ODD. For instance, in a longitudinal study assessing emotion dysregulation among children with ADHD, 57% of children with ADHD who displayed high rates of emotion dysregulation at baseline continued to exhibit severe emotion dysregulation at four-year follow-up (Biederman et al., 2012). These children who continued to demonstrate severe emotion dysregulation at follow-up demonstrated significantly higher rates of ODD compared to children with ADHD only (Biederman et al., 2012). Taken together, these findings implicate emotion dysregulation as a potential transdiagnostic risk factor that may increase risk for internalizing and/or externalizing comorbidity among children with ADHD.

**Emotion dysregulation as a transdiagnostic factor.** Transdiagnostic factors are useful to understand “fundamental processes underlying multiple disorders” (Nolen-Hokesma & Watkins, 2011). With the development of the Research Domain Criteria (RDoC), transdiagnostic factors have emerged as crucial to “bridge psychiatric
phenomena and biological substrates of behavior” (Krueger & Eaton, 2015). Additionally, transdiagnostic factors are useful to explain high rates of comorbidity between disorders (Nolen-Hokesma & Watkins, 2011). In a recent study, McLaughlin et al. (2011) examined the longitudinal relation between emotion dysregulation and psychopathology in adolescents. Results revealed that emotion dysregulation predicted both internalizing problems (e.g., anxiety symptoms) and externalizing problems (e.g., aggressive behavior) over time. Interestingly, no psychopathological symptoms predicted increased emotion dysregulation over time, suggesting a temporal relationship between emotion dysregulation and psychopathology development in which emotion dysregulation predicts subsequent development of psychopathological symptoms. This study provided preliminary evidence that emotion dysregulation may span across multiple internalizing and externalizing disorders of childhood. Given the transdiagnostic nature of ER and research revealing high rates of emotion dysregulation among children with ADHD and comorbid disorders (Anastopoulous et al., 2011; Leaberry et al., 2017; Seymour et al., 2014; Spencer et al., 2011), it is likely that emotion dysregulation may serve as a risk factor for both internalizing and externalizing pathology among children with ADHD. As discussed, temperament may serve as a distal risk factor for ER; yet, the relationship between temperament, ER, and internalizing and externalizing pathology has yet to be explored in a child ADHD sample.

**Temperament, ER Mechanisms, and Psychopathology in ADHD**

The link between low AC, ER, and internalizing pathology. In the child literature, internalizing pathology is a broad term to encompass symptoms of internal distress including problems with depression, dysthymia, low mood, guilt, fear, anxiety,
nervousness, and somatic concerns (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). Children encounter fearful stimuli, negative stimuli, and stimuli that could be interpreted as threatening in their daily lives. An abundance of evidence has indicated attentional biases toward threat confer risk for the development of internalizing pathology in youth (Bar-Haim et al., 2007; Bradley, Mogg, White, Groom, & Bono, 1999; Cisler, Bacon, & Williams, 2009). Several mechanisms have been hypothesized to link threat biases to internalizing pathology. Threat biases are thought to activate a “primal threat mode” (Beck & Clark, 1997). Activation of the “threat mode” leads to subsequent activation of cognitive (e.g., worry, rumination), behavioral (e.g., avoidance), and physiological systems (e.g., autonomic nervous system) in order to maintain safety and reduce threat (Beck & Clark, 1997). According to the multidimensional model of attention, threatening stimuli trigger physiological arousal, which then leads to the allocation of cognitive resources toward threat (Bar-Haim et al., 2007). If a situation is judged as a “high threat situation,” attention is maintained on threat, which increases anxiety. Emerging research suggests that among children who exhibit deficits in information processing (e.g., bias toward threatening stimuli), only children with deficient AC develop internalizing pathology (Susa, Pitica, Benga, and Miclea, 2012). The ability to control and refocus attention away from potential threat appears to be an important self-regulatory function that protects against the development of internalizing pathology.

*ER may be another important mechanism linking temperamental reactivity and regulatory dimensions to internalizing pathology.* Research suggests that children high in temperamental reactivity 1) are more prone to experience feelings of sadness, frustration, and fear and 2) are more easily aroused in the face of emotional stimuli (Hankin et al.,
2017; Muris et al., 2007). These children are at greater risk of experiencing heightened physiological arousal, emotionality, and a depletion of cognitive resources to modulate negative emotions such as sadness and worry. Difficulties regulating sadness and worry may result in the expression of subsequent internalizing pathology such as low mood, nervousness, and social withdrawal. Children with ADHD who exhibit temperamental negative affect are also hypothesized to be more reactive and aroused in the face of emotional stimuli. Increased physiological arousal and stress reactivity to negative emotional stimuli leads to a “fight or flight” response and heightened negative emotions (Compas et al., 2004). According to Gross’s process model (1998), the ability to control and deploy attention is an important “upstream” ER process that then influences a “downstream” cognitive response process. Children who exhibit difficulties in AC may attend to and focus on negative stimuli, emotions, or hyperarousal cues; thus, they exhibit fewer resources to utilize adaptive cognitive ER strategies such as cognitive appraisal or positive refocusing. The inability to implement adaptive ER strategies may result in the experience of intense negative affect. Children with ADHD who are highly aroused, exhibit poor AC, and have fewer cognitive resources to utilize adaptive cognitive strategies may attempt to utilize maladaptive, overcontrolled ER strategies such as avoidance strategies, catastrophizing, or ruminative strategies to cope with intense negative emotions such as worry or sadness. These strategies are known to reduce intense negative emotions in the moment (e.g., momentarily avoid distress); however, over time, continued use of avoidance and other maladaptive strategies (e.g., rumination) increase risk for internalizing pathology (Garnefski et al., 2005). Thus, high temperamental negative affect and low temperamental AC may lead to an overcontrolled and
maladaptive pattern of emotion dysregulation that increases risk for internalizing pathology in children with ADHD; however, this has yet to be explored within an ADHD sample. Given executive functioning deficits inherent in ADHD, poor AC may be more salient in this population; thus, the relationship between AC, ER, and internalizing pathology is important to explore.

The link between IC, ER, and externalizing pathology. Externalizing pathology is a broad term used to encompass disruptive behavior symptoms such as oppositionality, defiance, and aggression. For the purpose of this study, externalizing pathology does not refer to conduct symptoms/antisocial behavior, given research suggesting conduct disorder may arise from callous/unemotional traits, rather than a broad negative affect dimension (Frick & White, 2008). Research has indicated children with conduct disorder may exhibit shallow or deficient affect rather than negative affect (APA, 2013). Alternatively, children who exhibit oppositional defiant disorder or children who display high rates of reactive aggression and behavior problems are more likely to experience externalizing symptoms that are driven by negative emotionality (Singh & Waldman, 2010; Stringaris, Maughan, & Goodman, 2010). This research suggests that oppositionality and conduct pathology may occur as a result of differential affective processes.

Neurobiological research indicates children with externalizing disorders demonstrate impaired cognitive control, altered punishment processing, and dysfunctional reward processing as a result of impairments in social learning (Matthys et al., 2012; Matthys, Louk, Vanderschuren, & Schutter, 2013). Due to alterations in punishment processing, children with externalizing disorders are thought to be less
sensitive to punishment and fear cues (Matthys et al., 2012). Sensitivity to punishment
and fear cues allow children to learn to refrain from inappropriate behaviors. Thus,
children with externalizing disorders who demonstrate deficiencies in punishment
processing have more difficulties learning to refrain from engaging in inappropriate
behavior (Matthys et al., 2012). Reward sensitivity is another process that is important in
shaping and reinforcing appropriate behavior. Due to deficiencies in reward processing,
children with externalizing behavior may demonstrate less motivation to obtain natural
rewards (Matthys et al., 2012). Additionally, children who demonstrate deficits in
cognitive control processes have difficulties inhibiting behavioral responses, which may
lead children to act on, rather than inhibit, inappropriate responses. In their review on
these three mechanisms (i.e., punishment processing, reward processing, cognitive
control), Matthys et al. (2013) indicated children with externalizing behavior are
impaired on all three social learning domains. These impairments lead to difficulties
making associations between behavior and consequences, prevent learning of appropriate
behavior, and lead to increased inappropriate behavior and impaired problem solving
(Matthys et al., 2013).

Research suggests children with externalizing pathology exhibit poor IC,
particularly in situations that provoke affective and reward systems (Matthys et al., 2012;
Hobson et al., 2011). Studies investigating IC and psychopathology in broad samples of
children have indicated children high in negative emotionality and low in IC experience
elevated rates of externalizing pathology because they are more likely to act on, rather
than inhibit behavioral responses when encountering emotional stimuli (Eisenberg et al.,
2005). The ability to self-regulate behavior (i.e., inhibitory control) appears to be
protective against the development of externalizing pathology (Eisenberg et al., 2004). For children with ADHD who exhibit deficient motivation and reward processing and subsequent impulsivity, the ability to regulate behavior is particularly important during highly emotional situations in which children may be prone to act on negative emotions.

**Negative urgency, ER, and externalizing pathology.** One potential mechanism that may explain high rates of externalizing pathology, that may be particularly salient in children with ADHD, is *negative urgency*. Negative urgency has been defined as “the tendency to engage in rash action in response to extreme negative affect” (Cyders & Smith, 2008). Negative urgency provides an explanation for how *emotionality/affect is linked with behavior*. According to the theory of urgency, emotions are linked to rash behavior through several mechanisms (Cyders & Smith, 2008). First, experiencing extreme emotions lead individuals to focus on the immediate, emotional situation. Focus on the immediate may be adaptive in some scenarios (e.g., to avoid a threat). However, focus on an immediate emotional situation can also be maladaptive, as it can lead to the depletion of cognitive resources necessary for rational decision-making. Thus, heightened extreme negative affect leads to increased risky or maladaptive behavior through depletion of cognitive resources necessary for decision-making. Additionally, this theory purports that rash acts are often reinforcing by either reducing distress (i.e., negative reinforcement) or satisfying an urge (i.e., positive reinforcement); thus, maladaptive responding to emotion is reinforced over time.

The theory of negative urgency has received minimal attention in the child literature; however, Cyders and Smith (2008) have suggested that temperament may serve as a risk factor for negative urgency. They posit that a temperamental disposition
toward negative affect (also termed “emotionality”) may interact with poor behavioral regulation (e.g., IC) to predict emotion-based rash action over time (Cyders & Smith, 2008). The authors also posit that temperamental negative affect and poor behavioral regulation impedes the ability to learn adaptive ER strategies (Cyders & Smith, 2008). Based on this theory, it is highly plausible that children with a biological vulnerability to experience emotions with high arousal (i.e., temperamental negative affect) that demonstrate a limited repertoire of cognitive and behavioral strategies to regulate arousal and extreme emotions adaptively, experience a pattern of severe emotion dysregulation.

Thus, temperamental negative affect and poor behavioral control (i.e., IC) may interact to produce emotion dysregulation and subsequent externalizing pathology; however, this process has also yet to be explored in a child ADHD sample.

**The Current Study**

Research has indicated that temperament and ER are both important predictors of children’s attentional, behavioral, and mental health outcomes (Steinberg & Drabick, 2015). While the link between temperamental traits and psychopathology is well established (Rettew & McKee, 2005), it is unclear what specific mechanisms explain this relationship. It is likely that ER may serve as one mechanism linking reactive and regulative temperament traits to psychopathology. ER appears to be a transdiagnostic process; the inability to regulate emotional reactivity is related to both internalizing and externalizing pathology in children (McLaughlin et al., 2011). Most of the research on temperament and psychopathology development has been conducted in broad populations of children. Few attempts have been made to apply this research to children with ADHD, despite the high rates of comorbid internalizing and externalizing pathology in this
population. Such studies are needed as ER processes may either confer risk for or serve as protective factors against the development of psychopathology among children with ADHD who exhibit difficult temperaments.

The current study examined the relationship between temperament, ER, and concurrent internalizing and externalizing pathology among children with ADHD. Pre- and early-adolescence appears to be a critical period for developing the ability to regulate emotions across multiple systems (i.e., cognitive, physiological, behavioral; Zeman et al., 2006). This also appears to be a developmental period in which internalizing and externalizing pathology crystalize. Temperamental reactivity and regulation dimensions likely contribute to differences in children’s ER capacity. Children with ADHD are at increased risk for deficits in effortful control due to executive control and motivational deficits inherent in ADHD (Nigg, 2006). Understanding how children with ADHD who exhibit high negative affect and poor regulatory abilities are able to modulate their emotions may inform our understanding of how temperament and internalizing and/or externalizing pathology relate. The current study aimed to address a significant gap in the ADHD field, as few models currently exist to account for internalizing and externalizing psychopathology development within an ADHD sample.

**Study Aims and Hypotheses**

**Aim 1.** Examine the relationship between reactive (i.e., negative affect, negative urgency) and regulative (i.e., IC, AC) temperament dimensions and emotion dysregulation in children with ADHD.

**Hypothesis 1a.** Temperamental negative affectivity (broad reactive dimension) and trait negative urgency will estimate emotion dysregulation among
children with ADHD.

**Hypothesis 1b.** Temperamental AC and IC will estimate emotion dysregulation among children with ADHD.

**Aim 2.** Examine the correlation between temperamental AC and IC to task-based AC and IC. Examine the relationship between parent and child report of emotion dysregulation to laboratory task-based AC and IC.

**Hypothesis 2a.** Parent and child-report of temperamental AC will correlate with performance on a task of AC. Parent and child-report of temperamental IC will correlate with performance on a task of IC.

**Hypothesis 2b.** Parent and child report of emotion dysregulation will estimate task-based performance on the AC and IC tasks.

**Aim 3.** Test the hypothesized link between ER and internalizing and externalizing pathology in children with ADHD.

**Hypothesis 3a.** Emotion dysregulation will emerge as a transdiagnostic factor. Emotion dysregulation will estimate both internalizing and externalizing pathology among children with ADHD.

**Aim 4.** Test the hypothesized link between temperament, ER, and psychopathology. The proposed theoretical model appears in Figure 1.

**Hypothesis 4a.** Indicators of temperament (negative affect, AC/IC), and indicators of emotion dysregulation will estimate higher internalizing pathology among children with ADHD. ER will emerge as a mechanism to explain the relationship between temperament and internalizing pathology. The hypothesized model is depicted in Figure 2.
**Hypothesis 4b.** Indicators of temperament (negative affect and/or negative urgency, AC/IC) and emotion dysregulation will estimate higher externalizing pathology among children with ADHD. ER will emerge as a mechanism to explain the relationship between temperament and externalizing pathology. They hypothesized model is depicted in Figure 3.
CHAPTER II

METHOD

Participants

Fifty children between the ages of 9-14 years with ADHD and their parents were recruited from community populations in Louisville, Kentucky. Parents completed a phone screening to determine eligibility before they were invited to participate in the study in the lab. Eligibility was limited to children who had a previous diagnosis of ADHD or who were showing clinically concerning symptoms for ADHD. Participants were excluded if they had a pre-existing diagnosis of autism spectrum disorder or intellectual disability, as these disorders would interfere with the child’s ability to understand all instructions and complete all tasks. Additionally, children were excluded from the study if they had visual impairment (e.g., color blindness) that prevented them from being able to complete computerized assessment tasks. For parents and children invited to participate in the study, a diagnostic screening was conducted to determine if children met criteria for a diagnosis of ADHD. Three children did not meet full criteria for ADHD and were excluded from further analyses. Only one 14-year-old child completed the study; thus, this child was excluded from any further analyses, given this child’s age was not representative of the sample. Thus, the final sample of children included in data analyses were 46 children (n= 26 males, n= 20 females) ages 9-13 (M age 10.65) with ADHD.
The ethnic composition of the sample (67.4% Non-Hispanic White/Caucasian, 21.7% African American/Black; 2.2% Hispanic/Latino; 2.2% Asian/Pacific Islander; 6.5% Biracial) was reflective of the larger Louisville/Jefferson County population. The ethnic composition of Louisville/Jefferson County is as follows: 68.3% Non-Hispanic White/Caucasian, 22.9% African American/Black, 4.5% Hispanic/Latino, and 2.3% Asian/Pacific Islander (United States Census Bureau, 2010). Demographic characteristics of the sample are presented in Table 1.

Twenty-one children (45.7%) met criteria for at least one comorbid internalizing or externalizing disorder; 16 (34.8%) children met full criteria for an internalizing disorder (social anxiety disorder, separation anxiety disorder, generalized anxiety disorder, major depressive disorder, dysthymia) and 14 children (30.4%) met full criteria for comorbid oppositional defiant disorder. The breakdown by disorder is presented in Table 2.

