

A MULTI-METHOD INVESTIGATION OF INTRINSIC AND EXTRINSIC  
EMOTION REGULATION IN ATTENTION-DEFICIT/HYPERACTIVITY  
DISORDER

by

ERICA DAWN MUSSER

A DISSERTATION

Presented to the Department of Psychology  
and the Graduate School of the University of Oregon  
in partial fulfillment of the requirements  
for the degree of  
Doctor of Philosophy

September 2013

DISSERTATION APPROVAL PAGE

Student: Erica D. Musser

Title: A Multi-Method Investigation of Intrinsic and Extrinsic Emotion Regulation in Attention-Deficit/Hyperactivity Disorder

This dissertation has been accepted and approved in partial fulfillment of the requirements for the Doctor of Philosophy degree in the Department of Psychology by:

Jeffrey R. Measelle	Chair
Jennifer C. Ablow	Member
Sanjay Srivastava	Member
Jane Squires	Outside Member
Joel T. Nigg	Non-UO Member

and

Kimberly Andrews Espy	Vice President for Research & Innovation/Dean of the Graduate School
-----------------------	--

Original approval signatures are on file with the University of Oregon Graduate School.

Degree awarded September 2013

© 2013 Erica Dawn Musser

## DISSERTATION ABSTRACT

Erica Dawn Musser

Doctor of Philosophy

Department of Psychology

September 2013

Title: A Multi-Method Investigation of Intrinsic and Extrinsic Emotion Regulation in Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) likely involves fundamental alterations in self-regulation. These problems typically have been viewed as involving disruptions in the regulation of cognition and behavior. However, they also have been hypothesized to involve disruptions in emotion regulation. If so, parenting behaviors may take on renewed importance in ADHD, because parents play an essential role in children developing the ability to regulate their emotions independently.

Three studies examined the association between emotion regulation and ADHD. Study 1 examined autonomic nervous system functioning during the experience and regulation of both positive and negative emotions. Study 2 examined coherence among autonomic and behavioral emotional systems. Finally, Study 3 examined the roles of parenting behavior, parental expressed emotion, and child autonomic nervous system functioning. In Studies 1 and 2, participants with ADHD and typically developing youth aged 7 to 11 years old completed an emotion induction and suppression procedure. For Study 3, participants completed a parent-child interaction task coded for parental behavior, and parents completed a five-minute speech sample coded for expressed emotion. Electrocardiogram and impedance cardiography were monitored for children

across all three studies.

The following results were notable. In Study 1, children with ADHD showed atypical autonomic inflexibility (i.e., elevated parasympathetic and sympathetic responding across task conditions). Additionally, children with ADHD were divided according to levels of prosocial behavior. Unlike other children with ADHD, children with very low levels of prosocial behavior displayed blunted autonomic activity across task conditions. In Study 2, specific patterns of reduced coherence among emotion regulatory systems (i.e., facial affective behavior and autonomic nervous system reactivity) were observed among children with ADHD. Finally, in Study 3, high levels of parental expressed emotion were associated uniquely with ADHD, even after controlling for comorbid symptoms. In contrast, parental intrusiveness was associated uniquely with child oppositional defiant and low prosocial behavior, even after controlling for ADHD symptoms. Furthermore, specific, different patterns of autonomic reactivity during the parent-child interaction were associated with ADHD and oppositional defiant behaviors. Across these studies, it is concluded that intrinsic and extrinsic emotion and emotion regulatory systems are disrupted among children with ADHD.

## CURRICULUM VITAE

NAME OF AUTHOR: Erica Dawn Musser

### GRADUATE AND UNDERGRADUATE SCHOOLS ATTENDED:

University of Oregon, Eugene  
University of Rochester, Rochester, New York

### DEGREES AWARDED:

Doctor of Philosophy, Psychology, 2013, University of Oregon  
Master of Science, Psychology, 2008, University of Oregon  
Master of Science, Human Development, 2004, University of Rochester  
Bachelor of Arts, Psychology and Biology, 2003, University of Rochester

### AREAS OF SPECIAL INTEREST:

Physiological Regulation of Emotion  
Emotion Regulation and the Development of Psychopathology  
Development of Externalizing Behaviors and Disorders

### PROFESSIONAL EXPERIENCE:

Research Assistant/Coordinator, Oregon Health & Science University: Heterogeneity of ADHD Longitudinal Study, Portland, OR, August 2008 to present

Research Assistant/Coordinator, University of Oregon: Developmental Sociobiology Lab, Eugene, OR, August 2006 to June 2012

Research Assistant, Mt. Hope Family Center: University of Rochester, Rochester, NY, September 2004 to September 2006

Instructor, University of Oregon, June 2007-July 2011

Graduate Teaching Fellow, University of Oregon, Eugene, OR, 2006-2009

Undergraduate Teaching Assistant, University of Rochester, 2000-2003

Clinical Interviewer, Adult ADHD Research Study: Oregon Health & Science University, Portland, OR, June 2011 to January 2012

Clinical Interviewer, Heterogeneity of ADHD Longitudinal Study: Oregon Health & Science University, Portland, OR, August 2009 to January 2012

Doctoral Student Neuropsychology Intern, Child Psychiatry: Doernbecher Children's Hospital: Oregon Health & Science University, Portland, OR, July 2009 to July 2011

Practicum Student, Early Intervention and Infant Mental Health Practicum: University of Oregon, Eugene, OR, September 2008 to May 2009

Clinical Interviewer, ADHD and Working Memory Study: University of Oregon, Eugene, OR, September 2007 to July 2009

Therapist, Oregon Social Learning Center, Eugene, OR, April 2008 to November 2008

Practicum Student, Cognitive Behavioral Therapy Practicum: University of Oregon Psychology Clinic, Eugene, OR, September 2007-May 2008

#### GRANTS, AWARDS, AND HONORS:

Beverly C. Fagot Memorial Psychology Fellowship, Psychology Department, University of Oregon, 2012

Marthe E. Smith Memorial Science Fellowship, College of Arts and Sciences, University of Oregon, 2012

Norman D. Sundberg Memorial Psychology Fellowship, Psychology Department, University of Oregon, 2012

Early Career Travel Award, National Institute of Child Health & Human Development, Executive Function in Preschool Children Workshop, 2010

Graduate Research Travel Award, Graduate School, University of Oregon, 2007-2010

Early Career Travel Award, International Society Magnetic Resonance in Medicine, 2008

Excellence in Campus Leadership Award, American Psychological Association of Graduate Students, 2008

*Cum Laude*, University of Rochester, 1999-2004

Xerox Scholarship for Excellence in the Social Sciences and Humanities, University of Rochester, 1999-2003

## PUBLICATIONS:

- Musser, E.D., Galloway-Long, H.S., Frick, P.J., & Nigg, J.T. (under review). Autonomic subtypes of attention-deficit/hyperactivity disorder in children, *Journal of the American Academy of Child and Adolescent Psychiatry*.
- Bernstein, R.E., Measelle, J.R., Laurent, H.K., Ablow, J.C., & Musser, E.D. (under review). Sticks and stones may break my bones but words relate to adult physiology? Child abuse experience and women's sympathetic nervous system response while self-reporting trauma, *Journal of Aggression, Maltreatment & Trauma*.
- Costa Dias, T.G., Wilson, V.B., Bathula, D.R., Iyer, S., Mills, K.L., Thurlow, B.L., Stevens, C.A., Musser, E.D., Carpenter, S.D., Mitchell, S.H., Nigg, J.T., Fair, D. A. (under review). Reward circuit connectivity relates to delay discounting in children with attention-deficit/hyperactivity disorder. *European Neuropsychopharmacology*.
- Wild, K.V., & Musser, E.D. (in press). The CANTAB battery to assess executive functioning. In S. Goldstein & J. Naglieri (Eds.), *Handbook on Executive Functioning*. New York: Guilford Press.
- Musser, E.D., Laurent, H.K., & Ablow, J.C. (2012). Neural correlates of maternal sensitivity: An fMRI study, *Developmental Cognitive Neuroscience*.
- Mills, K.L., Bathula, D., Costa Dias, T.G., Fenesy, M.C., Musser, E.D., Stevens, C.A., Thurlow, B.L., Carpenter, S.D., Nagel, B.J., Nigg, J.T., Fair, D.A. (2012). Altered cortico-striatal-thalamic connectivity in relation to spatial working memory capacity in children with ADHD, *Frontiers in Neuropsychiatric Imaging and Stimulation*.
- Musser, E.D., Measelle, J.R., & Ablow, J.C. (2011). Predicting maternal insensitivity: The effects of postnatal depression and respiratory sinus arrhythmia, *Infant Mental Health Journal*.
- Musser, E. D., Baks, R.W., Schmitt, C.F., Ablow, J.C., Measelle, J.R & Nigg, J.T. (2010). Emotion regulation via the autonomic nervous system in children with attention-deficit/hyperactivity disorder (ADHD), *Journal of Abnormal Child Psychology*.
- Wilson, V.B., Mitchell, S.H., Musser, E.D., Schmitt, C.F., Nigg, J.T. (2010). Delay discounting of reward in ADHD: Application in young children, *Journal of Child Psychology and Psychiatry*.

## ACKNOWLEDGMENTS

I thank Drs. Abow, Measelle, Nigg, Srivastava, and Squires for their assistance in the completion of this dissertation. Special thanks are also due to Drs. Richard Backs, Paul Frick, and Joann Wu Shortt, whose familiarity with this literature and these methods was helpful during the planning phases of this undertaking, as well as Drs. Cara Bohon, Sarah Karalunas, and Ida Moadab, and Ms. Colleen Schmitt, whose proofreading, consultation, and support were helpful throughout multiple phases of this undertaking. Additionally, this work could not have been accomplished without the assistance of Ms. Hilary Galloway-Long, Ms. Rebecca Feldman, Ms. Grace Icenogle, and other members of the Heterogeneity of ADHD Lab at the Oregon Health & Science University, who graciously assisted with data collection and preparation. This research was supported in part by grant number 2R01MH 059105 from the National Institutes of Mental Health, awarded to Dr. Joel T. Nigg at the Oregon Health & Science University.

For Dad and Mom, who taught me the value of hard work, determination, and to follow my heart.

## TABLE OF CONTENTS

Chapter	Page
I. INTRODUCTION.....	1
Specific Aims.....	1
Specific Aims of Study 1 .....	3
Specific Aims of Study 2 .....	4
Specific Aims of Study 3 .....	5
Background and Significance for All Three Studies .....	6
Theoretical Basis and Conceptual Framework for Proposed Studies.....	13
Innovation .....	21
II. STUDY 1: PHYSIOLOGICAL EMOTION REGULATION IN ADHD .....	23
Methods.....	23
Summary.....	23
Participants.....	23
Procedures.....	24
Recruitment and Identification Procedures.....	24
Final ADHD and Other Diagnoses .....	25
Identification of Callous/Unemotional Traits .....	26
Exclusion Criteria .....	27
Medication Washout .....	27
Emotion Induction and Suppression Procedure .....	27
Facial Action Coding System .....	29
Physiological Recording.....	29

Chapter	Page
Overview .....	29
Cardiac Preejection Period (PEP) .....	30
Respiratory Sinus Arrhythmia (RSA) .....	30
Data Analysis .....	31
Analytic Plan .....	31
Power Calculations .....	32
Results .....	32
Preliminary Analyses .....	32
Descriptive and Diagnostic Overview of Sample .....	32
Evaluation of Possible Confounds and Task Validity .....	35
Effectiveness of Emotion Induction by Self-Report .....	35
Effectiveness of Emotion Induction by Facial Affective Behavior .....	36
Baseline Physiological Effects .....	38
Task Habituation and Order Effects .....	38
Primary Analyses: Emotion Induction and Suppression on PEP and RSA .....	39
Effects on PEP .....	39
Effects on RSA .....	41
Follow-up Checks on PEP and RSA Results using Continuous CU .....	42
Continuous Effects of CU on PEP .....	42
Continuous Effects of CU on RSA .....	43
Follow-up Checks on PEP and RSA Results by Group .....	43
Discussion .....	44

Chapter	Page
III. STUDY 2: EMOTIONAL RESPONSE COHERENCE IN ADHD.....	50
Methods.....	50
Summary.....	50
Participants.....	51
Procedures.....	51
Recruitment and Identification Procedures.....	52
Final ADHD and Other Diagnoses .....	52
Identification of Callous/Unemotional Traits .....	52
Exclusion Criteria .....	52
Medication Washout .....	52
Emotion Induction and Suppression Procedure .....	52
Facial Action Coding System .....	53
Physiological Recording.....	53
Data Analysis .....	53
Analytic Plan.....	53
First Model.....	53
Second Model .....	54
Third Model .....	56
Power Calculations .....	58
Results.....	59
Preliminary Analyses .....	59
Descriptive and Diagnostic Overview of Sample.....	59

Chapter	Page
Data Reduction for Primary Analyses .....	61
Baseline Effects .....	62
Model 1: Preliminary Analyses .....	63
Model 2: Preliminary Multilevel Analyses.....	64
Model 2 for RSA.....	66
Model 2 for PEP.....	67
Summary of Preliminary Results for Models 1 and 2.....	68
Model 3: Correspondence Analyses .....	69
Model 3 for RSA.....	71
Model 3 for PEP.....	73
Effects of Callous/Unemotional Behavior .....	75
Discussion.....	76
<b>IV. STUDY 3: PARENTING FACTORS IN EMOTION REGULATION IN ADHD.....</b>	<b>86</b>
Methods.....	86
Summary .....	86
Participants.....	87
Procedures.....	87
Recruitment and Identification Procedures.....	87
Final ADHD and Other Diagnoses .....	87
Identification of Callous/Unemotional Traits .....	87
Exclusion Criteria .....	88

Chapter	Page
Medication Washout .....	88
Parent-Child Interaction Task Procedure .....	88
Five Minute Speech Sample and Expressed Emotion Coding.....	89
Physiological Recording .....	90
Data Analysis .....	91
Analytic Plan.....	91
Power Calculations .....	92
Results .....	92
Preliminary Analyses .....	92
Descriptive and Diagnostic Overview of Sample .....	92
Data Reduction for Primary Analyses .....	94
Baseline Effects .....	96
Examination of Task Condition Differences .....	96
Examination of Prerequisites for Mediation and Moderation.....	97
Mediational Analysis .....	101
Moderation Analysis.....	101
Follow-up on Moderation Analysis with ADHD Diagnosis.....	103
Independent Associations Models .....	104
Further Exploration of Independent Associations Models .....	105
Other Explanations for Independent Effects.....	106
Independent Associations Models with Comorbid Symptoms .....	108
Discussion.....	109

Chapter	Page
V. OVERALL DISCUSSION AND CONCLUSIONS.....	118
APPENDICES .....	129
A. TABLES.....	129
B. FIGURES .....	155
REFERENCES CITED.....	171

## LIST OF TABLES

Table	Page
1. List of Studies and Measures Included in Each .....	129
2. Study 1: Descriptive and Diagnostic Statistics for ADHD and Control Groups.....	130
3. Study 1: Self-Assessment Manikin Scores (SAM) Scores across Task Conditions for ADHD and Control Groups .....	132
4. Study 1: Self-Reported Primary Emotion Rating Scores across Task Conditions for ADHD and Control Groups .....	133
5. Study 1: Facial Action Coding System Rating Scores According to Task Condition for ADHD and Control Groups.....	134
6. Study 1: Respiratory Sinus Arrhythmia (RSA; ms <sup>2</sup> ) and Pre-ejection Period (PEP; ms) by Task Epochs for ADHD and Control Groups.....	136
7. Study 2: Descriptive and Diagnostic Statistics for ADHD and Control Groups.....	137
8. Study 2: Hierarchical Linear Model Variables Presented According to Model Tested.....	139
9. Study 2: Raw Facial Action Coding System Ratings According to Task Condition by Group .....	141
10. Study 2: Approach and Avoidance Factor Scores According to Task Conditions by Group.....	143
11. Study 2: Respiratory Sinus Arrhythmia (RSA; ms <sup>2</sup> ) and Pre-ejection Period (PEP; ms) by Task Epochs for ADHD and Control Groups.....	144
12. Study 2: Hierarchical Linear Model Results Presented According to Model Tested.....	145
13. Study 3: Descriptive and Diagnostic Statistics for ADHD and Control Groups.....	147
14. Study 3: Differences in Parent-Child Interaction System Codes According to Group .....	149

Table	Page
15. Study 3: Differences in Parent Expressed Emotion Codes from the Five Minute Speech Sample According to Group .....	151
16. Study 3: Child Respiratory Sinus Arrhythmia (RSA; $ms^2$ ) and Pre-ejection Period (PEP; ms) by Task Epoch for ADHD and Control Groups.....	152
17. Study 3: Correlations among Psychophysiological, Parenting Expressed Emotion, Parenting Behavior, Child Symptoms, and Child Behavior .....	153

## LIST OF FIGURES

Figure	Page
1. Unifying Theoretical Model of the roles of ANS, Emotion, Emotion Regulation, Temperament, Parenting, and Behavior in the Development of Externalizing Pathology .....	155
2. Descriptive Model Examining the Mediating Role of Autonomic Nervous System Activity in the Association between Parenting Behavior and ADHD Symptoms. ....	156
3. Study 1: Mean Cardiac Pre-ejection Period (PEP) Raw Scores for Each of the Task Epochs: Negative Induction (NI), Negative Suppression (NS), Positive Induction (PI), and Positive Suppression (PS) for Control and Full ADHD (Non-divided) Groups .....	157
4. Study 1: Mean Cardiac Pre-ejection Period (PEP) Raw Scores for Each of the Task Epochs: Negative Induction (NI), Negative Suppression (NS), Positive Induction (PI), and Positive Suppression (PS) for Control, ADHD, and ADHD+CU Groups .....	158
5. Study 1: Mean Respiratory Sinus Arrhythmia (RSA) Change Scores for Each of the Task Epochs: Negative Induction (NI), Negative Suppression (NS), Positive Induction (PI), and Positive Suppression (PS) for Control and Full ADHD (Non-divided) Groups .....	159
6. Study 1: Mean Respiratory Sinus Arrhythmia (RSA) Change Scores for Each of the Task Epochs: Negative Induction (NI), Negative Suppression (NS), Positive Induction (PI), and Positive Suppression (PS) for Control, ADHD, and ADHD+CU Groups .....	160
7. Study 2: HLM Results of Correspondence (Standardized Beta Weights) between Facial Affective Behavior (Approach and Avoidance) and Respiratory Sinus Arrhythmia (RSA) during Induction Compared to during Suppression .....	161
8. Study 2: HLM Results of Correspondence (Standardized Beta Weights) between Facial Affective Behavior (Approach and Avoidance) and Cardiac Pre-ejection Period (PEP) during Induction Compared to during Suppression .....	162
9. Study 2: HLM Results of Correspondence (Standardized Beta Weights) between Avoidance Facial Affective Behavior type and Respiratory Sinus Arrhythmia (RSA) across Induction Conditions (Negative and Positive Collapsed) .....	163

Figure	Page
10. Study 2: HLM Results of Correspondence (Standardized Beta Weights) between Avoidance Facial Affective Behavior type and Respiratory Sinus Arrhythmia (RSA) during Positive Induction Compared to Negative Induction .....	164
11. Study 2: HLM Results of Correspondence (Standardized Beta Weights) between Avoidance Facial Affective Behavior type and Cardiac Pre-ejection Period (PEP) during Positive Induction Compared to Negative Induction .....	165
12. Study 3: Regression of Parental Expressed Criticism on Child Attention-Deficit/Hyperactivity Disorder Symptoms .....	166
13. Study 3: Regression of Child Cardiac Pre-ejection Period (PEP) on Child Attention-Deficit/Hyperactivity Disorder Diagnosis .....	167
14. Study 3: Regression of Parent Intrusiveness on Child Callous/Unemotional Behavior Symptoms .....	168
15. Study 3: Regression of Parent Intrusiveness on Child Oppositional Defiant Disorder Symptoms .....	169
16. Study 3: Regression of Child Respiratory Sinus Arrhythmia on Child Oppositional Defiant Disorder Symptoms .....	170

# CHAPTER I

## INTRODUCTION

### *Specific Aims*

What are the roles of intrinsic (e.g., child behavior and psychophysiology) and extrinsic (e.g., parenting behavior and parent expressed emotion) mechanisms of emotion regulation in attention-deficit/hyperactivity disorder (ADHD)? Recent theories identifying emotion dysregulation as an important element in ADHD have the potential to reframe understanding of the nature of the disorder. Yet, this important hypothesis has not been accompanied by empirical studies directly examining emotion regulation, using both behavioral and autonomic nervous system indices, to investigate the exact nature of this disruption. Furthermore, emotion regulation develops in context. Children initially begin to regulate their emotions by observing and being reinforced by their parents, and they gradually take on this role themselves. Although there is empirical evidence to support the influence of parenting behavior on young children's development of the behavioral and physiological regulation of emotion, clarification of whether these processes are altered in ADHD is surprisingly sparse. The present series of studies aimed to evaluate autonomic indices of child emotion and its regulation (including parasympathetic and sympathetic contributions); evaluate the correspondence among autonomic and facial affective behavioral indices of emotion and its regulation; and assess the roles of parenting behavior and parent expressed emotion in shaping the associations between the autonomic regulation of emotion and the number of and types of symptoms occurring and co-occurring with ADHD in childhood.

Across the three studies, four primary hypotheses guided the work, and several

secondary hypotheses were also examined. First, for Study 1, it was hypothesized that ADHD (DSM-IV combined subtype) would be related to alterations in emotion induction and suppression, as assessed through facial affective behavior, self-report, and parasympathetic and sympathetic nervous system functioning. Specifically, it was hypothesized that children with ADHD would display elevated parasympathetic and sympathetic reactivity from homeostasis, which would be especially evident during positive emotions. Second, also for Study 1, it was hypothesized that children with ADHD and co-occurring low levels of prosocial behavior (i.e., callous/unemotional traits), serving here simply as one way to clinically characterize children with uniquely altered emotion processes within the ADHD group, would display blunted autonomic reactivity (i.e., reduced reactivity in parasympathetic and sympathetic activity across emotion-based task conditions).

Turning to Study 2, the third hypothesis was that ADHD would be characterized by low correspondence among the facial affective behavioral and physiological systems of emotion regulation. Low correspondence among the systems was expected among children with ADHD, in part, because previous work has demonstrated that a reduced correspondence among facial affective behavior and autonomic reactivity is characteristic of children with externalizing behavior. However, this has not been addressed for children with ADHD, specifically.

Finally, for Study 3, the fourth hypothesis was that autonomic nervous system activity would mediate the association between parenting behaviors, including expressed emotion and parental sensitive behavior with ADHD and comorbid disruptive behavior symptoms; such that children with parents displaying high levels of expressed emotion

and insensitivity would display specific patterns of autonomic dysregulation, and this dysregulation would be associated with greater behavior and attention problems, including co-occurring symptoms of oppositional defiant disorder. However, children with parents displaying low levels of expressed emotion and sensitivity would display age-appropriate levels of autonomic regulation of emotions, and this regulation would be associated with fewer behavior and attention problems. This dissertation, which utilized data collected at Oregon Health & Science University, was organized as three parallel studies with three concurrent aims.

### **Specific Aims of Study 1**

The specific aim of Study 1 was to evaluate autonomic indices of emotion regulation in children with ADHD.

#### *Hypothesis 1a*

Children with ADHD will have a pattern of parasympathetic and sympathetic reactivity that is augmented (or increased) from homeostatic (or baseline levels of) functioning across the changing affective and regulatory demands of induction and suppression, whereas typically developing children will show autonomic flexibility in the regulation of emotion. Specifically, typically developing children will display increases in parasympathetic activity during negative emotion conditions, as well as during suppression conditions, while displaying parasympathetic withdrawal during the induction of positive emotions.

#### *Hypothesis 1b*

Children with ADHD and co-occurring callous/unemotional traits will display blunted parasympathetic and sympathetic reactivity. Specifically, children with ADHD

and callous/unemotional traits will display little change in either parasympathetic or sympathetic activity from homeostatic functioning across each of the task conditions.

*Hypothesis 1c*

Children with ADHD will display the greatest level of parasympathetic and sympathetic increase from baseline during the positive emotion conditions, as several theories have proposed a specific dysfunction in the approach (positive) system among children with ADHD.

**Specific Aims of Study 2**

The aim of Study 2 was to evaluate the correspondence between autonomic and facial affective behavioral indices of emotion and its regulation in children with ADHD. Functionalist theories of emotion propose that synchrony among emotional response systems is associated with emotional health, while desynchronization of these systems contributes to the development and maintenance of psychopathology (Ekman, 1992a; Mauss, Evers, Wilhelm, & Gross, 2006; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Wilhelm, Grossman, & Roth, 2005).

*Hypothesis 2a*

Congruent with Study 1, this sub-sample of children with ADHD will show elevated parasympathetic and sympathetic nervous system activity across task conditions when compared to typically developing controls.

*Hypothesis 2b*

Children with ADHD will show increased emotionality (particularly in positive or approach emotional domains) across task conditions, as indexed by more frequent displays of facial affective behavior.

### *Hypothesis 2c*

According to functionalist theory, in the full sample, there will be a statistical correspondence among measures of emotion and its regulation, including: autonomic reactivity and facial affective behavior, during induction. However, this correspondence will be weaker during suppression.

### *Hypothesis 2d*

Children with ADHD will show weaker correspondence among measures of emotion regulation including: autonomic reactivity and facial affective behavior, during both negative and positive emotion induction. However, typically developing children will show developmentally- and task-appropriate levels of correspondence between these measures during both negative and positive emotion induction.

### **Specific Aims of Study 3**

The final specific aim of this dissertation was to evaluate parenting behavior in relation to children's autonomic-based arousal and emotion regulation in children with ADHD.

### *Hypothesis 3a*

Parents of children with ADHD will display higher levels of expressed emotion (e.g., criticism and emotional over-involvement) as coded during the parent five minute speech sample, as well as more intrusive and less sensitive behaviors as coded during the parent-child interaction task than parents of typically developing youth.

### *Hypothesis 3b*

Children's disruptions in autonomic reactivity (i.e., augmented reactivity in both the parasympathetic and sympathetic branches) in response to parent-child interactions

will be associated with specific presentations of ADHD (i.e., number of ADHD symptoms and the number of comorbid ODD and CD symptoms).

### *Hypothesis 3c*

The association between parenting behavior (e.g., parental emotion expression and insensitivity) and ADHD symptom presentations (i.e., number of ADHD symptoms, number of ODD and CD symptoms) will be mediated by autonomic nervous system reactivity. Specifically, children with parents displaying the highest levels of expressed emotion and insensitivity will show an augmented, inflexible autonomic regulatory pattern, and this pattern of autonomic dysregulation will be associated with more severe behavior and attention problems, whereas children with parents displaying the lowest levels of expressed emotion and insensitivity will show age-appropriate autonomic flexibility, which will be associated with low levels of behavior and attention problems.

### ***Background and Significance for All Three Studies***

Attention-deficit/hyperactivity disorder (ADHD) has been previously theorized to involve dysfunctions in the physiological and neural systems supporting behavioral inhibition and control (Barkley, 1997; Nigg, 2001), with an emphasis on the inability to regulate behavior (Pennington & Ozonoff, 1996) and cognition (Barkley, 1997).

Concomitantly, and more recently with the recognition that these same control processes are also involved in emotion and its regulation, interest has crystallized in the role of emotion regulation in ADHD (Barkley & Fischer, 2010; Martel, 2009; Wehmeier, Schacht, & Barkley, 2010). Given how crucial this idea is for conceptualizing heterogeneity in ADHD, it warrants further empirical evaluation. Yet, adding specificity to these claims is essential to avoid an excessively broad theory, because emotion

dysregulation in some form is likely related to nearly all forms of psychopathology. For example, if emotion includes both positive (or approach) and negative (or avoidance) systems, it is unclear which system would be most affected in ADHD, though several theories have proposed dysfunction in the positive (approach) system (for a review see Nigg, 2006).