**Recruitment**

Children who had been diagnosed with ADHD or who were showing symptoms of ADHD were recruited. Flyers describing the study were distributed to child health service and mental health service providers and organizations (i.e., pediatricians, child evaluation clinics, child and family mental health clinics, etc.), child and family community-based organizations, and school counselors. Flyers were sent directly to providers/organization for distribution to parents of children within the study’s targeted age range and range of clinical difficulty. Flyers were distributed by the organization/provider to the parents, and referred parents to contact study staff directly to receive additional information regarding the study. Study personnel did not have any
direct contact with children or students during the flyer distribution process. All flyers were worded in such a way that specifically recruited children with diagnosed or suspected ADHD. Study staff also contacted parents of children who had participated in previous studies in the lab and had provided consent to be contacted for additional studies. Four children in the current study participated in previous lab studies. All parents completed a phone screening process to determine study eligibility before scheduling an appointment in the lab.

**Procedures**

All study procedures were approved by University of Louisville’s Institutional Review Board. Parents and children completed a single session in the lab lasting approximately 2.5 to 3 hours in duration. During the session, parents provided informed consent prior to the initiation of study procedures. Children provided assent for study participation. Parents were administered the Diagnostic Interview Schedule for Children Parent-Report (DISC-P; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) to determine if their child met criteria for ADHD and internalizing and/or externalizing diagnoses. While parents completed the diagnostic interview with the experimenter, children completed questionnaires regarding their temperament, ER, and internalizing and externalizing symptoms. Questionnaires were completed on RedCap, a secure web-based application for collecting research data. After the administration of the diagnostic interview, parents completed questionnaires on RedCap to assess their child’s ADHD symptoms, temperament, ER, and internalizing and externalizing symptoms. While parents completed questionnaires individually, children completed the computerized attentional and inhibitory control tasks with the experimenter. The order of presentation
of the two computerized tasks was counterbalanced. Participating families received a $10 prepaid card and children were provided with a small prize as a reward for participation.

**Measures**

**Demographic Measures.**

**Demographic Questionnaire.** The demographic questionnaire was designed specifically for this study. The demographic questionnaire was completed by parents to provide child demographic data including child age/date of birth, race, family socioeconomic status, and relevant psychiatric/neuropsychiatric history (i.e., previous psychiatric diagnoses, active medication status.)

**Pubertal Development Scale (PDS, Peterson et al., 1998).** The PDS is a 5-item parent report measure that assesses children’s pubertal development stage. The PDS consists of separate items to assess pubertal development in males and females. Items are averaged to create an average pubertal development rating. The PDS has been extensively used in a wide range of studies as a non-invasive means of assessing children’s pubertal development (Peterson et al., 1998).

**Diagnostic Interview.**

**Diagnostic Interview Schedule for Children-Version IV, Parent Report (DISC-P).** Parents were administered the DISC-P (Shaffer et al., 2000), a diagnostic structured interview using parent responses to determine whether children met DSM-IV criteria for a number of psychological disorders. The DISC-P has not yet been updated to reflect DSM-5 criteria. The DISC-P contains algorithms to generate diagnoses, based on rules similar to those published in the American Psychiatric Association’s Diagnostic and Statistical Manual, Fourth Edition (American Psychiatric Association, 1994). The DISC-
P was used to determine children’s ADHD diagnostic status by assessing for the presence of inattentive, hyperactive, and impulsive symptoms and the degree of impairment caused by symptoms. The DISC-P required parents to report on whether symptoms of ADHD are present across multiple settings (e.g., home, school, other.) The DISC-P was also used to determine if children met criteria for internalizing and/or externalizing disorder diagnoses. Parents were administered DISC-P modules for the following internalizing and externalizing disorders: separation anxiety disorder, social phobia, generalized anxiety disorder, depression, dysthymia, and oppositional defiant disorder. Research indicates that the DISC-P is considered reliable and valid across numerous settings (Shaffer et al, 2000).

**Temperament Measures.**

*Early Adolescent Temperament Questionnaire Revised-Parent and Child Report* (EATQ; Capaldi & Rothbart, 1992; Ellis & Rothbart, 2001). The EATQ is a well-validated questionnaire that assesses child temperament. The EATQ consists of parallel child self-report and parent report forms (62 items for parent report; 65 items for child report) that ask parents and children to rate on a 5-point scale ranging from 1 (almost always untrue) to 5 (almost always true) the extent to which each temperamental trait is true. The EATQ yields three major subscales, Negative Affect, Surgency, and Effortful Control. Negative Affect and Effortful Control major subscales (i.e., Attentional Control and Inhibitory Control subscales) were utilized for the current study. For each subscale, items are averaged to create a subscale score that represents the average item score. The revised EATQ has been validated for use in children ages 8-15 and has demonstrated good internal consistency and test-retest stability (Ellis & Rothbart, 2001).
In previous studies, scale alphas on the revised EATQ ranged from .65 to .86 (Capaldi & Rothbart, 1992; Ellis & Rothbart, 2001; Muris & Meesters, 2009). In the current study, scale alpha’s for the EATQ-parent measures were EATQ Negative Affect \( \alpha = .74 \), Effortful control \( \alpha = .74 \); scale alpha’s for the EATQ-child report were cEATQ Negative Affect \( \alpha = .85 \) and cEATQ Effortful Control \( \alpha = .74 \).

_Urgency, Premeditation, Perseverance, Sensation Seeking, and Positive Urgency_ Impulsive Behavior Scale – Child Report (UPPS; Zapolski, Stairs, Settles, Combs, & Smith, 2010). The UPPS Impulsive Behavior Scale – Child report is an adaptation of the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001). The total number of items was reduced, and the language of remaining items was modified to meet a fourth grade reading level. The UPPS is a 40-item measure assessing negative urgency, lack of premeditation, lack of perseverance, and sensation seeking. Children are asked to rate on a 4-point Likert scale (“Agree Strongly,” “Agree Some,” “Disagree Some,” or “Disagree Strongly”) how much they believe each statement is true of them. For each subscale, items are averaged to create a subscale score that represents the average item score. Cronbach’s alphas in previous studies were .90 (sensation seeking), .87 (negative urgency), .84 (lack of planning), and .81 (lack of perseverance; Zapolski, Stairs, Settles, Combs, & Smith, 2010). In ADHD samples, the UPPS has been shown to differentiate between ADHD subtypes (Miller, Derenfinko, Lynam, Milich, & Filmore, 2010). In the current study, only the negative urgency subscale was used, \( \alpha = .79 \).

_Emotion Regulation Measures._

_Cognitive Emotion Regulation Questionnaire-kids_ (CERQ-k; Garnefski & Kraaij, 2005; Garnefski, Kraaij, & Spinhoven, 2002). The CERQ-k is a 36-item self-
report inventory that asks children to rate on a five-point scale ("almost never" to "almost always") how frequently they use cognitive ER strategies to cope with unpleasant or negative events. Nine subscales are derived from this measure. Each subscale represents a cognitive coping strategy: self-blame, acceptance, rumination, positive refocusing, refocus on planning, positive reappraisal, putting into perspective, catastrophizing, and other blame. Items are summed to create subscale scores. In previous studies, Cronbach’s alpha coefficients for the nine subscales range from .68 to .83 (Garnefski et al., 2002). The CERQ-k has demonstrated reliability and validity in the assessment of cognitive ER strategies (Garnefski & Kraaij, 2005). The CERQ-k has not yet been validated in an ADHD sample; thus, Cronbach’s alpha was calculated to determine subscales with adequate internal consistency. In the current study, Cronbach’s alpha coefficients ranged from α= .50 to .84. The acceptance (α=.51) and rumination (α=.50) subscales had poor Cronbach’s alpha coefficients and were removed from further analyses. Seven subscales were retained; coefficient alphas ranged from .61 to .84, consistent with previous studies (Garnefski et al., 2002).

Given the large number of subscales and the need to ensure adequate power to conduct analyses using the CERQ-k measure, the subscales were composited to create two higher order factors, an Adaptive cognitive ER factor and a Maladaptive cognitive ER factor. This methodology has been utilized in other studies to provide more parsimonious adaptive and maladaptive ER factors (Aldao, & Nolen-Hoeksema, 2011). Correlations emerged as significant (r’s > .3 and p’s < .05) between four subscales, Positive Refocusing, Refocus on Planning, Positive Reappraisal, and Putting into Perspective, and were composited by averaging the subscales to create an Adaptive
factor. Additionally, correlations emerged as significant \((r's > .3\) and \(p's < .05\) among the three subscales, Self-blame, Catastrophizing, and Other Blame, that were composited by averaging the subscales to create the Maladaptive subscale. These correlations appear in Table 3. Internal consistency was good for the Adaptive scale, \(\alpha = .84\) and excellent for the Maladaptive scale, \(\alpha = .91\).

*Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997).* The ERC is a 24-item parent-report questionnaire that assesses parents’ perceptions of their children’s emotional negativity and ER abilities. Parents are asked to rate items on a four-point Likert scale regarding their child’s emotional responses, and responses yield the subscales Emotional Lability/Negativity, Emotion Regulation, and Total. The ERC has been used to assess ER in a wide variety of studies, and has demonstrated substantial reliability and validity in previous studies (Shields, & Cicchetti, 1997). Items are averaged to create an average total score. The Cronbach’s alpha coefficient in the current study for the ERC total score was \(\alpha = .85\).

*Emotion Regulation Index for Children and Adolescents (ERICA; MacDermott, Gullone, Allen, King, & Tonge, 2010).* The ERICA is a self-report adaptation of the ERC designed to assess children’s perceptions of their ability to regulate and manage their emotions. The ERICA is a 16-item self-report inventory that asks children to rate their ER on a 5-point Likert scale (“Strongly Disagree” to “Strongly Agree”) and yields a general composite and three subscales: Emotional Control, Emotional Self-Awareness, and Situational Responsiveness. The ERICA has been extensively validated with children in the age range of the proposed study (MacDermott
et al., 2010). The Cronbach’s alpha coefficient in the current study for the ERICA general composite was \( \alpha = .79 \).

**Internalizing and Externalizing Pathology.**

*Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001).* The CBCL is a 113-item parent-report measure of children’s socioemotional and behavioral functioning that yields two composites (internalizing problems and externalizing problems.) The Internalizing Problems scale is composed of anxious/depression, somatic problems, and withdrawn concerns subscales. The Externalizing Problems scale is composed of aggressive behavior and rule-breaking behavior subscales. The CBCL-Internalizing and CBCL-Externalizing scales were used in the current study to assess child internalizing and externalizing pathology. The CBCL has demonstrated high test-retest reliability and good internal consistency. The coefficient alpha on the internalizing and externalizing problem scales in previous studies were .90 and .94 (Achenbach & Rescorla, 2001). In the current study coefficient alpha’s were \( \alpha = .88 \) on the internalizing problem scale and \( \alpha = .87 \) on the externalizing problem scale.

*Children’s Depression Inventory-10 Item Short Form (CDI-S; Kovacs, 1992).* The CDI is a well-established self-report inventory designed to assess depression in children ages 7 to 17. Children rate symptoms of depression on a three point rating scale. Items are summed to create a total score. The CDI-S is a 10 item short form of the CDI that provides a brief assessment of depression symptoms in children. The CDI and CDI-S have demonstrated substantial reliability and validity across a range of studies (Kovacs, 1992). In the current study, the Cronbach’s alpha coefficient for the total score was \( \alpha = .85 \).
**Multidimensional Anxiety Scale for Children – 10 Item Short Form (MASC; March, Sullivan, & Parker, 1990).** The MASC is a well-established self-report inventory designed to assess anxiety in children ages 8 to 16. Children rate symptoms of anxiety on a four point rating scale. Items are summed to create a total score. The MASC-10 is a 10-item short form of the MASC that provides a brief assessment of anxiety symptoms in children. The MASC and MASC-10 have demonstrated substantial reliability and validity over a broad range of studies (March et al., 1990). In the current study, the Cronbach’s alpha coefficient for the total score was $\alpha = .76$.

**Reactive-Proactive Anger Questionnaire (RPAQ; Raine et al., 2006).** The RPAQ is a 23 item self-report inventory that asks participants to rate on a three-point scale (‘never’ to ‘often’) how frequently they act in an angry or aggressive manner towards other children. Two subscales are derived from this measure: Reactive Aggression and Proactive Aggression. Only the Reactive Aggression subscale was used in the current study. The RPAQ has demonstrated reliability and validity in the assessment of emotional reactivity and behavior in a wide variety of studies (Bas & Yurdabakan, 2017; Ollendick, Jarrett, Wolff, & Scarpa, 2009; Raine et al., 2006). In the current study, the Cronbach’s alpha coefficient for the reactive aggression subscale was $\alpha = .88$.

**Attentional and Inhibitory Control Tasks.**

**Stroop Color and Word Test (Stroop Test).** The Stroop Test is a well-validated computerized paradigm that has been used extensively to assess AC in both children and adults; it is often considered the “gold standard” method of assessing attention (MacLeod, 1992). In the classic Stroop Test, participants are presented with lists of words
for colors (e.g., BLUE, GREEN, YELLOW, RED). During congruent trials, the word “BLUE” will appear in blue colored font. During incongruent trials, the word “BLUE” will appear in an incongruent font color (e.g., green.) Thus, for incongruent trials, participants are required to control their attention in order to respond exclusively to the color of the font, and not the written word. Participants must shift their attention to changing demands. On incongruent trials, participants are both more prone to errors and take longer to respond. The task has been designed to run in E-Prime, a psychology software tool used to collect behavioral experimental data (Psychology Software Tools, Inc., Pittsburgh, PA; Schneider, et al., 2002).

Participants completed a classic Stroop paradigm to assess AC. Participants completed one practice block composed of 48 trials, which lasted for approximately 2 minutes in duration. Participants then completed a trial block, composed of 140 trials, presented in a randomized order. The trial block lasted approximately 6 minutes in duration. Color word stimuli were presented every 2500 ms. Four colors (RED, BLUE, GREEN, YELLOW) were presented. There were three stimulus conditions. Stimuli were either congruent (e.g., word YELLOW presented in yellow colored font), incongruent (word YELLOW presented in red colored font), or neutral, color/non-color word (e.g., the word LEAF presented in yellow.) Participants were prompted to press a button on the keypad corresponding to the appropriate color. If participants did not respond to a trial, a message stating, “No response detected” appeared on the screen and the next trial began. The Stroop test has been utilized extensively to examine executive functioning in children with ADHD (Homack & Riccio, 2004).
Task administration. After completing self-report measures, children completed the Stroop Test. Children were seated at a small table across from the examiner. The examiner provided the child with the computerized Stroop Test using E-Prime. After placing the computer in front of the participant, the examiner stated, “You’re going to play a computer game where your job is to pay attention to colors and press the correct button on this keypad. First, read this list of words to me out loud.” Participants read the list of color words to ensure reading ability necessary for the task. Then, participants were shown pictures of the four colors and were asked to name the four colors to ensure correct color naming. Then, participants were shown cue cards of color words. They were also shown the buttons on the keypad corresponding to each color. A colored sticker was placed on each button (red, blue, yellow, green.) The experimenter then stated, “When you see a word appearing in the color red, press this button here (points to red button.)” This instruction were repeated for each of the four colors (red, blue, green, yellow.) Participants were asked to demonstrate that they understood which button they were required to push on the keypad.

Once the participant demonstrated understanding of this part of the task, the examiner stated, “Sometimes the color and the word will match (show relevant cue card.) Sometimes the color and the word will not match (show relevant cue card.) Sometimes the word will not be a color word (show relevant cue card.) Your job is to only respond to the color, so what matters is the color of the word.” Participants were asked to demonstrate which button they would press for each example.

Once the participant had established understanding of the task directions, the participant completed practice trials. After completion of the practice trials, the examiner
stated, “Great, now you understand how to do the task. Now you will complete the game. Try to respond to the words as quickly as you can, but not so fast that you make many mistakes.” Participants completed the test trials on their own. The entire task duration, with instructions, lasted about 10 minutes in duration.

Data reduction. Accuracy and reaction time were recorded for each trial type (congruent, incongruent, neutral.) A Stroop interference effect was calculated as an index of AC. Numerous studies support the use of the interference effect as a measure of AC (Bush et al., 1999; Eysenck, Derakshan, Santos, & Calvo, 2007; Moore, Gruber, Derose, & Maliniwski et al., 2012). Several methodologies have been utilized to calculate an interference effect on the Stroop task (Lansbergen, Kenemans, & van Engeland, 2007; Scarpina & Tagini, 2017). In a meta-analysis exploring various methods of calculating the Stroop effect among children with ADHD, Lansbergen et al. (2007) highlight the importance of utilizing a ratio score as this allows the researcher to control for reaction time. The interference ratio score was calculated by taking the difference between the mean reaction times of the incongruent condition and the neutral condition. This score was then divided by the reaction time for the neutral condition, which resulted in a ratio score that controlled for general differences in reaction time (Lansbergen et al., 2007).

Emotional Go/No Go Task. The Emotional Go/No-Go task is a computerized behavioral inhibition task that utilizes emotional stimuli. Most computerized behavioral inhibition tasks use a “classic go/no-go paradigm” in which participants are continuously presented with frequent (i.e., occur for 75% of trials) “go” stimuli and less frequent (i.e., occur for 25% of trials) “no go,” stimuli (Schulz et al., 2007). Participants are instructed to respond as quickly as possibly to “go” stimuli, and are instructed to not respond to “no
“go” stimuli while reaction time is monitored via a computerized program. For the classic go/no go paradigm, commonly used stimuli include letters or pictures. The Emotional Go/No-Go utilizes emotional stimuli to assess affective behavioral inhibition. Thus, this task permits for the assessment of emotional modulation of inhibition while assessing participants’ response to a variety of emotional valences (i.e., happy, sad, etc.; Schultz et al., 2007). Many variants of this task have been utilized to assess emotional processing in both healthy and clinical child and adult populations (Schultz et al., 2007).

Recently, the emotional go/no-go paradigm was utilized to assess response inhibition toward several facial cues in children with and without ADHD (Kochel, Leutgeb, & Schienle, 2014). Emotional stimuli, from 4 affective categories (i.e., happy, sad, anger, neutral) were presented in 4 randomized blocks (3 emotional and 1 neutral block.) For the current study, a similar (adapted from Kochel et al., 2014) emotional go/no task was created. Emotional stimuli from 4 affective categories (i.e., happy, afraid, anger, neutral) were presented in 4 randomized blocks (3 emotional, 1 neutral). At the beginning of each block, the “go” condition was displayed, followed by the “no go” condition, and then followed by a fixation cross. For each block, 60 faces were presented at a rate of 1000ms with an interstimulus interval of 1000ms. The ratio of go to no/go stimuli was 2/3 (e.g., 40 “go” stimuli and 20 “no/go” stimuli.”) This task was adapted to shorten the task, given all participants have ADHD or suspected ADHD and exhibit difficulties sustaining attention.

Task administration. The administrator provided the child with the computerized Emotional Go/No-Go task using E-Prime (Psychology Software Tools, Inc., Pittsburgh, PA; Schneider, et al., 2002). The experimenter remained seated across from the child.
The procedure was similar to the procedures outlined in Kochel et al. (2014). The stimuli consisted of children’s faces from the National Institute of Mental Health Child Emotional Faces Picture Set (NIMH-ChEFS; Egger et al., 2011). Stimuli from the different emotion categories (happy, afraid, anger, neutral) were presented in a randomized order via the E-Prime computer program in 4 blocks (happy, afraid, anger, neutral.) Block order was also counterbalanced and specific instructions were provided for each block. Children were instructed to click a button on the computer for “go” stimuli and were instructed to refrain from clicking the button for “no go” stimuli.

At the beginning of the experiment, the experimenter provided the following instruction: “On this computer, I have lots of pictures of children feeling different emotions: either happy, afraid, angry, or nothing. You will complete different tasks. For each task, I will give you instructions on when to click the “x” button. You will try to click as quickly as you can while being as careful as you can not to make a mistake. Let’s practice.” Children completed a practice trial before beginning the task.

At the beginning of each block, participants were first shown an example of “go” stimuli and then “no go” stimuli on the computer screen. For instance, for the “happy” emotional block, participants were presented with a picture on the computer screen of angry, afraid, and neutral faces. They were instructed, “Press the button when you see an angry, afraid, or neutral girl or boys face.” Then the participant was presented with a picture of a happy face. They were instructed, “Do not press the button when you see a happy girl or boys face.” Then, children were provided with a reminder, “Now you will see the pictures of children feeling different emotions. Do not click the “x” button if you see a child with a happy face. Click the “x” button for all other emotions.” Then, the task
was initiated. Children were provided with the same instructions for the happy, afraid, anger, and neutral blocks (with specific instructions for each “no go” stimuli.) The entire task took participants approximately 10 minutes to complete.

Data reduction. For the purpose of this study, one overall accuracy score was calculated for no-go stimuli on the four trial blocks (happy, angry, afraid, neutral blocks). No-go stimuli do not require the press of a button. The number of incorrect reactions (i.e., respond to no-go stimulus), also known as commission errors, was indexed as a measure of IC. An extensive body of research has indicated that commission errors on go/no-go tasks provide a measure of IC (Eagle, Bari, & Robbins, 2008; Kochel et al., 2014; Schultz et al., 2007). For the current study, omission errors were not analyzed, given research suggests omission errors provide an estimate of sustained attention rather than IC (Trommer et al., 1988).
CHAPTER III
RESULTS

Data Reduction and Analytic Plan

All questionnaire data were completed on RedCap, a secure online web-based application. Questionnaire data were transferred from RedCap to IBM SPSS Statistics, version 25. For ePrime task-based assessments, data were extracted from e-Prime, using the e-Prime DataAid tool, into excel and then transferred to SPSS. Given the paucity of research examining the relationship between temperament, ER, and psychopathology among children with ADHD, data were analyzed using an exploratory approach.

Bivariate correlations were conducted for measures where parallel child and parent report was available (e.g., child and parent EATQ scales and child and parent ERC/ERICA scales) to determine if composite scores could be created. Several other researchers have utilized the composite score methodology to permit for a more valid assessment of parent and child perceptions of constructs of interest (Kolak & Vernon-Feagans, 2008; Oh, Volling, Gonzalez, Rosenberg, Song, 2017). This methodology reduces the limitations of a single reporter. Previous studies have suggested correlations between parallel child and parent report measures must emerge as moderately significant (i.e., $r = .3$ or above) to be composited (Kolak & Vernon-Geagans, 2008; Oh et al., 2017). In the current study, correlations between parallel child and parent report measures did not emerge as significant (i.e., all $r’s < .3$, $p’s > .05$). Thus, parent and child report of
each construct were examined independently, rather than composited. This finding was consistent with previous research suggesting the correlation between parent and child report of child psychopathological symptoms is generally low (Agnold et al., 1987; Van Roy, Groholt, Heyerdahl, & Clench-Aas, 2010).

Given parent and child report data could not be composited; cross-rater analyses (e.g., parent report estimating child report; child report estimating parent report) were conducted for each hypothesis, as this would also allow for a more valid assessment of relationships of interest, less reliant on a single reporter. Additionally, within-rater analyses (e.g., parent report estimating parent report; child report estimating child report) were also conducted for each hypothesis to examine independent parent and child perceptions of relationships of interest. All bivariate correlations are displayed in tables.

Bivariate correlations between constructs were examined to determine covariates and independent variables to include in multivariate analyses. This approach ensured only essential variables were included in multivariate analyses, which increased the power of the analyses to detect significant effects.

**Preliminary Analytical Procedures**

The assumption of normal distribution of variables was evaluated by examining boxplots, histograms, and skewness statistics. Outliers were evaluated to determine whether participant data would be included or excluded from analyses. Cases were considered outliers and excluded if they were three standard deviations or more above the mean for the construct of interest. Outliers were removed, case wise. For the Stroop task, 3 cases were removed (i.e., 1 task failure, 2 participants removed due to low accuracy on the task.) For the emotional go/no go task, 5 cases were removed (i.e., 2 task failures, 1
child too distressed to continue the task, 2 children with high omission errors on the task.)

Skewness was examined for each variable. Skewness was determined by dividing the skewness statistic for each variable by the skewness standard error (Malgady, 2007). Once outliers were removed, all skewness statistics were within accepted limits.

Multicollinearity was assessed by examining correlations between predictor variables and by examining collinearity statistics (i.e., Tolerance and VIF.) All collinearity statistics were within accepted limits; thus, the assumption of multicollinearity was met. Residual and scatter plots indicated assumptions of normality, linearity, and homoscedasticity were all satisfied.

**Exploration of Potential Covariates.** Point-biserial correlations were conducted between demographic variables and dependent variables to determine covariates to be included in further analyses. Age was explored as a covariate given research suggesting psychopathology increases with age (Costello, Copeland, & Angold, 2011) and given suspected differences in ER dependent upon child age (Silvers et al., 2012). Additionally, research suggests pubertal development is related to psychopathology, such that children who have undergone puberty may exhibit higher rates of psychopathology (Graber, 2013). Thus, pubertal development was explored as a covariate. Sex was also explored as a covariate, given known sex differences in ER capacity and psychopathological symptoms (Chaplin & Aldao, 2013; Kistner, 2009; Zimmerman & Iwanski, 2014).

ADHD medication status (yes/no) was explored as a covariate, given it is hypothesized that children prescribed ADHD medications would show improved ER and less psychopathology (Hinshaw, Henker, Whalen, Erhardt, & Dunnington, 1989; Posner, Kass, & Hulvershorn, 2014). ADHD medication status during the lab-based task was
explored to assess for the role of current medication status in predicting task performance.

Point-biserial correlations between potential covariates and dependent variables appear in Table 4. Sex was significantly positively correlated with child report of ERICA emotion dysregulation \( (r = .37, p = .012) \) and child report of CDI depressive pathology \( (r = .38, p = .01) \) indicating female children reported greater emotion dysregulation and depressive pathology. Age was only significantly negatively correlated with percentage of commission errors on the emotional go/no go task \( (r = -.43, p = .005) \) indicating older children made fewer commission errors on the task. Pubertal development was only significantly positively correlated with externalizing pathology \( (r = .38, p = .02) \) indicating children further in the pubertal development process demonstrated more externalizing pathology. ADHD medication status during the lab-administered tasks was not significantly correlated with task performance (all \( p \)'s > .05) indicating no differences in task performance based on ADHD medication status. Thus, ADHD medication status on the task was not included as a covariate in analyses assessing task performance.

ADHD medication status was significantly positively correlated with parent report of ERC emotion dysregulation \( (r = .32, p = .028) \), child report of CERQ Maladaptive ER \( (r = .37, p = .014) \), and parent report of CBCL externalizing pathology \( (r = .40, p = .006) \) indicating children currently prescribed ADHD medications demonstrated greater emotion dysregulation and externalizing pathology. This was contrary to hypothesized (i.e., children prescribed ADHD medications would have less emotion dysregulation and less psychopathology). Indeed, research suggests stimulant medications are associated with decreased emotion dysregulation and comorbid...
symptoms in children with ADHD (Gamil & Tahiroglu, 2018; Hinshaw et al., 1989). Thus, it appeared that ADHD medication status may be serving as a proxy for ADHD diagnostic history/severity (i.e., children prescribed ADHD medication had previously been diagnosed with ADHD and thus, may have been exhibiting more severe comorbid symptoms.) This is consistent with previous research indicating an earlier age of onset of ADHD is related to higher rates of comorbid psychopathology (Connor et al., 2003). Given the direction of the hypothesized relationship between ADHD medication status and comorbid symptomatology was contrary to the hypothesis (i.e., ADHD medication status predicted more comorbid symptoms rather than less), ADHD medication status was not explored as a covariate.

**Hypothesis 1**

**Bivariate Analyses.** To test hypothesis 1 (i.e., temperament variables will estimate emotion dysregulation), bivariate correlations were conducted between temperament variables (i.e., EATQ negative affect, UPPS negative urgency, EATQ AC, EATQ IC) and indicators of emotion dysregulation (ERC, ERICA). No significant cross-rater correlations emerged (i.e., child report temperament variables were not correlated with parent report of emotion dysregulation and vice versa, all p’s >.05.) Within-rater correlations emerged and appear in Table 5. Greater parent reported EATQ negative affect and lower parent reported EATQ IC was correlated with greater parent reported ERC emotion dysregulation.

For bivariate analyses examining the association between child reported temperament variables and child reported emotion dysregulation, higher EATQ negative affect and higher UPPS negative urgency was significantly positively correlated with
greater child report of ERICA emotion dysregulation. Temperamental AC and IC were negatively correlated with ERICA emotion dysregulation (i.e., lower AC and IC associated with higher emotion dysregulation.) Given both child report of AC and IC were negatively correlated with ERICA emotion dysregulation, a bivariate correlation was conducted between EATQ Effortful Control, a higher order factor encompassing both AC and IC, and ERICA emotion dysregulation. Child reported Effortful Control was significantly negatively correlated with ERICA emotion dysregulation indicating lower effortful control was related to increased emotion dysregulation.

**Multivariate Analyses.** Given the lack of significant cross-rater correlations between indicators of temperament and indicators of emotion dysregulation, two within-rater regressions (i.e., one regression examining parent perception and one examining child perception) were conducted to examine the relationship between temperament and emotion dysregulation. The first regression explored parent reported ERC emotion dysregulation as the dependent variable. No demographic variables were correlated with ERC in bivariate analyses; thus, no covariates were included in the model. Parent reported EATQ negative affect and EATQ IC were entered into step 1 of the regression model. The interaction of parent reported EATQ negative affect and EATQ IC was entered into step 2. Step 1 of the model was significant, $F(2, 43) = 12.55, p < .001$. Both EATQ negative affect ($\beta = .39, p = .003$) and EATQ IC ($\beta = -.37, p = .005$) contributed significantly to the model and explained 36.9% of the variance in ERC emotion dysregulation. The addition of the interaction of EATQ negative affect and EATQ IC in step 2 did not explain additional variance in emotion dysregulation ($p > .05$). Thus, these results suggest that among children with ADHD, greater negative affect and low IC each
independently estimated ERC emotion dysregulation, according to parent perception. Results are depicted in Table 6.

The second hierarchical regression analysis explored child reported ERICA emotion dysregulation as the dependent variable. Child sex emerged as a significant covariate in bivariate analyses and thus, was entered into step 1 of the regression model. Child reported EATQ negative affect, EATQ effortful control, and UPPS negative urgency were entered into step 2 of the model. Regression statistics are presented in Table 7. At step 1, sex contributed significantly to the regression model, $F(1, 43) = 6.83$, $p = .012$, and accounted for 13.7% of the variance in ERICA emotion dysregulation. The addition of child reported EATQ negative affect, EATQ effortful control, and UPPS negative urgency explained an additional 49.2% of variance in ERICA emotion dysregulation, and this change in $R^2$ was significant, $F(3, 40) = 17.70$, $p < .001$. Both EATQ effortful control ($\beta = -.458$, $p = .001$) and UPPS Negative Urgency ($\beta = .329$, $p = .002$) contributed significantly to the model. EATQ negative affect did not significantly contribute to the model ($\beta = .14$, $p > .05$). Thus, these results suggest that among children with ADHD, greater negative urgency and lower effortful control explained 62.9% of the variance in emotion dysregulation, according to child perception.

**Hypothesis 2**

**Bivariate Analyses.** Bivariate correlations were conducted to determine if parent and child report of temperamental AC and IC on the EATQ correlated with task-based performance on the AC task (i.e., interference ratio on the Stroop task) and IC task (i.e., percent of commission errors on the emotional go/no go task.) Means and standard deviations for the Stroop task variables and emotional go/no go task variables appear in
Table 8. Contrary to the hypothesis, parent and child-report of temperamental AC on the EATQ did not correlate with the Stroop interference ratio (i.e., task-based measure of AC.) Additionally, parent and child-report of temperamental IC on the EATQ did not correlate with the percent of commission errors on the emotional go/no go task (i.e., task-based measure of IC.) Correlations appear in Table 9.

Bivariate correlations were conducted to determine the correlation between indicators of emotion dysregulation (i.e., ERICA and ERC) and performance on the laboratory AC and IC tasks. Parent report of ERC emotion dysregulation did not correlate with AC or IC task performance. However, child-report of ERICA emotion dysregulation was significantly correlated with laboratory task-based IC performance ($r = .32, p = .046$) indicating greater child reported emotion dysregulation was correlated with greater commission errors (i.e., greater disinhibition or lower IC) on the emotional go no/go task. ERICA was not significantly correlated with AC task performance. Correlations appear in Table 9.

**Multivariate Analyses.** To explore the hypothesis that emotion dysregulation would estimate performance on a laboratory-based task of affective IC, a hierarchical regression analysis was conducted with emotional go/no go commission errors as the dependent variable. Age was entered into step 1 and child report of ERICA emotion dysregulation was entered into step 2. Regression statistics are presented in Table 10. At step 1, age ($\beta = -.46, p = .003$) contributed significantly to the regression model, $F(1, 38) = 10.24, p = .003$, and accounted for 21.2% of the variance in emotional go/no go commission errors. The addition of ERICA emotion dysregulation ($\beta = .30, p = .03$) explained an additional 9.1% of variance in emotional go/no go commission errors and
this change in $R^2$ was significant, $F(1, 37)= 4.83, p = .03$. In sum, age and ERICA emotion dysregulation accounted for 30.3% of the variance in emotional go/no go commission errors. Thus, greater child reported emotion dysregulation estimated lower IC (i.e., as indexed by greater commission errors) on the emotional go/no go task.

**Exploratory Bivariate Analyses.** Given parent and child report of emotion dysregulation did not correlate with Stroop task performance, the author conducted further post-hoc analyses. In the review of the literature, low AC was theorized to predict increased internalizing pathology. Thus, the author conducted exploratory analyses to determine whether parent and child report of internalizing symptoms (rather than emotion dysregulation) may correlate with task-based AC performance (i.e., interference ratio on the Stroop task.) Bivariate correlations were conducted. Child reported MASC anxiety symptoms ($r = .34, p = .02$) was correlated with task-based AC performance such that children who reported greater anxiety symptoms had greater interference scores on the Stroop task (i.e., lower AC). Neither child reported CDI depressive symptoms nor parent reported internalizing symptoms ($p > .05$) were correlated with Stroop task performance. Results of a linear regression analysis indicated that MASC anxiety symptoms ($\beta = .34, p = .02$) contributed 11.8% of the variance in task-based AC performance ($F (1, 41) = 5.51, p = .02$).