Viewing ADHD through the lens of emotion regulation has been motivated by findings linking temperament with ADHD (Martel & Nigg, 2006) and by the high comorbidity of ADHD with mood disorders (Chronis-Tuscano et al., 2010; Elia, Ambrosini, & Berrettini, 2008) and oppositional behavior, which has been linked with negative emotionality (Martel, 2009). With regard to emotion *per se*, ADHD has been associated with emotional reactivity and with reduced emotional inhibition, recognition, and empathy (Cadesky, Mota, & Schachar, 2000; Maedgen & Carlson, 2000; Walcott & Landau, 2004). However, few studies have examined directly emotion regulation in ADHD (but see Melnick & Hinshaw [2000] for an initial behavioral examination), and almost none have done so using autonomic nervous system functioning as a physiological index of emotion regulation under regulation-demanding conditions, leaving whether *regulation*, in particular, is impaired unclear.

Emotion regulation is conceptualized herein as described by Gross and colleagues (Gross, 1998; Gross & John, 2003). According to this framework, emotions are biologically-based reactions and are brief and malleable, resulting in changes in physiology, subjective experience, and expressive behavior. Emotion regulation is the manipulation of the physiological, subjective, or behavioral components of the emotional response. Suppression is one type of emotion regulation that involves consciously

inhibiting one's expression behaviors during emotional arousal (Gross & Levenson, 1993; Gross, 1998). It has the attractive features of being readily manipulated in the laboratory and of being similar to real world demands on children. Methodologically, a successful approach with adolescents and adults has been to induce emotional arousal via clips taken from emotionally evocative films. Participants are instructed to exhibit (or enhance) the emotion displayed in the film and/or to suppress it. In typically developing adults, suppression has been shown to influence subjective ratings of emotion, expressive behavior, physiological reactivity, and neural responding (Crowell et al., 2006; Goldin, McRae, Ramel, & Gross, 2008; Gross & Levenson., 1993).

Physiological indices of emotion and its regulation have been developed on the basis of a substantive theoretical and empirical literature (Berntson et al., 1997; Porges, 1995, 2007). Parasympathetic-linked cardiac activity is indexed by respiratory sinus arrhythmia (RSA), which has been associated with emotion regulation in response to regulation challenge or contextual demands (Beauchaine, 2001; Berntson, et al., 1997). Sympathetic-linked cardiac reactivity has been indexed by cardiac pre-ejection period, which has been associated with emotional arousal, mental effort, sensitivity to reward, and in some contexts, approach and avoidance behaviors (Beauchaine, 2001; Berntson, et al., 1997; Kelsey, Ornduff, McCann, & Reiff, 2001).

These physiological indices have been used to examine emotion regulation in the context of externalizing behaviors and disorders in children, generally, but to a lesser degree to do so among children with ADHD, specifically. This is important because it is not clear that ADHD and externalizing problems are the same in this regard, despite their frequent behavioral co-occurrence and despite theories suggesting that ADHD may be a

precursor of more severe disruptive behavior disorders. With regard to externalizing behaviors in children, autonomic dysregulation at rest has been associated with aggression (Mezzacappa et al., 1997), inattention (Suess, Porges, & Plude, 1994), low behavioral inhibition (Kagan & Snidman, 1991), decreased empathy (Fabes, 1994; Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994), emotion lability (Beauchaine, 2001), hostility (Sloan et al., 1994), temperamental reactivity (Calkins, 1997), and callous/unemotional behavior (Frick, 1999, 2003). Additionally, boys with externalizing symptoms tend to have lower baseline heart rate and RSA than controls (Mezzacappa, et al., 1997). Although most of these studies did not examine physiological reactivity during emotion induction or emotion-based challenge tasks, results from two studies suggest that child externalizing disorders (Beauchaine, Gatzke-Kopp, & Mead, 2007) and externalizing behaviors in typically developing populations (Calkins, 1997) are associated with reduced parasympathetic responding during negative emotion induction and reduced sympathetic nervous system when responding to rewards. However, neither study used an induction/suppression paradigm. Thus, the exact role of *regulation* during a challenging task, like suppression, remains unclear.

In short, the association between altered autonomic functioning at *baseline* and externalizing behavior is rather well established, but the functioning of autonomic *regulatory* response is less clear in ADHD. Indeed, very few studies have examined emotion *induction* using physiological methods in relation to child externalizing behavior (Boyce et al., 2001; Eisenberg et al., 1997; Herpertz et al., 2005; Marsh, Beauchaine, & Williams, 2008; Mead et al., 2004). However, none of these studies did so with ADHD and none used emotion *suppression* paradigms to challenge regulatory capacities.

Induction, while relevant, does not alone allow for a full examination of the *regulation* of emotion.

Taken together, whether emotion regulation is abnormal among children with ADHD remains unclear. Additionally, whether the conclusions drawn about externalizing behaviors apply to ADHD, when accounting for comorbid externalizing behavior, also remains unclear. Beauchaine and colleagues (Beauchaine, Katkin, Strassberg, & Snarr, 2001) found that compared with controls, both adolescents with ADHD and with conduct disorder exhibited reduced skin conductance (sympathetic nervous system responding) during a reward task, but nearly all remaining autonomic nervous system effects were confined to the conduct disorder group. In pre-school aged children, ADHD and oppositional disorder were characterized by reduced electrodermal responding and lengthened PEP (decreased sympathetic functioning) during a reward task, whereas heart rate changes in these groups were mediated exclusively by parasympathetic withdrawal, unlike controls who displayed heart rate changes associated with parasympathetic and sympathetic activation (Crowell et al., 2005).

In addition to examining the roles of emotion and its regulation in ADHD via induction and suppression, the functionalist theory of emotion has suggested that the *coherence* among the different aspects of the emotional response systems (facial affective behavior, subjective experience, and physiological responding) plays a critical role in emotional health, while desynchronization contributes to the development of psychopathology (Ekman, 1992a; Mauss, et al., 2006). Functionalist theory proposes that emotions have evolved to maintain the safety of the organism and to organize and provide an explanation of our actions. As such, reduced coherence among the systems

may result in the experience of conflicting emotional signals and maladaptive responding to emotional cues (Ekman, 1992a; Marsh et al., 2008; Mauss, et al., 2006). Previous work has shown that a reduced correspondence among facial affect and autonomic reactivity is characteristic of boys with disruptive behavior disorders (Marsh, et al., 2008); however, this type of correspondence of emotional regulation systems has not been examined in children with ADHD. This study sought to do so.

Finally, emotion regulation develops in context. This is relevant because only some children with ADHD go on to develop conduct disorder and related externalizing behavioral outcomes. Previous work has established that parenting behaviors (in particular, criticalness, intrusiveness, loose limit-setting, reduced warmth, and excessive expectations for independence) predict specific developmental outcomes among children with ADHD, including the development of antisocial behavior and later school-based problems/failure (Hinshaw, Zupan, Simmel, Nigg, & Melnick, 1997; Melnick & Hinshaw, 2000). It has also been proposed that parents shape children's acquisition of regulatory capabilities through coaching, modeling, reinforcement, and scaffolding (Melnick & Hinshaw, 2000). Furthermore, parental expressed emotion, assessed during the five minute speech sample, has been used as an index of the emotional climate of the home environment (Peris & Baker, 2000; Peris & Hinshaw, 2003). Expressed emotion is a two-dimensional construct composed of criticism and emotional over-involvement domains. In particular, the criticism domain is designed to assess feelings of negativity or resentment directed toward the child, while the emotional over-involvement category indexes behaviors which are overprotective or overly self-sacrificing (Peris & Baker, 2000; Peris & Hinshaw, 2003). In the context of child psychopathology, high expressed

emotion has been shown to be a risk factor for both internalizing (Vostanis & Nicholls, 1992) and externalizing disorders (Faraone & Rosenbaum, 1997; Peris & Baker, 2000; Peris & Hinshaw, 2003). Furthermore, parental expressed emotion in the criticism domain is predictive of child externalizing behavior two years later (Peris & Baker, 2000), and high levels of criticism have been shown to be uniquely predictive of ADHD symptoms among adolescent girls even after accounting for other externalizing and internalizing symptoms (Peris & Hinshaw, 2003). Thus, parental insensitivity and expressed emotion have been shown to be associated with the development, presentation, and severity of externalizing pathology, and ADHD, specifically. Therefore, it is important to determine the role of parenting and the emotional climate of the home in the development of both behavioral and physiological emotion regulation in ADHD, as these factors may be mediated by autonomic activity in predicting ADHD symptoms and other externalizing symptoms. This study was one of the first studies to examine both the behavioral and physiological regulation of emotion in the context of parenting behavior and expressed emotion in children with ADHD.

The novel contributions of these studies are meaningful because, these studies serve to clarify the role of the individual branches of the autonomic nervous system in ADHD, the role of parenting behavior and expressed emotion in the development of the physiological regulation of emotion in ADHD, the role of both autonomic nervous system flexibility and emotion regulatory system coherence in the development of ADHD, as well as the independent and interactive contributions of multiple systems of emotion regulation and parenting behavior to the presentation of ADHD, including co-occurring behavior problems and severity of symptoms. In turn, these data enable

evaluation and revision of existing theory about the place of emotion and its regulation in ADHD. As such, the examination of these behaviors and biological indexes is important, because answers to these questions, using these methods, provide empirical evidence to support or refute theories relating emotion and emotion dysregulation to ADHD in the literature. If these theories are supported, then a reformulation of the mechanisms of ADHD may be appropriate, which may directly influence prevention and intervention, as well as future research efforts. Specifically, if these theories are supported, then dysregulation as a core of ADHD becomes enhanced, as it extends further to emotional dysregulation (in addition to behavioral and cognitive dysregulation), even when emotional symptoms like ODD are controlled. However, it may also be that there is a subgroup (or subgroups) with emotional dysregulation, which would further serve to elucidate mechanisms of underlying heterogeneity in ADHD. Finally, if these theories are supported, it allows for translation back to basic developmental theory by revealing that cognitive and emotional dysregulation are both affected in ADHD, which suggests a more unitary understanding of self-regulation.

### ***Theoretical Basis and Conceptual Framework for Proposed Studies***

Addressing the preceding issues requires an organizing conceptual framework by which sympathetic and parasympathetic systems can be considered, as well as the component behavioral domains of ADHD (i.e., inattention and hyperactivity/impulsivity) and associated problems (e.g., disruptive behavior, callous/unemotional behavior). To that end, a theoretical model to guide the planned studies was proposed (**Figure 1**; all figures are located in Appendix B, beginning on page 150). The model examines the roles of autonomic nervous system functioning, temperament (including emotionality and

regulatory domains), and parenting behavior in the development of externalizing behaviors and psychopathology in children. The concept builds on multiple sources as cited in what follows.

Beginning at the left of Figure 1, the autonomic nervous system is the portion of the nervous system which is responsible for unconscious activities. This branch of the nervous system is further subdivided into the sympathetic and parasympathetic branches. The sympathetic system is associated with arousal and emotion, while the parasympathetic system is associated with growth, restorative functions, and regulatory phenomena. Sympathetic-linked cardiac reactivity has been indexed by both cardiac pre-ejection period (PEP) and galvanic skin conductance (GSK), both of which have been associated with emotional arousal, mental effort, sensitivity to reward, and in some contexts, approach and avoidance behaviors (for basis of these ideas see Beauchaine, 2001; Berntson, et al., 1997; Kelsey, Ornduff, & Alpert, 2007). PEP is a commonly used index of sympathetic cardiac control of the heart through the beta-adrenal system (Berntson et al., 2004). PEP represents the time between the depolarization of the left ventricle of the heart and the onset of ejection of blood into the aorta. Shorter PEP values have been associated with increased sympathetic activation which is believed to be influenced by the beta-adrenal system, and increases in adrenergic activation are associated with shortened PEP and increased sympathetic activation (Cacioppo et al., 1994).

In contrast, one of the primary roles of the PNS at rest is to maintain homeostasis and allow for restorative and regulatory functions. Parasympathetic-linked cardiac activity is indexed by respiratory sinus arrhythmia (RSA), which has been associated

with emotion regulation (Beauchaine, 2001; Berntson, et al., 1997). RSA is the naturally occurring change in heart rate which corresponds to breathing. Specifically, heart rate is known to increase during inhalation and to decrease during exhalation.

Thus, as shown in Figure 1, SNS activity is seen as directly influencing the emotionality domain of temperament, while PNS activity is seen as indexing the regulatory domain of temperament (Beauchaine, 2001; Berntson et al., 1997). In particular, the model proposed by Rothbart and colleagues (e.g. Rothbart et al., 2001; Rothbart & Sheese, 2007) defines temperament as “constitutionally-based individual differences in reactivity and self-regulation in the domains of affect, activity, and attention”. Thus, temperament is proposed by Rothbart and colleagues to be constitutionally-based; however as she and others have acknowledged, the term “constitutionally-based” needs more specification (for a review see Nigg, 2006). Others have proposed that temperament should be conceptualized as specific behaviors, for which there should be physiological indicators, which are strongly influenced by heredity, maturation, and environmental experience (Kagan 1991, 1997). Therefore, in order to more fully operationalize the behavioral domains of temperament outlined by Rothbart, laboratory based-behavioral measures and parent-/self-report measures have been developed (Rothbart et al., 2001). Additionally, sympathetic and parasympathetic nervous system activity has been associated with measures of temperament behavior and may serve as a physiological index of temperament (Calkins et al., 2001, 2004; Eisenberg et al., 1997, 2001, 2005).

According to Rothbart’s model, reactivity can be defined as an infant’s or child’s latency to respond, threshold for responding, and the intensity of the response to a

particular sensory stimulus, while regulation is rooted in the attention and inhibitory skills which control these reactive processes (Rothbart et al., 2001; Rothbart & Sheese, 2007). The regulation domain allows individuals to modulate reactivity through either conscious or unconscious processes. The two major factors in this model map nicely on to an emotional arousal factor (reactivity) and an emotion regulatory factor, and these two factors each may differ among individuals. Thus, according to Rothbart and colleagues (2001, 2007), emotion regulation and temperament are not distinct entities, but rather mutually influential behaviors, both with roots in physiological processes of the central and autonomic nervous systems.

Moving to the right portion of Figure 1, the behavioral emotionality domain of temperament can be further divided into negative, positive, and blunted emotionality sub-domains (Eisenberg et al., 2004; Rothbart et al., 1998). Additionally, the regulatory domain of temperament can be further subdivided into behaviors of self-regulation and effortful control (Eisenberg et al., 2004; Rothbart et al., 1998). Both negative and positive emotionality have been indexed by a heightened or increased change in SNS activity, as measured by both PEP and skin conductance, from baseline and that a predisposition toward heightened emotionality in these domains is indexed by elevated SNS activity at baseline (Calkins et al., 1997, 1998; Gross & Levenson, 1993, 1997). Additionally, blunted or flattened affect and very low levels of both positive and negative emotionality are indexed by reduced SNS activity both during baselines and during stressors/emotionally provocative events (Beauchaine et al., 2001; Frick et al., 1999; Raine et al., 2001).

With respect to the regulatory domain of temperament, effortful control includes activational control (i.e., the ability to activate new behaviors), attentional control (i.e., the ability to attend to stimuli), and inhibitory control (i.e., the ability to inhibit behaviors). Effortful control allows a child to voluntarily orient away from threatening stimuli and simultaneously increase attention toward calming inputs (Derryberry & Rothbart, 1997). In contrast, self-regulation has been defined as complex systems of reactive control, which may include more rigid self-regulation associated with greater avoidance or less rigid self-regulation associated with increased approach (Derryberry & Rothbart, 1997). Furthermore, disruptions in both effortful control and self-regulation have been indexed by abnormal PNS activity, as assessed by RSA levels, both at baseline and in response to tasks which require high levels of regulation. In particular, Calkins and colleagues (1997, 1998, 2001, 2004) and Beauchaine and colleagues (2001, 2002, 2004, 2007) as well as others have shown that both low baseline PNS activity and reduced PNS reactivity in response to stressful tasks index both behavioral observations of self-regulation and effortful control (Eisenberg et al., 2001, 2004).

Both (1) behavioral observations and parental reports of behavioral aspects of temperament and (2) physiological indicators of behavioral presentations of emotionality and emotion regulation have been directly linked to specific externalizing behaviors and disorders. However, externalizing behavior domains are neither unitary, nor is the factor structure of these behaviors fully agreed upon. Specifically, reviews have proposed dimensions based on types of aggression, age of onset, the presence or absence of callous/unemotional behavior, as well as other ways of categorizing these behaviors (Frick, 1999; Lahey & Loeber, 1994; Lahey et al., 1999; Loeber et al., 2000). For

example, support has emerged for two distinct profiles of early-onset and late-onset subtypes of conduct problems, with early-onset being associated with more severe behavior problems and an increased risk of antisocial behavior in adulthood (Achenbach, 1966, Achenbach et al., 1989; Frick et al., 1999; Hinshaw et al., 2010; Lahey et al., 1995; Moffit et al., 2000). The early-onset subtype is associated with oppositional behavior, defiance, reactive aggression, and impulsivity, while the late-onset subtype is more associated with property crimes, status offenses, and the use of illegal substances (Frick et al., 1999; Lahey et al., 1995; Hinshaw et al., 2010). Others have conceptualized the distinction between early- and late-onset externalizing behavior as being perhaps better thought of as distinguished by aggression and rule-breaking, respectively, rather than age of onset, with an additional, separate dimension of callous/unemotional behavior (Burt et al., 2008, 2012). One method that attempts to integrate these different approaches to categorizing externalizing behavior is to examine individual behavioral domains of callous/unemotional behavior, proactive aggression, reactive aggression, oppositional behaviors, hyperactivity/impulsivity, inattention, property crimes, and status offenses (Frick, 1999; Lahey et al., 1995, 2005). A simplified version of this association is proposed in last two sections of Figure 1.

Beginning with callous/unemotional behavior and proactive/instrumental aggression, both have been linked to underactive SNS activity at baseline, during emotionally evocative tasks, and during reward and punishment (Fabes et al., 1994; Frick et al., 1999; Kagan & Snidman, 1991; Raine et al., 2002). Additionally, these symptoms appear to have a very strong genetic component, to run a lifelong course, and to be relatively uninfluenced by environmental factors such as parenting (Beauchaine et al.,

2010), and it has been suggested that children exhibiting these types of behaviors may constitute a very specific subgroup of children with early-onset conduct disorder who go on to develop more serious antisocial behaviors in adulthood (Frick et al., 1999; Lahey et al., 1995). While the callous/unemotional trait has mostly been studied among children with conduct disorder, this domain may also have utility even without the presence of co-occurring conduct problems; that is, children with the callous/unemotional trait are more likely to have later behavior problems even without a diagnosis of conduct disorder (Moran et al., 2008). Finally, interventions have proven to be relatively ineffective in treating or preventing these kinds of symptoms and behaviors (Gatzke & Raine, 2001). Thus, it may be that this specific trait or behavioral profile helps to explain some of the heterogeneity observed in externalizing disorders and ADHD, specifically.

Moving down Figure 1, oppositional/defiant behaviors, as well as reactive/impulsive aggression, have been linked to negative emotionality and increased SNS activity in response to negative emotional stimuli and at baseline (Burgess et al., 2003; Mezzacappa et al., 1997; Martel, 2009, 2011; Nigg, 2006). In contrast, hyperactivity and impulsivity have been linked with excess positive emotionality, approach, and increased SNS activity at baseline and in the face of emotional stimuli (particularly positive emotional stimuli; Musser et al., 2010; Nigg, 2006). Similarly, hyperactivity and impulsivity (as well as reactive aggression) have also been linked to deficits in self-regulation (or unconscious regulation) and reduced PNS flexibility during emotionally provocative tasks (Martel, 2009; Musser et al., 2010). Uniquely, inattention appears to be associated almost exclusively with disruptions in effortful control, which has been indexed by dysregulated parasympathetic activity during attentionally

demanding tasks (Suess, Porge, & Plude, 1994). This is in line with theoretical suggestions and empirical evidence that the DSM-IV inattentive dimension is partially distinct etiologically from the DSM-IV hyperactive/impulsive dimension of ADHD (Hinshaw et al., 1997; Nigg, 2006; Nikolas & Burt, 2010; Sonuga-Barke et al., 2002).

This study utilizes this theoretical frame work to inform the proposed hypotheses. According to this model, it was expected that children with ADHD would display heterogeneous profiles of autonomic reactivity and regulation based on co-occurring behavioral and trait profiles. Specifically, it was expected that children with ADHD combined type would display elevated SNS activity across all task conditions, as well as augmented from baseline PNS activity across task conditions. In addition, children with ADHD and callous/unemotional traits would display blunted responding in both autonomic branches across task conditions, and typically developing children would respond with autonomic flexibility based on the demands of the task conditions.

Additionally, the proposed studies expand upon this conceptual framework by examining correspondence across emotional systems, as well as the roles of parenting behavior and emotion, in the association between emotion regulation and child presentation of ADHD symptoms and externalizing behavior. Specifically, it was expected that the association between parenting factors (i.e., expressed emotion and sensitivity) and ADHD symptom presentation would be mediated by autonomic nervous system activity for children with ADHD (see Figure 2). That it, it was expected that parenting behavior and expressed emotion would directly influence child autonomic functioning at both branches, which would directly affect child behavioral outcomes, including number of ADHD and co-occurring behavior problem symptoms.

## ***Innovation***

While numerous studies have used film clips to examine the behavioral, experiential, and physiological correlates of emotion induction and suppression among adolescent and adult populations (Crowell, et al., 2006; Goldin, et al., 2008; Gross & Levenson, 1997; Gross & Levenson., 1993; Hutcherson et al., 2005), few studies have examined emotion *induction* among child populations (for exceptions see: (Fabes, et al., 1994; Marsh, et al., 2008), and *no* prior studies have examined the correlates of emotion *suppression* with children (with the exception of Musser et al., 2010). Thus, the examination of emotion suppression in children is novel and could open new lines of investigation for the development of emotion regulation generally, as well as for studying emotion regulation in child psychopathology beyond ADHD.

Additionally, prior studies using induction have emphasized negative emotions. This is the first study to examine the induction and suppression of *positive* emotions in children in this age range (again, with the exception of Musser et al., 2010). Thus, the use of an induction/suppression paradigm to study positive emotion regulation in ADHD is novel. This is particularly important in children with ADHD, as several theories have hypothesized a specific dysfunction in the approach (positive) system among these children (Beauchaine, 2001, 2002, 2003; Nigg, 2006).

Only one prior study has examined correspondence among emotional systems in children with disruptive behavior disorders, and none have done so with ADHD. This is the first study to examine correspondence of these systems in ADHD, as well as during positive emotion induction. This is particularly important as several theories have hypothesized that correspondence among emotional systems may be particularly

important to emotional and psychological health (Beauchaine, 2001, 2002, 2003; Frijda, 2002).

Finally, only one previous study has examined the role of parenting sensitivity as it pertains to emotion regulation among children with ADHD (Melnick & Hinshaw, 2000), and no studies have done so using physiological indexes of emotion regulation. Prior work has addressed the independent roles of parenting and emotion regulation in the development of externalizing behavior, but not in ADHD, specifically (El-Sheikh et al., 2009). Furthermore, while two prior studies have examined the effects of parental expressed emotion on ADHD severity and the presence of comorbid psychopathology (Peris & Baker, 2000; Peris & Hinshaw, 2005), neither of these studies examined the role of parental expressed emotion on child emotionality or regulation. Thus, the present convergence of approaches with children is novel, as is their application in understanding ADHD, and if successful this set of approaches may serve to inform new theories, empirical work, assessment, and prevention and intervention approaches for children with ADHD and other externalizing problems.

## CHAPTER II

### STUDY 1: PHYSIOLOGICAL EMOTION REGULATION IN ADHD

#### *Methods*

#### **Summary**

The purpose of Study 1 was to evaluate physiological indices of emotion and emotion regulation in children with ADHD during conditions of negative and positive emotion induction and suppression. To address the main hypotheses of Study 1 that children with ADHD would display a pattern of dysregulated physiological emotional arousal and regulation with both parasympathetic and sympathetic indexes elevated from baseline, a novel emotion induction and suppression task with four conditions: (a) negative induction, (b) negative suppression, (c) positive induction, and (d) positive suppression, was used. Continuous electrocardiogram and impedance cardiography recordings were collected across task conditions. Behavioral codes of facial affective behavior and self-reported experience of emotion served as a validity check for task manipulations. A table providing a list of assessment measures for screening and diagnosis, Study 1, Study 2, and Study 3 is provided (Table 1, page 123; all tables are located in Appendix A beginning on page 123).

#### **Participants**

All participants either meet DSM-IV criteria for ADHD combined type (ADHD) or were typically developing control youth, aged 7 to 11 years of age. The presence of both inattentive and hyperactive/impulsive symptoms provided by the combined-type diagnosis allowed for a secondary exploration of the nature of the heterogeneity of this disorder, including investigation of factors associated uniquely with inattention,

hyperactivity/impulsivity, and other comorbid externalizing behaviors and traits.

Study 1 included 150 participants; half of whom met DSM-IV criteria for ADHD (combined type, N=75), and the remainder of whom were non-ADHD (typically developing) comparison youth (N=75).

## **Procedures**

The following procedures were utilized in Study 1.

### *Recruitment and Identification Procedures*

Families volunteering for the study were passed through a multi-gate screening process to establish eligibility and diagnostic group assignment. At stage one, parents of potential participants completed a phone interview to rule out the use of ineligible medications (including all long acting psychoactive medications that could not be washed out for the study), neurological impairments, seizures, traumatic brain injury, major medical conditions, mental retardation, autism spectrum disorders, or pervasive developmental disorders, as reported by the participating parent.

At stage two, a parent and a teacher of remaining eligible youth completed the ADHD Rating Scale (DuPaul, Power, Anastopoulos, & Reid, 1998), Conner's Rating Scale-3<sup>rd</sup> Edition (Conners, 2008), and the Strengths & Difficulties Questionnaire (Goodman, 2001). A parent also completed the Kiddie Schedule for Affective Disorders and Schizophrenia version E (KSADS-E; (Puig-Antich & Ryan, 1996), with a master-degree level clinician. Interviews were monitored for inter-interviewer reliability; and all interviewers achieved adequate inter-interviewer reliability ( $k > 0.85$  for ADHD). Children completed an IQ screening, consisting of the Block Design, Vocabulary, and Information sub-tests from the Wechsler Intelligence Scales for Children, Fourth Edition (WISC-IV;

(Wechsler, 2003). Children also completed the Word Reading, Math Reasoning, and PseudoWord subscales from the Wechsler Individual Achievement Test, Second Edition (WIAT-II;(Wechsler, 2005).

#### *Final ADHD and Other Diagnoses*

Results from the KSADS-E, all of the parent and teacher rating scales, and child IQ and achievement tests were presented to a diagnostic team (a board-certified child psychiatrist and licensed neuropsychologist) each of whom arrived independently at a “best estimate” diagnosis for ADHD, ADHD DSM-IV subtype, and all other disorders assessed by the KSADS-E, using DSM-IV criteria. Agreement rates were satisfactory ( $k > .70$  for all disorders with a  $> 5\%$  base rate in the sample) based on a sample of 547 children screened in 2009-2011. Disagreements were resolved by conference. If consensus was not easily achieved, the case was excluded from participation.

A diagnosis of ADHD required that the child’s symptoms had a cross-situational presentation, evidence of impairment, and were not accounted for by another disorder. Symptoms were counted as present if endorsed by the parent on the KSADS-E or the teacher on the ADHD Rating Scale, provided that the child had elevated teacher and parent ratings of at least  $T > 65$  on at least one major sub-scale of ADHD. If both parent and teacher provided standardized ratings of at least the 90<sup>th</sup> percentile on a rating of inattention or hyperactivity using the above rating scales, then symptoms were counted as present if endorsed by the parent on the KSADS-E or the teacher on the ADHD Rating Scale. The teacher was allowed to contribute a maximum of two additional symptoms (that is, at least four symptoms had to be identified on the KSADS-E). This procedure was similar to that used in the DSM-IV field trials and the MTA study (MTA

Cooperative Group, 1999). The “or” algorithm was used in 15% of cases.