In sum, these results suggest child reported emotion dysregulation is significantly related to poorer performance on a laboratory task of affective IC. Emotion dysregulation was not related to performance on a laboratory task of AC. Rather, child report of anxiety symptoms was associated with increased interference on a laboratory task of AC.

**Hypothesis 3**
**Bivariate Analyses.** Bivariate correlations were conducted to assess the transdiagnostic nature of ER (i.e., ERC, ERICA, CERQ-k) in estimating both internalizing (i.e., CBCL internalizing, CDI depression, MASC anxiety) and externalizing pathology (i.e., CBCL externalizing, RPAQ reactive aggression.) All bivariate correlations appear in Table 11. Results of cross-rater bivariate analyses revealed that parent report of ERC emotion dysregulation was significantly positively correlated with child report of CDI depressive symptoms ($r = .32, p = .03$). No other significant cross-rater correlations emerged (all $p$’s $>.05$). Results of parent within-rater bivariate analyses revealed that ERC emotion dysregulation was positively correlated with both CBCL internalizing pathology and CBCL externalizing pathology. Results of child within-rater bivariate analyses revealed ERICA emotion dysregulation, CERQ-k adaptive cognitive ER, and CERQ-k maladaptive cognitive ER was significantly correlated with child reported CDI depressive symptoms. ERICA emotion dysregulation was significantly positively correlated with RPAQ reactive aggression symptoms and CERQ-k maladaptive ER. Only ERICA emotion dysregulation was significantly positively correlated with MASC anxiety pathology.

**Multivariate Analyses.** Several regressions were conducted to examine the relationship between emotion dysregulation and psychopathology. For the first model, a linear regression was conducted to examine the relationship between ERC emotion dysregulation and CBCL internalizing pathology, according to parent report. ERC emotion dysregulation significantly estimated internalizing pathology ($\beta = .556, p < .001$). This model was significant, $F (1,43) = 19.28, p < .001$, and accounted for 31% of the variance in CBCL internalizing pathology.
A hierarchical regression explored the relationship between parent reported ERC emotion dysregulation and CBCL externalizing pathology. Model coefficients appear in Table 12. Pubertal development emerged as a significant covariate (i.e., correlated with externalizing pathology) in bivariate analyses and thus, was entered into step 1 of the regression model. Parent reported ERC emotion dysregulation was entered into step 2 of the model. At step 1, pubertal development ($\beta = .38, p = .02$) contributed significantly to the regression model, $F(1, 35) = 5.92, p = .02$, and accounted for 14.5% of the variance in CBCL externalizing pathology. The addition of parent reported ERC emotion dysregulation ($\beta = .57, p < .001$) in step 2 explained an additional 32.2% of variance in CBCL externalizing pathology, and this change in $R^2$ was significant, $F(1, 34) = 20.52, p < .001$. The overall model explained 46.7% of the variance in parent reported CBCL externalizing pathology. Overall, results of parent reported within-rater analyses supported the hypothesis that emotion dysregulation would emerge as a transdiagnostic factor, estimating both increased internalizing and externalizing pathology.

Three regressions were conducted to examine the relationship between child reported emotion dysregulation to indicators of child reported psychopathology (i.e., depression, anxiety, reactive aggression.) For the first regression analysis examining the relationship between emotion dysregulation and child reported CDI depressive symptoms, sex was entered into step 1 of the model. ERICA emotion dysregulation, ERC emotion dysregulation, and CERQ-k Adaptive and Maladaptive scales were entered into step 2 of the model. Parent reported ERC emotion dysregulation was entered into the model, given the significant correlation between ERC and depressive symptoms in bivariate analyses. The inclusion of the ERC permitted for cross-rater assessment of the
relationship of interest. At step 1, sex ($\beta = .38, p = .01$) contributed significantly to the regression model, $F(1, 43) = 7.12, p = .01$, and accounted for 14.2% of the variance in CDI depressive symptoms. The addition of child reported ERICA emotion dysregulation, ERC emotion dysregulation, and CERQ-k Adaptive and Maladaptive scales explained an additional 38.8% of variance in CDI depressive symptoms, and this change in $R^2$ was significant, $F(4, 40) = 8.06, p < .001$. Both CERQ-k Adaptive ($\beta = -.33, p = .007$) and CERQ-k Maladaptive ($\beta = .39, p = .003$) cognitive ER contributed significantly to the model. ERICA emotion dysregulation ($\beta = .17, p > .05$) and ERC emotion dysregulation ($\beta = .02, p > .05$) did not significantly contribute to the model. Model coefficients appear in Table 13. The overall model explained 53.0% of the variance in child reported depressive symptoms. Thus, these results suggest that children with ADHD who endorse utilizing more maladaptive and less adaptive cognitive ER strategies report greater depressive symptoms.

A linear regression analysis examined the relationship between child reported ERICA emotion dysregulation and child reported MASC anxiety symptoms. This model was significant, $F(1, 43) = 13.20, p = .001$; ERICA emotion dysregulation explained 23.5% of the variance in MASC anxiety symptoms. ERICA emotion dysregulation ($\beta = .49, p = .001$) contributed significantly to the model.

A multiple regression was conducted to assess the relationship between indicators of emotion dysregulation (ERICA, CERQ-k Maladaptive) and RPAQ reactive aggression symptoms. Model coefficients appear in Table 14. This model was significant, $F(2, 42) = 15.23, p < .001$ and explained 42.0% of the variance in RPAQ reactive aggression symptoms. However, only ERICA emotion dysregulation ($\beta = .52, p < .001$) and not
CERQ-k Maladaptive scale ($\beta = .21$, $p = .12$) contributed significantly to the model. Overall, the results of child report analyses also supported hypotheses that emotion dysregulation would correlate with both internalizing (i.e., anxiety, depression) and externalizing (i.e., aggression) symptoms. Of note, cognitive ER deficits explained significant variance in the estimation of depressive symptoms, but not anxiety or reactive aggression symptoms.

**Hypothesis 4**

**Data Analytic Plan.** The final exploratory analyses utilized direct and indirect effect path analyses to examine the hypothesis that ER would emerge as a mechanism to explain the link between temperament and both broad internalizing and broad externalizing pathology. For each analysis, only parent reported variables were utilized, given the parent reported CBCL was the only measure that provided broad internalizing and externalizing pathology dependent variables. Only symptom-specific measures existed for child report (i.e., depression, anxiety, aggression) rather than broad internalizing/externalizing pathology measures.

Previous analyses established the relationship between parent report of temperament and emotion dysregulation and between emotion dysregulation and psychopathology. In previous analyses, both parent reported temperamental negative affect and temperamental IC (but not temperamental AC) were correlated with parent reported emotion dysregulation. Additionally, emotion dysregulation was correlated with both parent reported broad internalizing and externalizing pathology. Bivariate analyses were first conducted to establish the relationship between temperament variables and broad internalizing and externalizing pathology variables before the relationship between
temperament, ER, and psychopathology was explored further in path analyses. To ensure adequate power to detect a significant effect, only one temperament, one ER, and one psychopathology variable were entered into each analysis. For each analysis, an indirect effects analysis was conducted using ordinary least squares path analysis in IBM Statistics version 25 using the PROCESS macro (Hayes, 2012). For each model, ER was hypothesized to emerge as a mechanism to explain the link between temperament and psychopathology.

**Bivariate Analyses.** Bivariate correlations explored the relationship between parent reported temperament variables and parent reported internalizing and externalizing pathology. EATQ temperamental negative affect was significantly correlated with both internalizing ($r = .58, p < .001$) and externalizing pathology ($r = .35, p = .017$) indicating parents who described their children as exhibiting higher negative affect also reported their children had greater internalizing and externalizing problems. EATQ temperamental IC was significantly negatively correlated with both internalizing ($r = -.36, p = .015$) and externalizing pathology ($r = -.51, p < .001$) indicating parents who described their children as exhibiting lower IC also reported their children had greater internalizing and externalizing problems. Contrary to the hypothesis, EATQ temperamental AC was not significantly correlated with either internalizing or externalizing pathology ($p's > .05$). All correlations appear in Table 15.

Four exploratory path analyses were conducted. The following analyses were conducted:

1) The effects of EATQ temperamental negative affect on CBCL internalizing pathology directly and indirectly through ERC emotion dysregulation
2) The effects of EATQ temperamental IC on CBCL internalizing pathology directly and indirectly through ERC emotion dysregulation

3) The effects of EATQ temperamental negative affect on CBCL externalizing pathology directly and indirectly through ERC emotion dysregulation

4) The effects of EATQ temperamental IC on CBCL externalizing pathology directly and indirectly through ERC emotion dysregulation.

These analyses permitted for the exploration of the relationship between both reactive (i.e., negative affect) and regulative (i.e., IC) temperament dimensions and both broad internalizing and externalizing pathology through emotion dysregulation.

**Multivariate Analyses.** The first model estimated the effects of EATQ temperamental negative affect on CBCL internalizing pathology directly and indirectly through ERC emotion dysregulation. The influence of the antecedent variable (X), temperamental negative affect, on the proposed indirect effect variable (M), emotion dysregulation, and the consequent variable (Y), internalizing pathology, was examined. No demographic variables were associated with CBCL internalizing pathology in bivariate analyses; thus, no covariates were included in this model. Results indicated temperamental negative affect was a significant estimator of emotion dysregulation (a path, $\beta = .42, p = .004$). In turn, emotion dysregulation was a significant estimator of internalizing pathology (b path, $\beta = .38, p = .004$), while controlling for temperamental negative affect. The direct path between temperamental negative affect and internalizing pathology was significant (c path, $\beta = .58, p < .001$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab, B = 1.92$) based on 10,000 bootstrap samples was entirely above zero (.47 to 4.26). The direct effect of temperamental negative affect on
internalizing pathology remained significant (c’ path, \( \beta = .43, p = .002 \)), yet reduced, with emotion dysregulation in the model, indicating a direct effect of temperamental negative affect on the estimation of internalizing pathology existed independent of its effect through emotion dysregulation. Contrary to the hypothesis, ER did not fully statistically account for the relationship between temperamental negative affect and internalizing pathology. Rather, both temperamental negative affect and emotion dysregulation directly estimated internalizing pathology. Model coefficients appear in Table 16.

The second model estimated the effects of EATQ temperamental IC on CBCL internalizing pathology directly and indirectly through ERC emotion dysregulation. The influence of the antecedent variable (X), temperamental IC, on the proposed indirect effect variable (M), emotion dysregulation, and the consequent variable (Y), internalizing pathology, was examined. Results indicated that temperamental IC was a significant estimator of emotion dysregulation (a path, \( \beta = -.49, p < .001 \)). In turn, emotion dysregulation was a significant estimator of internalizing pathology (b path, \( \beta = .50, p = .001 \)), while controlling for temperamental IC. The direct path between temperamental inhibitory control and internalizing pathology was significant (c path, \( \beta = -.36 p = .01 \)). A bias-corrected bootstrap confidence interval for the indirect effect (\( ab, B = 1.92 \)) based on 10,000 bootstrap samples was entirely below zero (-5.31 to -1.11). The direct effect of temperamental IC on the on internalizing pathology was not significant (c’ path, \( \beta = -.12, p = .42 \)) with emotion dysregulation in the model, indicating no direct effect of temperamental IC on the estimation of internalizing pathology existed independent of its indirect effect through emotion dysregulation. Thus, in support of hypotheses, ER fully statistically account for the relationship between temperamental IC and internalizing
pathology such that the data were best fit by an indirect effect of temperamental IC on internalizing pathology through emotion dysregulation. Model coefficients appear in Table 17. Figure 4 depicts this relationship.

The third model estimated the effects of EATQ temperamental negative affect on CBCL externalizing pathology directly and indirectly through ERC emotion dysregulation. The influence of the antecedent variable (X), temperamental negative affect, on the proposed indirect effect variable (M), emotion dysregulation, and the consequent variable (Y), externalizing pathology, was examined. Results indicated that temperamental negative affect was a significant estimator of emotion dysregulation (a path, $\beta = .49, p < .001$). In turn, emotion dysregulation was a significant estimator of externalizing pathology (b path, $\beta = .66, p < .001$). The direct effect of temperamental negative affect on externalizing pathology was significant (c path, $\beta = .35, p = .02$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab, B = 3.88$) based on 10,000 bootstrap samples was entirely above zero (.73 to 7.03). The direct effect of temperamental negative affect on the estimation of externalizing pathology was not significant (c’ path, $\beta = .03, p = .85$) with emotion dysregulation in the model, indicating no direct effect of temperamental negative affect on the estimation of externalizing pathology existed independent of its indirect effect through emotion dysregulation. Thus, in support of hypotheses, ER fully statistically accounted for the relationship between temperamental negative affect and externalizing pathology such that the data were best fit by an indirect effect of temperamental negative affect on externalizing pathology through emotion dysregulation. Model coefficients appear in Table 18. This model is depicted in Figure 5.
The fourth model estimated the effects of EATQ temperamental IC on CBCL externalizing pathology directly and indirectly through ERC emotion dysregulation. The influence of the antecedent variable (X), temperamental IC, on the proposed indirect effect variable (M), emotion dysregulation, and the consequent variable (Y), externalizing pathology, was examined. Pubertal development was included as a covariate in the model. Results indicated that temperamental IC was a significant estimator of emotion dysregulation (a path, $\beta = -0.46$, $p = .005$). In turn, emotion dysregulation was a significant estimator of externalizing pathology (b path, $\beta = 0.50$, $p = .001$), while controlling for temperamental IC and pubertal development status ($\beta = 0.27$, $p = .04$). The direct path between temperamental inhibitory control and externalizing pathology was significant (c path, $\beta = -0.39$, $p = .01$) while controlling for the effect of pubertal development status ($\beta = 0.31$, $p = .04$). A bias-corrected bootstrap confidence interval for the indirect effect ($ab$, $B = -2.42$) based on 10,000 bootstrap samples was entirely below zero ($-4.97$ to $-0.53$). The direct effect of temperamental IC on the estimation of externalizing pathology was not significant ($c'$ path, $\beta = -0.16$, $p = .26$) with emotion dysregulation in the model, indicating no direct effect of temperamental IC on the estimation of externalizing pathology existed independent of its indirect effect through emotion dysregulation. Thus, in support of hypotheses, ER fully statistically accounted for the relationship between temperamental IC and externalizing pathology such that the data were best fit by an indirect effect of temperamental IC on externalizing pathology through emotion dysregulation. Model coefficients appear in Table 19. This model is depicted in Figure 6.
In sum, results suggest the reactive temperament dimension, negative affect, directly estimated internalizing pathology. However, negative affect indirectly estimated externalizing pathology, through emotion dysregulation. Thus, ER explained the link between temperamental negative affect and externalizing pathology. Additionally, the regulative temperament dimension, IC, estimated internalizing and externalizing pathology, indirectly through emotion dysregulation. Thus, ER also explained the link between temperamental IC and both internalizing and externalizing pathology.

**Post-Hoc Exploratory Analyses**

Post-hoc analyses were conducted to examine how AC and IC task performance may estimate broad internalizing and externalizing pathology. In the review of the literature, the author hypothesized AC may interact with temperamental negative affect to produce emotion dysregulation and subsequent internalizing pathology among children with ADHD. Results of hypothesis four revealed that emotion dysregulation did not account for the relationship between temperamental negative affect and internalizing pathology; instead, temperamental negative affect directly estimated internalizing pathology. Thus, the author conducted a post-hoc hierarchical regression analysis to examine whether negative affect may interact with task-based AC to directly estimate internalizing pathology. It was hypothesized that only children with higher levels of negative affect who also demonstrated greater interference on the AC task would show increased internalizing pathology.

Parent reported EATQ negative affect and Stroop interference ratio were entered into step 1 of the regression model estimating CBCL internalizing pathology. At step 2, the interaction term (negative affect by Stroop interference) was entered into the model.
At step 1, EATQ negative affect ($\beta = .59$, $p < .001$), but not Stroop interference, contributed significantly to the regression model $F(2, 39) = 10.20$, $p < .001$, and accounted for 34.3% of the variance in CBCL internalizing problems. At step 2, the interaction of EATQ negative affect and Stroop interference did not explain significant variance in internalizing symptoms ($\Delta R^2 < .001$). Thus, contrary to hypothesized, negative affect, rather than the interaction of negative affect and AC indexed by Stroop interference, estimated internalizing pathology. Model coefficients appear in Table 20.

Results of hypothesis four revealed that emotion dysregulation fully statistically explained the relationship between temperamental negative affect and externalizing pathology. Additionally, emotion dysregulation also fully statistically explained the relationship between parent reported IC and externalizing pathology. However, the author was interested in examining how task-based IC may estimate externalizing pathology. Given temperamental variables indirectly estimated externalizing pathology through emotion dysregulation, a post-hoc hierarchical regression analysis explored the interaction of emotion dysregulation and task-based IC in the estimation of externalizing pathology. It was hypothesized that only children with high emotion dysregulation that also demonstrated greater commission errors on the IC task would show greater externalizing pathology.