*Identification of Callous/Unemotional Traits.* Both a parent and a teacher of eligible youth completed the 25-item Strengths and Difficulties Questionnaire (Goodman, 2001); the Prosocial Behavior scale served as an indicator of callous/unemotional behavior (when reverse coded). Previous factor analytic work suggests that the items from the Prosocial Behavior subscale load on a callous/unemotional factor when reverse coded (Dadds, Fraser, Frost, & Hawes, 2005). Additionally, definitions of callous/unemotional behavior have utilized these items from the Strengths and Difficulties Questionnaire (Viding et al., 2005, 2008). The Prosocial Behavior subscale of the Strengths and Difficulties Questionnaire was used to assign children to the callous/unemotional group as follows.

In order to be assigned the CU specifier, both the parent and teacher had to rate the child with an overall clinical impairment score of zero (i.e., very low prosocial behavior) on the Prosocial Behavior subscale. That is, *both* the parent and teacher had to endorse a deficit in prosocial behaviors in the 90<sup>th</sup> percentile for a given child in order for that child to receive the CU specifier. This method was intended to be conservative in that only the most severe cases of CU traits were captured. Among the children with ADHD, 21 (28% of those with ADHD) were assigned the CU specifier; none of the control participants were assigned the CU specifier. Additional clinical features of the groups are provided in the results section and in Table 2.

In order to provide further validity for assignment to groups based on the presence or absence of the CU specifier, a subsample of parents of participants (n = 90; 52 ADHD, 38 control) completed the Inventory of Callous/Unemotional Traits (ICU; Kimonis et al.,

2008). Total scores on the ICU were correlated negatively with the parent Strengths and Difficulties Questionnaire Prosocial Behavior domain at reliability corrected  $r = 0.853$  ( $p < 0.001$ ), which is consistent with previous work (Fan, 2003, Kimonis et al., 2008).

*Exclusion Criteria.* Exclusion criteria include an estimated Full Scale IQ  $< 75$ , current major depressive episode, lifetime mania or psychosis, pervasive developmental disorder, learning disability, or any of the medical conditions described above. Other disorders were free to vary. Additionally, as described above children were excluded if taking certain psychoactive medications, which could not be washed out for the study, or if a consensus-diagnosis was not achieved by the diagnostic team.

#### *Medication Washout*

Prior to completing the tasks, children taking stimulant medication completed a 24-48 hour medication washout, dependent on the type of preparation. This was equivalent to a minimum of five half-lives (24-48 hours, depending on the type of stimulant preparation they were prescribed). Other long-acting psychotropic medications were considered exclusion criteria for the study, as described above. As an extra precaution, stimulant prescription status (present or absent) was covaried to remove the effect of the medication washout in all analyses.

#### *Emotion Induction and Suppression Procedure*

Each child was video-taped as they completed the emotion induction and suppression procedure using both a negative and a positive emotion-laden film clip. This involved watching four, two-minute film clips selected from *Homeward Bound*, a film about two dogs and a cat who are separated and reunited with their human family.

Pilot data indicate that the first two clips elicit negative emotions, while the last

two segments elicit positive emotions. To continue to evaluate the validity of these conditions, children completed the Self-Assessment Manikin (SAM) valence and arousal scales (Bradley & Lang, 1994) for each clip. The SAM is a pictorial assessment, which measures self-reported valence and arousal on 5-point scales.

In the induction condition, children facially mimicked the emotion of the main character. This instruction was given for the first negative and first positive segment. In the suppression condition, the child was instructed to imagine what the main character was feeling, but to keep his or her face still, masking (suppressing) the emotion. This instruction was given for the second negative and second positive segment. These conditions were not counterbalanced as: (a) it was important to end with positive emotion for human subjects' welfare, (b) the film is a continuous story, and changing the order would have likely confounded suppression with cognitive challenge to interpret the story, (c) putting induction prior to suppression maximized the suppression challenge. Thus, the same sequence of the four task conditions was presented to each child: (a) negative induction, (b) negative suppression, (c) positive induction, and (d) positive suppression. Data from a prior study also suggested that there were few carry-over effects (Musser et al., 2010).

A resting baseline period of two minutes was presented before the task, and neutral baseline periods of two minutes were presented before the negative task condition and before the positive condition. Each neutral baseline required the child to observe a set of ten neutral pictures from the International Affective Picture System (IAPS; (P. J. Lang, Bradley, & Cuthbert, 1997). This type of baseline accounts for the physiological response of orienting and attending (Jennings, van der Molen, & Somsen, 1998). The SAM was

used to assess self-reported valence and arousal during these baselines. Thus, the overall presentation order for each participant was: (a) resting baseline, (b) neutral pictures baseline 1, (c) negative induction, (d) negative suppression, (e) neutral pictures baseline 2, (f) positive induction, and (g) positive suppression.

### *Facial Action Coding System*

Using the videotapes, after data collection was complete, the child's facial affect was coded using a modified version of the Facial Action Coding System (FACS) to assess the child's affective facial behavior during each segment of the task (Ekman, 1992b) by two blinded research assistants trained by the author for reliability. The blinded research assistants coded a sub-sample of 100 tapes, and 40% were coded for reliability. Reliabilities for each of the six facial affective behavior frequency domains coded (i.e., surprise, happiness, anger, fear, anxiety, and sadness) were acceptable with all ICCs  $>0.85$ . This full Facial Action Coding System has been well validated with standard reliabilities of  $\alpha = .89$  (Gross & Levenson, 1993).

### *Physiological Recording*

*Overview.* Disposable silver/silver-chloride electrodes were placed in a standard lead II electrocardiogram (ECG) and impedance cardiography (ICG) configuration. The ECG electrodes were placed at the right collar bone and the tenth-left rib with a ground electrode placed at the tenth-right rib. For ICG, two voltage electrodes were placed below the suprasternal notch and xiphoid process, and two current electrodes were placed on the back 3 to 4 cm above and below the placement of the voltage electrodes. ECG and ICG were recorded continuously throughout each of the baselines and task epochs. The R-R series was sampled at 1000 Hz. Heart rate (HR), inner-beat-interval (IBI), and respiration

rate (RR) were derived using the ECG and ICG data after data collection. Artifacts were examined and removed using the software and visual inspection completed by two raters; pilot data suggests that satisfactory inter-rater agreement was readily achieved ( $k > 0.82$  for each epoch).

*Cardiac Pre-ejection Period (PEP)*. PEP, derived from ECG and ICG, represents the time between the depolarization of the left ventricle of the heart and the onset of ejection of blood into the aorta. PEP has satisfactory long-term temporal consistency (Burlison et al., 2003; Cacioppo, 1994). PEP was derived using 30 second epochs, using MindWare Impedance Cardiography V. 2.6 (MindWare, 2008a), allowing for simultaneous editing of the data obtained from ECG and ICG. PEP was indexed as the time interval in milliseconds from the onset of the Q-wave to the B point of the dZ/dt wave, using the method outlined by Berntson and colleagues (Berntson, Lozano, Chen, & Cacioppo, 2004).

*Respiratory Sinus Arrhythmia (RSA)*. RSA was indexed by extracting the high frequency component ( $> 0.15$  Hz) of the R-R peak time series. RSA has good long-term temporal consistency (Berntson, Cacioppo, & Quigley, 1993), and predicts vagal control during pharmacological blockade (Hayano et al., 1991). R-R waves were examined for artifacts and outliers using MindWare® Heart Rate Variability software V. 2.6 (MindWare, 2008b). Artifacts were removed using the software and visual inspection completed by two raters for reliability.

RSA was derived using spectral analysis (Berntson, et al., 1997), in 30 second epochs. Spectral analysis was performed on the R-R time series from the ECG (Berntson, et al., 1997). The time series was detrended and submitted to a Fourier transformation.

The high frequency band ( $\ln(\text{ms}^2)$ ) was set over the respiratory frequency band of 0.24 to 1.040 Hz. Respiratory rates and amplitudes were derived from the impedance cardiograph signal (Z0) ensuring that these signals remained within the analytical bandwidth.

### ***Data Analysis***

The following data analysis methods were utilized.

### **Analytic Plan**

All variables were examined for adherence to the distributional requirements of the planned analyses. Resting baseline, neutral picture baseline, and film segment differences in SAM scores were examined with repeated measures ANOVA to determine whether there were differences in self-reported emotion during the different conditions for both the ADHD and typically developing groups, as a quality check to determine whether task manipulations were valid. Additionally, to determine whether task manipulations were valid, results of the FACS behavioral coding were examined with repeated measures ANOVA to determine whether the anticipated differences in facial affect were congruent with the task conditions for both the ADHD and typically developing groups. To evaluate presentation order and task acclimation effects, all baselines were examined for differences in both RSA and PEP using repeated measures ANOVA to test for specific time-based trends in the autonomic measures, which may be suggestive of acclimation to the task. These analyses examined whether the task manipulations worked as intended.

Finally, in order to test the main hypotheses of Study 1, repeated measures ANOVA were used to examine group differences (first for ADHD [both groups

collapsed] and control, then for ADHD, ADHD with callous/unemotional behavior, and control) in RSA and PEP reactivity scores across the emotion induction and suppression conditions.

### **Power Calculations**

To determine the sample size needed for a repeated measures ANOVA, G\*Power (Faul, Erdfelder, Lang, & Buchner, 2007) was used to determine sample size for the current study. Based on the pilot study (Musser et al., 2010), the minimal Cohen's  $d$  representing the difference in RSA between the groups were expected to be .8, which is considered to be a large effect size (Cohen, 1992). Thus, a total sample size of 66 participants was required for adequate (.90) power to detect a significant group difference ( $p < .05$ ) in RSA between ADHD and typically developing youth. Based on the pilot data, the minimal Cohen's  $d$  representing the difference in PEP between the groups were expected to be .7, which is a moderate effect size (Cohen, 1992). Thus, a total sample size of 98 participants was required for adequate (.90) power to detect a significant group difference ( $p < .05$ ) in PEP. However, it was anticipated that the power needed to examine simple task x group effects would be somewhat greater; thus, a sample size of 150 participants was utilized.

### ***Results***

#### **Preliminary Analyses**

##### *Descriptive and Diagnostic Overview of Sample*

Descriptive statistics and their comparisons are reported according to group in **Table 2**. Groups did not differ with respect to age, race, family income, parent marital status, or IQ. Inclusion of these variables as covariates did not affect results reported.

Results are therefore reported without these variables treated as covariates in order to conserve power. However, groups differed significantly in gender ratio (more boys in the ADHD+CU group than the other two groups). Although gender was unrelated to physiological parameters, and therefore was unlikely to affect results, for clarity gender was covaried in all results reported below. Additionally, as expected, the ADHD groups did not differ from one another with respect to the use of stimulant medications (29.5% in the ADHD only group; 26.3% in the ADHD+CU group); unsurprisingly, both groups displayed significantly more stimulant medication use than the control group (with 0.0% of the control group prescribed stimulant medication). Thus, stimulant medication use was also treated as a covariate in all subsequent analyses, and therefore, was unlikely to account for results.

Clinical characteristics are also provided in **Table 2**. As expected according to group assignments, both the ADHD groups differed significantly from the control group on the parent and teacher rated problems on all clinical scales on the Strengths and Difficulties Questionnaire (i.e., conduct problems, emotion problems, hyperactivity, peer problems, total difficulties, or impact scales), with the exception of the emotion problems scale (see Table 2). None of the groups differed significantly in scores on the emotion problems scale (see Table 2). As intended, the ADHD and control groups displayed significantly more prosocial behaviors than the ADHD+CU group (see Table 2). This pattern of differences suggests that the CU (reverse coded prosocial) indicator is conveying unique information, distinct from conduct or other problems for ADHD subgroups. Furthermore, both ADHD groups differed from the control group on each of the parent- and teacher-rated sub-scales of the ADHD-Rating Scale and Conner's 3<sup>rd</sup>

Edition (i.e., ADHD-RS inattentive, hyperactive, and total symptoms; and Conner's inattention, hyperactive, learning problems, peer problems, and aggression); however, the ADHD and ADHD+CU groups did not differ on any of the scales (Table 2). Again, this supports the group assignments, suggesting that the two ADHD groups differed only on the callous/unemotional dimension.

With regard to clinical comorbidity, as shown in **Table 2**, groups did not differ in lifetime or current mood, sleep, conduct, or tic disorders. The low rate of comorbidity in this sample reflects both the young age of most of the children and the caution with which children whose ADHD might have been better explained by a comorbid disorder were excluded (per DSM-IV; APA, 2000). Although, as is typical in ADHD samples, the two ADHD groups had a significantly greater prevalence of oppositional defiant disorder than the control group, though the two ADHD groups did not differ on prevalence of ODD (23.8% of the ADHD+CU group, 24.7% of the ADHD group). Unsurprisingly, the ADHD+CU group had significantly less prevalence of anxiety disorders than either the control or ADHD only group (4.7% of the ADHD+CU group, 23.9% of the ADHD group, and 21.3% of the control group; see Table 2). The inclusion of comorbid disorders including anxiety, conduct, and oppositional defiant disorders as covariates (individually or collectively) did not affect any of the main study results. Covarying of total ODD symptoms, total CD symptoms, or total anxiety symptoms (all from the KSADS-E) also did not alter findings or conclusions. For clarity, results are shown with KSADS-E ODD and anxiety total symptoms covaried. CD symptoms were not ultimately treated as a covariate, due to their low prevalence across groups (see **Table 2**).

## Evaluation of Possible Confounds and Task Validity

### *Effectiveness of Emotion Induction by Self-Report*

Before examining group differences, the validity of our task manipulation were considered by examining self-report of the mood induction. Groups did not differ in their SAM ratings of the neutral pictures, all  $F < 1.0$ , all  $p > 0.10$  (see **Table 3**). Thus, the intended neutral conditions were reported to be neutral for all groups, and any differences in autonomic reactivity during the tasks could not be attributed to differences in tendency to over or under self-report mood related to the task. To assess the SAM ratings for the different experimental conditions, a 2x2x3 repeated-measures ANOVA (valence [negative/positive] x regulation [induction/suppression] x group [control/ADHD/ADHD+CU]) was conducted. Partial eta<sup>2</sup> ( $\eta^2$ ) was included as a measure of effect size (Cohen, 1992). The analysis supported the validity of the positive/negative emotion induction and suppression manipulations in **Table 3**.

Beginning with valence ratings, scores for the full sample differed according to both the valence condition  $F(1,149) = 434.81$ ,  $p < 0.001$ ,  $\eta^2 = 0.75$ , and the regulation condition,  $F(1,149) = 8.01$ ,  $p < 0.005$ ,  $\eta^2 = 0.06$ . The interaction of valence and regulation conditions was significant, consistent with the interpretation that the experience of each emotion stimulus valence (negative and positive) was greater in the suppression than induction instruction conditions, particularly for negative emotion stimuli,  $F(1,149) = 21.95$ ,  $p < 0.001$ ,  $\eta^2 = 0.13$ . Analysis of the simple effects confirmed that the four conditions differed as indicated by our labeling of the conditions: higher (more positive) ratings for positive than negative emotion stimuli, all  $p < 0.001$ . Additionally, higher (more positive) ratings for induction than suppression instructions

were also observed, all  $p < 0.001$ , see **Table 3** for the task condition descriptive data according to group. Also reassuringly, the ADHD, ADHD+CU, and control groups were similar with respect to SAM valence rating scores, as none of the interactions involving group status were significant (all  $F < 1.0$ , all  $p > 0.10$ ), suggesting that interpretation of changes in autonomic reactivity were not confounded by differences in self-reported valence appraisal of the task conditions. Furthermore, groups did not differ with respect to valence during any of the task conditions, all  $F < 1.0$ , all  $p > 0.10$  (see **Table 3**).

The arousal rating scores showed a similar picture (see Table 3). There was a significant main effect for arousal scores between positive and negative segments,  $F(1,149) = 107.31$ ,  $p < 0.001$ ,  $\eta^2 = 0.43$ . Additionally, the interaction of valence and regulation conditions was significant,  $F(1,149) = 8.35$ ,  $p < 0.005$ ,  $\eta^2 = 0.06$ . However, the suppression versus induction difference was not significant,  $F(1,149) = 1.52$ ,  $p = 0.22$ ,  $\eta^2 = 0.01$ , and none of the interactions involving group status were meaningful (all  $F < 1.0$ ), see **Table 3** for the task condition descriptive data according to group. Specifically, groups did not differ with respect to arousal level during any of the task conditions, all  $F < 1.0$ , all  $p > 0.10$  (see Table 3).

Furthermore, the groups did not differ in the emotion which they rated as feeling most prominently (with choices of surprise, happy, angry, fear, anxiety, or sadness) during any of the task conditions (all  $\chi^2 < 11.920$ , all  $p > 0.05$ ; see **Table 4**). Thus, these data suggest that the task manipulations were interpreted in the same way across all groups.

#### *Effectiveness of Emotion Induction by Facial Affective Behavior*

Before examining group differences the validity of our task manipulation were also considered by examining facial affective behavior during each of emotion-induction

and suppression task conditions. Groups did not differ in their FACS frequency ratings of any of the six facial affective behaviors rated during the neutral pictures, all  $F < 1.0$ , all  $p > 0.10$ . Thus, any differences in autonomic reactivity during the tasks could not be attributed to differences in tendency to over or under display of facial affective behavior related to the task. To assess the FACS ratings for the different experimental conditions, a 4x3 repeated-measures ANOVA (condition [negative induction/negative suppression/positive induction/positive suppression] x group [control/ADHD/ADHD+CU]) was conducted. Partial eta<sup>2</sup> ( $\eta^2$ ) was included as a measure of effect size (Cohen, 1992). The analysis supported the validity of the positive/negative emotion induction and suppression manipulations across groups according to the amount of facial affect displayed for each of the six facial affective behaviors coded examined separately in **Table 5**. Additionally, a simple examination of the means of each of the facial affective behaviors coded in each condition reveals that these were in the appropriate direction (i.e., greater levels of happiness for all groups during the positive conditions, greater levels of surprise, anger, anxiety, fear, and though not significantly different, sadness, during the negative conditions; see Table 5).

Specifically, scores for the full sample differed according to condition for each of the six facial affective behaviors rated all  $F > 4.2$ ,  $p < 0.05$ , with the exception of sadness  $F(1,149) = 2.17$ ,  $p > 0.05$ , which did not differ significantly according to task condition. Also reassuringly, the ADHD, ADHD+CU, and control groups were similar with respect to facial affective rating scores for each of the six facial affective behaviors coded, as neither the main effect of group (all  $F < 2.0$ , all  $p > 0.10$ ) nor the interaction involving group status were significant (all  $F < 2.0$ , all  $p > 0.10$ ), suggesting that interpretation of

changes in autonomic reactivity were not confounded by differences in behavioral engagement in the task conditions (see **Table 5**).

#### *Baseline Physiological Effects*

It was important to show that there was stability in the physiology measures throughout each baseline condition to confirm that these were stable periods of rest or recovery compared to the experimental conditions, and to show that changes in physiological indexes were not due to simple acclimation to the task over time. The mean scores of RSA, PEP, interbeat interval, and respiration rate were calculated for each of the four 30-second epochs of both the resting and the neutral pictures baselines (overall mean and standard deviations of these values are listed by group in Table 6). None of these physiology parameters differed from the first 30 second epoch to the last 30 second epoch (all  $F < 1.02$ ,  $p = 0.37$ ,  $\eta^2 = 0.01$ ). The mean of the two 30 second epochs of the second minute of each baseline is used in calculations of change scores for the subsequent analyses, in order to allow for acclimation to the task conditions and recovery from the emotion-task, respectively.

#### *Task Habituation and Order Effects*

Task habituation and order effects were evaluated by comparing physiological measures across the resting and neutral baseline conditions. Repeated measures ANOVA indicated that RSA, interbeat interval, and PEP differed across the baselines, all  $F > 8.76$ ,  $p < 0.01$ , suggesting systematic changes in physiology when comparing rest to the demands imposed by attending to and orienting to a neutral task, as expected (see Table 6). Second, a polynomial repeated measures ANOVA for the full sample revealed that the linear effect of time on RSA across all task conditions was significant,  $F(1,149) = 9.82$ ,  $p$

= 0.002,  $\eta^2 = 0.06$ , as was the quadratic effect,  $F(1,149) = 8.24$ ,  $p = 0.00$ ,  $\eta^2 = 0.05$ , as was the cubic effect,  $F(1,149) = 32.92$ ,  $p < 0.001$ ,  $\eta^2 = 0.18$ . This is consistent with participants responding to the task manipulations and inconsistent with a habituation effect. Simple examination of the means (**Table 6**) confirms that RSA did not simply decrease across task conditions. Table 6 displays the exact means and standard deviations, by group, for specialists wishing to make detailed comparisons.

A different pattern was observed for PEP: here, no significant effect was seen for the linear, quadratic, or cubic effect (all  $F < 1.0$ , all  $p > 0.05$ ), see Table 6. This suggests there was no change in PEP across the baseline (rest and neutral) task conditions and that group main effects of PEP would be the likely focus of results. Interbeat interval and respiration rate displayed a similar consistency across conditions all linear, quadratic, and cubic effects (all  $F < 2.3$ , all  $p > 0.05$ ).

Additionally, group comparisons revealed that during the neutral and resting baselines there were no significant differences in RSA, heart rate, or respiration rate, according to group status, all  $F < 1.0$ , all  $p > 0.10$  (see Table 6). However, groups did differ with respect to PEP during the neutral baseline only, during which the ADHD and control groups showed higher arousal (shorter PEP) than the ADHD+CU group,  $F(2,148) = 3.7$ ,  $p < 0.05$  (see Table 6). Thus, it is concluded that habituation and simple time-based effects were trivial relative to task manipulation effects, and proceeded to our primary analyses and hypothesis testing.

### **Primary Analyses: Emotion Induction and Suppression on PEP and RSA**

#### *Effects on PEP*

A 2x2x3 repeated-measures ANCOVA examined the effects of task condition on

raw scores for PEP with gender, stimulant medication status, and ODD and anxiety symptoms treated as covariates. The interaction of valence by condition was non-significant,  $F < 1.0$ . However, in line with our hypothesis that the ADHD and ADHD+CU groups would differ with respect to overall arousal, there was a significant group main effect,  $F(1,149) = 3.16, p < 0.05, \eta^2 = 0.05$ . Specifically, the grand mean of PEP for the control group (97.85,  $SD=7.42$ ) was longer than for the ADHD group (95.22,  $SD=7.91$ ), but shorter than for the ADHD+CU group (99.10,  $SD=5.71$ ), as predicted by the hypothesis that CU would be associated with lower sympathetic arousal (see **Figures 3 and 4**). **Figure 3** shows effects for both the ADHD groups collapsed compared to the control group to illustrate overall differences in PEP across the task conditions, when the CU trait is not taken into account. However, **Figure 4** shows effects for both the ADHD and ADHD+CU groups compared to control to illustrate differences in PEP across task conditions.

Here, group comparisons showed that there was a significant difference in PEP level at each of the 4 task conditions when comparing groups, all  $F > 3.2$ , all  $p < 0.05$ . In particular, the ADHD+CU group (101.17,  $SD = 5.73$ ) had a longer PEP value than the control group (97.59,  $SD = 7.49$ ), which was longer than the ADHD only group (94.40,  $SD = 8.02$ ) during the negative induction condition,  $F(2, 148) = 3.79, p < 0.05$  (Figure 4). During the negative suppression condition, the ADHD+CU group (99.31,  $SD = 5.44$ ) had a longer PEP value than the control and ADHD groups (97.45,  $SD = 7.59$  and 95.09,  $SD = 7.71$ , respectively), which did not differ from one another,  $F(2, 148) = 3.51, p < 0.05$  (Figure 4). However, as shown in **Figure 3** and **Figure 4**, during the positive condition, the ADHD only group displayed shorter PEP values than both the control and

ADHD+CU groups, which did not differ from one another  $F(2, 148) = 3.34, p < 0.05$  and  $F(2, 148) = 3.35, p < 0.05$ , respectively for positive induction and suppression (see Table 6; Figure 3 and 4).

#### *Effects on RSA*

The means of the RSA reactivity scores for each task epoch are listed in **Table 6**, and the results of the factorial decomposition are depicted in **Figure 5** and **6**. **Figure 5** shows effects for both the ADHD groups collapsed compared to the control group to illustrate overall differences in RSA reactivity scores across the task conditions, when the CU trait is not taken into account. However, **Figure 6** shows effects for both the ADHD and ADHD+CU groups compared to control to illustrate differences in RSA reactivity across task conditions. The mixed model 2x2x3 repeated-measures ANCOVA (with gender, stimulant medication status, and ODD and anxiety symptoms treated as covariates) revealed condition main effects for both emotion stimulus condition (i.e., negative vs. positive)  $F(1,149) = 16.49, p < 0.01, \eta^2 = 0.10$  and regulation instruction condition (i.e., induction vs. suppression),  $F(1,149) = 7.327, p < 0.01, \eta^2 = 0.05$ . Significant interactions were seen for both group by emotion stimulus condition  $F(1,149) = 6.448, p < 0.01, \eta^2 = 0.08$  and group by regulation instruction condition,  $F(1,149) = 6.619, p < 0.05, \eta^2 = 0.05$ . Simple effects revealed that CU was associated with under regulation, as the ADHD+CU group showed a smaller increase than the ADHD and control group during the negative induction and negative suppression task segments ( $F > 1.0$ , all  $p < 0.05$ ; see Figure 6). Thus, the children in the ADHD+CU group did not increase their parasympathetic regulation during the experience of or suppression of negative emotion.

Furthermore, effects were seen in the positive emotion stimulus conditions. In the positive induction condition, the ADHD only group's RSA increased from baseline, while the control and ADHD+CU group's RSA decreased from baseline,  $F(2,148) = 3.65, p < 0.05$ , supporting our hypothesis that children with ADHD would have a more challenging time regulating positive emotions in the absence of CU (see Figure 6). Additionally, during the positive suppression condition, RSA increased from baseline for the control and ADHD group and this differed from the ADHD+CU group, which did not change from baseline,  $F(2,148) = 3.52, p < 0.05$  (see Figure 6).

### **Follow-up Checks on PEP and RSA Results using Continuous CU**

The CU trait was treated as a categorical variable above for ease of interpretation, and those children assigned to the CU group were assigned conservatively, as both the parent and teacher had to rate them as greater than the 90<sup>th</sup> percentile of impaired prosocial behavior. However, as CU traits are perhaps better understood as a dimensional phenomenon, with individuals falling on a continuum of being more or less callous/unemotional, these results were also examined with CU included as a continuous variable. These results are presented separately for PEP, then RSA below.

#### *Continuous Effects of CU on PEP*

A 2x2x2 repeated-measures ANCOVA examined the effects of task condition on raw scores for PEP with between-subject factors of ADHD status and continuous scores of CU, as well as the interaction of ADHD group status and CU level, in the model. Again, gender, stimulant medication status, and ODD and anxiety symptoms were treated as covariates. The interaction of valence by condition was non-significant,  $F < 1.0$ , as were each of the 3-way interactions involving valence by condition by ADHD group status

(and by CU level),  $F < 0.7$ , as was the 4-way interaction of valence by condition by ADHD status by CU level,  $F < 0.05$ .

While there was a no significant ADHD group main effect,  $F < 1.0$ , there was a significant main effect of CU level,  $F(1,149) = 5.52, p < 0.05, \eta^2 = 0.07$ , as well as a significant interaction of ADHD group status by CU level,  $F(1,149) = 4.16, p < 0.05, \eta^2 = 0.04$ . This is consistent with the hypothesis that CU would be associated with lower sympathetic arousal even when CU was treated as a continuous variable.

#### *Continuous Effects of CU on RSA*

The mixed model 2x2x2 repeated-measures ANCOVA with between subjects factors of ADHD group status, CU level, and the interaction of ADHD group status with CU level (with gender, stimulant medication status, and ODD and anxiety symptoms treated as covariates) was examined. This model revealed condition main effects for both emotion stimulus condition (i.e., negative vs. positive)  $F(1,149) = 7.835, p < 0.01, \eta^2 = 0.09$  and regulation instruction condition (i.e., induction vs. suppression),  $F(1,149) = 7.571, p < 0.01, \eta^2 = 0.07$ . Significant interactions were seen for both ADHD group by emotion stimulus condition  $F(1,149) = 6.107, p < 0.01, \eta^2 = 0.06$  and ADHD group by regulation instruction condition,  $F(1,149) = 4.026, p < 0.05, \eta^2 = 0.04$ . Furthermore, a significant 3-way interaction of ADHD group by CU level by Emotion instruction type was observed,  $F(1,149) = 3.426, p < 0.05, \eta^2 = 0.14$ , as was a significant ADHD group by CU level by Instruction type interaction,  $F(1,149) = 3.026, p < 0.05, \eta^2 = 0.10$ .

#### **Follow-up Checks on PEP and RSA Results by Group**

The hypothesis was that there would be specific responses in sympathetic and parasympathetic systems associated with the induction and suppression of both negative

and positive emotions described above, and that these response patterns would be moderated by group status. However, an alternative explanation for the PEP and RSA results might be that they were due to global fluctuations in the participants' overall physiology, such as changes in interbeat interval or respiration rate, rather than to isolated physiological changes that are specific to the sympathetic or parasympathetic systems. If so, the results should emerge similarly whether simply examining interbeat interval or respiration rate.

To test this possibility, the main effects and interactions for interbeat interval and respiration rate were examined using the same 2x2x3 repeated-measures ANCOVA. The main effects of task emotion stimulus (positive/ negative) and regulation-based instruction condition (suppression/induction) for interbeat interval were significant  $F(1,149) = 5.392, p < 0.05, \eta^2 = 0.04$  and  $F(1,149) = 21.555, p < 0.001, \eta^2 = 0.13$ , respectively, with interbeat interval changing in the task-appropriate direction. However, none of the interactions by group were significant, all  $F < 2.10$  and  $p > 0.10$ . Additionally, there were no significant effects with respect to respiration rates (all  $F < 3.10, p > 0.10$ ). Thus, groups did not differ on general, multi-system determined physiological parameters, suggesting it was appropriate to interpret the PEP and RSA effects as specific indexes of sympathetic and parasympathetic activity, respectively, as intended.

### ***Discussion***

Grouping children with ADHD on the basis of high and low CU traits revealed distinct patterns of autonomic reactivity during emotional arousal and regulation, within the ADHD cohort. This same pattern held whether the low prosocial score (CU) was

treated as a category or a dimension. For simplicity, the results are discussed with CU treated as a category. The effect held even in sample with a low prevalence of comorbid conduct problems and after controlling for such conduct problems; thus, CU was not simply a proxy for conduct problems in this sample. When CU traits were low, children with ADHD displayed a pattern of sympathetic activity that was elevated in comparison to typically developing children both during attending/orienting and across the changing affective and regulatory demands of induction and suppression. Furthermore, children with ADHD and low levels of CU traits displayed the greatest level of parasympathetic increase from baseline during the positive emotion induction condition, suggesting a specific dysfunction in the approach (positive) system among these children when compared to typically developing youth. Finally, children with ADHD and high levels of co-occurring CU traits displayed both blunted parasympathetic and sympathetic activity across all task conditions, consistent with a reduction in arousal and regulation both during attending/orienting and in the face of emotion-based demands.

Several alternative interpretations of these data were ruled out. Specifically, neither child gender nor history of stimulant medication use nor comorbid diagnoses nor symptoms accounted for the effects. Additionally, sympathetic nervous system activity differed across the groups during an emotionally neutral task, as expected given previous reports on SNS activity in individuals with CU traits (Raine et al., 1996, 1997, 2002). Additionally, parasympathetic nervous system activity did not differ during conditions of emotional neutrality or rest, suggesting that these differences were not due to preexisting differences in homeostatic functioning, but rather limited to differences in physiological emotion regulation. Furthermore, children's self-reported emotion valence and arousal

levels during the emotionally neutral task suggested no preexisting differences in mood among the groups. Additionally, these changes do not appear to be due to group differences in engagement in the task demands, as the groups did not differ in facial affective behavior during any of the task conditions. Importantly, these results also held when examining the CU trait as a continuous variable, further underlining the validity our results. Finally, the autonomic differences among the groups were not accounted for by more general changes in overall physiological reactivity (i.e., heart rate or respiration rate), which is consistent with previous research in this age group (Beauchaine, 2001).

The current results are consistent with past research showing that CU traits moderate emotional responding in children with conduct problems (Frick & Viding, 2009; Frick & White, 2008). However, this study is innovative in several ways. First, CU traits in children were examined without conduct disorder and while controlling for ODD symptoms, allowing us to get more specifically at the nature of this trait in ADHD in the absence of clinically significant conduct problems. This design was intended to facilitate parsing the unique heterogeneity of ADHD by examining a specific sub-group of children with ADHD and co-occurring CU traits.

Second, autonomic reactivity in both branches (parasympathetic and sympathetic) was measured during both emotion induction *and* suppression in response to an emotionally arousing film-clip. This revealed group differences in sympathetic functioning across conditions, suggesting specifically that children with ADHD may have a tendency to experience over-arousal, while children with ADHD and co-occurring CU traits have a tendency to experience under-arousal, both at rest and during emotionally evocative situations. Effects in parasympathetic activity also emerged. Specifically,

children with ADHD and low CU scores showed increased parasympathetic activity, whereas children with ADHD and high CU scores showed blunted parasympathetic activity more generally across all conditions. These findings build upon studies by Beauchaine et al., (2007) and Crowell et al., (2006) that reported differences in sympathetic functioning among children with ADHD and comorbid conduct problems when compared to typically developing youth, but which found attenuated sympathetic activity in response to reward-based tasks.

Third, the use of both positive and negative emotion stimulus conditions further helped to explore differences in autonomic functioning among these groups, as children in the ADHD only group displayed ineffective parasympathetic responding specifically in the positive emotion domain. In particular, children with ADHD showed a pronounced *increase* in RSA from baseline during the positive induction condition in contrast to the decrease observed in typically developing children. This finding is in line with previous theory suggesting that ADHD is uniquely associated with disruptions in the positive (approach) system (Martel, 2009; Nigg, 2006). Prior findings (Marsh et al., 2008) have suggested alterations in the anger response among children with ADHD, which is not a positive emotion but is an approach emotion (Canli et al., 2001, 2004). This effect was reduced in the presence of high CU scores, where general blunting of response was seen across task conditions. Thus, mixed findings and heterogeneous results might be expected when regulation or positive emotion processing are assessed in ADHD without considering the level of CU traits.

Several limitations in the current results should be noted. First, the sample size was relatively small for detecting moderator effects between groups (e.g., diagnosis x sex

interactions), and too small to examine the effects of ADHD subtype (e.g., inattentive compared to combined). While the effects reported here cannot be explained by the presence of comorbid disorders or behaviors, it will be interesting in future research to examine children with comorbid CD and ADHD. Furthermore, as this sample was recruited from the community, the severity of the both CU traits and ADHD may be less than a sample recruited from clinics or forensic samples. It should also be noted that the use of generic positive and negative emotion valence stimuli rather than specific emotion-based stimuli, such as sadness or anger, limits more specific conclusions and requires follow-up. Finally, it will be of interest to assess whether the patterns observed are stable over time, or are predictive of course, impairment, response to treatment, the development of comorbid disorders, and other clinical outcomes.

In conclusion, this study revealed that when children with ADHD were divided according to their level of CU traits, distinct patterns of autonomic response within the ADHD population are revealed. This clarifies both that emotion and emotion regulation are features of ADHD and that the nature of this problem is meaningfully heterogeneous within ADHD subpopulations. Failure to consider standing on CU traits may confound evaluation of emotion regulation in ADHD. Additionally, CU traits were distinct from the assessment of oppositional or conduct problems in children with ADHD. Thus, there may be meaningful sub-groups of children with ADHD with a distinct pattern of autonomic dysfunction in comparison to typically developing youth. Although ADHD is associated with emotion dysregulation during positive emotions, (as indexed by overactive parasympathetic activity) this effect was masked when CU traits were elevated. These findings are consistent with hypotheses that ADHD is a heterogeneous disorder,

involving alterations in both emotion and its regulation, in addition to difficulties with cognition and behavior. Further, if future research replicates these findings in other samples, it would suggest that the CU traits specifier, which is currently being considered in DSM-5 for the diagnosis of conduct disorder, might also be informative for ADHD. However, it should be noted that while the CU traits specifier may be informative for ADHD, additional consideration of both its clinical utility and issues related to potential stigmatization associated with the label will need to be fully addressed (Frick & Nigg, 2012). Given this new understanding of the roles of physiological emotional arousal and regulation in specific sub-groups of children with ADHD, attention is now turned to the role of coherence among of multiple emotional systems, including facial affective behavior and autonomic nervous system functioning, in ADHD.

## CHAPTER III

### STUDY 2: EMOTIONAL RESPONSE COHERENCE IN ADHD

#### *Methods*

#### **Summary**

To address the main hypotheses of Study 2, children with ADHD would display reduced correspondence among emotional systems, the same emotion induction and suppression task utilized in Study 1 was used. However, using hierarchical linear modeling, the correspondence among physiological indexes of emotion/emotion regulation and facial affective behavior was examined. Specifically, hierarchical linear modeling was used to confirm that the findings of Study 1 held for this sub-sample of 100 participants (67% of the original 150 participants in Study 1). That is, it was used to determine whether the ADHD and control groups differed with respect to overall frequencies of facial affective behavior, as well as overall levels of autonomic activity, across task conditions. Additionally, hierarchical linear modeling was used to determine whether correspondence between facial affective behavior and autonomic activity would be stronger during the induction condition than the suppression condition. Finally, to assess the primary hypothesis that children with ADHD would show weaker correspondence of emotional systems than typically developing youth, hierarchical linear model was used to determine whether ADHD was characterized by reduced correspondence among emotional systems of facial affective behavior and autonomic activity during the experience of both negative and positive emotions.

The important role of CU traits in influencing psychophysiological responses (i.e., CU traits, when treated both dimensionally and categorically, were associated with blunted parasympathetic and sympathetic nervous system activity across conditions of

negative and positive emotion induction and suppression) observed in Study 1 should be noted. Thus, it was possible that CU traits would also affect the primary results of Study 2. As such, CU traits were also treated as a covariate in all of the primary analyses of Study 2. In Study 1, the CU trait was treated as a categorical variable for ease of interpretation. However, CU traits are perhaps better understood as a dimensional phenomenon (Frick, 1999), with individuals falling on a continuum of being more or less callous/unemotional. As such, and because no a priori hypotheses were made regarding the effects of CU traits on the primary results, the results of Study 2 are presented with CU included as a continuous covariate. Additionally, it should be noted that the inclusion of CU traits in these analyses did not yield different results, when treated as a covariate, as described in detail in Study 2 Results below.

Again, a table providing a list of assessment measures for screening and diagnosis, Study 1, Study 2, and Study 3 is provided (Table 1, page 123). Most of the remaining data collection methods and procedures were the same as in Study 1 as noted below.

### **Participants**

Study 2 included 100 participants (a sub-set of the original 150 participants from Study 1, ADHD,  $n=50$ ; control,  $n=50$ ). This study evaluated the correspondence between physiological and facial affective behavioral indices of emotion and its regulation in children with ADHD. A summary of the demographic and diagnostic characteristics of the participants of Study 2 are presented in Table 7, page 132.

### **Procedures**

The following procedures were used for Study 2.

### *Recruitment and Identification Procedures*

For a full description of Study 2 recruitment and identification procedures, see Study 1, page 23. These procedures were identical for Study 1, as Study 2 utilized a subset of these same participants.

### *Final ADHD and Other Diagnoses*

For a full description of Study 2 ADHD and other diagnostic procedures, see Study 1, page 24.

*Identification of the Callous/Unemotional Traits.* For a full description of Study 2 assessment of the callous/unemotional trait, see Study 1, page 24. However, note that group assignments were not made in Study 2, but rather, the continuous scores from the reverse coded Prosocial Scale of the Strengths and Difficulties questionnaire were utilized. Specifically, this continuous score was utilized as a covariate in all of the primary analyses of Study 2 to rule out the explanation that the presence of callous/unemotional traits explained the primary results of Study 2.

*Exclusion Criteria.* For a description of Study 2 exclusion criteria, see Study 1, page 25.

### *Medication Washout*

For a full description of Study 2 medication washout procedures, see Study 1, page 26.

### *Emotion Induction and Suppression Procedure*

For a full description of the emotion induction and suppression procedure utilized in Study 2, see Study 1, page 26.

### *Facial Action Coding System*

For a full description of Study 2 facial affective coding procedures, see Study 1, page 27.

### *Physiological Recording*

For a full description of Study 2 physiological recording procedures, see Study 1, page 28.

### ***Data Analysis***

#### **Analytic Plan**

Hierarchical linear modeling (HLM) was used to analyze the levels and levels of coherence among indexes of emotion regulation, including facial affective behavior (modified Facial Action Coding System ratings) and physiological measures (RSA and PEP, in separate models). To do this, three separate models were examined in order to examine three separate groups of hypotheses. Finally, it should be noted that each of the predictors in each of the models described below were entered into the models as variables which had been centered around the appropriate means.

#### *First Model*

First, the hypothesis that children with ADHD will show increased emotionality (i.e., a greater frequency of facial affective behaviors) across task conditions was examined. In order to do so, two separate models were constructed, one for *Approach* facial affective behaviors and one for *Avoidance* facial affective behaviors, which were determined to be higher-order factors of the six facial affective behaviors assessed (see Data Reduction for Primary Analyses below). For each model (i.e., for *Approach* and *Avoidance*), Level 1 included facial affective behavior as an outcome (i.e., either

*Approach* or *Avoidance*, examined in separate models), and Level 2 included group status. Again, this model was examined for both forms of facial affective behavior (i.e., *Approach* and *Avoidance*), separately. For a summary of the variables examined in this model see Table 8 (page 134). Full maximum likelihood models were composed in the following way for facial affect:

$$\textbf{Level-1: } \text{AFFECT RATING}_{ij} = \beta_{0j} + \beta_{0j} (\text{TIME}_{ij}) + r_{ij}$$

$$\textbf{Level-2: } \begin{aligned} \beta_{0j} &= \gamma_{00} + \gamma_{01}(\text{GROUP}_j) + u_{0j} \\ B_{1j} &= \gamma_{10} + \gamma_{11}(\text{GROUP}_j) + u_{1j} \end{aligned}$$

where AFFECT RATING<sub>ij</sub> represents *Approach* facial affective behavior (and in a second, separate model, *Avoidance* facial affective behavior), TIME<sub>ij</sub> represents the 16 continuous 30 second epochs of the task, and GROUP<sub>j</sub> represents a dummy coded variable representing ADHD=1 or control=-1 group status.

### *Second Model*

Next, in order to examine the hypothesis that there would be a statistical correspondence among measures of emotion and its regulation, during induction, but not during suppression, a second, different model was examined. For this model, at Level 1, three types of variables were included as predictors of ANS activity (with the outcome variables of RSA and PEP, included in separate models). Each of these variable types was entered in the model simultaneously. First, the two facial affective behaviors (both *Approach* and *Avoidance* in the same model) were treated as predictors of ANS activity (first for RSA as an outcome, then in subsequent analysis for PEP as an outcome). Second, the type of instruction (induction or suppression) was included as a dummy coded (-1=induction, 1=suppression) predictor of ANS activity. Third, the interaction

term of the instruction type with each of the facial affective behavior ratings (the product term of the each of the first two variable types) were included at Level 1 to examine the prediction of autonomic nervous system activity (i.e., RSA, then PEP) by *Approach* and *Avoidance* facial affective behaviors during the induction and suppression conditions.

At Level 2 of this model, group effects were tested using a dummy coded time invariant variable (1=ADHD, -1=control). It was expected that the associations between autonomic nervous system activity (i.e., RSA and PEP) and facial affective behavior (i.e., *Approach* and *Avoidance*) would be moderated by a 2-way, within level, Instruction Condition Type (i.e., induction vs. suppression; Level 1)\*Facial Affective Behavior (i.e., *Approach* or *Avoidance*, Level 1) interaction. For a summary of the variables examined in this model see Table 8 (page 134). For each of the two psychophysiological outcome measures (i.e., RSA, PEP) the following model was constructed (Bryk & Raudenbush, 1992).

**Level-1:**

$$ANS_{ij} = \beta_{0j} + \beta_{1j}(TIME_{ij}) + \beta_{2j}(APPROACH_{ij}) + \beta_{3j}(AVOID_{ij}) + \beta_{4j}(INDvSUP_{ij}) + \beta_{5j}(APPRO*INDvSUP_{ij}) + \beta_{6j}(AVOID*INDvSUP_{ij}) + r_{ij}$$

**Level-2:**

$$\begin{aligned} \beta_{0j} &= \gamma_{00} + \gamma_{01}(GROUP_j) + u_{0j} \\ \beta_{1j} &= \gamma_{10} + \gamma_{11}(GROUP_j) \\ \beta_{2j} &= \gamma_{20} + \gamma_{21}(GROUP_j) \\ \beta_{3j} &= \gamma_{30} + \gamma_{31}(GROUP_j) \\ \beta_{4j} &= \gamma_{40} + \gamma_{41}(GROUP_j) \\ \beta_{5j} &= \gamma_{50} + \gamma_{51}(GROUP_j) \\ \beta_{6j} &= \gamma_{60} + \gamma_{61}(GROUP_j) \end{aligned}$$

where  $ANS_{ij}$  represents RSA (and in a separate analysis PEP),

$APPROACH_{ij}$  represents *Approach* facial affective behavior,  $AVOID_{ij}$  represents

*Avoidance* facial affective behavior,  $INDSUP_i$  is a dummy coded variable

representing whether the instruction condition required induction=-1 or

suppression=1,  $APPRO*INDSUP_{ij}$  represents the interaction of *Approach* facial affective behavior and instruction type (induction vs. suppression),  $AVOID*INDSUP_{ij}$  represents the interaction of *Avoidance* facial affective behavior and instruction type (induction vs. suppression) and  $GROUP_j$  is a dummy coded variable representing ADHD=1 or control=-1 group status.

### *Third Model*

To assess the primary hypotheses of this study that children with ADHD would show weaker correspondence among measures of emotion regulation including: autonomic reactivity and facial affective behavior, whereas typically developing children will show developmentally- and task-appropriate levels of correspondence among these measures during emotion both negative and positive emotion induction. The outcome variable was ANS activity (i.e., RSA, and separately, PEP), as in Model 2. At level one, three types of variables were included in the model simultaneously. First, facial affective behavior (*Approach* and *Avoidance*) was treated as a predictor of ANS activity (first for RSA as an outcome, then in subsequent analysis with PEP as an outcome). Second, the valence of the stimulus condition (i.e. negative and positive stimulus valence) was included as a dummy coded variable (Negative=-1, Positive=1). Note that this model examined these stimulus conditions only within induction (negative induction versus positive induction). The scores for suppression were omitted from this model, as the hypothesis involving suppression was examined in Model 2. Third, the interaction term of the type of the valence stimulus condition type with the coded facial affective behavior rating (the product terms of the each of the first two variable types) was included at Level 1 to examine the prediction of autonomic nervous system activity (i.e., RSA, then PEP)

by *Approach* emotions according to stimulus valence, as well as for *Avoidance* facial affective behaviors. Thus, Models 2 and 3 were alike in many respects. The key difference being that while Model 2 examined correspondence of facial affect behavior and ANS activity during conditions of induction compared to conditions of suppression, Model 3 examined this correspondence during conditions of negative induction compared to conditions of positive induction. Again, suppress conditions were excluded from Model 3.

At level 2, group effects were tested using a dummy coded time invariant variable (1=ADHD, 0=control). It was expected that there would be a main effect of ADHD group status on both RSA and PEP in each of the separate models, as is consistent with the results of Study 1. Additionally, it was expected that the associations between autonomic nervous system activity (i.e., RSA and PEP) and facial affective behavior (i.e., *Approach* and *Avoidance*) would be moderated by an across-level 3-way interaction of Group (i.e., ADHD or control, Level 2) \*Stimulus Valence Condition Type (i.e., negative or positive, Level 1) \*Facial Affective Behavior (i.e., *Approach* or *Avoidance*, Level 1) interaction. Moderation was expected to be present for both *Approach* and *Avoidance* facial affective behaviors in the prediction of both RSA and PEP, in separate models. That is, the 3-way interaction is what would be expected if there were, in fact, a valence-specific response pattern, which differed according to group status. Thus, it was expected that *Approach* would predict ANS activity under positive induction, while *Avoidance* would predict ANS activity under negative induction for the control group, while these associations would be diminished in the ADHD group. For a summary of the variables examined in this model see Table 8 (page 134).

For each psychophysiological measure (i.e., RSA, PEP) a two-level model was constructed (Bryk & Raudenbush, 1992), as follows:

**Level-1:**

$$ANS_{ij} = \beta_{0j} + \beta_{1j}(TIME_{ij}) + \beta_{2j}(APPROACH_{ij}) + \beta_{3j}(AVOID_{ij}) + \beta_{4j}(NEGvPOS_{ij}) + \beta_{5j}(APPROACH*NEGvPOS_{ij}) + \beta_{6j}(AVOID*NEGvPOS_{ij}) + r_{ij}$$

**Level-2:**

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(GROUP_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(GROUP_j)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(GROUP_j)$$

$$\beta_{3j} = \gamma_{30} + \gamma_{31}(GROUP_j)$$

$$\beta_{4j} = \gamma_{40} + \gamma_{41}(GROUP_j)$$

$$\beta_{5j} = \gamma_{50} + \gamma_{51}(GROUP_j)$$

$$\beta_{6j} = \gamma_{60} + \gamma_{61}(GROUP_j)$$

where  $ANS_{ij}$  represents RSA (and in a separate analysis PEP),

$APPROACH_{ij}$  represents *Approach* facial affective behavior,  $AVOID_{ij}$  represents *Avoidance* facial affective behavior,  $NEGvPOS_{ij}$  is a dummy coded variable representing whether the stimulus valence condition involved negative=-1 or positive =1 emotional stimuli,  $APPRO*NEGvPOS_{ij}$  represents the interaction of *Approach* facial affective behavior and stimulus valence condition type (negative vs. positive),  $AVOID*NEGvPOS_{ij}$  represents the interaction of *Avoidance* facial affective behavior and stimulus valence (negative vs. positive). Finally,  $GROUP_j$  is a dummy coded variable representing ADHD=1 or control=-1 group status.

**Power Calculations**

Castello and O'Brien (2000) assert that there is no generally accepted standard for power or sample size analyses for HLM. Specifically, the power level in HLM is affected by effect size, sample size, and the variable covariance structure, which can be difficult to estimate (Fang, 2006). However, according to Fang's (2006) work with Monte Carlo

simulations, it has been shown that when the sample size is 200 per group, the power level approaches .8, given a medium effect size (.5). In the case of this set of analyses, 100 participants with two samples (measures: RSA/PEP, facial affect) per group (ADHD and control) were proposed, which suggests that there was adequate power to detect both main and interaction effects. Finally, it should be noted that previous work by Marsh and colleagues (2008) using similar methods, utilized 54 participants with two samples (RSA/PEP, facial affect) per group (disruptive behavior disorders vs. controls) with adequate power (.90), given a medium effect size (.5) to detect the main and interaction effects assessed.

## ***Results***

### **Preliminary Analyses**

#### *Descriptive and Diagnostic Overview of Sample*

Descriptive and diagnostic statistics are reported for Study 2 by group in Table 7. Again, Study 2 was composed of 100 participants (67% of the 150 participants from Study 1). Prior to analyses, distributions of all variables were examined for adherence to the assumptions of the statistics utilized. Preliminary analyses examined the significance of group differences in each of the demographic, diagnostic, and psychopathology scales using analyses of variance (ANOVA) or Chi-square analyses, as appropriate. Effects sizes (partial eta-squared) are also reported (see Table 7).

Based on these analyses, groups did not differ with respect to age, race, parent marital status, or IQ. Inclusion of these variables as covariates did not affect results reported. Results are therefore reported without these variables treated as covariates for clarity and to reduce the chances of multicollinearity in the models. However, groups

differed in gender ratio (more boys in the ADHD group than the control group), family income (with families of ADHD children earning less than control children), and the prescription of stimulant medication (with the prescription of stimulant medication confined to the ADHD group). However, gender, family income, and stimulant use were each unrelated to facial affective behavior and physiological parameters, and therefore, did not affect results. Additionally, the primary results were not affected when gender, family income, or stimulant use were treated as covariates. Results are presented with gender treated as a covariate, as this was the most theoretically relevant to the primary questions of interest.

Clinical characteristics are also provided in **Table 7**. As expected according to group assignments, the ADHD group had more symptoms of inattention and hyperactivity than the control group on the parent and teacher rated problems on all clinical T-scored scales of the ADHD-Rating Scale (i.e., Hyperactivity/Impulsivity, Inattention, Total symptoms) and the Conner's 3<sup>rd</sup> edition (i.e., Inattention, Hyperactivity, Learning Problems, Aggression, and Peer Relations). Furthermore, the control and ADHD group differed significantly on each of the parent and teacher Strengths and Difficulties Questionnaire scales (i.e., conduct problems, emotion problems, hyperactivity, peer problems, total difficulties, and impact scales; see Table 7).

With regard to clinical comorbidity, as shown in **Table 7**, groups did not differ in the presence of lifetime conduct disorder. However, the ADHD group displayed significantly higher rates of oppositional defiant disorder than the control group. Additionally, the ADHD group had significantly more symptoms of ODD, CD, and anxiety than the control group. The inclusion of comorbid disorders including anxiety,

conduct, or oppositional defiant disorders as covariates (individually or collectively) did not affect any of the main study results. Covarying of total ODD symptoms, total CD symptoms, or total anxiety symptoms (all from the KSADS-E) also did not alter findings or conclusions for Study 2. Results are presented below without covarying comorbid symptoms; however, these results are available from the author upon request.

#### *Data Reduction for Primary Analyses*

Study 1 used the frequency of each of the individual six coded facial affective behaviors, in order to determine whether the task manipulations were successful for both groups. However, as more specific hypotheses regarding facial affective behavior were made for Study 2, an exploratory principal components analysis was performed to determine the underlying factor structure of the coded facial affective behaviors. This was done to examine the dimensionality of the frequency ratings of the six facial affective behaviors which were coded during each of the four task conditions, as well as to reduce multi-collinearity in the models.

The item set tended to be bi-dimensional for this sample. This was supported by the fact that the first eigenvalue was 1.50, while the second was all subsequent eigenvalues being less than 0.75. The two factors were labeled as *Approach* and *Avoidance*. The *Approach* factor had five items which loaded on it: happiness (0.75), anger (0.69), surprise (0.24), fear (0.12), and anxiety (-0.51). *Avoidance* factor had two items, anxiety (0.75) and sadness (0.61). However, as anger and fear had low factor-loadings and were coded infrequently (see **Table 9** for a description of the frequencies of individual indices of facial affective behavior), anxiety loaded more strongly on the *Avoidance* factor, these items were removed

the final solution for *Approach*. Thus, it was determined that the overall solution composed of an *Approach* factor (anger and happiness) and an *Avoidance* factor (sadness and anxiety), which is consistent with previous reviews of emotion (Beauchaine, 2001). Thus, this 2-factor solution was utilized in the multi-level modeling computing an overall factor score for each factor. To compute the two factor standardized score for each variable (happiness and anger for the *Approach* sadness and anxiety for the *Avoidance* factor) was multiplied by the corresponding loading and these products were added together to create a final factor score for *Approach* and for *Avoidance*. For rates of each of the *Approach* and *Avoidance* scores according to task condition and group see **Table 10**.

#### *Baseline Effects*

During the two minute Resting Baseline there were no significant differences in indexes of autonomic activity (i.e., respiration rate, interbeat interval, or RSA; all  $F_s < 1.10$ ,  $p > 0.10$ ) with the exception of PEP, with the ADHD group showing significantly lower PEP than the control group ( $F=4.52$ ,  $p < 0.01$ ). Additionally, no significant differences in autonomic activity for any of the measures were observed during the two minute Neutral Pictures Baseline (all  $F_s < 2.30$ ,  $p > 0.07$ ; see table 11). Finally, it should be noted that there were no group differences in the expression of facial affect for any of the six individual emotions assessed (i.e., surprise, happiness, anger, anxiety, fear, or sadness; Table 9), nor were group differences observed for either of the larger emotion factors (i.e. *Approach* or *Avoidance*, described above) during baseline with all  $F < 1.50$ ,  $p > 0.10$  (see Table 10).