A hierarchical regression analysis explored the interaction of emotion dysregulation and task-based IC on externalizing pathology. Pubertal Development status was entered into step 1 of the model. Parent reported ERC emotion dysregulation and Emotional Go/No Go Commission Errors were entered into step 2 of the regression model. At step 3, the interaction term (emotion dysregulation by commission errors) was
entered into the model estimating CBCL externalizing pathology. At step 1, pubertal development ($\beta = .37 \ p = .036$) contributed significantly to the regression model $F(1, 31) = 4.80 \ p = .036$, and accounted for 13.4% of the variance in CBCL externalizing pathology. At step 2, ERC emotion dysregulation ($\beta = .54 \ p = .001$), but not commission errors, contributed significantly to the regression model $F(2, 29) = 7.25 \ p = .003$, and accounted for an additional 28.9% of the variance in CBCL externalizing pathology. At step 3, the interaction of ERC emotion dysregulation and commission errors ($\beta = .30 \ p = .09$) did not explain significant variance in externalizing pathology ($\Delta R^2 = .06, \ p = .09$); however, this was approaching significance. Model coefficients appear in Table 21.
CHAPTER IV

DISCUSSION

The current study investigated an initial conceptual model examining the effects of temperament and ER on concurrent internalizing and externalizing psychopathology among a sample of preadolescent youth with ADHD. Various components of a conceptual model were empirically tested. The conceptual model posited 1) temperamental reactivity and regulatory dimensions would estimate emotion dysregulation, and in turn, 2) emotion dysregulation would estimate concurrent internalizing and externalizing pathology among youth with ADHD. One specific aim of the current study was to determine if ER would emerge as a mechanism to account for the relationship between temperament and concurrent psychopathology. Additionally, the study investigated whether parent and/or child report of emotion dysregulation would predict performance on laboratory-based AC and IC regulation tasks. Currently, few models exist to explicate the development of psychopathology among children with ADHD, despite high rates of comorbid psychopathology in this population (Cuffe et al., 2017; Larson, Russ, Kahn, & Halfon, 2011). The current study empirically evaluated a theoretical model of comorbid psychopathology development to identify potential transdiagnostic risk factors that may inform the development of novel treatment interventions for youth with ADHD.
Overall, results from the current study lend support to hypotheses positing both temperament and ER would estimate concurrent internalizing and externalizing pathology among youth with ADHD. Both reactive (i.e., negative affect, negative urgency) and regulative (i.e., IC, AC) temperament dimensions emerged as salient predictors of psychopathology among children with ADHD. Additionally, emotion dysregulation emerged as a transdiagnostic factor estimating both internalizing and externalizing pathology. Results of direct and indirect effects analyses revealed that temperamental negative affect and emotion dysregulation each independently directly estimated internalizing pathology among youth with ADHD. Of note, this study presents novel findings that ER explains the link between temperamental negative affect and externalizing pathology among youth with ADHD. ER also explained the relationship between temperamental IC and broad psychopathology (i.e., internalizing and externalizing pathology.) Interestingly, child report of emotion dysregulation predicted child performance on a laboratory-based affective IC task. Children reporting higher emotion dysregulation demonstrated a decreased ability to regulate and inhibit their responses to emotional stimuli on the laboratory-based affective IC task. Overall, the findings of the study highlight the robust and mechanistic role of ER in contributing to the development of psychopathology among youth with ADHD. Results that pertain to individual hypotheses are presented below.

**Concordance Between Parent and Child Report**

**Findings.** In an effort to provide a more valid assessment of temperament and ER constructs of interest, less reliant on a single reporter, correlations were conducted between parallel child and parent report measures to determine if composite scores could
be created for constructs of interest. However, agreement between parent and child report of symptoms was low, indicating differences existed in parent and child perception of child temperament and emotion dysregulation. This finding is consistent with a large body of literature revealing discrepancies in parent and child report of emotional and behavioral problems (Agnold et al., 1987; Van Roy et al., 2010).

**Theoretical Implications.** Research suggests early adolescents may actually report more emotional and behavioral symptoms than parents, but less impact of symptoms on functioning, whereas parents may be more valid reporters of overall early adolescent functioning (Van Roy et al., 2010). Research investigating the etiology of parent/child report discrepancies has highlighted the relevance of contextual factors in predicting discrepancies in reporting. In a study exploring parents and children who were highly discrepant in their report of emotional and behavioral symptoms, Van Roy and colleagues (2010) found that children who reported significantly more symptoms than their parents were more likely to have parent-child relationship difficulties and disrupted family systems. However, demographic characteristics such as low parental education level, low income, and male child sex were associated with discrepancies in which parents reported higher symptoms than youth (Van Roy et al., 2010).

Research has also indicated a subset of children with ADHD exhibit a positive illusory bias in which they demonstrate a tendency to overestimate their emotional and social competence (Owens, Goldfine, Evangelista, Hoza, & Kaiser, 2007). Thus, discrepancies in parent and child report may be influenced by various contextual factors such as psychosocial difficulties and self/observer report biases. These findings highlight the need for future research identifying contextual/sociocultural factors and biases that
may influence parent child report discrepancies in emotional symptoms among preadolescents with ADHD. This is particularly important given findings that parent/child relationships are more impaired among ADHD families (Pressman et al., 2006). Despite the origin of reporting discrepancies, the current study explored parent and child perception of relationships of interests separately, given the lack of agreement observed between parents and children.

**The Relationship Between Temperament and ER**

**Findings.** In support of hypotheses, both reactive (i.e., negative affect, negative urgency) and regulative (i.e., IC, overall effortful control) temperament dimensions estimated emotion dysregulation. Interestingly, results of analyses investigating parent perception of the relationship between temperament and emotion dysregulation indicated temperamental negative affect and IC did not interact to produce higher emotion dysregulation. Rather, both high temperamental negative affect and low IC independently estimated higher emotion dysregulation. Additionally, contrary to expected, parental report of AC did not estimate emotion dysregulation. In bivariate analyses exploring child perception of the relationship between temperament and emotion dysregulation, negative urgency, negative affect, and low effortful control estimated emotion dysregulation. However, in multivariate analyses, negative urgency and effortful control emerged as the most salient predictors of emotion dysregulation. Thus, negative affect did not significantly contribute to the model estimating emotion dysregulation, according to child perception.

**Theoretical Implications.** Numerous theories exist to explain the etiology of ER. Dominant theories of ER highlight the biological basis of emotion dysregulation. For
instance, emotion dysregulation is theorized to occur as a result of neurobiological deficiencies in frontal-striatal circuitry (Banks et al., 2007; Morawetz et al., 2017) and dysfunction in vagal systems responsible for modulating cardiac output necessary for promoting self-regulatory behavior (Porges, 2001). Results of the current study suggest both temperamental reactivity (i.e., negative affect, negative urgency) and regulation dimensions (i.e., IC, effortful control) also play an important role in the etiology of ER. In the current study, children with either greater negative affect/negative urgency or lower IC/effortful control reported greater emotion dysregulation. Thus, either high temperamental reactivity or low temperamental regulation abilities alone were sufficient to produce emotion dysregulation among youth with ADHD. The findings of the current study illustrate the importance of the inclusion of temperamental reactivity and regulation dimensions in conceptual models of emotion dysregulation.

Children who exhibit temperamental negative affect demonstrate high trait level reactivity, arousal, and a tendency to display negative moods such as sadness and irritability (Hankin et al., 2017). Higher baseline negative affect and arousal likely contributes to “emotional overload,” which may limit children’s capacity to regulate negative emotions. Children with ADHD who show greater impulsivity and general self-regulation deficits in conjunction with high negative affect may act on negative emotions, which may result in emotion dysregulation. Indeed, children with ADHD who reported higher trait negative urgency, or a tendency to act rashly when encountering strong negative emotions, also reported greater emotion dysregulation. Thus, although not explored in the current study, urgency may actually serve as a mechanism to explain the link between negative affect and emotion dysregulation. Future research would benefit
from a more thorough investigation of the relationships between negative affect, urgency, and regulation temperament dimensions to further disentangle the relationship between temperament dimensions and ER.

Parent report of IC rather than AC independently estimated emotion dysregulation. Children with low IC exhibited greater emotion dysregulation regardless of their level of trait negative affect. There may be several explanations for this. First, the inability to inhibit and control behavior may lead children to act on negative emotions. Even if children do not exhibit high trait level negative affect, they likely experience situations that elicit negative emotions and emotional arousal in their daily lives. Consequently, these children with ADHD who exhibit low trait IC may act on the negative emotions leading to a pattern of emotion dysregulation. Second, children with low IC may act impulsively, which may elicit negative reactions from the environment. As children respond to the environment and the consequences of failing to inhibit behavioral responses, they may experience negative affect and negative emotions. Then, they may act on negative emotions, which may also result in subsequent emotion dysregulation. This pattern of responding could become cyclical or may become reinforced by the environment, leading to a recurrent experience of emotion dysregulation. Thus, environmental factors (e.g., parental reactivity, social disapproval) may in part, explain the relationship between IC and emotion dysregulation. However, the current initial investigation did not examine the interaction of dispositional and contextual factors in the estimation of emotion dysregulation. Future research would benefit from the inclusion of contextual factors in models of emotion dysregulation, as
this would shed light on dispositional and contextual risk factor interactions in the prediction of ER.

Of note, the measure used to assess parent report of emotion dysregulation assessed the behavioral dimension of ER rather than the cognitive dimension of ER. Parents may be more valid reporters of behavioral emotion dysregulation than cognitive emotion dysregulation, given parents can directly observe child emotional expression and behavioral dysregulation. Parents have a more limited ability to report on child use of cognitive ER strategies, given the internal nature of these processes. Previous research has indicated difficulties regulating attention may increase risk for use of maladaptive and overcontrolled, ER strategies (catastrophizing, self-blame; Hilt, Leitzke, & Pollak, 2014; O’Bryan, Kraemer, Johnson, McLeish, & McLaughlin, 2017) thus, AC and/or cognitive control may be more predictive of cognitive ER than behavioral ER. The inability to assess cognitive ER according to parent perception may explain null findings in the current study that parent reported AC was not related to parent report of emotion dysregulation. The relationship between AC and cognitive ER is likely best understood by examining child perception. Regardless, the overall results of hypothesis one implicate temperament as a distal, biological risk factor for emotion dysregulation among youth with ADHD. These findings suggest there may be multiple pathways from temperament to emotion dysregulation among youth with ADHD.

The Relationship Between ER and Laboratory Task Performance

**Findings.** No significant correlations emerged between child/parent report of AC and IC and performance on the AC and IC tasks. These results indicated discrepancies between parent and child report of temperamental regulation and performance on tasks of
regulation. Interestingly, in partial support of hypotheses, child report of emotion dysregulation, but not parent report of emotion dysregulation, predicted performance on a laboratory-based task of affective IC. Children who reported more difficulties regulating emotions demonstrated lower IC on a task in which they were required to inhibit their responses to emotional stimuli. Contrary to expected, child and parent report of emotion dysregulation did not predict performance on the laboratory-based task of AC. However, post hoc analyses revealed that child report of anxiety predicted performance on the AC task. Children who reported higher anxiety demonstrated greater interference on a task in which they were required to control their attention and inhibit their behavioral responses.

**Theoretical Implications.** Results indicating parent and child report of AC and IC temperament dimensions do not correlate with AC and IC laboratory-based task performance were not surprising. Numerous studies have indicated that the correlation between self-report of executive functioning and performance on executive functioning tasks is generally low (Baars, Bijvank, Tonnaer, & Jolles, 2015; Nordvall, Jonsson, & Neely, 2017). Although the results of the current study did not reveal significant correlations between AC and IC child/parent report measures and AC and IC task performance, the tasks estimated higher emotional symptoms. One of the most intriguing findings from the current study was that children with ADHD who reported greater emotion dysregulation made more errors on the emotional go/no go task. Additionally, although emotion dysregulation was not related to AC task performance, child report of anxiety symptoms estimated AC task performance. Children reporting greater anxiety pathology demonstrated greater interference on the AC task. In the ADHD literature, executive functioning tasks have traditionally been utilized to assess sustained attention.
and disinhibition (Ridderinkhof et al., 2004). However, the results of the current study suggest executive functioning tasks may also be useful for assessing emotional and internalizing problems among youth with ADHD.

There may be several explanations for why children with greater emotional symptoms (e.g., emotion dysregulation, anxiety) demonstrate worse performance AC and IC tasks. The emotional go/no go adaptation presents children with multiple challenges. Children are required to sit and focus their attention for approximately 10 minutes, engage emotion recognition executive processes, set shift for each trial block (e.g., inhibit for angry emotional valence and then switch to inhibit for happy emotional valence), and inhibit dominant behavioral responses. For children with ADHD, any one of these tasks is challenging and could lead to cognitive overload and emotional distress. Children with ADHD must regulate both their behavioral responses and their emotional distress in order to perform well on this task. Although emotional intensity and dysregulation were not measured during the task, it is plausible that this task produced greater emotion dysregulation in children. Indeed, several children’s data were excluded either because they became too distressed to complete the task or because they appeared taxed and quit responding, resulting in invalid data. Children who exhibit higher rates of emotion dysregulation in their daily lives will likely perform worse on this task given this task requires both behavioral and emotional inhibition.

Additionally, the emotional go/no go task requires children to recognize emotional valences (e.g., happy, angry, fear, neutral). Difficulties with emotion recognition and emotion processing are common in children with ADHD who exhibit emotion dysregulation (Razavi, Tehranidoost, Ghassemi, Purabassi, & Taymourtash,
2017; Shaw et al., 2014). Thus, it is also plausible that poor performance on the emotional go/no go task may be a function of emotion recognition deficits. Further research is necessary to empirically test mechanisms that may be related to poorer performance on the emotional go/no go task among youth with ADHD. However, it is evident that among youth with ADHD, emotion dysregulation confers risk for deficits in IC of affective stimuli, measured in a controlled laboratory environment. Children who report more difficulties regulating emotions show deficits in their ability to inhibit responses to emotional stimuli.

The Stroop task, used to assess AC, may have been less distressing to children. Although this task also required children to sit and attend for approximately 10 minutes in duration and inhibit a dominant attentional response, the directions remained the same for the entire task duration (i.e., children did not have to set shift). Additionally, there were no emotional stimuli in this task; thus, there was no emotion recognition component. Children did not have to engage in as many competing responsibilities as compared to the emotional go/no go task. It is plausible that they did not experience cognitive overload and/or emotional distress while completing this task, so their performance on this task may be less correlated with emotion dysregulation.

Interestingly, results of the study suggested anxiety estimated Stroop task performance. These findings supported previous research indicating individuals with anxiety demonstrate greater interference on the Stroop task (Becker, Rinck, Margraf, & Roth, 2001; Williams, Mathews, & MacLeod, 1996) and extended these results to an ADHD only sample. On incongruent trials of the Stroop task, children were required to inhibit a dominant attentional response and activate a subdominant response. Research
suggests children with anxiety exhibit biases to attend to negative or threatening stimuli in conjunction with a decreased ability to control attention and allocate attention away from threat (Bar-Haim et al., 2007; Susa et al., 2012). Attention deficits inherent in ADHD may place children with ADHD at greater risk for exhibiting low AC. The results of the current study suggest children with ADHD who demonstrate more difficulties allocating and controlling attention during the Stroop task also report more anxiety. Thus, these findings implicate AC as a mechanism that may predict anxiety among youth with ADHD. Emotion dysregulation was not related to Stroop task performance; this suggests AC may be directly associated with anxiety symptoms rather than influencing anxiety symptoms through ER.

In sum, the Stroop and emotional go/no go tasks may not actually serve as a proxy for temperamental AC and IC. Rather, parent and child report of temperamental AC and IC may assess different processes than AC and IC tasks. Or, parents and children may not be adequate reporters of IC and AC regulatory processes. Multi-method assessment permits for a more ecologically valid assessment of temperamental regulation. The results of the current study present novel findings that AC and IC tasks may be useful tools for assessing emotional deficits among youth with ADHD. Future studies are needed to better and more fully understand what mechanisms may drive the observed relationships between emotional symptoms and executive functioning task performance among youth with ADHD.

The Relationship Between ER and Psychopathology

Findings. In support of hypotheses, ER estimated both internalizing and externalizing pathology. According to parent perception, greater broad emotion
dysregulation estimated higher broad internalizing pathology and higher broad externalizing pathology, while controlling for the influence of child pubertal development. Children who were further in pubertal development demonstrated higher externalizing pathology, according to parent perception.