## Model 1: Preliminary Analyses

First a multilevel model was used to test the hypothesis that children with ADHD would display a greater number of facial affective behaviors, including the *Approach* and *Avoidance* behavior factor scores described above. This was done in part to assess whether overall group differences in the amount of *Approach* or *Avoidance* facial affect might explain the primary results, as well as to examine whether there were group differences in overall behavioral expressions of emotionality.

Analyses exploring group differences in each of the coded facial affective behavior factors (i.e., the *Approach* and *Avoidance* factors) for the entire task (i.e., across all task conditions) were conducted using HLM 6.0 (Raudenbush, Bryk, Cheong, & Congdon, 2004). Multilevel modeling is advantageous because it allows for simultaneous estimates of within-participant (i.e. the sixteen repeated observations of *Approach* and *Avoidance* for each individual participant) and between-participant effects (i.e., between groups, or individual differences between individuals). Full maximum likelihood models were composed in the following way for facial affect (for the *Approach* and *Avoidance* factors, separately):

$$\text{Level-1: } \text{AFFECT RATING}_{ij} = \beta_{0j} + \beta_{1j}(\text{TIME}_{ij}) + r_{ij}$$

$$\text{Level-2: } \begin{aligned} \beta_{0j} &= \gamma_{00} + \gamma_{01}(\text{GROUP}_j) + u_{0j} \\ \beta_{1j} &= \gamma_{10} + \gamma_{11}(\text{GROUP}_j) + u_{1j} \end{aligned}$$

At Level 1, the repeated observations for each participant were modeled. Specifically, in the above equation,  $\text{AFFECT RATING}_{ij}$  represents the eight repeated observations of facial affect (*Approach* or *Avoidance*, with  $\text{TIME}_{ij}$  representing the continuous observations of one per 30 seconds across each of the two minutes of the four task conditions) for each participant. The  $\text{AFFECT RATING}_{ij}$  variable represents the

*Approach* and *Avoidance* factors assessed individually in two separate models with the above structure.

At Level 2, group comparisons were conducted using a dummy coded time invariant  $GROUP_j$  variable (1=ADHD, -1=control). This was completed to compare the ADHD group to control with respect to the amount of *Approach* and *Avoidance* facial affective behavior displayed across the task conditions in separate models. As such, in the Level 2 equation displayed above, the  $\gamma_{0i}$  term (which is similar to a standardized beta coefficient in regression analyses) captures whether there were differences between these groups in average frequencies of *Approach* or *Avoidance* facial affective behavior. Again, this set of analysis was run with the *Approach* factor, and then the *Avoidance* factor, separately. For a summary of the results of the primary model see Table 12.

With respect to the first hypothesis that children with ADHD would display a greater number of facial affective behaviors across task conditions, no significant group differences were observed when comparing ADHD with typically developing youth on either the two facial affective behavior factors across all task conditions (*Approach*:  $\gamma_{0i} = -0.01, p=0.833$ ; *Avoidance*:  $\gamma_{0i} = -0.09, p=0.295$ ). Thus, the first hypothesis was not supported, as no significant group differences in *Approach* or *Avoidance* facial affective behavior were observed. For a summary of the results of the primary model see Table 12.

### **Model 2: Preliminary Multilevel Analyses**

Next, a different multilevel model was used to test the hypothesis that there would be a statistical correspondence among measures of emotion and its regulation, including autonomic reactivity and facial affective behavior, during induction, but not during suppression. Specifically, analyses exploring group differences in levels of

correspondence between facial affective behavior (*Approach* and *Avoidance*) and autonomic nervous system activity (RSA and PEP, separately) according to instruction type (induction vs. suppression) were also conducted using HLM 6.0 (Raudenbush, Bryk, Cheong, & Congdon, 2004). It should be noted that in this analysis the model used a different outcome measure than in Model 1. Whereas Model 1 used facial affect behavior (*Approach* and *Avoidance* factors) as outcome measures, the following models (Models 2 and 3), facial affective behavior was used as a predictor with both in the same model, and autonomic nervous system activity was used as the outcome with RSA for one model and PEP for a second model. For each psychophysiological measure (i.e., RSA, PEP; examined as outcomes separately) the following model was constructed (Bryk & Raudenbush, 1992).

**Level-1:**

$$ANS_{ij} = \beta_{0j} + \beta_{1j}(TIME_{ij}) + \beta_{2j}(APPROACH_{ij}) + \beta_{3j}(AVOID_{ij}) + \beta_{4j}(INDvSUP_{ij}) + \beta_{5j}(APPRO*INDvSUP_{ij}) + \beta_{6j}(AVOID*INDvSUP_{ij}) + r_{ij}$$

**Level-2:**

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(GROUP_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(GROUP_j)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(GROUP_j)$$

$$\beta_{3j} = \gamma_{30} + \gamma_{31}(GROUP_j)$$

$$\beta_{4j} = \gamma_{40} + \gamma_{41}(GROUP_j)$$

$$\beta_{5j} = \gamma_{50} + \gamma_{51}(GROUP_j)$$

$$\beta_{6j} = \gamma_{60} + \gamma_{61}(GROUP_j)$$

At Level 1, the repeated observations for each participant were modeled.

Specifically,  $ANS_{ij}$  represents the eight repeated observations of either RSA or PEP (with one observation per 30 seconds across each of the two minutes of the four task conditions), which were examined in two separate multilevel models with this structure. Additionally,  $APPROACH_{ij}$  represents the eight repeated observations of facial affect frequency for *Approach* (with one observation per 30 seconds across each of the two

minutes of the four task conditions), while  $AVOID_{ij}$  represents these same observations but for the *Avoidance* factor for each participant. The  $INDvSUP_{ij}$  variable is a dummy coded variable representing the instruction conditions of induction and suppression (i.e., 1=induction, -1=suppression) Finally, the  $APPRO*INDSUP_{ij}$  and  $AVOID*INDSUP_{ij}$  variables represent the interaction between the induction vs. suppression dummy code variable and facial affective coding of *Approach* or *Avoidance*, respectively.

At Level 2, individual variation among participants was assessed, and group comparisons were conducted using a dummy coded time invariant  $GROUP_j$  variable (1=ADHD, -1=control). This was completed test the hypothesis that for typically developing individuals, correspondence between autonomic functioning and facial affect would be present during emotion induction, but not suppression, and the interactive effect of ADHD group status was also examined at Level 2. Identical procedures were followed for PEP. To aid in interpretation and for clarity, RSA is addressed first, followed by PEP.

#### *Model 2 for RSA*

With respect to the hypothesis that coherence would be observed during induction but not during suppression, significant interaction effects of both the *Approach* and *Avoidance* facial affective behaviors by the induction or suppression instruction dummy code were observed in the prediction of RSA ( $Approach*IndSup: \gamma_{40} = -0.05, p=0.045$ ;  $Avoidance*IndSup: \gamma_{50} = -0.03, p=0.022$ ). However, no significant 3-way interaction of group status by facial affective behavior type by instruction type was observed ( $Approach*IndSup*Group: \gamma_{41} = 0.001, p=0.951$ ;  $Avoidance*IndSup*Group: \gamma_{51} = -0.02, p=0.192$ ). It should be noted that only those portions of the model which were predicted

by hypotheses are reported here. For full results for each of the parameters of the model see Table 12.

Analyses of the simple effects of the 2-way interactions of the *Approach* (and in a second analysis the *Avoidance*) facial affective behavior by the induction or suppression instruction dummy code were completed. These follow-up analyses supported the hypothesis that there would be stronger associations between facial affective behavior (*Approach* and *Avoidance*) and autonomic nervous system functioning (RSA) during the induction condition than during the suppression condition. Specifically, there was a larger association between RSA and *Approach* during induction ( $\gamma_{10} = -0.07$ ) than during suppression ( $\gamma_{10} < -0.01$ ;  $t(540) = 2.15$ ,  $p = 0.03$ ). There was also a larger association between RSA and *Avoidance* during induction ( $\gamma_{20} = 0.05$ ) than during suppression ( $\gamma_{20} < -0.01$ ;  $t(540) = 2.03$ ,  $p = 0.04$ ), see also Figure 7.

#### *Model 2 for PEP*

A similar picture emerged for the model predicting PEP. Specifically, there was a significant main effect of ADHD group status on PEP ( $\gamma_{01} = -2.24$ ,  $p = 0.03$ ). Additionally, significant interaction effects of both the *Approach* and *Avoidance* facial affective behaviors by the induction or suppression instruction dummy code were observed (*Approach*\*IndSup:  $\gamma_{40} = -0.57$ ,  $p = 0.031$ ; *Avoidance*\*IndSup:  $\gamma_{50} = -0.45$ ,  $p = 0.041$ ). However, no significant 3-way interaction of group status by facial affective behavior type by instruction type was observed (*Approach*\*IndSup\*Group:  $\gamma_{41} = 0.39$ ,  $p = 0.099$ ; *Avoidance*\*IndSup\*Group:  $\gamma_{51} = 0.23$ ,  $p = 0.121$ ). Thus, there was no statistically significant moderation by group status of any of the aforementioned associations between the interaction of facial affective behavior and instruction type in prediction ANS

activity. Again, it should be noted that while only those portions of the model which were predicted by hypotheses are reported here. For full results for each of the parameters of the model see Table 12.

Similar results were observed for the 2-way interaction predicting PEP by *Approach* and *Avoidance*, respectively, with each being stronger during induction ( $\gamma_{10}=0.51$  and  $\gamma_{20}=-0.43$ , for *Approach* and *Avoidance*, respectively) than suppression ( $\gamma_{10}=0.29$  and  $\gamma_{20}=-0.19$ , for *Approach* and *Avoidance*, respectively) with all  $t(540) > 2.02$ ,  $p < 0.04$ ), see also Figure 8.

### **Summary of Preliminary Results of Models 1 and 2**

In contrast to the initial hypothesis, the results from Model 1 show that children with ADHD did not differ significantly from typically developing children with respect to the number of *Approach* or *Avoidance* facial affective behaviors they displayed across the task conditions. Thus, the following results cannot simply be explained by the two groups engaging in the task differently.

Furthermore, as hypothesized, there was a statistical correspondence among measures of emotion and its regulation, including: autonomic reactivity (i.e., RSA and PEP) and facial affective behavior (i.e., *Approach* and *Avoidance*), during induction, but not during suppression. This association was not moderated by group status. Thus, the primary hypothesis for Study 2 was able to be tested, as it was determined that the associations between the specific facial affective behaviors and autonomic activity would most fruitfully be examined under conditions of induction. That is, it was originally hypothesized that children with ADHD would show weaker correspondence among measures of emotion regulation including: autonomic reactivity (i.e., RSA and PEP) and

facial affective behavior (i.e., *Approach* and *Avoidance*), whereas typically developing children would show developmentally- and task-appropriate levels of correspondence among these measures during emotion *induction*, with specific patterns of correspondence observed with *Avoidance* during negative induction and with *Approach* during positive induction.

### **Model 3: Correspondence Analyses**

Finally, to test the primary hypothesis that children with ADHD would show weaker correspondence among measures of emotion regulation including: autonomic reactivity and facial affective behavior, whereas typically developing children would show developmentally- and task-appropriate levels of correspondence among these measures during emotion induction, HLM with full maximum likelihood estimation was again used in a third, separate model. Specifically, this model looked at differences in the associations between *Approach* and *Avoidance* with autonomic nervous system activity (again, RSA and PEP were examined in separate models) according to group status (ADHD compared to control) during specific stimulus valence conditions. It was hypothesized that reduced correspondence between *Approach* and ANS activity (both RSA and PEP) would be more salient in the positive induction condition, while reduced correspondence between *Avoidance* and ANS activity would be more salient in the negative induction condition (for both RSA and PEP). For each psychophysiological measure (i.e., RSA, PEP) a two-level model was constructed (Bryk & Raudenbush, 1992), the model was as follows:

#### **Level-1:**

$$ANS_{ij} = \beta_{0j} + \beta_{1j}(TIME_{ij}) + \beta_{2j}(APPROACH_{ij}) + \beta_{3j}(AVOID_{ij}) + \beta_{4j}(NEGvPOS_{ij}) + \beta_{5j}(APPROACH*NEGvPOS_{ij}) + \beta_{6j}(AVOID*NEGvPOS_{IND}_{ij}) + r_{ij}$$

**Level-2:**

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(GROUP_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(GROUP_j)$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(GROUP_j)$$

$$\beta_{3j} = \gamma_{30} + \gamma_{31}(GROUP_j)$$

$$\beta_{4j} = \gamma_{40} + \gamma_{41}(GROUP_j)$$

$$\beta_{5j} = \gamma_{50} + \gamma_{51}(GROUP_j)$$

$$\beta_{6j} = \gamma_{60} + \gamma_{61}(GROUP_j)$$

As a reminder, from the Data Analysis section, at Level 1,  $ANS_{ij}$  represents the outcome variables of autonomic nervous system activity. That is, in one model  $ANS_{ij}$  represents repeated observations of RSA, and in the second, PEP. Again,  $APPROACH_{ij}$  represents the eight repeated observations of facial affect frequency for *Approach* (with one observation per 30 seconds across each of the two minutes of the four task conditions), while  $AVOID_{ij}$  represents these same observations but for the *Avoidance* factor for each participant. The  $NEGvPOS_{ij}$  variable is a dummy coded variable representing the conditions of negative induction and positive induction (i.e., negative induction=-1, positive induction=1) Finally, the  $APPRO*NEGvPOS_{ij}$  and  $AVOID*NEGvPOS_{ij}$  variables represent the interaction between the negative vs. positive induction dummy code variable and facial affective coding of *Approach* or *Avoidance*, respectively.

At Level 2, individual variation among participants was assessed, and group comparisons were conducted using a dummy coded time invariant  $GROUP_j$  variable (1=ADHD, -1=control). This was completed test the hypotheses that for typically developing individuals, correspondence between autonomic functioning and *Avoidance* would be present during negative emotion induction and correspondence between autonomic functioning and *Approach* would be present during positive induction. The interactive effect of ADHD group status was also examined at Level 2, where again,

$GROU P_j$  was a dummy coded time invariant variable (1=ADHD, -1=control). Identical procedures were followed for PEP. For a summary of the results of the primary model see Table 12.

### *Model 3 for RSA*

When the above model was examined for RSA, in line with hypothesis, a significant main effect of group was observed on RSA ( $\gamma_{01}=0.30, p=0.03$ ; see also Table 11 for means according to group and task condition), and an examination of the means suggests overall higher levels of RSA for the ADHD group than the control group across task conditions. Additionally, and somewhat surprisingly, there was a significant 2- way group by *Avoidance* interaction ( $\gamma_{011}=-0.17, p=0.01$ ), suggesting that the association between *Avoidance* and RSA was moderated by group status when collapsed across the negative and positive valence conditions. Specifically, in the decomposition of the significant group by *Avoidance* interaction ( $\gamma_{011}=-0.17, p=0.01$ ), the typically developing children ( $\gamma_{10}=0.08$ ) had a stronger association between *Avoidance* and RSA than the ADHD group ( $\gamma_{10}=-0.03$ ;  $t(380)=4.03, p<0.01$ ), across induction-based task conditions, see Figure 9.

However, there were no significant main effects of *Approach* or *Avoidance* scores (Level 1) or group x *Approach* (Level 1\*Level2) on RSA (all  $\gamma_{xx}<0.03$ , all  $p>0.596$ ). Additionally, there was no significant main effect of stimulus valence condition (i.e., negative vs. positive induction) on RSA ( $\gamma_{311}<0.01, p=0.346$ ), nor was there a significant interaction of the stimulus valence condition by group ( $\gamma_{31}=0.01, p=0.458$ ).

Finally, the primary hypotheses that the association between RSA and facial affective behavior response would be disrupted in children with ADHD were explored.

Specifically, this analysis was to test the hypothesis that the association between facial affective behavior and autonomic nervous system activity would be disrupted with respect to *Avoidance* facial behavior in the negative induction condition and with respect to *Approach* facial behavior in the positive induction condition. Therefore, of central interest were the interactions of *Approach* (and of *Avoidance*) with the stimulus valence. Presence of one or both could also trigger a 3-way interaction of *Approach*\*valence\*group and *Avoidance*\*valence\*group were also examined.

Recall that both *Avoidance* and *Approach* are predictors and RSA is the outcome. Results for *Avoidance* were in contrast to hypothesis, as they showed no significant interaction effect of *Avoidance* by stimulus valence on RSA ( $\gamma_{60} < 0.01$ ,  $p = 0.839$ ), nor was there a significant 3-way interaction by group ( $\gamma_{61} < 0.01$ ,  $p = 0.430$ ).

However, moving on to *Approach*, there was a significant interaction effect of *Approach* by stimulus valence ( $\gamma_{50} = 0.05$ ,  $p = 0.04$ ), as well as a significant 3-interaction according to group ( $\gamma_{51} = -0.03$ ,  $p = 0.03$ ), which is consistent with the primary hypothesis that the correspondence of *Approach* facial behavior and RSA would be moderated by both stimulus valence condition type and group status. For a summary of the results of the primary model see Table 12.

Next, analyses of the simple effects of the significant 3-way interaction of the *Approach* facial affective behaviors by stimulus valence condition type dummy code by ADHD group status were completed. Specifically, the association between *Approach* and RSA was stronger for the typically developing group during positive induction ( $\gamma_{10} = -0.10$ ) than during negative induction ( $\gamma_{20} = 0.02$ ;  $t(540) = 2.15$ ,  $p = 0.03$ ). However, there was no

significant difference in correspondence between the positive ( $\gamma_{10}=0.08$ ) and negative induction conditions for the ADHD group ( $\gamma_{20}=0.04$ ;  $t(540)=1.15$ ,  $p=0.23$ ).

Additionally, there was a significant difference between groups in the association between *Approach* and RSA during the position induction condition ( $t(540)=2.05$ ,  $p=0.04$ ). Specifically, during the positive induction condition, the control children showed a negative association between *Approach* facial behavior and RSA ( $\gamma_{10}=-0.10$ ), while the ADHD group showed a positive association between *Approach* facial behavior and RSA ( $\gamma_{10}=0.08$ ). Thus, it appears that during the experience of positive emotions, typically developing children experience RSA withdrawal during the expression of *Approach*-based emotions, while children with ADHD display RSA increase during the expression of *Approach*-based emotions, see Figure 10.

### *Model 3 for PEP*

When the same model was examined, but with PEP as the outcome, children with ADHD displayed elevated PEP across task conditions when compared to control children ( $\gamma_{01}=-2.07$ ,  $p<0.03$ ; see also Tables 11 and 12). However, no significant main effect of *Avoidance* or *Approach* facial behavior was observed on PEP ( $\gamma_{20}=0.32$ ,  $p=0.229$  and  $\gamma_{20}=-0.48$ ,  $p=0.313$ , respectively), nor was there a significant interaction of either of these facial affective behaviors by group ( $\gamma_{11}=-0.48$ ,  $p=0.606$  and  $\gamma_{21}=0.163$ ,  $p=0.512$ , respectively). Furthermore, there was no significant main effect of stimulus valence condition ( $\gamma_{30}=0.23$ ,  $p=0.547$ ); nor was there a significant group by stimulus valence condition type interaction ( $\gamma_{20}=-0.14$ ,  $p=0.03$ ), which is explored in more detail below (see PEP follow-up contrasts and Table 11).

Finally, the primary hypotheses for PEP were examined. Specifically, it was hypothesized that children with ADHD would show reduced correspondence between PEP and *Approach* during positive induction and *Avoidance* during negative induction when compared to controls. Beginning with *Avoidance*, there was no significant effect of *Avoidance* by stimulus valence condition type on PEP ( $\gamma_{60} < 0.10$ ,  $p = 0.429$ ); however, there was a significant 3-way interaction with group ( $\gamma_{61} = 0.18$ ,  $p = 0.04$ ), which is consistent with the primary hypothesis that the correspondence of *Avoidance* facial behavior and PEP would be moderated by both stimulus valence condition type and group status (see Table 12), and the nature of this interaction is explored below (see PEP follow-up contrasts and Table 12).

Simple effects were examined for this significant 3-way interaction next. Specifically, the association between *Avoidance* and PEP was stronger for the typically developing group during negative induction ( $\gamma_{10} = -0.80$ ) than during positive induction ( $\gamma_{20} = -0.09$ ;  $t(540) = 2.15$ ,  $p = 0.03$ ). However, there was no statistically significant difference in correspondence between the positive ( $\gamma_{10} = 0.13$ ) and negative induction conditions for the ADHD group ( $\gamma_{20} = 0.09$ ;  $t(540) = 0.95$ ,  $p = 0.46$ ), see Figure 11. Additionally, there was a significantly greater association between *Avoidance* and PEP during the negative condition for the typically developing group ( $\gamma_{10} = -0.80$ ) than for the ADHD group ( $\gamma_{20} = 0.09$ ;  $t(540) = 1.15$ ,  $p = 0.24$ ).

There was also a significant interaction effect of *Approach* by stimulus valence condition type ( $\gamma_{50} = -0.53$ ,  $p = 0.04$ ), as well as a significant 3-way interaction with group ( $\gamma_{51} = 0.39$ ,  $p = 0.03$ ), which is consistent with the primary hypothesis that the

correspondence of *Approach* facial behavior and PEP would be moderated by both stimulus valence condition type and group status (see Table 12).

Analyses of the simple effects of the interactions of the *Approach* facial affective behaviors by stimulus valence condition type dummy code by group were completed. Specifically, the association between *Approach* and PEP was stronger for the typically developing group during positive induction ( $\gamma_{10}=1.40$ ) than during negative induction ( $\gamma_{20}=0.42$ ;  $t(540)=3.15$ ,  $p<0.01$ ). However, there was no statistically significant difference in correspondence between the positive ( $\gamma_{10}=-1.02$ ) and negative induction conditions for the ADHD group ( $\gamma_{20}=-0.54$ ;  $t(540)=1.05$ ,  $p=0.26$ ).

Again, it was interesting to note that there was also a significant difference between groups in the association between *Approach* and PEP during the position induction condition ( $t(540)=3.15$ ,  $p<0.01$ ). Specifically, during the positive induction condition, the control children showed a positive association between *Approach* facial behavior and PEP ( $\gamma_{10}=1.4$ ), while the ADHD group showed a negative association between *Approach* facial behavior and PEP ( $\gamma_{10}=-1.02$ ). Thus, it appears that during the experience of positive emotions, typically developing children experience increases in PEP during the expression of *Approach*-based emotions, while children with ADHD display decreases in PEP during the expression of *Approach*-based emotions, see Figure 11.

#### *Effects of Callous/Unemotional Behavior*

It should be noted that each of these models (i.e., Models 1-3) were also rerun with continuous scores of the callous/unemotional trait variable from Study 1 treated as a

Level 2 covariate, in order to rule out these effects being due to the levels of this trait in the Study 2 sample.

For example, Model 1 with callous/unemotional traits was as follows:

$$\textbf{Level-1: } \text{AFFECT RATING}_{ij} = \beta_{0j} + \beta_{0j} (\text{TIME}_{ij}) + r_{ij}$$

$$\textbf{Level-2: } \begin{aligned} \beta_{0j} &= \gamma_{00} + \gamma_{01}(\text{Group}_j) + \gamma_{02}(\text{CU}_j) + u_{0j} \\ \beta_{1j} &= \gamma_{10} + \gamma_{11}(\text{Group}_j) + \gamma_{12}(\text{CU}_j) + u_{1j} \end{aligned}$$

As in Model 1, at Level 1, the repeated observations for each participant were modeled. Specifically,  $\text{AFFECT RATING}_{ij}$  represents the eight repeated observations of facial affect (*Approach* or *Avoidance*, with  $\text{TIME}_{ij}$  representing the continuous observations of one per 30 seconds across each of the two minutes of the four task conditions) for each participant. The  $\text{AFFECT RATING}_{ij}$  variable represents the *Approach* and *Avoidance* factors assessed individually in two separate models with the above structure.

At Level 2, group comparisons were conducted using a dummy coded time invariant  $\text{GROUP}_j$  variable (1=ADHD, -1=control), and  $\text{CU}_j$  was a continuous covariate representing the overall level of callous/unemotional behavior in each participant.

However, the inclusion of CU level in Models 1, 2, or 3 did not affect any of the primary results. Full results with callous/unemotional level as a covariate are available upon request from the author.

### ***Discussion***

Study 2 examined the correspondence of physiological and facial affective indices of emotion regulation in children with and without ADHD. However, prior to examining the primary hypotheses for Study 2, an analysis of the facial affective behavior coding

(i.e., the six emotions coded across the task conditions, including anger, anxiety, fear, happiness, sadness, and surprise), revealed a two factor structure labeled as (a) *Approach* (happiness and anger) and (b) *Avoidance* (anxiety and sadness). This was similar to previous literature on the functional nature of emotion (Beauchaine, 2001; Ekman, 1992a, 1992b; Kring et al., 1993; Levenson, 1994; Mauss et al., 2005).

It was hypothesized the children with ADHD would show reduced amounts of all facial affect across all task conditions. However, this was not observed. This was somewhat surprising as previous research has suggested that children with ADHD and other disruptive behavior disorders have difficulty with emotional empathy (Eisenberg et al., 1997; Zahn-Waxler, Cole, Welsh, & Fox, 1995). However, these previous studies have mostly relied on self-report, rather than facial affective coding. Furthermore, these results were congruent with the results of Study 1 for the larger sample, as both Study 1 and this study did not find evidence of group differences in facial affective behavior.

Additionally, children with ADHD were hypothesized to display increased parasympathetic activity (i.e., increased RSA) and sympathetic activity (i.e., shortened PEP) across all task conditions. This hypothesis was supported, and the results of this analysis were congruent with the results reported in Study 1. Children with ADHD displayed higher RSA and lower PEP than non-ADHD youth. These results support the primary hypotheses of study 1, while showing that the subsample utilized in the primary analyses of Study 2 displayed the same overall results observed in Study 1. That is, the finding that facial affective behavior did not differ between the ADHD and control groups suggests that the emotion induction and suppression task was not differentially successful for both groups, and the results of Study 2 are not better explained by effects

associated with lack of compliance or engagement in the task by the ADHD group. Additionally, the group differences in RSA and PEP suggest group differences in emotion regulation and emotional arousal, respectively.

The primary hypotheses of this study were rooted in functionalist theory. In particular, it was hypothesized that there would be a statistical correspondence among the measures of emotion and its regulation, during induction, but not during suppression. This hypothesis was supported. Specifically, it was determined that increases in both avoidance and approach facial affective behavior were associated with increased RSA and shortened PEP during induction when examined across the full sample. However, these associations were diminished significantly during the suppression condition when examined across the full sample. These results are similar to those reported by Mauss et al. (2005) in adults.

Further, it was hypothesized that children with ADHD would show weaker correspondence among measures of emotion regulation including autonomic reactivity and facial emotionally expressive behavior, whereas typically developing children would show developmentally- and task-appropriate levels of correspondence among these measures during induction of both negative and positive emotions. Functionalist theories of emotion propose that synchrony among emotional response systems is associated with emotional health, while desynchronization of these systems contributes to the development and maintenance of psychopathology (Ekman, 1992a; Mauss, Evers, Wilhelm, & Gross, 2006; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Wilhelm, Grossman, & Roth, 2005). This set of hypotheses was also supported. Specifically, typically developing children showed greater correspondence between

approach emotions and both RSA and PEP during positive induction than children with ADHD. Additionally, typically developing children showed greater correspondence between avoidance emotions and PEP during negative induction.

Additionally, it was hypothesized that reduced correspondence between approach and ANS activity (both RSA and PEP) would be more salient in the positive induction condition, while reduced correspondence between avoidance and ANS activity would be more salient in the negative induction condition (for both RSA and PEP). Several interesting results emerged. Specifically, during the positive emotion induction condition, facial expressions of approach-based emotions were associated with PNS withdrawal (i.e., decreased RSA) and decreased SNS activity (i.e., lengthened PEP) for typically developing children. However, the opposite pattern emerged for children with ADHD, such that approach-based emotions were associated with increased PNS activity (i.e., increased RSA) and increased SNS activity (i.e., shortened PEP). Furthermore, unlike the typically developing controls, there were no statistically significant differences in the association between approach and RSA (or approach and PEP) during the positive or negative induction conditions. Furthermore, unlike typically developing children, children with ADHD showed no significant differences in the association between avoidance and PEP during the negative and positive conditions. Specifically, typically developing children showed a significantly greater association between avoidance and PEP during the negative induction condition than they did during the positive induction condition or than the children with ADHD did across either valence condition. Additionally, there was a statistical correspondence between avoidance-based emotions and RSA across the induction conditions for the typically developing children; however,

this association was reduced significantly among children with ADHD. Specifically, for typically developing children avoidance emotions were associated with increased RSA, while avoidance emotions were associated with RSA withdrawal for children with ADHD.

As mentioned above, these results cannot be attributed to a lack of emotional responding among the children with ADHD or lack of engagement in the task, given that both groups displayed similar levels of both approach and avoidance facial affective behavior throughout the task, and given that inclusion of approach and avoidance affective behavior in the models did not affect the overall results. Additionally, as outlined in study 1, both groups displayed significant changes in both RSA and PEP from baseline, suggesting a physiological reaction to the emotional and regulation demands of the task. Thus, one way to interpret these findings is that when children with ADHD communicate via facial affective behaviors of approach or avoidance emotions, their autonomic nervous systems are not responding adaptively to promote physiological homeostasis or to promote social engagement behaviors (Porges, 1995, 1997, 2001). Furthermore, these results cannot be explained by child gender, the use of stimulant medications, or the presence of other psychological diagnoses, including ODD, CD, or anxiety, as each of these factors were covaried. Finally, the primary results of this study can also not be attributed to the differential levels of callous/unemotional traits observed between the ADHD and typically developing groups, as the inclusion of callous/unemotional behavior as a continuous covariate did not alter any of the primary results of this study. However, it should be noted that high levels of callous/unemotional behavior were associated with reduced avoidance-based facial affective behavior, as well

as longer PEP across task conditions, which is consistent with the results of Study 1.

Interestingly, in children with ADHD correspondence between facial and physiological indices of avoidance was diminished across task conditions, which was contrary to the expectation that it would be only in the negative emotion induction condition. However, given that RSA is believed to be a physiological index of emotion regulation abilities (Beauchaine, 2001; Beauchaine et al., 2007; Porges, 2001, 2007), discordance between RSA and avoidance-based facial affective behavior suggests that physiological dysregulation may play a central role in the inappropriate avoidance-based affect displayed by some children with ADHD. That is, this diminished association between physiological emotion regulation and avoidance-based affect irrespective of emotional context, may help to explain when some children with ADHD are prone to displays of inappropriate negative affect. Past studies have shown that, while in typically developing samples emotion expression is accompanied by changes in RSA, children with disruptive behavior disorders and adults with antisocial behavior display aberrant patterns of RSA and RSA reactivity (Beauchaine et al., 2001, 2007; Marsh et al., 2008; Mauss et al., 2005). The present study extends this study to children with ADHD, showing that they tend to display a reduction in the coordinated changes in RSA which tends to be associated with facial reactions of avoidant behavior among typically developing children. With decoupled physiological and behavioral responses to affective challenges, children with ADHD may lack resources to effectively modulate physiological emotional arousal, resulting in inappropriate displays of affect, as well as reduced ability to respond appropriately to the emotional displays of others (Fabes et al, 1994). A similar picture was observed for PEP, during the negative induction condition

only, with avoidance-behaviors being associated with increased PEP for the control children only, while the ADHD children displayed reduced coordination between avoidance-behaviors and PEP during the negative induction condition.

Differences in the correspondence between facial expressions of approach emotions and both parasympathetic and sympathetic activity during the induction of positive emotions were also observed between children with and without ADHD. With respect to PEP, the control group displayed increases in PEP associated with the display of approach-based emotions, while this association was diminished among the ADHD group, as shown by the significant 3-way interaction of approach by positive induction by group. However, of particular interest, while typically developing children displayed little association between RSA and facial displays of approach emotion, children with ADHD displayed significant increases in RSA in response to approach emotions, as shown by the significant 3-way interaction of approach by positive induction by group. This is congruent with theories that have suggested that unlike children with other forms of disruptive behavior disorders, children with ADHD may misinterpret approach (or positive emotions) and treat them as something aversive, which needs to be regulated (Braaten et al., 2000; Cohen & Strayer, 1996; Izard et al., 2001; Shin et al., 2009). Additionally, these results are supportive of several theories of the roles of temperament in ADHD have suggested that disruptions in the approach (or positive emotion domain) may be particularly salient to ADHD (Martel, 2009; Nigg et al., 2005).

Together, these results replicate prior research with adults suggesting that correspondence among emotional response systems are adaptive (Mauss et al., 2005). Additionally, these results extend those reporting disruptions in the coherence between

autonomic activity and negative emotions among boys with disruptive behavior disorder (Marsh et al., 2008). As with study 1, examining these systems separately may also be indicative of disruptions of autonomic activity; however, no differences in facial affective behavior emerged, which is illustrative of the importance of exploring multiple measures of emotional reactivity concurrently. Furthermore, deficits in the coordination of facial affect and both PNS and SNS activity were observed, which also illustrates the importance of examining multiple physiological systems of emotion and its regulation. Overall, these results indicate that in addition to the disruptions in autonomic nervous system activity observed in Study 1, this system may not be efficiently or appropriately coupled with facial affect both in negative and positive emotional domains. These findings are consistent with the functionalist theory of emotion, which states that coordination across emotional response systems should be associated with psychological adjustment and adaptive social responding.

While Study 2 advances understanding of the association between facial affective behavior in avoidance- and approach-based emotions with autonomic nervous system reactivity, there are several limitations to consider. First, the sample was too small to examine additional moderating factors, such as ADHD subtype, and the study was cross-sectional in design. Thus, additional longitudinal studies are needed to examine how patterns of response coherence among these emotional systems changes across childhood and into adolescence. Additionally, studies of young children may reveal important information about the nature of these associations with genetic, environmental, and dually influenced factors. That is, it maybe that over time children with ADHD may learn to misidentify emotional signals or they may be predisposed to this type of reduced

correspondence genetically, which can only be elucidated with longitudinal studies of infants and very young children. Second, the stimuli used to elicit “negative” and “positive” valenced emotions were somewhat generic. While the majority of children rated the negative clip as eliciting sadness and the majority of children rated the positive clip as eliciting happiness, there were some individual differences in these reports, though no significant differences emerged according to group. Using emotional stimuli designed to elicit more “pure” and specific emotions may yield different results (Levenson, 1992). Additionally, eliciting other approach, but “negative” emotions, particularly anger, may further elucidate issues related to coherence among children with ADHD. Third, while this study focused on a specific disorder, ADHD, it may be that disruptions in the coherence among distinct emotional systems may provide information about other forms of psychopathology, as well. In particular, work by Marsh and colleagues (2008) found that disruptions in the coherence between facial sadness and RSA were uniquely associated with internalizing symptoms, while these disruptions with PEP were uniquely associated with externalizing symptoms. While the current study did rule out explanatory power of ODD, CD, and anxiety symptoms in the primary hypotheses of interest, the sample size may not have allowed for statistical power to fully examine these associations. Future studies may wish to examine correspondence in other emotional response systems, as well, such as self-report measures or central nervous system activity. Finally, a time-lagged analysis may reveal alternate patterns of response coherence. For example, the children with ADHD may exhibit slowed physiological responses to emotional stimuli.

While Studies 1 and 2 explored the roles of physiological emotional arousal and

regulation of emotion in specific groups of children with ADHD and role of coherence among of multiple emotional systems in ADHD, it is established that emotion regulation develops in context. Specifically, parenting behavior and emotion has the potential to shape autonomic arousal and regulatory systems in children. Next, in Study 3, the roles of parenting behavior and expressed emotion in the physiological regulation of emotion in children with and without ADHD are explored.

## CHAPTER IV

### STUDY 3: PARENTING FACTORS IN EMOTION REGULATION IN ADHD

#### *Methods*

#### **Summary**

To address the main hypotheses of Study 3, a sub-sample of 100 of the 150 participants from Study 1 were used. However, these were not necessarily the same 100 participants from Study 2 (72% overlap between Studies 2 and 3). The specific hypotheses included that autonomic nervous system activity would mediate the association between parenting behavior and number of ADHD symptoms, child participants along with a parent completed a parent-child interaction task designed to require cooperation, negotiation, and to elicit mild levels of frustration. Continuous electrocardiogram and impedance cardiography recordings were collected across task conditions. Parent behavior was coded from these video-taped interactions. Additionally, parents of child participants completed a five minute speech sample, coded for parental expressed emotion. Parental sensitivity and expressed emotion were examined as possible predictors of child autonomic activity. Child indexes of parasympathetic and sympathetic nervous system functioning, during the task, were used to predict ADHD and comorbid behavior problem symptoms. Additionally, child parasympathetic and sympathetic nervous system activity were examined as possible mediators and moderators of the association between parent sensitivity/expressed emotion and ADHD and comorbid symptoms.

It should be noted that given the important role of CU traits in influencing psychophysiological responses observed in Study 1, CU traits were also treated as a covariate in all of the primary analyses of this study just as they were in Study 2.

However, no a priori hypotheses were made regarding the nature of the effects expected in association with callous/unemotional traits in these analyses. Again, a table providing a list of assessment measures for screening and diagnosis, Study 1, Study 2, and Study 3 is provided (Table 1, page 123).

## **Participants**

Study 3 includes another, separate subset of 100 participants from the original 150 participants in Study 1 (ADHD, N=50; control N=50; 72% overlap with Study 2). This study evaluates parenting behavior and expressed emotion in relation to children's physiological emotion regulation in children with ADHD. The remaining procedures were mostly identical therefore to Study 1, as summarized next.

## **Procedures**

The procedures for Study 3 are described as follows.

### *Recruitment and Identification Procedures*

For a full description of Study 3 recruitment and identification procedures, see Study 1, page 23.

### *Final ADHD and Other Diagnoses*

For a full description of Study 3 ADHD and other diagnostic procedures, see Study 1, page 24.

*Identification of the Callous/Unemotional Traits.* For a full description of Study 3 assessment of the callous/unemotional trait, see Study 1, page 24. However, note that group assignments were not made in Study 3, but rather, the continuous scores from the reverse coded Prosocial Scale of the Strengths and Difficulties questionnaire were utilized. Specifically, this continuous score was utilized as a covariate in all of the

primary analyses of Study 3 to rule out the explanation that the presence of callous/unemotional traits explained the primary results of Study 3.

*Exclusion Criteria.* For a full description of Study 3 exclusion criteria, see Study 1, page 25.

#### *Medication Washout*

For a full description of Study 3 medication washout procedures, see Study 1, page 26.

#### *Parent-Child Interaction Task Procedure*

Children were also video-taped with a parent (the child's biological mother) as they completed two five-minute tasks designed by Deater-Deckard (Deater-Deckard & O'Connor, 2000). The tasks have been structured to require cooperation while inducing mild to moderate levels of frustration. In the first task, the mother and child were instructed to copy a line-drawing of a house using a toy Etch-A-Sketch (a drawing toy with two dials, one each to draw horizontal and vertical lines). The mother was instructed to use only the vertical dial, and the child was instructed to use only the horizontal dial. To complete the drawing within the five-minute time limit, both parties must collaborate. They were told that most families complete the task within five minutes. In the second half of the task, the mother and child completed a more challenging version of the task, in which they were asked to move a marble through a tilting-maze with dead ends and holes through which the marble could drop, requiring the dyad to begin again. The tilting and maneuvering action was again controlled by two dials, one of which was controlled by the mother and the other of which was controlled by the child. Again, the dyad was told that most families complete the maze task in the five minute limit; however, in reality the

task is meant to be quite challenging and nearly impossible to finish within five minutes. Parents and children were debriefed about the nature of these tasks after they completed participation.

Both parent-child interaction tasks were coded by two blinded research assistants trained by the applicant for reliability, using the Parent-Child Interaction System (PARCHISY; (Deater-Deckard, Pylas, & Petrill, 1997). This coding system includes global ratings on a seven-point scale. Previous studies using this coding system have developed sub-constructs to examine parenting and child behavior, and this study intends to do the same. Specifically, (Deater-Deckard & O'Connor, 2000) included a composite of the parent responsiveness/sensitivity to child and parental intrusiveness domains. Two child behavior problems composite based on child noncompliance and lack of engagement were also coded.

Thirty percent of the parent-child interactions were coded for reliability, which was determined to be acceptable for all variables coded,  $k > .85$ , for all parent, child, and dyadic codes. This coding system has been validated with global reliabilities of  $\alpha = .87$  (Deater-Deckard, 2000; Deater-Deckard & Petrill, 2004).

#### *Five Minute Speech Sample and Expressed Emotion Coding*

The FMSS is a measure which requires parents to describe their child and their relationship with their child during a 5-minute, recorded monologue. They are instructed to talk about their child in their own words, without interruption. The FMSS was audio-recorded and coded with respect to both content and emotional tone, with ratings that include both the involvement of the parent as derived from emotions, feelings and

attitudes expressed in the monologue and the parent's level of criticism of the child (Magana et al., 1986).

Two, independent raters coded the FMSS for expressed emotion. Specifically, the two raters were Sybil Zaden, Ph.D. (a senior criterion rater in the nation) and Tara Peris, Ph.D. (a reliable doctoral level researcher) at the University of California; Los Angeles Medical Center, who have been coding the FMSS in this manner for over ten years. Full training in FMSS coding requires passing a training in which an overall reliability rating (across all dimensions) of  $r=.90$  is reached.

The final classifications could be either High EE, high-level of emotional expression, characterized by an excessive presence or intensity of the emotions, often beyond the control of the subject and scarcely modulated; or, Low EE, low level of expressed emotion, characterized by a well-modulated and balanced level of communicated emotion. The FMSS measure comprises two distinct components: criticism (CRIT) and emotional over-involvement (EOI) that are subsumed under the more general labels of High EE and Low EE. For this sample, 10% of all tapes were coded by both raters, disagreements were resolved via conference. For this sample, percentages of agreement were 92% ( $k=.81$ ) for the overall final score, 87% ( $k=.78$ ) for Criticism, and 84% ( $k=.74$ ) for emotional over-involvement.

#### *Physiological Recording*

These were the same as Study 1, as described on page 28.

## *Data Analysis*

### **Analytic Plan**

Mediation was evaluated by estimating confidence intervals around the hypothesized indirect effect, using regression-based path analysis (Preacher & Hayes, 2004). This procedure yields a path model that directly estimates the significance of the indirect effect for relatively small samples (Preacher et al., 2004). It therefore is similar to but more powerful than other procedures (e.g., Baron & Kenny, 1986).

Specifically, to determine whether mediation is present regression-based path analysis using the methods outlined by Preacher and Hayes (2004) was used (**Figure 2**). This method is similar to the procedures outlined by Baron and Kenny (1986), as it uses regression to determine the total effect of the independent variable on the dependent variable (path c in Figure 2), the effect of the independent variable on the proposed mediator (path a in Figure 2), the effect of the proposed mediator on the dependent variable, while controlling for the independent variable (path b in Figure 2), as well as the direct effect of the independent variable on the dependent variable, while controlling for the mediator ( $c'$  in Figure 2). However, this method also estimates the indirect effect of parenting behavior on ADHD symptoms through ANS activity (both a and b in Figure 2) as well as the change of the association between parenting behavior on ADHD symptoms with ANS activity in the model ( $c-c'$  in Figure 2), and this method results in a 95% confidence interval for the size of this indirect effect using a bootstrapping procedure. The SPSS macro provided in Preacher and Hayes (2004) was utilized.

It should be noted where the preconditions for mediational analyses were not present per the suggestions made by Kraemer et al. (2001), moderation and/or independent

predictors analyses were conducted. Specifically, moderation was tested using the procedures proposed by Preacher and Matthes (2009) and Preacher (2012), while the independent nature of the association of these predictors was assessed using a multivariate regression model. Specifically, a single regression predicting ADHD severity by parent expressed emotion, parent behavior during the parent-child interaction task, and child psychophysiological indexes (i.e., RSA and PEP) was assessed. Then, each of these groups of predictors was tested individually in three separate models predicting ADHD severity.

### **Power Calculations**

As the Preacher & Hayes (2004) approach expands upon the methods used by Baron & Kenny (1986; a multi-step multiple regression approach) with the addition of bootstrapping, a multiple regression approach to power calculation was utilized. G\*Power (Faul, Erdfelder, Lang, & Buchner, 2007) was used to determine sample size. With a Cohen's *d* of .5, which is considered to be a medium effect size (Cohen, 1992), a total sample size of 87 participants was required for adequate (.90) power to detect a significant group differences ( $p < 0.05$ ) in each of the proposed regression paths.

### ***Results***

#### **Preliminary Analyses**

##### *Descriptive and Diagnostic Overview of Sample*

As this is a different sub-sample of 100 of the original 150 participants from Study 1 and not identical to the sub-set from Study 2, descriptive and diagnostic statistics are reported by group in Table 13 for this sub-sample. Preliminary analyses examined the significance of group differences in each of the demographic, diagnostic, and

psychopathology scales using analyses of variance (ANOVA) or Chi-square analyses, as appropriate. Effects sizes (partial eta-squared) are also reported (Table 13).

Based on these analyses, groups did not differ with respect to age, race, parent marital status, or IQ. Inclusion of these variables as covariates did not affect results reported. Results are therefore reported without these variables treated as covariates. However, groups differed in gender ratio (more boys in the ADHD group than the control group) and unsurprisingly the use of stimulant medication (with the use of stimulant medication being significantly higher in the ADHD group). However, gender and stimulant use were each unrelated to parent behavior and child physiological parameters. Additionally, when covaried, neither stimulant use nor gender affected the primary study results, and therefore, these variables were not included in the final reported results.

Clinical characteristics are also provided in **Table 13**. Additionally, these characteristics were not fully identical to those in Studies 1 or 2, and so, are presented here. As expected, the ADHD group differed significantly from the control group on the parent and teacher rated problems on all clinical T-scored scales of the ADHD-RS (i.e., Hyperactivity/Impulsivity, Inattention, Total symptoms) and the Conner's 3<sup>rd</sup> edition (i.e., Inattention, Hyperactivity, Learning Problems, Aggression, and Peer Relations). Furthermore, the control and ADHD groups differed significantly on each of the parent and teacher SDQ scales (i.e., conduct problems, emotion problems, hyperactivity, peer problems, total difficulties, and impact scales; see Table 13).

With regard to clinical comorbidity, as shown in **Table 13**, groups did not differ in the presence of lifetime mood or conduct disorder or current anxiety disorder. However, the ADHD group displayed significantly higher rates of oppositional defiant

disorder than the control group. The inclusion of comorbid disorders as covariates (individually or collectively) did not affect any of the main study results. The ADHD group also displayed significantly larger number of anxiety, conduct, and oppositional defiant disorder symptoms than the control group. Additional examinations of total ODD, CD, and anxiety symptoms (all from the KSADS-E) are reported below.

#### *Data Reduction for Primary Analyses*

An exploratory principal components analysis was first performed to dimensionality of the PARCHISY ratings of the eight parenting behaviors which coded during the two task conditions (Etch-A-Sketch and Marble Maze). The standard deviations of the originally coded variables are presented in Table 14. set was determined to be bi-dimensional for this sample. This interpretation was supported by the fact that the first eigenvalue was 4.194, while the second was with all subsequent eigenvalues being less than 0.80. Four items loaded on the factor, with loadings ranging from 0.69 to 0.85 for Parent Responsiveness, Positive Control, Parent Reciprocity, and Parent Cooperation, respectively. Negative Control, Parent Negative Affect, and Parent Conflict loaded more highly second factor, which loaded at 0.75, 0.81 and 0.85, respectively. As Parent Affect had a low factor-loading, this item was removed from the final solution. factor solution was utilized in the subsequent mediation and moderation analyses. The factors were labeled as *Sensitive Parenting* or parental warmth (Positive Control, Responsiveness, Reciprocity, and Cooperation) and *Intrusive Behavior* or parental negativity (Negative Content, Negative Affect, and Conflict), were correlated at  $r=.431$ ,  $p<0.05$ . These factors have also emerged in previous

theoretical and empirical examinations of parenting behavior (Murray et al., 1999, Musser et al., 2009), as well as in factor analyses of the PARCHISY codes (Deater-Deckard, Petrill, & Thompson, 2009). As such, these factors were used in subsequent analyses.

A similar analysis was completed to examine the dimensionality of the PARCHISY ratings of the six child behaviors which were coded during the two task conditions (Etch-A-Sketch and Marble Maze). This item set also tended to be bi-dimensional for this sample. This interpretation was supported by the fact that the first eigenvalue was 2.182, while the second was 1.267, with all subsequent eigenvalues being less than 0.70. Four items loaded on the first factor, with loadings ranging from 0.55 to 0.86 for Child Positive Affect, Child Negative Affect, Child Responsiveness, and Child Compliance. Child Autonomy and Child Activity loaded more highly on the second factor, which loaded at 0.76 and 0.81, respectively. As Child Cooperation and On Task behavior had low factor-loadings, these items were removed from the final solution. Thus, a 2-factor solution was utilized in the subsequent analyses. The two factors were labeled as *Agreeableness* (e.g., Child Positive Affect, Reversely Coded Child Negative Affect, Child Responsiveness, and Child Compliance) and *Engagement* (Child Autonomy and Child On Task Behavior), which were correlated at  $r=.372$ ,  $p<0.05$  (see Table 14). These factors were used as covariates in all relevant subsequent analyses (i.e., those involving the *Sensitive Parent Behavior* and *Intrusive Parenting* subscales). As a primary score of child behavior, as well as a secondary classification of low, borderline, or high on both Criticism and

Over-involvement, are built into the FMSS Expressed Emotion coding solutions, scores were utilized in all subsequent analyses involving parenting affective and no further data reduction was completed. Percentages of each expressed domain coded are presented by group in Table 15.

### *Baseline Effects*

During the two minute Resting Baseline there were no significant differences in autonomic activity for interbeat interval, respiration rate, or RSA (all  $F_s < 1.10$ ,  $p > 0.10$ , see Table 16). However, there was a significant group difference in PEP at baseline, with the ADHD group showing significantly lower PEP than the control group ( $F=6.99$ ,  $p < 0.01$ ).

### *Examination of Task Condition Differences*

2\*2 repeated measures ANOVA was used to examine group- and task-differences, as well as the interaction of group\*task-based differences in physiological measures; the same approach was used for the coded parent (i.e., *Sensitive* and *Intrusive Parent Behavior*) and child behavior (i.e., *Agreeableness* and *Engagement*). This analysis was completed in order to examine whether there were differences in autonomic and behavioral variables according to task-demands (as the Etch-A-Sketch portion is designed to induce less stress and frustration than the Marble Maze portion).

Physiological indices were examined as outcome variables first. There was a significant main-effect of group on PEP ( $F=4.174$ ,  $p < 0.01$ ) with children with ADHD displaying shorter PEP (i.e., elevated sympathetic activation) than control children. However, all other effects of condition, group, and interactive effects related to PEP were non-significant ( $F < 1.2$ ,  $p > 0.05$ ; see Table 16). None of the RSA effects were significant

(all  $F < 1.5$ , all  $p > 0.05$ ; see Table 16).

Parental indices were examined next. There was a significant main-effect of task condition on both parental *Sensitive Behavior* and *Intrusive Behavior* ( $F = 81.92$ ,  $p < 0.001$  and  $F = 140.50$ ,  $p < 0.001$ , respectively), as well as significant main effects of group on parent behavior ( $F = 7.26$ ,  $p < 0.01$  and  $F = 3.86$ ,  $p < 0.05$ , respectively). However, there was not a significant interaction of group by task condition ( $F = 1.45$ ,  $p > 0.06$  and  $F = 2.34$ ,  $p > 0.05$ , respectively, for *Sensitive* and *Intrusive Behavior*).

Child behavior was examined third. There was a significant main-effect of task condition on both child *Agreeableness* and *Engagement* ( $F = 69.26$ ,  $p < 0.001$  and  $F = 31.07$ ,  $p < 0.001$ , respectively). However, there was no significant main-effect of group, nor was there a significant interaction effect (all  $F > 1.13$ , all  $p > 0.05$ , see Table 14). Thus, as the parenting and child behaviors were determined to differ according to task condition, but without a significant interaction of group by task condition, only the Marble Maze results are reported in the subsequent mediation and moderation analyses, as this task pulled for more challenging child behavior and the need for more sensitive parenting. However, results of the analyses using data from the Etch-A-Sketch portion of the task are available upon request from the author.

#### *Examination of the Prerequisites for Mediation and Moderation*

Correlational analyses were used to examine associations among (a) levels of expressed emotion (low vs high, criticism, and emotional over-involvement), (b) levels of parenting behavior during the Marble Maze (*Sensitive Behavior*, *Intrusive Behavior*), (c) indexes of ANS activity (RSA, PEP), (d) the severity of ADHD inattention and hyperactive symptoms (inattentive, hyperactive/impulsive, total ADHD symptoms T-

scores from ADHD-RS), (e) ODD and CD symptom counts from the K-SADS E, as well as (f) diagnostic-team diagnosis of ADHD (i.e., ADHD group status). This analysis ensured that any subsequent analyses of mediation and/or moderation were warranted given the recommendations of Kramer, Stice, Kazdin, Offord, and Kupfer (2001). Specifically, it was initially hypothesized that child RSA (and PEP) levels during the Marble Maze task would mediate the association between parental expressed emotion (and behavior during the task) with ADHD symptoms outside the task (see Figure 2). However, as with all mediational models, several important caveats had to be considered prior to completing these analyses.

In particular, Kraemar et al. (2001) suggest that mediational models must meet several criteria. The first criterion, temporal precedence, states that the mediated factor must occur prior to the mediating factor in time. The second criterion, correlation, states that the two predictors in the model must be correlated with one another. The final criterion, domination, states that for full mediation to be present the association between the predictor and the criterion must be accounted for fully by the mediated factor. Temporal precedence of the predictors being tested in this study could not be established with complete certainty, as they were assessed concurrently. However, in keeping with previous literature examining the temporal relationship between parenting behavior and child psychophysiological activity (Calkins et al., 1999, 2005; Porges et al., 1999, 2002), it was assumed that parenting behavior preceded physiological predispositions toward regulation (RSA) and arousal (PEP). However, no significant correlations (criterion 2) were observed between the parenting behaviors of interest and RSA or PEP (see Table 17). Thus, mediation could not be examined

With respect to potential moderation models, it was also possible that parenting expressed emotion (and behavior during the task) may operate as a moderator of the association between child ANS (RSA and PEP) response and ADHD symptoms. As proposed by Kraemer et al. (2001), moderation occurs when two predictors are uncorrelated (or when moderation is not simply due to such a correlation), as is the case for each of the parenting variables of interest (i.e., criticism, emotional over-involvement, *Sensitive Behavior*, and *Intrusive Behavior*) with neither RSA nor PEP (see Table 17). Additionally, Kraemer and colleagues (2001) argue that the moderator typically precedes the moderated construct, which was established above. As such the moderation models assessed whether high or low levels of each of the parental expressed emotion (criticism and emotional over-involvement) and behavior during the parent-child interaction task (*Sensitive* and *Intrusive Behavior*) variables may help to clarify for which children ANS dysregulation (RSA or PEP disruptions) is associated with ADHD symptoms. In this test of moderation (model 2), it was hypothesized that higher levels of parental expressed emotion on the FMSS (criticism and emotional over-involvement), as well as low levels of *Sensitive* and high levels of *Intrusive Behavior* during the parent-child task, would exacerbate the association between dysregulated ANS activity (high RSA, low PEP) and ADHD symptoms.

Additionally, it may be that there is neither a mediational nor a moderation-based relationship among these variables. In such a case, the predictors would not be correlated with one another, as in the moderation criteria. Thus, it may be that both parental expressed emotion (and task-based behavior) and ANS dysregulation are independently

associated with ADHD symptoms. Such a model was also examined as it was determined that moderation was not present.