An interesting pattern of results emerged when examining the relationship between child perception of emotion dysregulation and symptom-specific psychopathology (i.e., anxiety, depression, aggression.) Children completed a broad emotion dysregulation measure and a cognitive emotion dysregulation measure. This permitted for an assessment of “upstream” (i.e., catastrophizing, refocusing) ER processes (Gross, 1998) in addition to broader ER. Results revealed that cognitive ER emerged as a unique predictor of depressive symptoms. Children who reported greater use of maladaptive cognitive ER strategies such as catastrophizing and blame and less use of adaptive cognitive ER strategies such positive reappraisal and positive refocusing also reported elevated depressive pathology. These cognitive ER strategies did not significantly estimate anxiety or aggression pathology in multivariate analyses. Rather, the broad based measure of emotion dysregulation estimated both anxiety and reactive aggression symptoms.

**Theoretical Implications.** Numerous studies have investigated the associations between ADHD and ER (Anastopoulos et al., 2011; Shaw et al., 2014) and between ADHD and comorbid psychopathology (Cuffe et al., 2017; Larson et al., 2011) separately. However, few attempts have been made to integrate both lines of research to establish a relationship between emotion dysregulation and psychopathology among an ADHD sample. The results of the current study implicate ER as a transdiagnostic factor
that estimates both broad internalizing and broad externalizing pathology among youth with ADHD. Emerging research has contrasted bifactor psychopathology models that posit psychopathology is composed of two separate internalizing and externalizing factors to general “p” psychopathology models that theorize psychopathology is explained by one higher order factor (i.e., includes internalizing and externalizing pathology; Hankin et al., 2017; Martel et al., 2016; Murray, Eisner, & Ribeaud, 2016). Results of the current study suggest that among youth with ADHD, emotion dysregulation may confer risk for a higher order general “p” factor that constitutes both internalizing and externalizing pathology.

Although, emotion dysregulation was related to both broad internalizing and externalizing pathology, preliminary findings indicated specific ER processes may predict symptom-specific pathology (e.g., depression, anxiety, aggression.) For instance, according to child report, cognitive ER as opposed to broad ER, was a more salient estimator of depressive internalizing pathology. Youth who endorsed utilizing more maladaptive and less adaptive cognitive ER strategies reported higher depressive symptoms. Alternatively, broad ER was a stronger estimator of anxiety and aggression pathology. Previous research has theorized children with internalizing problems may be more prone to engage in “overcontrol” strategies to regulate negative emotions (e.g., rumination, catastrophizing) whereas children with externalizing problems may be more prone to emotional undercontrol in that they fail to inhibit responses to negative emotions (Garnefski et al., 2005; Southam-Gerow & Kendall, 2002). Thus, emotional over versus undercontrol may differentiate children who exhibit internalizing versus externalizing pathology.
The theory of emotional overcontrol/undercontrol was only partially supported in the current study. Although emotional overcontrol, as indexed by deficits in cognitive ER, estimated depressive internalizing pathology, cognitive ER did not estimate anxiety internalizing pathology. There could be several explanations for this finding. First, in the current study, the Cronbach’s alpha for the rumination subscale of the CERQ-k had low internal consistency and was thus, not included in the maladaptive cognitive ER factor. This could have provided an underestimate of cognitive ER deficits, which may have contributed to the lack of a significant relationship observed between cognitive ER and anxiety pathology. It is also possible that specific maladaptive cognitive ER processes (e.g., catastrophizing, blame, rumination) may be more predictive of anxiety as opposed to the general maladaptive cognitive ER factor explored in the current study. This maladaptive cognitive ER factor was created for parsimony; however, this may have diminished potential significant findings. It is also likely that different ER mechanisms predict depressive versus anxiety pathology among youth with ADHD. As previously mentioned, there may be multiple pathways from temperament to emotion dysregulation. The results of the current study indicate there may also be differential pathways from emotion dysregulation to symptom-specific pathology among youth with ADHD. Most importantly, the current study implicates the significance of broad emotion dysregulation in conferring risk for broad internalizing and externalizing pathology, lending support to general “p” psychopathology models.

**The Relationship Between Temperament, ER, and Psychopathology**

**Findings.** In the current study, several models investigated the mechanistic role of ER in accounting for the relationship between temperament (e.g., negative affect, IC) and
broad internalizing and externalizing pathology. The reactive temperament dimension, negative affect, directly estimated internalizing pathology. Emotion dysregulation also directly estimated internalizing pathology. Thus, emotion dysregulation did not explain the relationship between temperamental negative affect and internalizing pathology. Rather, internalizing pathology was better explained by either negative affect or emotion dysregulation independently.

In support of hypotheses, ER emerged as a mechanism that fully accounted for the relationship between temperamental negative affect and externalizing pathology. As hypothesized, children high in temperamental negative affect experienced greater emotion dysregulation, and in turn, children with higher emotion dysregulation exhibited more externalizing pathology. However, the direct path between temperamental negative affect and externalizing pathology was non-significant once emotion dysregulation was entered into the model, indicating temperamental negative affect exerted an indirect effect on externalizing pathology through emotion dysregulation. Overall, these results suggest that the temperamental reactivity dimension, negative affect, is a direct and strong estimator of internalizing pathology. Alternatively, negative affect only indirectly estimated externalizing pathology. ER emerged as a more salient estimator of externalizing pathology that accounted for the relationship between temperamental negative affect and externalizing pathology.

As explained previously, the regulatory dimension, AC was not associated with either internalizing or externalizing pathology, according to parent perception. IC estimated both internalizing and externalizing pathology. However, IC only indirectly estimated internalizing and externalizing pathology through emotion dysregulation. Thus,
emotion dysregulation fully statistically accounted for the relationship between IC and internalizing pathology and the relationship between IC and externalizing pathology. In support of hypotheses, ER emerged as a mechanism to explain the link between IC and broad psychopathology.

Post-hoc analyses investigated the role of AC and IC task performance in estimating psychopathology. It was hypothesized that only children with higher temperamental negative affect who demonstrated greater interference on the AC task would show greater internalizing pathology. However, this hypothesis was not supported. Only temperamental negative affect, rather than the interaction of negative affect and interference control, directly estimated internalizing pathology. Given, emotion dysregulation emerged as the most robust predictor of externalizing pathology, post hoc analyses explored whether children with higher emotion dysregulation who demonstrated more commission errors on the IC task would show higher externalizing pathology. This hypothesis was not supported. Emotion dysregulation, rather than the interaction of emotion dysregulation and IC task performance, estimated externalizing pathology.

**Theoretical Implications.** Although the link between temperament dimensions and psychopathology has been well established in the broad child psychopathology literature (Rettew & Mckee, 2005), there has been minimal research exploring this relationship within an ADHD sample. Additionally, few attempts have been made to identify mechanisms that may explain the link between temperament and psychopathology. The results of the current study present new and exciting findings implicating ER as a mechanism to explain the link between temperament dimensions and psychopathology among youth with ADHD. These findings have important theoretical
implications for future research investigating the development of psychopathology among youth with ADHD.

An abundance of research has indicated temperamental negative affect is a robust predictor of both internalizing and externalizing pathology in broad samples of youth (Rettew & McKee, 2005; Ormel et al., 2005) which would suggest temperamental negative affect is a strong and direct risk factor for psychopathology. The findings of the current study lend partial support to this theory. Indeed, temperamental negative affect directly estimated internalizing pathology among youth with ADHD. However, temperamental negative affect only indirectly estimated externalizing pathology through emotion dysregulation. These findings suggest the externalizing behavior problems, experienced by many children with ADHD, may result from failures to regulate negative emotions, rather than as a direct result of a dispositional negative affect.

It is not surprising that ER did not account for the relationship between temperamental negative affect and internalizing pathology among youth with ADHD. According to the tripartite model of internalizing disorder (i.e., anxiety, depression) development, both anxiety and depressive disorders share a nonspecific component, “general affective distress” (Clark & Watson, 1991), also commonly known as general negative affect. This body of research suggests negative affect is a large component of internalizing disorders; thus, it is justifiable that temperamental negative affect was a direct estimator of internalizing pathology among youth with ADHD. Although ER did not explain the link between temperamental negative affect and internalizing pathology, results indicated ER was also a direct estimator of internalizing pathology among youth with ADHD. High negative affect and difficulties regulating negative emotions both
appear to influence the development of internalizing pathology among youth with ADHD. These findings implicate the importance of including both distal, dispositional temperament dimensions and proximal developmental processes, such as ER, in models of internalizing pathology development among youth with ADHD.

Findings of the current study implicate ER as a mechanistic factor that plays a significant role in the development of externalizing pathology among youth with ADHD. Dispositional negative affect alone does not uniquely estimate externalizing pathology. Rather, temperamental negative affect may predispose children with ADHD to exhibit deficient ER, which then increases risk for co-occurring externalizing pathology. As described previously, the construct temperamental negative affect encompasses traits such as a tendency to display negative moods as well as heightened physiological arousal and reactivity (Oldehinkel et al., 2004; Santucci et al., 2008). Research suggests children with ADHD may actually experience greater overall frustration and more intense frustration in their daily lives (Fogelman, Leaberry, Rosen, Walerus, & Slaughter, 2018). Additionally, they may also demonstrate greater negative emotional reactivity when recalling frustrating events (Fogelman et al., 2018). Thus, it is plausible that children with ADHD both encounter more frustrating situations in their daily lives and exhibit greater reactivity to these frustrating situations (Fogelman et al., 2018; Jensen & Rosen, 2004). Greater frustration and higher emotional reactivity may contribute to emotional and cognitive overload. As a result, children with ADHD may possess fewer resources to cope with and manage distress, which may then result in emotion dysregulation. Children who express negative emotions rather than utilizing adaptive strategies to regulate
emotional arousal may act on negative emotions, leading to a pattern of emotionally-driven externalizing behavior.

The results of the current study support previous findings in the general child psychopathology literature that externalizing behavior may be partially explained by dysregulated negative emotion and emotional undercontrol (Nigg, 2006; Southam-Gerow & Kendall, 2002). Of note, it was hypothesized that only children with high emotion dysregulation who also demonstrated greater deficits in IC on the emotional go/no go task would display greater externalizing pathology. However, this was not the case. ER, rather than the interaction of ER and task-based IC deficits estimated externalizing pathology. In sum, these novel findings highlight the vital role of ER in explaining the relationship between dispositional traits and psychopathology in youth with ADHD.

Another interesting finding that emerged was that ER also explained the link between a temperamental regulation dimension, IC, and both internalizing and externalizing pathology. Thus, ER was implicated as a mechanistic factor to explain the relationship between IC and broad psychopathology. The ability to inhibit and control responses does not directly predict broad psychopathology among youth with ADHD. Instead, IC predisposes children to exhibit emotion dysregulation, which then leads to co-occurring broad psychopathology. Thus, the ability to engage in inhibition is important because it influences ER capacity. Children who exhibit poor IC likely have difficulties inhibiting reactions to negative emotions experienced in their daily lives, leading to a pattern of acting on negative emotions (i.e., emotion dysregulation.) More recent research has been in favor of including ER in models of psychopathology development (Aldao, Gee, De Los Reyes, Seager, 2016). The results of the current study provide strong
support for the inclusion of ER in conceptual models of psychopathology development, particularly among youth with ADHD.

**Clinical Implications.**

The results of the current study have important clinical implications for the assessment and treatment of youth with ADHD. The diagnostic criteria for ADHD in the DSM-5 do not include a consideration of ER deficits (APA, 2013), despite the fact emotion dysregulation is hypothesized to occur in 25-45% of youth with ADHD (Shaw et al., 2014). Researchers have begun to advocate for the addition of an “Emotional Dysregulation” Type in the next text revision of the DSM-5, given the high co-occurrence of ADHD and emotion dysregulation and the impact of emotion dysregulation on functional impairment (Hattatoglu & Mustafa, 2014). In the current study, ER emerged as a transdiagnostic factor estimating both internalizing and externalizing pathology among youth with ADHD, indicating emotion dysregulation may confer risk for comorbid psychopathology among youth with ADHD. Thus, the results of the current study indicate that a thorough assessment for ADHD should include an ER component. The inclusion of an ER component will provide clinicians with useful information about which children may be at risk for future internalizing and externalizing pathology. Additionally, by assessing ER, clinicians may be able to more effectively tailor treatment interventions to address emotionally-driven internalizing or externalizing pathology among youth with ADHD who exhibit comorbid psychopathology.

In the current study, an association between temperament dimensions and emotion dysregulation was observed, indicating children with ADHD who exhibit difficult temperaments such as dispositional negative affect and/or low effortful control
may be predisposed to experience emotion dysregulation. Thus, these findings highlight the importance of screening for temperament early in a child’s life as a means of curtailing the development of future emotion dysregulation. Early, preventative interventions may be implicated for children with ADHD who exhibit difficult temperaments. The Incredible Years Program, a preventative parent/child intervention aimed at promoting child social competence and ER as well as reducing behavior problems, has been adapted for use in youth with ADHD (Webster-Stratton, Reid, & Beauchaine, 2011). Results of a randomized controlled trial evaluating the efficacy of this program in youth with ADHD ages 4-6 revealed that following treatment, youth receiving treatment compared to waitlist controls had lower aggressive, hyperactive, and oppositional symptoms as well as improvements in social competence and ER (Webster-Stratton et al., 2011). These results are promising and suggest that early childhood interventions may prove useful in increasing emotional competence and ER abilities among youth with ADHD who are vulnerable to experiencing emotion dysregulation.

According to Division 53 Society of Clinical Child and Adolescent Psychology Guidelines (2017), the only evidence-based interventions that have received “level one” best support for the treatment of ADHD in youth include behavioral interventions and organization training. The efficacy of cognitive behavioral and emotion-regulation focused interventions have received considerably less attention than behavioral interventions in the ADHD field, and they have yet to receive “level one” best support. The results of the current study revealing the critical role of ER in conferring risk for comorbid psychopathology in youth with ADHD highlight the need for novel emotion-regulation focused interventions for this population of youth.
Traditionally, evidence-based interventions have been developed to target specific disorders (e.g., anxiety, depression) rather than transdiagnostic symptoms or processes (e.g., avoidance, impulsivity, emotion dysregulation; Barlow et al., 2017). Disorder-specific evidence-based interventions are useful for individuals with one primary psychiatric diagnosis; yet, these interventions are not designed to target comorbidities. A reliance on disorder-specific interventions becomes problematic when treating youth with ADHD, given 40-70% of youth with ADHD exhibit at least one comorbid disorder (Larson et al., 2011). Even youth with ADHD who do not meet full criteria for a DSM-5 disorder are likely to exhibit comorbid internalizing and externalizing pathology.

There has been a movement in the intervention research field to develop interventions targeting transdiagnostic factors underlying multiple disorders (Barlow et al., 2017; Chu, 2012). The Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (UP), an emotion-focused cognitive behavioral intervention, was specifically designed to target “temperamental characteristics, particularly neuroticism, and resulting emotion dysregulation, underlying all anxiety, depressive, and related disorders” (Barlow et al., 2017). The UP has now been adapted and validated to target high negative emotion, emotional reactivity, and ER deficits in youth (Bilek & Ehrenreich-May, 2012; Ehrenreich, Goldstein, Wright, & Barlow, 2009; Seager, Rowley, & Ehrenreich-May, 2014; Kennedy, Bilek, & Ehrenreich-May, 2019). Research evaluating the efficacy of the UP for youth has primarily included children with diagnoses of anxiety and/or depression (Kennedy et al., 2019). The UP for youth has been found to reduce anxiety and depressive symptoms and improve ER and cognitive reappraisal abilities (Bilek & Ehrenreich-May, 2012). It is unclear whether the UP is a feasible or efficacious
intervention for the treatment of emotion dysregulation and concurrent internalizing and externalizing pathology in youth with ADHD. However, findings from the current study implicating ER as a transdiagnostic mechanism linking temperamental negative affect to psychopathology among youth with ADHD provide a rationale for further investigation of the efficacy of unified transdiagnostic treatments for ADHD youth.

One of the most clinically relevant and important findings from the current study was that externalizing pathology, common in youth with ADHD, are in part, emotionally-driven. Thus, strict behavioral interventions, commonly employed for the treatment of youth with ADHD, may not target externalizing problems that are influenced by underlying deficits in ER. Indeed, findings from one of the largest treatment studies conducted in youth with ADHD, the Multimodal Treatment Study, revealed that although improvements in externalizing problems are often observed immediately after behavioral treatment, improvements in behavioral functioning are often not maintained over time (Molina et al., 2009). Children with ADHD continue to function significantly worse than their typically developing peers despite receiving intensive behavioral interventions. These findings illustrate the need for alternate treatments for youth with ADHD that may lead to improvements in functioning over time. Results of a recent pilot study evaluating the initial feasibility and efficacy of the Managing Frustration for Children treatment, an emotion-regulation focused intervention for youth with ADHD, found that children with ADHD demonstrated clinically significant improvements in internalizing, externalizing, and ER deficits following treatment (Rosen, 2018). Although it is unclear whether this emotion regulation focused treatment leads to long-term improvements in emotional and behavioral functioning, it provides preliminary evidence that transdiagnostic, emotion
regulation focused treatments may be feasible and efficacious for treating comorbid
symptoms among youth with ADHD.