Finally, correlations between the parenting expressed emotion and behavioral variables, as well as the ANS variables, and comorbid symptoms of ODD, CD, and anxiety were examined. This was completed in order to examine whether additional associations may exist between the predictors and specific presentations of ADHD (i.e., the number of ADHD symptoms; the presence of additional externalizing or internalizing symptoms). Results also indicated significant correlations of ODD symptoms with RSA and parental criticism ( $r=.344$ ,  $p<0.01$ ,  $r=.296$ ,  $p<0.01$ ) as well as between CD symptoms and criticism ( $r=.233$ ,  $p<0.05$ ). Finally, there was also a significant correlation between anxiety symptoms and overall expressed emotion ( $r=.244$ ,  $p<0.01$ ); however, the correlations for criticism and EOI were non-significant. Therefore, additional analyses examining ODD, CD, and anxiety symptoms as outcomes were also examined in the primary analyses below. Finally, as with Study 2, the important role of CU traits in influencing psychophysiological responses (i.e., CU traits, when treated both dimensionally and categorically, were associated with blunted parasympathetic and sympathetic nervous system activity across conditions of negative and positive emotion induction and suppression) observed in Study 1 should be noted. Thus, it was possible that CU traits would also affect the primary results of Study 3, as such CU traits were treated as a continuous covariate in all the analyses reported below. Furthermore, CU traits were treated as a continuous outcome variable when indicated by its predictive power in several of the independent association models.

### **Mediational Analysis**

Again, none of the parent expressed emotion (criticism or emotional over-involvement) or behavior (*Sensitive* or *Intrusive Behavior*) factors were correlated significantly with either ANS indicator (RSA or PEP; all  $r < 0.196$ , all  $p > 0.05$ ). Thus, mediation was not evaluated, as the preconditions proposed by Kraemer et al. (2001) were not met.

### **Moderation Analysis**

Using the methods outlined in Hayes and Matthes (2012), regression analyses were used to examine the main and interactive effects of parental expressed emotion (criticism and emotional over-involvement) then, in separate analyses parent behavior (*Sensitive* and *Intrusive Behavior*) and child RSA (then, in a separate analysis PEP) during the Marble Maze task on child ADHD symptoms (and in a separate analysis ADHD diagnosis).

Beginning with parent criticism as a moderator of the association of RSA during the Marble Maze task on child ADHD symptoms, from parent-report on the ADHD Rating Scale, the overall model explained 15.2% of the variance in ADHD symptoms ( $R^2 = 0.152$ ,  $p < 0.01$ ). However, the interaction effect of parent criticism and RSA was non-significant ( $\beta = 0.09$ ,  $p = 0.47$ ); thus, moderation was not present. When the same model was considered, but examining PEP rather than RSA during the Marble Maze Task, the overall model accounted for 17.2% of the variance in ADHD symptoms ( $R^2 = 0.172$ ,  $p < 0.003$ ). However, the interaction term of criticism and PEP was not a significant predictor of child ADHD symptoms ( $\beta = 0.07$ ,  $p = 0.67$ ) so again, moderation was not observed. Thus, while each of the overall models including parent criticism

explained a significant portion of the variance in ADHD symptoms, there was no evidence for moderation in either of the models examined.

A different picture emerged upon examining the other domain of expressed emotion, emotional over-involvement. Specifically, the overall model including parent emotional over-involvement and child RSA and the interaction term during the Marble Maze task did not significantly predict ADHD symptoms ( $R^2=0.031$ ,  $p=0.50$ ). When the same model was tested for child PEP during the Marble Maze task, again the overall model was non-significant ( $R^2=0.06$ ,  $p=0.21$ ). As such, it appears that parental emotional over-involvement is not a significant predictor of child ADHD symptoms, and there was no evidence for moderation in either of the models examined.

When similar models were tested examining parenting behavior during the Marble Maze task as a moderator of the association between ANS task-based reactivity and child ADHD symptoms, the models containing parental *Sensitive Behavior*, ANS activity (RSA and PEP, respectively), and the interactions of these factors were significant overall ( $R^2=0.124$ ,  $p<0.01$  and  $R^2=0.116$ ,  $p<0.01$ , respectively); however, neither the interaction term containing RSA nor the interaction term containing PEP were significant (all  $\beta<-0.19$  all  $p>0.43$ ). Similar results were observed for models including: parental *Intrusive Behavior*, RSA, and the interaction of *Intrusive Behavior*, and RSA ( $R^2=0.122$ ,  $p<0.01$ ). This was also the case for the model including parental *Intrusive Behavior*, PEP, and the interaction of *Intrusive Behavior* and PEP ( $R^2=0.126$ ,  $p<0.01$ ). However, again, neither the interaction term containing RSA, nor the interaction term containing PEP, were significant (all  $\beta<0.02$  all  $p>0.90$ ). Thus, while each of the overall models examining parenting behavior during the task and autonomic

functioning explained a significant portion of the variance in ADHD symptoms, there was no evidence for moderation in any of the models examined.

#### *Follow-up on Moderation Analyses with ADHD Diagnosis*

It should be noted that the pattern of overall results of the moderation analyses was the same whether ADHD was treated dimensionally (as above) or categorically (i.e., dividing the sample into control and ADHD groups based on diagnostic team conclusions). Specifically, when each of the moderation models were tested again, but with ADHD group status as the outcome variable instead of number of ADHD symptoms, each model examined explained a significant portion of the variance in ADHD group status (again with the exception of the models examining emotional over-involvement). However, as the interaction term was not a significant predictor of ADHD group status in any of the moderation models tested, moderation was determined not to be present in the case of ADHD group status, as well (all  $F < 1.0$ , all  $p > 0.34$ ). Full details of the analyses examining ADHD group status are available from the author upon request.

Additionally, it should also be noted that the inclusion of callous/unemotional behavior did not affect the results of any of the moderation analyses, all  $F$ -values for the interaction terms with callous/unemotional behavior in the model were less than 1.0 (all  $p > 0.29$ ). Furthermore, ODD, CD, or anxiety symptoms as covariates separately or together did not affect the results of any of the reported moderation analyses (all  $F < 1.0$ , all  $p > 0.19$ ). Finally, neither the inclusion of parent IQ, parent ADHD or depressive symptoms, nor number of words stated during the FMSS, nor the inclusion of either of

the child behavior factors (*Agreeableness* and *Engagement*) significantly altered the results of the moderation models reported (all  $F < 1.0$ , all  $p > 0.24$ ).

### **Independent Associations Models**

Finally, a model to examine the overall independent predictive power of parent criticism, emotional over-involvement, *Sensitive Behavior*, *Intrusive Behavior*, and child RSA or PEP on child ADHD symptoms was examined, as there were no moderation effects detected. The model including all of these individual predictors (as well as child *Agreeableness* and *Engagement* as covariates) explained a 20.4% of the variance in child ADHD symptoms ( $R^2 = 0.204$ ,  $p < 0.01$ ). However, only parent criticism remained a significant predictor of ADHD symptoms with all other predictors in the model (beta = 0.333,  $p < 0.01$  for parent criticism, with all other beta < 0.143, all other  $p > 0.34$ ).

Callous/unemotional behavior, ODD, CD, and anxiety symptoms were each added individually (and then collectively) to the overall model as covariates. The addition of these symptoms did not alter the results, as parent criticism remained a significant predictor of ADHD symptoms even after controlling for comorbid psychopathology symptoms and behaviors (beta = 0.281,  $p < 0.01$ ). In this model, both callous/unemotional behavior and ODD symptoms were also a significant predictors of ADHD symptoms (beta = -0.419,  $p < 0.01$ ; beta = 0.286,  $p = 0.01$ , respectively), with the full model including comorbid symptoms explaining a significant 38.3% of the variance in ADHD symptoms ( $R^2 = 0.383$ ,  $p < 0.001$ , see Figure 12). Additionally, when this same model was tested but with ADHD group status as the outcome (rather than number of ADHD symptoms, as above), again the full model including comorbid symptoms explained a significant 37.1% of the variance in ADHD group status ( $R^2 = 0.371$ ,  $p < 0.001$ ).

Additionally, it should be noted that when this same model was tested but with ADHD group status as the outcome (rather than number of ADHD symptoms, as above), a similar but somewhat different picture emerged. Specifically, the model with parent criticism, emotional over-involvement, *Sensitive Behavior*, *Intrusive Behavior*, and child RSA or PEP explained a significant 27.7% of the variance in ADHD group status ( $R^2=0.277$ ,  $p<0.001$ ), with the only significant predictors of ADHD group status being parent criticism and notably child PEP during the Marble Maze task (beta=0.259,  $p<0.05$ ; beta=0.936,  $p=0.01$ , respectively). Furthermore, the addition of ODD, CD, anxiety, and callous/unemotional symptoms did not alter the primary results of the model predicting ADHD group status, as parent criticism and child PEP during the maze task remained significant predictors of ADHD group status after controlling for comorbid psychopathology symptoms and behaviors (beta=0.231,  $p<0.05$  and beta=0.714,  $p<0.05$ , respectively). In this model, both callous/unemotional behavior and ODD symptoms were also a significant predictors of ADHD group status (beta=-0.459,  $p<0.01$ ; beta=0.316,  $p=0.01$ , respectively).

#### *Further Exploration of Independent Associations Models*

Next, as parent expressed emotion can be considered separate from the task, models including only parent criticism and emotional over-involvement and only parental *Sensitive Behavior* and *Intrusive Behavior* were considered. In the model including both parent expressed emotion factors (criticism and emotional over-involvement), a significant 14.5% of the variance in ADHD symptoms was explained ( $R^2=0.145$ ,  $p<0.01$ ), and again, only criticism was significant predictor (beta=.383,  $p<0.001$ ), while emotional over-involvement was not (beta=.0245,  $p=0.826$ ). When a similar model was examined,

but with ADHD group status as the outcome variable, a similar pattern was noted ( $R^2=0.117$ ,  $p<0.01$ ;  $\beta=.462$ ,  $p<0.001$ ,  $\beta=.178$ ,  $p=0.544$ , for criticism and emotional over-involvement, respectively).

In the model including both parent *Sensitive Behavior* and *Intrusive Behavior*, the overall model explained a significant 6.8% of the variance in child ADHD symptoms ( $R^2=0.068$ ,  $p<0.05$ ); however, neither *Sensitive Behavior* nor *Intrusive Behavior* were independently predictive of ADHD symptoms ( $\beta=-.093$ ,  $p=0.465$  and  $\beta=-.180$ ,  $p=.161$ , respectively). This was also the case when the same model was examined, but with ADHD group status as the outcome ( $R^2=0.108$ ,  $p<0.05$ ;  $\beta=-.080$ ,  $p=0.265$  and  $\beta=-.151$ ,  $p=.078$ , for *Sensitive* and *Intrusive Behavior*, respectively).

Additionally, in order to examine the effects of ANS activity separately, a model was examined with only child RSA and PEP during the task as predictors of child ADHD symptoms. This model predict a significant 7.2% of the variance in ADHD symptoms ( $R^2=0.072$ ,  $p<0.05$ ); however, again neither RSA nor PEP were significant predictors ( $\beta=0.167$ ,  $p=0.052$  and  $\beta=-.169$ ,  $p=0.095$ , respectively). When the same model was tested but with ADHD group status as the outcome, the overall model predicted a significant 11.8% of the variance in ADHD group status ( $R^2=0.118$ ,  $p<0.05$ ); however, only PEP, and not RSA, was a significant predictor ( $\beta=.769$ ,  $p=0.009$  and  $\beta=0.137$ ,  $p=0.072$ , respectively, see Figure 13).

#### *Other Explanations for Independent Effects*

It should be noted that the addition of child gender, medication status, or the child behavioral domains of *Agreeableness* and *Engagement* in the task to any of the independent models as covariates did not significantly affect the primary results, and in

order to reduce model over saturation, these results have been presented without these covariates in the model. Additionally, several other explanations for these outcomes were examined. Specifically, it is possible that the independent effect of parent expressed emotion (i.e., criticism) on ADHD symptoms (or ADHD group status) may be better explained by the IQ of the parent completing the five minute speech sample or by the number of words used in the speech sample or by the presence of parent psychopathology (including ADHD or depression symptoms). However, probing each of these explanations did not account for the significant association between parent criticism and child ADHD symptoms (or group status). Specifically, no differences in parent full-scale IQ were observed between children with and without ADHD ( $F=2.93, p>0.13$ ) nor between parents with low or high levels of expressed emotion ( $F=0.29, p>0.59$ ) nor in the interaction between parent expressed emotion and child ADHD diagnosis ( $F=0.67, p>0.42$ ). With respect to the number of words utilized by the parent in the five minute speech sample, a significant main effect of expressed emotion (high vs. low) was observed ( $F=12.14, p<0.01$ ) with higher expressed emotion being associated with the use of a larger number of words, but no other significant effects were observed. Furthermore, there was a significant association between parent ADHD symptoms and child ADHD symptoms ( $F=2.392, p<0.01$ ); however, there was no association between parent criticism and parent ADHD symptoms ( $F=3.52, p=0.08$ ), and the interaction of child ADHD symptoms and parent criticism was not significantly associated with parent ADHD symptoms ( $F=1.791, p=0.122$ ). Additionally, the inclusion of any of these variables as covariates, individually or collectively, did not change the overall association between criticism and number of ADHD symptoms (or ADHD group status).

### *Independent Associations Models with Comorbid Symptoms*

As both callous/unemotional behavior and ODD remained significant predictors of ADHD symptoms in the model predicting ADHD symptoms from parent expressed emotion, parent behavior, and child ANS activity, the role of each of these factors in predicting both callous/unemotional behavior and ODD symptoms (in separate models), while controlling for ADHD symptoms, was explored. Beginning with callous/unemotional behavior, the model including parent criticism, emotional over-involvement, parent *Sensitive Behavior*, *Intrusive Behavior*, and child RSA, PEP, and ADHD and ODD symptoms, as well as child *Agreeableness* and *Engagement* accounted for a significant 48.5% of the variance in callous/unemotional behavior ( $R^2=0.485$ ,  $p<0.01$ ). However, only child ADHD symptoms ( $\beta=0.437$ ,  $p<0.01$ ), child ODD symptoms ( $\beta=0.277$ ,  $p=0.01$ ), and interestingly, parent *Intrusive Behavior* ( $\beta=0.296$ ,  $p=0.03$ ) were significant predictors of callous/unemotional behavior, see Figure 14.

Moving on to ODD, interestingly, the model including parent criticism, emotional over-involvement, parent *Sensitive Behavior*, *Intrusive Behavior*, and child RSA, PEP, callous/unemotional behavior, and ADHD symptoms, as well as child *Agreeableness* and *Engagement* accounted for a significant 43.4% of the variance in ODD symptoms ( $R^2=0.434$ ,  $p<0.01$ ). However, only child ADHD symptoms ( $\beta=0.458$ ,  $p<0.01$ ), callous/unemotional behavior ( $\beta=0.305$ ,  $p=0.02$ ), child RSA ( $\beta=0.254$ ,  $p<0.01$ ), and again, parent *Intrusive Behavior* ( $\beta=0.341$ ,  $p=0.01$ ), were significant predictors of ODD symptoms, see Figures 15 and 16.

## *Discussion*

Children initially begin to regulate their emotions by observing and being reinforced by their parents, and they gradually take this role on themselves (Cole, 2005; Eisenberg et al., 1997), and therefore, parent-child interactions may inform emotion regulation abilities. As this may be the case even in school age children with ADHD, Study 3 examined the roles of parenting behavior and autonomic nervous system reactivity to parent-child interactions in children with ADHD and typically developing youth.

It was originally hypothesized that parents of children with greater numbers of ADHD symptoms would display more expressed emotion (i.e., emotional over-involvement and criticism) during the parent five minute speech sample, as well as more intrusiveness and less sensitive parenting behaviors, during the parent-child interaction task than parents of typically developing youth. This hypothesis was supported partially. Specifically, the model including each of these predictors accounted for approximately 20% of the variance in ADHD symptoms and approximately 28% of the variance in ADHD diagnosis. However, only parent criticism (a sub-domain of parental expressed emotion) remained a significant predictor of number of ADHD symptoms (accounting for approximately 13% of the variance in ADHD symptoms) with all of the other parent-related predictors in the model. Furthermore, the association between ADHD and parental criticism held even after controlling for co-occurring ODD, CD, anxiety, and callous/unemotional behaviors. A similar picture emerged when ADHD diagnosis was considered.

However, turning to parent behavior during a frustrating interaction task, interestingly, parental intrusiveness was a significant predictor of both ODD and callous/unemotional (CU) behaviors, even after controlling for ADHD symptoms (when including CU in models of ODD and ODD in models of CU). Thus, it appears that parental criticism is uniquely associated with ADHD symptoms, while parental intrusiveness is uniquely associated with comorbid symptoms of ODD and callous/unemotional behavior in children with ADHD.

Additionally, it was hypothesized that children's autonomic reactivity in response to parent-child frustrating interactions would be associated with specific presentations of ADHD (i.e., the number of ADHD symptoms and the number of comorbid ODD and CD symptoms). Again, this hypothesis was partially supported. Specifically, overall number of ADHD symptoms was not significantly associated with autonomic reactivity (in either branch) during the parent-child interaction task when controlling for symptoms of ODD and callous/unemotional behavior. However, when attention was shifted to ADHD diagnosis, children with ADHD tended to have shortened PEP (i.e., elevated sympathetic activity) at rest and during the parent-child interaction task when compared to typically developing youth. This finding was congruent with the results of previous research, as well as Studies 1 and 2, and it suggests that children with ADHD tend to be hyper-aroused both at rest and in emotionally taxing situations. Furthermore, this hyper-arousal is present for children with ADHD, even when controlling for comorbid conduct problems. Although there was no significant association between child RSA and ADHD symptoms (or diagnosis), elevated child RSA during the parent child interaction task was associated with increased numbers of ODD symptoms after controlling for both ADHD

symptoms and callous/unemotional behavior symptoms. Thus, with respect to child autonomic functioning under conditions of parent-child negotiation, it appears that child sympathetic reactivity is uniquely associated with ADHD, while parasympathetic reactivity during such an interaction is uniquely associated with oppositional and defiant behaviors in children with ADHD.

Finally, it was hypothesized that the association between parenting behavior and expressed emotion with ADHD symptom presentation (i.e., severity, ODD, CD, and CU symptoms) would be mediated by autonomic nervous system reactivity in response to a frustrating parent-child interaction task. However, the preconditions for mediational analyses were not met, and an analysis of moderation was considered. Specifically, parent expressed emotion (e.g., criticism and emotional over-involvement), as well as parental sensitivity and intrusiveness during the task were examined as moderators of the association between autonomic nervous system reactivity (e.g., RSA and PEP) and ADHD symptoms. However, neither mediation- nor moderation-based models were supported. As such, the independent associations described earlier were pursued. Each of these independent associations is discussed below.

First, parental expressed emotion is considered. Consistent with the small number of studies on parental expressed emotion and ADHD (Peris & Baker, 2000; Peris & Hinshaw, 2003), high parental expressed emotion was associated with an ADHD diagnosis, as well as more child ADHD symptoms. Importantly, the association between ADHD and high parental expressed emotion remained significant even after controlling for ODD, CD, callous/unemotional behavior, and anxiety symptoms. However, the converse was not the case. Thus, these results suggest that oppositional, defiant,

callous/unemotional or anxious child behavior does not explain the link between parent expressed emotion and child ADHD diagnosis or symptoms.

Furthermore, consistent with previous literature, the criticism sub-domain, but not the emotional over-involvement sub-domain of expressed emotion, was associated with both child ADHD diagnosis and the number child of ADHD symptoms (Peris & Baker, 2000; Peris & Hinshaw, 2003). In fact, consistent with prior literature (Hibbs et al, 1991; Peris & Baker, 2000) the emotional over-involvement sub-domain was not significantly associated with ADHD, ODD, CD, or anxiety symptoms.

Additionally, criticism was the only significant predictor of ADHD symptoms when examining a model, including criticism, emotional over-involvement, and both parental sensitivity and intrusiveness coded during a parent-child interaction task designed to induce frustration and negotiation. Furthermore, the effect of criticism was not explained by parent IQ, parent ADHD symptoms, or parent symptoms of major depressive disorder. Finally, while the overall number of words utilized during the five minute speech sample was associated with overall expressed emotion rating and level of criticism, the number of words utilized by parents during the task did not explain the association between the overall level of criticism and number of child ADHD symptoms. Thus, it appears that the association between parental criticism and child ADHD symptoms is fairly robust and not easily explained by common possible artifacts.

While the unique association between parental criticism and child ADHD symptoms is consistent with previous literature and survived several validity checks, it may be that other forms of parenting problems, including parental intrusiveness are more strongly associated with comorbid child conduct problems in children with ADHD.

Specifically, with respect to parenting behavior observed during the task (i.e., sensitivity and intrusiveness), these parenting behaviors were not uniquely associated with child ADHD symptoms. However, importantly, parental intrusiveness was associated with both ODD and callous/unemotional behavior, even after controlling for co-occurring ADHD symptoms, parental expressed emotion (i.e., both criticism and emotional over involvement), and child behavior during the task (i.e., both child *Agreeableness* and child *Engagement*). The roles of these types of parent behavior in child adjustment are well established in infants and young children (Campbell et al., 1999, 2000; Murray et al, 1999, 2002, 2005). Additionally, parenting behavior characterized by high levels of intrusiveness may have an important impact on child development (Crowley et al, 2005; Murray et al., 2000).

These parenting behavior results are also consistent with previous literature that has reported on the association between harsh, critical, and controlling parenting and ADHD (Anderson et al., 1994; Campbell et al., 1991; Marshall et al., 1990). However, few prior studies have examined the unique associations of negative parenting and ADHD, specifically, while controlling for the unique associations of comorbid child behavior problems or the unique associations of other parent characteristics, such as parent IQ, ADHD symptoms, or depression symptoms. In fact, one such study that did control for aggressive child behavior found that while harsh, negative parenting may be correlated with ADHD, negative parenting may be more salient for children with other externalizing pathology, such as aggression, ODD, and CD (Johnston & Mash, 2001). Furthermore, due to the cross-sectional design of this study, a full assessment of the direction of effects, as well as a full assessment of child-driven and bi-directional effects,

was not possible. Finally, it must be noted that previous literature also provides support for these types of models (i.e., those models which support child-driven and bi-directional effects; Anderson et al., 1994; Marshall et al., 1990). That is, previous literature has consistently shown that the influence of conduct problems on changes in parenting behaviors tends to be just as strong as the influence of parenting behaviors on changes in conduct problems across development (Pardini et al., 2008). Thus, additional considerations of these bi-directional effects is needed in future research, and in particular, in longitudinal studies in order to parse the unique effects of parenting behavior on ADHD and comorbid symptoms, as well as the converse. Though some have shown more evidence for child-driven than parent-driven effects in ADHD (Burt & Nickolas, 2011).

In terms of autonomic nervous system activity at rest and reactivity during the parent-child interaction task, consistent with previous literature (and Studies 1 and 2) children with ADHD displayed shortened PEP at baseline, as well as shortened PEP during the task conditions, suggesting heightened sympathetic activity and arousal among children with ADHD. During the parent-child interaction task, no significant associations between RSA and ADHD symptoms or ADHD group status were observed. As such, it appears that in contrast to the hypothesis, overall levels of emotion regulation during frustrating parent-child interactions were not associated significantly with child ADHD severity in this sample. However, elevated RSA during the parent-child interaction was associated with increased numbers of ODD symptoms even after controlling for ADHD symptoms. Thus, poor emotion regulation, as indexed by dysregulated parasympathetic activity during a frustrating parent-child interaction, was independently associated with

child oppositional and defiant behaviors.

There may be several reasons for these differences. Specifically, while previous literature has linked dysregulated parasympathetic activity during both frustrating tasks (Crowell et al., 2005) and social tasks (El Shiekah, 2010) specifically to symptoms of ODD. However, these results in contrast to the hypotheses, as well as the findings of Studies 1 and 2. It may be that the overall emotional tone of the task used in Studies 1 and 2 differ substantially from that used in Study 3. It may also be that an interaction task, when compared to a solitary task, requires different types of regulation. That is, the interaction task may allow for more extrinsic forms of emotion regulation, while the induction and suppression task require more intrinsic forms of regulation, including child behavior and parasympathetic activity. Finally, it may also be that the two different task types (i.e., induction/suppression and parent-child interaction) may result in different appraisals of the task. Specifically, it may be that while the induction/suppression task is appraised as a *challenge*, the parent-child interaction task with its time-limit and instructions informing participants that “most families can complete the task within five minutes”, is more likely to be appraised as a *threat* (Mendes et al., 2008). Each of these possibilities is given more consideration in the Overall Discussion and Conclusions (see page 113).

With respect to limitations and future directions, while Study 3 is one of the first to examine the independent and joint roles of parenting behavior, parent expressed emotion, and child autonomic nervous system functioning in ADHD, certain limitations have to be considered to place these findings in context. Specifically, only very few fathers (less than 10% across both groups with no significant difference in the percentage

of fathers between the groups) completed the five minute speech sample and parent-child interaction tasks. Nearly all of the participating parents were mothers. This is a limitation, as paternal expressed emotion and parenting behavior may have equally important effects, and statistical power precluded tests of main effects of parent gender.

Furthermore, this design precluded an examination of parent gender by child gender-based interaction effects. That is, previous research has shown that parenting behavior of the parent of the same gender as the child may be particularly salient at this age (Fivush et al., 2000; Kerig et al., 1993; Starrels, 1994).

An additional limitation, of particular importance, the cross-sectional, non-experimental design precludes explorations of causality and directionality of effects, though one previous longitudinal study showed that parent expressed emotion during preschool was predictive of child ADHD diagnoses three years later (Peris & Baker, 2000). Furthermore, the established bi-directional nature of the association between child ADHD symptoms and parenting behavior patterns further complicates a linear, unidirectional interpretation (Johnston & Mash, 1990). That is, child ADHD symptoms may also bring out critical, harsh, intrusive parenting. Additionally, it may be that there are gene-by-environment correlational effects at play here. Specifically, it may be that parents with ADHD or ADHD symptoms may be more harsh or critical than parents without such symptoms. However, in this sample, the effect of parental expressed emotion and criticism specifically, held even after controlling for parent ADHD symptoms.

In conclusion, it appears that parental expressed emotion, and parental criticism in particular, was uniquely associated with child ADHD symptoms, even after controlling

for comorbid symptoms, in this sample. In contrast, parental intrusiveness appears to be uniquely associated with child symptoms of oppositional defiant disorder (ODD) and callous/unemotional traits, even after controlling for ADHD symptoms. Furthermore, children with ADHD displayed increased sympathetic reactivity during the parent-child interaction, while in contrast, increased child parasympathetic activity during the parent-child interaction was associated with increased ODD symptoms, even after controlling for ADHD symptoms. However, the reverse was not true. That is, no significant association was observed between child parasympathetic reactivity during the parent-child interaction task and ADHD symptoms after controlling for the presence of ODD symptoms. Thus, it appears that with respect to extrinsic emotion regulation (in the forms of parenting behavior and expressed emotion) in children with ADHD, there are distinct associations between the emotional climate of the home and the severity of ADHD, as well as between intrusive parenting behavior and the number of comorbid oppositional defiant and callous/unemotional behaviors. Furthermore, it appears that intrinsic emotion regulation (in the form of parasympathetic reactivity to a frustrating parent-child interaction) is uniquely disrupted in children with greater numbers of ODD symptoms, while overall emotionality and arousal in the face of a challenging parent-child negotiation is uniquely disrupted in children with ADHD.

## CHAPTER V

### OVERALL DISCUSSION AND CONCLUSIONS

The primary question addressed in different ways in each of these three studies was: What are the roles of intrinsic (child behavior and physiology) and extrinsic (parenting behavior and expressed emotion) mechanisms of emotion regulation in attention-deficit/hyperactivity disorder (ADHD)? Specifically, the studies described here aimed to evaluate (a) physiological indices of child emotion regulation (including parasympathetic and sympathetic contributions); (b) correspondence among physiological and facial affective behavior indices of emotion regulation; and (c) the role of parenting behavior and parental expressed emotion in shaping the associations between the physiological regulation of emotion and the severity of, and types of symptoms co-occurring with, ADHD.