Limitations

The current study presents evidence of the critical role of ER in development of
psychopathology among youth with ADHD; however, there are several limitations that
should be addressed. First, the current study served as an initial cross-sectional
investigation of the relations between temperament, ER, and psychopathology, measured
concurrently. It is not possible to determine the temporal relationship between constructs;
thus, effects should not be interpreted as causal. A body of research has indicated
temperament is a biological, dispositional trait preceding the development of ER and
psychopathology processes (Rettew, 2005; Zalewski, Lengua, Wilson, Trancik, &
Bazinet, 2011); however, this cannot be ascertained given the cross-sectional nature of
the current study. Although the temporal relationship between ER and psychopathology
has been debated in the literature, ER has been theorized as an early developmental
process that precedes the development of psychopathology (Chaplin & Cole, 2005).
Additionally, in a study that investigated the temporal relationship between ER and
psychopathology, ER predicted elevations in internalizing and externalizing pathology
over time; yet, psychopathological symptoms did not predict greater ER over time
(McLaughlin et al., 2011); thus, it is plausible that ER predicts psychopathology
development. However, it is also possible that psychopathology may predict deficiencies
in ER capacity. Future longitudinal research assessing constructs at multiple time points
would permit for an investigation of temporal precedence between constructs, which
would more clearly establish temperament and ER as risk factors for psychopathology.
Secondly, the relationship between temperament and psychopathology has often proved difficult to study, given high overlap between temperament and psychopathology constructs. Despite some potential overlap between these constructs, research has suggested temperament, ER, and psychopathology are distinct, but related theoretical constructs (Zalewski et al., 2011). In an attempt to control for symptom overlap, some studies investigating the relationship between temperament and psychopathology have removed confounding items; however, these studies suggest significant relationships still exist between temperament and psychopathology even when this methodology is employed (Lemery et al., 2002). The current study was underpowered to perform the analyses required to remove confounding items. However, the current study utilized well-validated assessment tools to measure constructs of interest; thus, constructs were theoretically distinct. Additionally, there were no problems with multicollinearity. Future investigations exploring the relationship between temperament and psychopathology may benefit from controlling for item overlap to ensure constructs are statistically distinct.

One strength of the current study is the use of AC and IC tasks to provide an assessment of self-regulation that was not subject to reporter biases. These tasks were correlated with child report of ER and internalizing pathology measures. However, tasks were not used to assess ER constructs. Several different methodologies have been utilized to assess ER in children including use of self/parent report measures, observations tasks, frustration induction tasks, biological/physiological indices, and ecological momentary assessment (Adrian, Zeman, & Veits, 2011). The current study relied on self and parent report measures to assess ER, which are subject to retrospective report biases. However, the current study did examine both parent and child report of symptoms to provide input
from multiple reporters. Future research would benefit from the inclusion of task-based assessments of ER to reduce the limitations of self-report and to permit for an assessment of multidimensional aspects of ER.

The current study empirically tested a proposed conceptual model of psychopathology among youth with ADHD. The conceptual model explored child-level variables (e.g., temperament, ER) theorized to relate to psychopathology. Although many of the empirically tested models explained significant variance in psychopathology, there are likely many other child-level variables and contextual risk factors related to the development of psychopathology in youth with ADHD. For instance, numerous studies have linked negative parenting, maternal depression, and stressful life events, among other factors, to psychopathology in youth (Goodman et al., 2014; Mesman & Koot, 2001; Morris et al., 2002). Thus, future studies would benefit from the inclusion of other risk factors in models of psychopathology development; however, this study takes an important first step in identifying potential risk factors and mechanisms for psychopathology among youth with ADHD.

Finally, to the author’s knowledge, this was one of the first studies investigating the relationship between temperament, ER, and psychopathology among an ADHD only sample. Thus, the sample size was relatively small ($n=46$). Although significant effects were observed, the study was likely underpowered to detect significant interactions between temperament dimensions in the estimation of ER and psychopathology. As described previously, the results of the current study have important implications for future research. This study should be replicated in a larger sample to determine if the findings are generalizable to the larger population of youth with ADHD.
Conclusions

Over half of youth with ADHD meet criteria for a comorbid internalizing or externalizing disorder. These children with ADHD who meet criteria for a comorbid disorder are at significantly greater risk for poorer outcomes across the lifespan; yet, conceptual models explicating the development of psychopathology, specifically among youth with ADHD, have not been empirically tested. The current study took an important first step in empirically evaluating a theoretical model of psychopathology development among an ADHD only sample.

Findings implicated the importance of both dispositional temperament factors and developmental ER processes in estimating concurrent internalizing and externalizing pathology among youth with ADHD. Of critical importance, this study identified ER as a mechanism that explains the relationship between temperamental negative affect and externalizing pathology. Temperamental negative affect does not directly predict externalizing pathology, but instead, appears to predispose children to emotion dysregulation, which then estimates subsequent externalizing pathology. ER also emerged as a mechanism to explain the relationship between IC and broad internalizing and externalizing pathology implicating ER as a robust and mechanistic transdiagnostic risk factor for psychopathology development. Overall, the results of this study advocate for the integration of both temperament and ER into conceptual models of psychopathology development among youth with ADHD. This study also highlights the critical need for novel emotion regulation focused cognitive behavioral interventions for youth with ADHD and comorbidity, given this population of youth currently have few treatment options that lead to sustained improvements in functioning over time.
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Table 1. *Demographic Characteristics of the Sample*

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<thead>
<tr>
<th>Variable</th>
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<th>Percent</th>
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<tr>
<td><strong>Sex</strong></td>
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<tr>
<td>Males</td>
<td>26</td>
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<td>Females</td>
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<td>African American</td>
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<td>Latino/Hispanic</td>
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</tr>
<tr>
<td>Asian</td>
<td>1</td>
<td>2.2</td>
</tr>
<tr>
<td>Biracial</td>
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<tr>
<td>$0,000-$10,000</td>
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<td>4.3</td>
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<tr>
<td>$25,001-$40,000</td>
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<td>$40,001-$75,000</td>
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<tr>
<td>Over 75,000</td>
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<td>60.9</td>
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<td><strong>ADHD Presentation</strong></td>
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<tr>
<td>Inattentive</td>
<td>28</td>
<td>60.9</td>
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<tr>
<td>H/I*</td>
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<td>2.2</td>
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<tr>
<td>Combined</td>
<td>17</td>
<td>37.0</td>
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### ADHD Meds

<p>| | | |</p>
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<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>No</td>
<td>32</td>
<td>69.6</td>
</tr>
<tr>
<td>Yes</td>
<td>14</td>
<td>30.4</td>
</tr>
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Note. *H/I* = Hyperactive/Impulsive
Table 2.  

*Comorbidity on the Diagnostic Interview Schedule for Children-Parent*

<table>
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<th>Diagnosis</th>
<th>$N$</th>
<th>Percent</th>
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<td>Social Phobia</td>
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<tr>
<td>Separation Anxiety</td>
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<td>Generalized Anxiety</td>
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<td>Dysthymia</td>
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<tr>
<td>Oppositional Defiant</td>
<td>14</td>
<td>30.4</td>
</tr>
</tbody>
</table>

*Note.* Total sample size $N = 46.$
Table 3.

*Bivariate Correlations Between Cognitive Emotion Regulation Questionnaire Subscales*

<table>
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<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Self Blame</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2. Positive Refocusing</td>
<td>-.13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Refocus on Planning</td>
<td>-.05</td>
<td>.79**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Positive Reappraisal</td>
<td>.20</td>
<td>.74**</td>
<td>.83**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Putting into Perspective</td>
<td>-.29</td>
<td>.48**</td>
<td>.51**</td>
<td>.51**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Catastrophizing</td>
<td>.63**</td>
<td>-.14</td>
<td>.04</td>
<td>-.04</td>
<td>-.02</td>
<td></td>
</tr>
<tr>
<td>7. Other Blame</td>
<td>.50**</td>
<td>-.21</td>
<td>-.06</td>
<td>-.08</td>
<td>-.11</td>
<td>.41**</td>
</tr>
</tbody>
</table>

*Note. *p < .05; **p < .01*
Table 4.

*Point-Biserial Correlations Between Demographic Variables and ER, Task, and Psychopathology Variables*

<table>
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<tr>
<th></th>
<th>Sex</th>
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*Note.* *p < .05; **p < .01
Table 5.

*Bivariate Pearson’s Correlations Between Parent and Child Reported Temperament and ER Variables*

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*Note. *p < .05; **p < .01*
Table 6.

*Multiple Regression: Parent Reported EATQ Temperamental Indicators Estimating Parent Reported ERC Emotion Dysregulation.*

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*Note. N= 46.*
Table 7.


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Note. $N = 45$
Table 8.

*Demographic Characteristics of Stroop and Emotional Go/No Go Tasks.*

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*Note. N Stroop Task = 43; N Emotional Go/No Go (EGNG) Task N = 41*
Table 9.

Bivariate Pearson’s Correlations Between Parent and Child EATQ Temperament Variables, Stroop and Emotional Go/No Go Tasks, Indicators of Emotion Dysregulation, and Internalizing Variables.

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Note. *p < .05; **p < .01
Table 10.

*Hierarchical Regression: Child Reported ERICA Emotion Dysregulation Estimating Affective IC on the Emotional Go/No Go Task.*

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*Note. $N = 40.$*
Table 11.

Bivariate Pearson’s Correlations Between Parent and Child Indicators of ER, Internalizing, and Externalizing Pathology.

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*Note.* *p < .05; **p < .01
Table 12.


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Note. $N = 37$. 
Table 13.

*Hierarchical Regression: ERICA, ERC, and CERQ-k Emotion Dysregulation Estimating Child Reported CDI Depressive Pathology.*

<table>
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<tr>
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<th>$\Delta F$</th>
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<th>$\beta$</th>
<th>$t$</th>
<th>$p$ value</th>
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<td>2.67</td>
<td>.011</td>
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<td>.210</td>
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<td>.16</td>
<td>.872</td>
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<td>CERQ Adaptive</td>
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<td>-.28</td>
<td>.007</td>
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<td>.20</td>
<td>.39</td>
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*Note. N= 45.*
Table 14.

*Multiple Regression: Child Reported ERICA and CERQ-k Emotion Dysregulation Estimating Child Reported RPAQ Reactive Aggression.*

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<td>.01</td>
<td>.52</td>
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<tr>
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</tr>
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<td>CERQ Maladaptive</td>
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<td>.02</td>
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*Note. $N = 45.$*
### Table 15.

_Bivariate Pearson’s Correlations Between Parent Indicators of Temperament, ER, Internalizing, and Externalizing Pathology._

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<td>3. EATQ IC</td>
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<td>4. EATQ AC</td>
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<tr>
<td>5. ERC Total</td>
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<td>.49**</td>
<td>-.48**</td>
<td>.17</td>
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<tr>
<td>6. CBCL Internalizing</td>
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<td>.58**</td>
<td>-.36*</td>
<td>.22</td>
<td>.56**</td>
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<tr>
<td>7. CBCL Externalizing</td>
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<td>.35*</td>
<td>-.51**</td>
<td>-.10</td>
<td>.68**</td>
<td>.43**</td>
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</table>

*Note.* *p* <.05; **p** <.01
Table 16.

*Direct and Indirect Effects of Temperamental Negative Affect on Internalizing Pathology Through Emotion Dysregulation*

<table>
<thead>
<tr>
<th></th>
<th>M (ER)</th>
<th>Y (Int)</th>
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<td>X (NA)</td>
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<td>.27</td>
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<td>M (ER)</td>
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<tr>
<td>Constant</td>
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</table>

*Note. N = 45.*
Table 17.

**Direct and Indirect Effects of Temperamental IC on Internalizing Pathology Through Emotion Dysregulation**

<table>
<thead>
<tr>
<th></th>
<th>M (ER)</th>
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<th>Y (Int)</th>
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<td>SE</td>
<td>p</td>
<td>β</td>
<td>Coeff.</td>
<td>SE</td>
<td>p</td>
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<tr>
<td>X (IC)</td>
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<tr>
<td>M (ER)</td>
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<td>--</td>
<td>-2.52</td>
<td>9.31</td>
<td>.790</td>
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*Note. N = 45.*
Table 18.

Direct and Indirect Effects of Temperamental Negative Affect on Externalizing Pathology Through Emotion Dysregulation

<table>
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<tr>
<th></th>
<th>M (ER)</th>
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<th>Y (Ext)</th>
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<tr>
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<td>Coeff.</td>
<td>SE</td>
<td>p</td>
<td>β</td>
<td>Coeff.</td>
<td>SE</td>
<td>p</td>
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<tr>
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<td>.08</td>
<td>&lt;.001</td>
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<td>.28</td>
<td>1.53</td>
<td>.85</td>
</tr>
<tr>
<td>M (ER)</td>
<td>--</td>
<td>--</td>
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<td>.66</td>
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Note. N = 46.
Table 19.

Direct and Indirect Effects of Temperamental IC on Externalizing Pathology Through Emotion Dysregulation

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<th>Y (Ext)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>β</td>
<td>Coeff.</td>
<td>SE</td>
<td>p</td>
<td>β</td>
<td>Coeff.</td>
<td>SE</td>
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<tr>
<td>X (IC)</td>
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<td>8.73</td>
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*Note. N = 37.*
Table 20.

*Hierarchical Regression: The Interaction of EATQ Negative Affect and Stroop Task Performance on the Estimation of CBCL Internalizing Pathology.*

<table>
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<th>Step/variable</th>
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<td>-.003</td>
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<td>25.57</td>
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*Note. N = 42.*
Table 21.


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<td>.30</td>
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Note. $N = 33$. 
Figure 1. A trait model of internalizing and externalizing pathology among children with ADHD
Figure 2. A risk factor model of the ADHD primarily internalizing pathology outcome
Figure 3. A risk factor model of the ADHD primarily externalizing pathology outcome.

A. Distal Risk Factors:
- Neurobiological abnormalities: Deficits “hot” EF (orbital and medial PFC; ventral striatum); deficiencies in mesolimbic dopamine system; altered reinforcement sensitivity, deficient motivation and reward processing
- Temperamental influences: temperamental frustration, approach

B. Possible Mechanisms
- Dysfunction in the ventral striatum and deficiencies in the mesolimbic dopamine system give rise to deficient motivation and reward processing, which then lead to poor inhibitory control in situations with high affective involvement or in situations in which reward systems are signaled
- Temperamental frustration and approach leads to emotional approach

C. Proximal Risk Factors:
- Inhibitory control

D. Mechanisms & Moderators
- Conditions decreasing adaptive emotional and behavioral regulation; in situations of high affective involvement or reward, trait frustration and increased reactivity, and poor inhibitory control interact to produce externalizing pathology
Figure 4. A Model of The Indirect Effect of IC on Internalizing Pathology Through Emotion Dysregulation
Figure 5. A Model of The Indirect Effect of Negative Affect on Externalizing Pathology Through Emotion Dysregulation

- a, $\beta = .49^{**}$
- b, $\beta = .66^{**}$
- c', $\beta = .03$
- c, $\beta = .35^{*}$
Figure 6. A Model of The Indirect Effect of IC on Externalizing Pathology Through Emotion Dysregulation

\[ a, \beta = -0.46^{**} \]

\[ b, \beta = 0.50^{**} \]

\[ c', \beta = -0.16 \]

\[ c, \beta = -0.39^* \]
CURRICULUM VITAE
Kirsten Leaberry, M.S.

Department of Psychological and Brain Sciences
University of Louisville | Louisville, KY 40292
Kirsten.leaberry@louisville.edu | (301) 606-3896

EDUCATION:

Doctor of Philosophy, Clinical Psychology (Anticipated 2020)
University of Louisville, Louisville, KY
Advisor: Paul Rosen, Ph.D.
Dissertation: Reactive and Regulative Temperament Dimensions, Emotion Regulation, And Concurrent Internalizing and Externalizing Pathology Among Youth with ADHD
Current GPA 4.00

Masters of Science, Clinical Psychology (December 2017)
University of Louisville, Louisville, KY
Advisor: Paul Rosen, Ph.D.
Cumulative GPA 4.00

Masters of Arts, Psychology, General Track (May 2015)
University of North Carolina Wilmington
Advisor: Kate Nooner, Ph.D.
Thesis: A Comparison of Behavioral and Cognitive Outcome Assessment from a Neurofeedback Clinic for Children with ADHD
Cumulative GPA 4.00

Bachelor of Arts, Psychology (December 2012)
University of North Carolina Wilmington
Advisor: Caroline Clements, Ph.D.
Magna Cum Laude, Cumulative GPA 3.86
HONORS & AWARDS

2019  Stanley A. Murrell Scientist-Practitioner Award, University of Louisville
2019  Award for Peer Mentorship, University of Louisville
2018  Preliminary Examination, Distinguished Pass, University of Louisville
2018  Award for Excellence in Research, University of Louisville
2017  Award for Excellence in Clinical Work, University of Louisville
2016  University Fellowship, University of Louisville
2015  University Fellowship, University of Louisville
2014  Neurocognitive Therapies/Translational Research Special Interest Group: Eighth Annual Student Poster Competition, ABCT, Third place
2011  Chancellor’s Achievement Award, University of North Carolina Wilmington

CLINICAL EXPERIENCE

2017-Present  Cognitive Behavior Therapy Treatment Team
Noble H. Kelley Psychological Services Center, Louisville, KY
Graduate Student Therapist
- Conduct therapy intake evaluations for adults and children with a variety of psychological disorders
- Formulate client case conceptualizations and treatment plans with a diverse client population; present grand rounds case formulations at team meetings
- Implement and tailor evidence-based interventions for the treatment of children and adults with GAD, SAD, selective mutism, OCD, and mood disorders
- Attend IEP and 504-Plan meetings to advocate for accommodations and modifications for clients
- Participate in weekly group supervision and peer supervision
- Supervisors: Janet Woodruff-Borden, Ph.D. & Jenny Petrie, Ph.D.

2017-Present  Noble H. Kelley Psychological Services Center (PSC), Louisville, KY
Graduate Clinical Teaching Assistant
- Coordinate initiation of treatment and assessment services for individuals contacting the clinic, including individuals in crisis
- Collaborate with external agencies to provide referrals, outreach, and client case management
- Conduct intake interviews
- Serve as first-line contact for management of crisis situations within the PSC
- Provide peer supervision to graduate students including assistance with intakes, therapy services, assessments, and managing crisis situations
- Facilitate adherence to clinic operating procedures
- Responsible for management of clinical operations including scheduling, payment records, database and chart audits
- Serve as Community Outreach Coordinator managing Depression and Anxiety Screening events, health fairs, and community presentations
- Supervisor: Bernadette Walter, Ph.D.

2017-2018

**Pediatric Psychology Inpatient Consultation/Liaison Service**
**Norton Children’s Hospital, Louisville, KY**

*Practicum Student*

- Evaluated children with acute and chronic medical illnesses at the request of attending physicians to assist with coping, treatment adherence, and communication between children, families, and hospital staff
- Implemented evidence-based interventions for children and families undergoing long term hospitalizations to promote ongoing development and optimal functioning post-discharge
- Conducted psychosocial pre-transplant evaluation and provided ongoing inpatient care for children awaiting solid organ transplants
- Conducted risk assessments for patients following suicide attempts with dispositions for psychiatric care following medical stabilization
- Collaborated with interdisciplinary treatment teams to formulate inpatient treatment plans and to provide dispositions for outpatient care
- Participated in weekly individual and live supervision
- Wrote comprehensive electronic medical record intake and progress notes in Epic medical records
- Supervisor: Bryan Carter, Ph.D.

2017-2018

**Pediatric Psychology Outpatient Clinic**
**Bingham Child Guidance Clinic**
**University of Louisville Department of Pediatrics, Louisville, KY**

*Practicum Student*

- Provided short-term after-care for medically ill children following hospital discharge to promote recovery and facilitate continuity in care
- Implemented and tailored evidence-based treatment on an outpatient basis for children with chronic medical conditions and chronic pain, psychosomatic disorders, and related anxiety and mood symptoms and disorders
- Co-facilitated the Chronic Health and Illness Recovery Program group intervention, a manualized intervention for adolescents with chronic illnesses
- Provided outpatient therapy for a patient following suicide attempt
- Conducted a psychoeducational post-transplant evaluation for a patient following heart transplant and wrote comprehensive evaluation report to facilitate appropriate school accommodations
- Supervisor: Bryan Carter, Ph.D.

2016-Present **Child Assessment and Testing Practicum**
**Noble H. Kelley Psychological Services Center, Louisville, KY**
**Graduate Student Therapist**
- Administer psychological test batteries for children and adolescents ages 4-17 for diagnostic and assessment purposes; write comprehensive assessments by integrating information from parents, children, teachers, and providers; assess for ADHD, learning disabilities, language disorders, mood disorders, and behavioral disorders.
- Conduct intellectual and advanced placement assessment for gifted and talented program
- Utilize assessment tools including WISC-V, WJ-III, WPPSI-IV, WIAT-III, EVT-2, & PPVT-IV
- Prepare comprehensive integrated reports and conduct feedback sessions
- Consultation to parents, schools, therapists
- Supervisor: Bernadette Walter, Ph.D.

2016-Present **Adult Assessment and Testing Practicum**
**Noble H. Kelley Psychological Services Center, Louisville, KY**
**Graduate Student Therapist**
- Conduct psychological and neuropsychological assessment batteries for diagnostic and assessment purposes for adults; assess for ADHD, mood disorders, and anxiety disorders
- Utilize assessment tools including the WAIS-IV, WJ-III, MMPI, MCMI, CPT
- Administer semi-structured interview for diagnostic assessment of adults
- Prepare comprehensive integrative reports and conduct feedback sessions
- Supervisor: David Winsch, Ph.D.

2016-2017 **Child Psychology Assessment and Treatment Private Practice Practicum**
**Square One: Specialists in Child and Adolescent Development**
**Practicum Student**
- Provided evidence-based treatment to children ages 3-17 including behavior therapy, cognitive behavioral therapy, parent-child interaction therapy, and habit reversal training
- Conducted comprehensive psychological evaluations for concerns including ADHD, learning disorders, anxiety, OCD, and mood disorders
- Utilized assessment tools including the WISC-V, WPPSI-IV, WAIS-IV, WJ-IV, WIAT-III, Beery Buktenica VMI-6, CPT, CTOPP-2, & GORT-5
- Participated in weekly supervision and team consultation
- Supervisor: David Causey, Ph.D.

2015-2017
**Children with ADHD & Related Difficulties (CARDS) Treatment Team**
**Noble H. Kelly Psychological Services Center, Louisville, KY**
*Graduate Student Therapist*
- Provided evidence-based assessment and treatment to children ages 6-17 with ADHD and related difficulties
- Utilized evidence-based interventions including behavior therapy, cognitive behavior therapy, collaborative & proactive solutions (Ross Greene), EX/RP
- Provided manualized treatment, Organizational Skills Training for Children with ADHD (Gallagher, Abikoff, & Spira)
- Co-therapist for the Managing Frustration for Children group, an 11-week manualized intervention for children exhibiting difficulties with emotion regulation
- Utilized assessment tools including the WISC-V, WAIS-IV, WJ-III
- Wrote comprehensive integrated evaluation reports
- Conducted school observations for assessment clients
- Attended 504-plan and IEP school meetings to facilitate school accommodations and modifications for therapy clients
- Participated in weekly individual and group supervision
- Supervisor: Paul Rosen, Ph.D.

2014
**Pediatric Neurofeedback Clinic**
**University of North Carolina Wilmington**
*Clinic Coordinator*
- Coordinated and managed daily clinic activities including recruitment and scheduling of new participants, conducting baseline screening visits, and scheduling
- Conducted semi-structured diagnostic clinical interviews (K-SADS-PL) to assess for ADHD and comorbid disorders
- Consulted weekly with principal investigator and co investigators
- Directed and trained research assistants
- Wrote standard operating clinic procedures
- Administered electroencephalography and neurofeedback to participants
- Managed online research database for collection of pre, post, and follow-up participant data
Supervisor: Kate Nooner, Ph.D.

2012-2013  **Child Advocacy and Parenting Place, Wilmington, NC**  
*Volunteer*  
- Provided childcare services to at-risk youth during a weekly grandparent support group for caregivers of children removed from parental custody  
- Managed and cared for children with developmental, emotional, and behavioral disorders

2013  **Strategic Behavioral Center, Leland, NC**  
*Psychiatric Residential Treatment Facility*  
*Residential Advisor*  
- Advisor at psychiatric residential treatment facility for adolescents ages 12-17  
- Provided care for residents in the facility with various mental health diagnoses including post-traumatic stress disorder, conduct disorder, mood disorders, anxiety, and ADHD  
- Utilized a token economy system for behavior management on the unit  
- Provided instruction on coping skills to facilitate adjustment with long-term residential stay  
- Facilitated completion of daily client goals  
- Supervised client visitation with family members; provided supervision of adolescents in the school classroom

2010  **The Arc, Frederick, MD**  
*Skills Educator*  
- Provided developmentally and intellectually disabled adults with skills training and instruction on basic daily living skills  
- Aided an adult with integration into her community

2008-2009  **Heartly House Domestic Violence Shelter, Frederick, MD**  
*Volunteer*  
- Provided childcare to children temporarily living in the domestic violence shelter  
- Organized shelter donations  
- Observed emergency hotline phone calls  
- Aided with community outreach program

**RESEARCH EXPERIENCE**

2015-Present  **Research on ADHD and Children’s Emotion Regulation (RACER) Lab**
University of Louisville
Graduate Research Assistant
- Recruitment and scheduling of research participants
- Wrote IRB protocol and protocol amendments for research studies
- Wrote standardized operating procedures for research studies and lab tasks
- Administered brief intellectual (WASI-II) and achievement tests (WRAT-IV) to participants
- Administered structured diagnostic clinical interview to parents (DISC-P)
- Administered electrocardiogram to participants
- Wrote comprehensive evaluation reports integrating information from structured interviews, parent and child-report questionnaires, teacher questionnaires, and intellectual and achievement tests
- Provided feedback to parents
- Managed ecological momentary assessment data
- Created and programmed laboratory tasks in ePrime, a psychology software tool
- Supervised and trained undergraduate and graduate students in the lab
- Aided in supervision of undergraduate honor’s thesis and individual research projects
- Research Advisor: Paul Rosen, Ph.D.

2013-2015 Trauma and Resilience Laboratory
University of North Carolina Wilmington
Graduate Research Assistant
- Coordinated the lab Pediatric Neurofeedback Clinic for children with ADHD
- Collected data from child and college student populations including children with ADHD and trauma exposure and college students with trauma exposure and substance use
- Administered semi-structured diagnostic clinical interviews (K-SADS-PL) to parents and children
- Administered electroencephalography and neurofeedback sessions to participants
- Trained undergraduate and graduate research assistants on laboratory procedures
- Managed online data collection database
- Research Advisor: Kate Nooner, Ph.D.

2011-2013 Experimental Psychopathology Laboratory
University of North Carolina Wilmington
Undergraduate Research Assistant
- Experimenter for a study assessing aggressive responding in child witnesses of intimate partner violence
- Experimenter in a study assessing intimate partner violence perpetration and aggressive personality traits in male and female undergraduate students
- Research Advisor: Caroline Clements, Ph.D.

AWarded Grants

2018-2019
Title of Project: *Reactive and Regulative Temperament Dimensions, Emotion Regulation, and Psychopathology in Youth with ADHD*
Role: PI (Faculty Mentor: Paul Rosen, Ph.D.)
Agency: University of Louisville College of Arts & Sciences Research & Creative Activities
Awarded Amount: $500.00
Study Aims: Comprehensively examine temperamental and emotion regulation deficits among children with ADHD utilizing multi-method assessment (i.e., behavioral parent report, child report measures, cognitive tasks) to understand the effects of these deficits on concurrent internalizing and externalizing pathology.

2017 Grant: Graduate Network in Arts and Sciences Research Grant
Agency: University of Louisville
Purpose: Travel allotment to present poster at Association for Behavioral and Cognitive Therapies Convention
Awarded Amount: $250

2016 Grant: Graduate Student Council Travel Grant
Agency: University of Louisville
Purpose: Travel allotment to present poster at Association for Behavioral and Cognitive Therapies Convention
Awarded Amount: $350

2014 Grant: Graduate Student Association Research Travel Grant
Agency: University of North Carolina Wilmington
Purpose: Travel allotment to present poster at Association for Behavioral and Cognitive Therapies Convention
Awarded Amount: $400.00

2012 Grant: Undergraduate Student Research Travel Grant
Agency: University of North Carolina Wilmington
Purpose: Travel allotment to present poster at Association for Psychological Science Convention
Awarded Amount: $1000
PUBLICATIONS

Original Articles: Peer-Reviewed Journals


**Book Chapters**


**Manuscripts under Review**


**PRESENTATIONS**

**Symposia:**

*presenting author

**Conference Presentations**


17. Leaberry, K. D., Fogleman, N. D., Walerius, D. M., & Rosen, P. J. (2015, November). Differences in Levels of Frustration Following a Narrative Task are Related to ADHD. Poster presented at Association for Behavioral and Cognitive Therapies Convention, Chicago, IL.


**TEACHING EXPERIENCE**

2017-Present  
**University of Louisville, Louisville, KY**  
**Department of Psychological and Brain Sciences, Clinical Psychology Program**  
*Clinic Graduate Teaching Assistant*  
Graduate Courses  
*Clinical Interviewing* (PSY 693), Lead Instructor: Barbara Stetson, Ph.D.  
*Intellectual and Cognitive Assessment* (PSY 680), Lead Instructor: Bernadette Walter, Ph.D.

2013 **University of North Carolina Wilmington**  
**Department of Psychology**  
*Guest Lecturer*  
Undergraduate Course  
*Honors Abnormal Psychology*, Lead Instructor: Caroline Clements, Ph.D.

2013-2015  
**Center for Teaching Excellence (CTE)**  
**University of North Carolina Wilmington**  
*Graduate Teaching Assistant*  
- Assisted in organizing workshops aimed at educating faculty on effective teaching practices, mentoring models, and applied learning  
- Assisted with new faculty orientation aimed at orienting faculty to university policies and best teaching practices  
- Graduate student representative at dean candidate luncheon  
- Authored newsletters for faculty and staff  
- Supervisor: Caroline Clements, Ph.D.

2013-2014  
**Applied Learning & Teaching Community (ALTC)**  
**University of North Carolina Wilmington**  
*Graduate Teaching Assistant*
- Assisted with implementing the quality enhancement plan at UNCW
- Collaborated with UNCW faculty members in implementation of the applied learning practices in the classroom
- Assisted in coordinating the Bringing Theory to Practice Association for American Colleges and Universities Grant assessing applied learning practices and psychosocial well-being in UNCW students
- Assisted in faculty workshops on applied learning practices
- Assisted in writing ALTC newsletters
- Supervisor: Jess Boersma, Ph.D.

CERTIFICATIONS AND TRAININGS

<table>
<thead>
<tr>
<th>Year</th>
<th>Course Description</th>
<th>Institution</th>
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<tbody>
<tr>
<td>2018</td>
<td>Safe Zone LGBTQ Ally Training, University of Louisville</td>
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<tr>
<td>2018</td>
<td>Transgender and Gender Creative Youth: Mental Health and Evidence Based Treatments, LGBT Health Care Summit, sponsored by Humana</td>
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<tr>
<td>2017</td>
<td>Graduate Teaching Assistant Training, University of Louisville</td>
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<tr>
<td>2017</td>
<td>Best Practices in Pediatric Pretransplant Psychosocial Evaluation training, Bingham Clinic</td>
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<tr>
<td>2017</td>
<td>Suicide and Risk Assessment Series Training, Bingham Clinic</td>
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<tr>
<td>2017</td>
<td>Children’s Health and Illness Recovery Program Workshop, Bingham Clinic</td>
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<td>2016</td>
<td>Intimate Partner Violence, The Center for Women and Families</td>
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<td>2016</td>
<td>Introduction to Dialectical Behavior Therapy, Xavier University Training, Louisville, KY</td>
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<tr>
<td>2015</td>
<td>Safe Zone LGBTQ Ally Training, University of Louisville</td>
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<tr>
<td>2015</td>
<td>Microaggressions, racial stress and trauma, Center for Mental Health Disparities Training, University of Louisville</td>
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PROFESSIONAL AFFILIATIONS

<table>
<thead>
<tr>
<th>Year</th>
<th>Organization</th>
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<tbody>
<tr>
<td>2018-Present</td>
<td>Bipolar Disorders Special Interest Group, Association for Behavioral &amp; Cognitive Therapies</td>
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<tr>
<td>2017-Present</td>
<td>Society for Research and Child Development (graduate student member)</td>
</tr>
<tr>
<td>2015-Present</td>
<td>Association for Behavioral and Cognitive Therapies (graduate student member)</td>
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<tr>
<td>2013-2014</td>
<td>Association for Psychological Science (graduate student member)</td>
</tr>
<tr>
<td>2012-2013</td>
<td>Association for Psychological Science (undergraduate student member)</td>
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PROFESSIONAL SERVICE
2019  Ad-Hoc Reviewer, *Children & Youth Services Review*
2018  Community Outreach Coordinator, Depression Screening Day, University of Louisville
2017  Community Talk, *Stress and Coping*, Gaining Early Awareness and Readiness for Undergraduate Programs (GEAR-UP)
2017  Community Talk, *Stress and Coping*, Professional Education Preparation Program
2017  Ad-Hoc Reviewer, *Journal of Abnormal Child Psychology*
2016  Ad-Hoc Reviewer *Journal of Abnormal Child Psychology*
2013-2014  Graduate Board Member, Experiencing Transformative Education through Applied Learning, University of North Carolina Wilmington