As hypothesized, but not demonstrated so clearly in previous literature, ADHD was associated with alterations in emotion and its regulation, as assessed through facial affective behavior, self-report, and parasympathetic and sympathetic nervous system functioning, during induction and suppression of both positive and negative emotions. Specifically, children with ADHD tended to display elevated RSA and shorter PEP (i.e., parasympathetic-based emotion dysregulation and sympathetic-based over-arousal, respectively). Additionally, the greatest difference was observed during the positive induction condition, suggesting that children with ADHD may have a particularly difficult time regulating positive or approach-based emotions.

A second key finding was that there may be a role for co-occurring callous/unemotional traits in ADHD, similar to that previously observed among certain children with conduct disorder and adults with antisocial personality disorder.

Furthermore, the presence of callous/unemotional traits among children with ADHD was associated with blunted parasympathetic and sympathetic nervous system functioning, which is congruent with studies of these traits in adults.

In addition to disruptions in individual systems of emotion (i.e., autonomic, behavioral), multiple instances of reduced correspondence between autonomic nervous system reactivity and facial affective behavior were observed in the ADHD group. This is important as functionalist theories of emotion propose that synchrony among emotional response systems is associated with emotional health, while desynchronization of these systems contributes to the development and maintenance of psychopathology (Ekman, 1992a; Mauss, Evers, Wilhelm, & Gross, 2006; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Wilhelm, Grossman, & Roth, 2005). Additionally, it should be noted that these correspondence effects were somewhat domain specific. That is, while an association between avoidance-based emotions and RSA was observed across both negative and positive emotion induction for the control group, this association was reduced for the ADHD group. While reductions in RSA were observed during the expression of approach-based during positive emotion induction for typically developing children, this association was reduced among children with ADHD with similar findings for PEP. This suggests that in addition to specific disruptions in specific emotion and regulation based systems, the interaction among these systems may also be important to psychological health. Furthermore, these results suggest that rather than ADHD being associated with simply disruptions in arousal or regulation, there appears to be a more meaningful and context-specific adaptation failure between the emotional systems of facial affective behavior and autonomic nervous system functioning.

Finally, parental expressed emotion, and criticism specifically, but not sensitivity or intrusiveness, were associated with the number of ADHD symptoms present, while parental intrusiveness during frustrating parent-child interactions were associated uniquely with both callous/unemotional and oppositional/defiant symptoms among children with ADHD. Additionally, again, elevated child sympathetic reactivity during a parent-child interaction was associated uniquely with child ADHD symptoms. In contrast, child parasympathetic dysregulation during frustrating parent-child interactions was associated with child ODD symptoms. Thus, it appears that parent expressed emotion and sympathetic reactivity may be uniquely associated with the presentation of ADHD with respect to severity, while parental intrusiveness and child parasympathetic-based emotion dysregulation during frustrating parent-child interactions may be uniquely associated with the number of comorbid behavior problems.

While previous literature has linked dysregulated PNS activity during both frustrating tasks (Crowell et al., 2005) and frustrating social tasks (El Shiekah, 2010) to symptoms of ODD, these specific results of Study 3 were somewhat in contrast to the hypotheses. It should also be noted that findings of Studies 1 and 2, where parasympathetic differences were observed when comparing children with ADHD to typically developing controls in response to an emotion induction/suppression task, did support the hypothesis that dysregulated PNS activity would be observed among children with ADHD. It may be that the overall tone of the task used in Studies 1 and 2 differ substantially from that used in Study 3. Specifically, Study 1 showed that inflexibly augmented RSA during an emotion induction/suppression task was associated uniquely with symptoms of ADHD, which was also observed in Study 2. However, it could be

argued that the emotional tone of the two types of tasks (e.g., emotion induction and suppression; parent-child interaction) were quite different. Specifically, the emotion induction and suppression task was rated as “sad” by the majority of participants during the negative condition and as “happy” by the majority of participants during the positive condition. In contrast, the parent-child interaction task was designed to instigate parent-child negotiations and to induce mild frustration in both parents and children participants (Deater-Deckard et al., 2000). Thus, it may be that the differences are somewhat emotion type specific.

However, it may also be that an interaction task, like the parent-child interaction task utilized in Study 3, requires a different approach to emotion regulation than a more independent (or intrinsic regulation-based) task, such as the induction and suppression task used in Studies 1 and 2. That is, the interaction task may allow for more extrinsic forms of emotion regulation. Specifically, in the context of the parent-child interaction task the child may rely more on parenting behavior and structuring for emotion regulation than on physiological processes. In contrast, in the context of the more independent (intrinsic) induction and suppression task, the child is only able to rely on more intrinsic forms of regulation, including child behavioral, cognitive, and autonomic regulation. Thus, parasympathetic dysregulation may be a more salient predictor of child ADHD symptoms in the context of tasks requiring more intrinsic types of emotion regulation.

Finally, it may also be that the two types of tasks (i.e., the emotion induction/suppression task of Studies 1 and 2 versus the parent-child interaction task of Study 3) were fundamentally different with respect to the way that they were appraised by participants. Specifically, previous work has suggested that arousal (i.e., sympathetic

activation) tends to increase in anticipation of the evaluations of others, for example during a social task (Cottrell, Wack, Sekerak, & Rittle, 1968). Furthermore, differential patterns of arousal and regulation (i.e., sympathetic reactivity and parasympathetic activation or withdrawal, respectively) tend to emerge under situations involving social challenge in contrast to social threat (Blascovich et al., 1999, 2008). Specifically, challenge and threat have both been proposed to occur in goal-relevant situations that relate to self- or other-evaluations when perceived as relatively important. Challenge occurs when the individual experiences sufficient resources to meet situational demands. In contrast, threat occurs when the individual experiences insufficient resources to meet demands (Blascovich et al., 1999, 2008). Thus, it may be that the relatively easy induction and suppression task is perceived by the child participants as a challenge, as the task is relatively easy and straightforward, thereby resulting in more salient involvement of the parasympathetic system. In contrast, the parent-child interaction task with its frustration and deception components (i.e., the dyad are told that “most participants finish within five minutes”, which is deceiving) may be appraised by participants as more of a social threat as they realize they do not actually have the resources required to complete the task, thereby resulting in more salient sympathetic involvement.

Thus, future research is needed to further explore the exact nature of the differences in these forms of emotion and its regulation (i.e., intrinsic and extrinsic). Specifically, to assess possible differences in emotional tone (i.e., the proposal that the induction/suppression and extrinsic regulation differ with respect to the types of emotions experienced) child facial affective behavior codes or child ratings of the emotional valence and type experienced during parent-child interaction could be acquired and

compared to those to those observed during induction and suppression. Furthermore, to assess the proposal that different types of emotion regulation strategies are being utilized (i.e., that induction/suppression requires more intrinsic strategies, while parent-child interactions requires more extrinsic strategies) child-reports of regulation strategies could be assessed. Finally, to examine possible differences in the appraisal of challenge and threat (i.e., that the induction/suppression would be viewed as more of a challenge, while challenging parent-child interactions would be viewed as more of a threat) child-reports of how capable they felt to complete the task could be assessed in future research.

Each of these studies has implications for refining current theories of ADHD, as well as implications for future research. Specifically, ADHD has been theorized traditionally to primarily to involve dysfunctions in the physiological and neural systems supporting behavioral and cognitive inhibition and control (Barkley, 1997; Nigg, 2001), with an emphasis on inability to regulate behavior (Pennington & Ozonoff, 1996) and attention. More recently, theories of the roles of emotion and its regulation in ADHD (Barkley & Fischer, 2010; Martel, 2009; Wehmeier, Schacht, & Barkley, 2010) have begun to re-emerge since early work by Wender and colleagues (1972) initially proposed such disruptions, suggesting a broader or more general dysregulation is involved in ADHD. However, until recently, emotion in ADHD has not been given much empirical evaluation. This series of studies was one of the first to do so utilizing modern tools for the investigation of emotion regulation.

Specifically, as described in the Introduction and **Figure 1**, a theoretical model guided this series of investigations. As proposed in this model, the emotionality domain of temperament can be divided into negative, positive, and blunted emotionality sub-

domains (Eisenberg et al., 2004; Rothbart et al., 1998). Both negative and positive emotionality have been indexed by a heightened or increased change in SNS activity, as measured by both PEP and skin conductance, from baseline and that a predisposition toward heightened emotionality in these domains is indexed by elevated SNS activity at baseline (Calkins et al., 1997, 1998; Gross & Levenson, 1993, 1997; Porges, 1997). With respect to specific externalizing behaviors, hyperactivity and impulsivity have been linked with excess positive emotionality, approach, and increased SNS activity at baseline and in the face of emotional stimuli (particularly positive emotional stimuli; Musser et al., 2010; Nigg et al., 2005). This portion of the model was supported by the results of Studies 1, 2, and 3, as children with ADHD displayed sympathetic over-arousal in response both the emotion induction/suppression and parent-child interaction tasks.

It is helpful now in light of these data to reconsider the conceptual model proposed at the outset (Figure 1). In accordance with the model proposed (Figure 1), blunted or flattened affect and very low levels of both positive and negative emotionality are indexed by reduced SNS activity both during baselines and during stressors/emotionally provocative events (Beauchaine et al., 2001; Frick et al., 1999; Raine et al., 2001). With respect to behavior being associated with blunted emotionality, callous/unemotional behavior has been linked to underactive SNS activity at baseline, during emotionally evocative tasks, and during reward and punishment (Fabes et al., 1994; Frick et al., 1999; Kagan & Snidman, 1991; Raine et al., 2002). This portion of the model was supported by the results of Study1, which showed that children with ADHD and co-occurring callous/unemotional traits displayed sympathetic under-arousal.

Also, as proposed by the model, oppositional/defiant behaviors and

reactive/impulsive aggression have been linked to negative emotionality and increased SNS activity both at rest and in response to negative emotional stimuli (Burgess et al., 2003; Mezzacappa et al., 1997; Martel, 2009, 2011; Nigg et al., 2005). However, this was not directly assessed by the studies in this dissertation, as oppositional/defiant behaviors were only observed in the context of ADHD. However, preliminary results of Studies 1 and 3 showed that symptoms of oppositional defiant disorder were not associated with elevated sympathetic activation after controlling for ADHD symptoms.

Moving on to the regulatory domain of temperament as outlined in the model (Figure 1), this domain can be further subdivided into behaviors of self-regulation and effortful control (Eisenberg et al., 2004; Rothbart et al., 1998). Furthermore, disruptions in both forms of regulation have been indexed by abnormal PNS activity, as assessed by RSA levels, both at baseline and in response to tasks which require high levels of regulation. In particular, Calkins and colleagues (1997, 1998, 2001, 2002) and Beauchaine and colleagues (2001, 2002, 2004, 2009) as well as others have shown that both low baseline PNS activity and reduced PNS reactivity in response to stressful tasks index both behavioral observations of self-regulation and effortful control (Eisenberg et al., 2002, 2004).

With respect to externalizing behavior, hyperactivity and impulsivity (as well as reactive aggression) have been linked to deficits in self-regulation behaviorally and as assessed by increased sympathetic reactivity and reduced parasympathetic flexibility during emotionally provocative tasks (Martel, 2009; Musser et al., 2010). Additionally, inattention appears to be associated almost exclusively with disruptions in effortful control, which has been indexed by dysregulated parasympathetic activity during

attentionally demanding tasks (Suess, Porge, & Plude, 1994). Again, the studies outlined in this dissertation provide support for this portion of the proposed model, as Studies 1 and 2 found inflexible parasympathetic reactivity to both negative and positive emotions among children with ADHD. However, the results of Study 3 suggest that oppositional/defiant behavior was uniquely associated with increased parasympathetic reactivity after controlling for symptoms of ADHD. However, it is important to note that several of the oppositional defiant behaviors which were examined as covariates (e.g., often loses temper) were likely best characterized as symptoms of reactive aggression, which has also been linked to dysregulated parasympathetic reactivity. Future research might usefully continue to tease apart the exact nature of the association between parasympathetic-linked, regulatory aspects of temperament and symptoms of ADHD in contrast to those of oppositional-defiant symptoms.

Furthermore, as described in the Discussion sections of each of the individual studies, due to the cross-sectional design of this dissertation an assessment of causality or the directionality of effects was not possible. This is particularly important for Study 3, as the assessment of child-driven and bi-directional effects, was not possible. However, previous literature also provides support for models including child-driven, parent-driven, and bi-directional effects in ADHD (Anderson et al., 1994; Marshall et al., 1990). That is, previous literature has shown that the influence of behavior problems on changes in parenting behaviors tends to be just as strong as the influence of parenting behaviors on changes in behavior problems across development for children with and parents of children with ADHD (Johnston & Marsh, 2000). Thus, additional considerations of these bi-directional effects is needed in future research, and in particular, in longitudinal studies

in order to parse the unique effects of parenting behavior on ADHD and comorbid symptoms, as well as the reverse.

While remaining cautious before application to assessment and intervention is warranted, each of these studies may have future implications for both the assessment and treatment of ADHD if findings continue to hold up. For example, biological assessments of autonomic nervous system functioning may be used to help distinguish specific profiles of ADHD symptoms. Additionally, as described above there is likely a transactional association between parenting behavior and child ADHD and co-occurring behavior problems, which should be explored further in longitudinally-designed studies. However, if it is the case that parenting influences the severity of child behavior problems in children with ADHD or if bi-directional effects exist, it may be that curbing specific behavior problems may be accomplished by manipulating environmental factors, including making changes in the parent-child relationship. Importantly, parent-management training is currently the gold-standard psychosocial intervention approach for children with ADHD. However, if future research continues to support the importance of emotion and its regulation in ADHD, then the incorporation of emotion-based therapy in the treatment of ADHD may be warranted. Specifically, intervention may benefit from training children with ADHD to identify their emotions and from building emotion regulation skills. Such an intervention might resemble a modified version of Kendall's "Coping Cat" (2000), a manualized, cognitive-behavioral treatment for children with anxiety. However, this emotion-based treatment could focus on regulatory skills associated with both negative and positive emotions.

Overall, the results of these three studies highlight the role of emotion and its

dysregulation in ADHD and provide support for theories suggesting that emotion and emotional dysregulation at multiple levels of analysis may play an important role in ADHD. In particular, children with ADHD and co-occurring behavior problems appear to have specific disruptions in the domains of emotionality, as well as in the domains of both intrinsic (parasympathetic-based regulation) and extrinsic (parenting-based) emotion regulation.

APPENDIX A

TABLES

**Table 1.** *List of studies and measures included in each.*

Study	Measures
Screening and Diagnosis across Studies	Kiddie Schedule of Affective Disorders and Schizophrenia ADHD Rating Scale Conner’s Rating Scale-3rd Edition Strengths and Difficulties Questionnaire Wechsler Intelligence Scales for Children, Fourth Edition— Vocabulary, Block Design, Information subscales Wechsler Individual Achievement Test, Second Edition-- Reading, Math Reasoning, and PseudoWord subscales
Study 1	Emotion Induction and Suppression Task (EIST) International Affective Pictures System (IAPS) Self-Assessment Manikin Respiratory Sinus Arrhythmia—Derived from EKG during EIST/IAPS/Rest Cardiac Pre-ejection Period—Derived from IMP during EIST/IAPS/Rest Facial Affective Coding System (FACS)
Study 2	Emotion Induction and Suppression Task (EIST) International Affective Pictures System (IAPS) Self-Assessment Manikin Respiratory Sinus Arrhythmia—Derived from EKG during EIST/IAPS Cardiac Pre-ejection Period—Derived from IMP during EIST/IAPS Facial Affective Coding System (FACS)
Study 3	Parent Child Interaction Task (PCIT) Parent Child Interactive Coding System (PARCHISY) International Affective Picture System (IAPS) Respiratory Sinus Arrhythmia—Derived from EKG during PCIT/IAPS Cardiac Pre-ejection Period—Derived from IMP during PCIT/IAPS Parent Five Minute Speech Sample Expressed Emotion Coding System

**Table 2. Study1: Descriptive and diagnostic statistics for ADHD and control groups**

Variable	Control (n=75)	ADHD	
		ADHD only (n=54)	ADHD+CU (n=21)
<b>Demographics</b>			
Age (months; mean, SD)	97.41 (6.91)	97.30 (7.08)	97.63 (8.83)
Gender (% male)	49.3% <sup>a</sup>	52.6% <sup>a</sup>	70.4% <sup>b</sup>
Race (% White)	88.0%	86.2%	89.5%
Fam. Income (\$K; mean, SD)	100.35 (46.23)	84.81 (41.43)	98.06 (51.48)
% 2 parent homes	86.7%	79.6%	75.7%
Stimulant Med. (% on med.)	0.00% <sup>a</sup>	29.6% <sup>b</sup>	26.3% <sup>b</sup>
WISC-IV <sup>1</sup> FSIQ <sup>2</sup> (mean, SD)	109.11 (5.19)	107.14 (6.84)	104.85 (5.91)
<b>ADHD-RS- T-scores-Parent (mean, SD)</b>			
Hyperactive/Impulsive	47.24 (13.31) <sup>a</sup>	69.58 (13.83) <sup>b</sup>	70.71 (19.14) <sup>b</sup>
Inattentive	46.66 (11.52) <sup>a</sup>	74.14 (12.40) <sup>b</sup>	69.58 (18.37) <sup>b</sup>
Total	46.70 (12.83) <sup>a</sup>	73.50 (12.44) <sup>b</sup>	71.69 (19.98) <sup>b</sup>
Hyperactive Symptoms	0.57 (1.50) <sup>a</sup>	5.32 (2.69) <sup>b</sup>	5.17 (3.07) <sup>b</sup>
Inattentive Symptoms	0.48 (1.41) <sup>a</sup>	6.57 (2.68) <sup>b</sup>	5.42 (3.34) <sup>b</sup>
<b>ADHD-RS- T-scores-Teacher (mean, SD)</b>			
Hyperactive/Impulsive	44.04 (4.65) <sup>a</sup>	59.36 (9.26) <sup>b</sup>	57.51 (11.57) <sup>b</sup>
Inattentive	42.49 (4.08) <sup>a</sup>	56.91 (8.29) <sup>b</sup>	54.88 (9.46) <sup>b</sup>
Total	42.76 (4.41) <sup>a</sup>	58.61 (8.14) <sup>b</sup>	56.46 (10.46) <sup>b</sup>
Hyperactive Symptoms	0.21 (1.00) <sup>a</sup>	4.75 (3.16) <sup>b</sup>	3.82 (3.61) <sup>b</sup>
Inattentive Symptoms	0.17 (0.87) <sup>a</sup>	4.68 (3.04) <sup>b</sup>	3.81 (3.28) <sup>b</sup>
<b>Conner's 3rd-T-score-Parent (mean, SD)</b>			
Inattention	48.59 (8.71) <sup>a</sup>	73.79 (10.17) <sup>b</sup>	70.08 (14.08) <sup>b</sup>
Hyperactivity	47.57 (10.21) <sup>a</sup>	76.64 (12.25) <sup>b</sup>	72.67 (14.76) <sup>b</sup>
Learning Problems	46.55 (7.03) <sup>a</sup>	61.32 (12.48) <sup>b</sup>	58.67 (12.91) <sup>b</sup>
Executive Functioning	48.16 (8.97) <sup>a</sup>	70.46 (11.20) <sup>b</sup>	67.92 (14.98) <sup>b</sup>
Aggression	48.52 (8.88) <sup>a</sup>	57.25 (13.95) <sup>b</sup>	60.41 (12.26) <sup>b</sup>
Peer Relations	52.54 (11.89) <sup>a</sup>	66.82 (19.26) <sup>b</sup>	66.92 (14.59) <sup>b</sup>
<b>Conner's 3rd-Teacher (mean, SD)</b>			
Inattention	47.11 (8.20) <sup>a</sup>	67.17 (9.61) <sup>b</sup>	68.92 (10.85) <sup>b</sup>
Hyperactivity	49.39 (10.59) <sup>a</sup>	74.96 (15.05) <sup>b</sup>	65.25 (15.39) <sup>b</sup>
Learning Problems	45.13 (4.73) <sup>a</sup>	58.14 (8.61) <sup>b</sup>	52.00 (9.12) <sup>c</sup>
Aggression	49.54 (10.25) <sup>a</sup>	58.83 (17.59) <sup>b</sup>	67.08 (17.41) <sup>b</sup>
Peer Relations	50.98 (10.03) <sup>a</sup>	64.68 (16.56) <sup>b</sup>	70.17 (15.47) <sup>b</sup>
<b>SDQ<sup>3</sup> Subscales-Parent (mean, SD)</b>			
Emotional Symptoms	1.34 (1.57)	1.86 (2.04)	2.11 (1.60)
Conduct Problems	0.76 (1.28) <sup>a</sup>	2.26 (1.60) <sup>b</sup>	2.89 (1.91) <sup>b</sup>
Hyperactivity	2.32 (2.40) <sup>a</sup>	7.72 (2.07) <sup>b</sup>	7.67 (2.93) <sup>b</sup>
Peer Problems	0.81 (1.26) <sup>a</sup>	2.18 (2.11) <sup>b</sup>	2.28 (1.45) <sup>b</sup>
Prosocial Behavior	8.97(1.43) <sup>a</sup>	8.24 (1.90) <sup>a</sup>	4.67 (1.46) <sup>b</sup>
Total Difficulties	5.23 (5.04) <sup>a</sup>	14.02(5.22) <sup>b</sup>	14.94 (4.68) <sup>b</sup>
Impact/Impairment score	0.23 (0.82) <sup>a</sup>	2.65 (1.98) <sup>b</sup>	3.12 (2.04) <sup>b</sup>

<b>SDQ<sup>3</sup> Subscales-Teacher (mean, SD)</b>			
Emotional Symptoms	1.23 (1.54)	1.44 (1.87)	1.56 (1.95)
Conduct Problems	0.33 (1.11) <sup>a</sup>	1.83 (1.91) <sup>b</sup>	2.22 (2.77) <sup>b</sup>
Hyperactivity	1.51 (1.93) <sup>a</sup>	7.15 (2.38) <sup>b</sup>	6.50 (3.17) <sup>b</sup>
Peer Problems	0.77 (1.06) <sup>a</sup>	1.91 (1.94) <sup>b</sup>	2.20 (2.21) <sup>b</sup>
Prosocial Behavior	8.19 (1.83) <sup>a</sup>	7.37 (2.47) <sup>a</sup>	4.29 (1.88) <sup>b</sup>
Total Difficulties	3.43 (3.95) <sup>a</sup>	12.23 (5.15) <sup>b</sup>	14.79 (7.13) <sup>b</sup>
Impact/Impairment score	0.14 (0.65) <sup>a</sup>	1.83 (1.45) <sup>b</sup>	1.89 (1.88) <sup>b</sup>
<b>Comorbid Disorders (%; K-SADS<sup>4,5</sup>)</b>			
Mood Disorder (lifetime)	2.7%	3.7%	3.7%
Anxiety Disorder	21.3% <sup>a</sup>	23.9% <sup>a</sup>	4.7% <sup>b</sup>
Conduct Disorder (CD)	0.0%	0.0%	0.0%
Oppositional Defiant Disorder (ODD)	8.1% <sup>a</sup>	24.7% <sup>b</sup>	23.8% <sup>b</sup>
Tic Disorder	0.0%	3.7%	0.0%
Sleep Disorder	5.4%	7.1%	4.7%
<b>CD Symptoms (mean, SD)</b>	<b>0.02 (0.13)<sup>a</sup></b>	<b>0.09 (0.25)<sup>ab</sup></b>	<b>0.18 (0.42)<sup>b</sup></b>
<b>ODD Symptoms (mean, SD)</b>	<b>0.44 (1.15)<sup>a</sup></b>	<b>1.29 (1.86)<sup>b</sup></b>	<b>1.42 (1.1.84)<sup>b</sup></b>
<b>Total Anxiety Sx (mean, SD)</b>	<b>1.31 (1.59)</b>	<b>2.31 (4.19)</b>	<b>1.19 (2.45)</b>

*Note.* Differing superscripts indicate pairwise comparisons that were significant after a modified Bonferroni correction for multiple groups ( $\alpha=0.016$ ) for continuous variables, including: age, family income, estimated full-scale IQ, SDQ parent and teacher sub-scales, and comorbid symptoms; and Chi-square comparisons for categorical variables, including: gender, race, parent marital status, child medication status, and comorbid disorders.

<sup>1</sup> WISC-IV: Wechsler Intelligence Scales for Children

<sup>2</sup> Full-Scale Intelligence Quotient (estimated)

<sup>3</sup> Strengths and Difficulties Questionnaire

<sup>4</sup> Kiddie Schedule of Affective Disorders and Schizophrenia

<sup>5</sup> 0% of the sample had autism, eating disorders, learning disorders, post-traumatic stress disorder, psychosis, or substance use disorders

**Table 3.** Study 1: Self-Assessment Manikin (SAM) scores across task conditions for ADHD and control groups

Variable	Control (n=75)	ADHD	
		ADHD only (n=54)	ADHD+CU (n=21)
<b>SAM<sup>1</sup> Valence/pleasure</b>			
Baseline 1	3.39 (0.67)	3.54 (0.79)	3.45 (0.81)
Negative induction	2.44(1.17)	2.48(1.31)	2.58 (1.08)
Negative suppression	1.73(0.88)	1.74 (1.07)	1.91 (1.05)
Baseline 2	3.38 (0.71)	3.45 (1.22)	3.46 (0.81)
Positive induction	4.47 (0.81)	4.44 (1.13)	4.68 (0.67)
Positive suppression	4.64(0.77)	4.67 (0.89)	4.79 (0.42)
<b>SAM<sup>1</sup> Intensity/arousal</b>			
Baseline 1	2.04 (0.72)	2.31 (0.80)	2.15 (0.76)
Negative induction	3.51 (1.25)	3.61(1.45)	3.37 (0.89)
Negative suppression	3.20 (1.14)	3.09 (1.46)	3.19 (1.11)
Baseline 2	1.93 (0.91)	2.23 (0.81)	1.85 (0.72)
Positive induction	2.11 (1.24)	2.20 (1.48)	2.11 (1.29)
Positive suppression	2.32 (1.36)	2.48 (1.63)	2.16 (1.26)

*Note.* For this table, no significant group differences were observed. Each row represents the next time point in design. Time increasing down table for the repeated measures design.

<sup>1</sup>SAM: Self-Assessment Manikin

**Table 4.** Study 1: Self-reported emotion primary ratings scores across task conditions for ADHD and control groups

Variable	Control (n=75)	ADHD		$\chi^2$
		ADHD only (n=54)	ADHD+CU (n=21)	
Negative induction				10.567
Sup <sup>1</sup>	13.1%	5.7%	11.1%	
Hap <sup>2</sup>	18.7%	13.2%	11.1%	
Ang <sup>3</sup>	0.0%	1.9%	0.0%	
Anx <sup>4</sup>	56.0%	62.3%	50.0%	
Fri <sup>5</sup>	6.7%	13.2%	11.1%	
Sad <sup>6</sup>	5.3%	3.8%	16.7%	
Negative Suppression				8.643
Sup <sup>1</sup>	4.0%	0.0%	0.0%	
Hap <sup>2</sup>	2.7%	5.7%	0.0%	
Ang <sup>3</sup>	0.0%	0.0%	0.0%	
Anx <sup>4</sup>	6.7%	0.0%	5.6%	
Fri <sup>5</sup>	5.3%	9.4%	0.0%	
Sad <sup>6</sup>	82.7%	83.0%	94.4%	
Positive induction				3.659
Sup <sup>1</sup>	2.6%	0.0%	0.0%	
Hap <sup>2</sup>	93.7%	98.1%	100.0%	
Ang <sup>3</sup>	0.0%	1.9%	0.0%	
Anx <sup>4</sup>	0.0%	0.0%	0.0%	
Fri <sup>5</sup>	0.0%	0.0%	0.0%	
Sad <sup>6</sup>	0.0%	0.0%	0.0%	
Positive Suppression				11.920
Sup <sup>1</sup>	4.0%	9.4%	0.0%	
Hap <sup>2</sup>	92.0%	90.6%	88.9%	
Ang <sup>3</sup>	0.0%	0.0%	0.0%	
Anx <sup>4</sup>	1.3%	0.0%	5.6%	
Fri <sup>5</sup>	1.3%	0.0%	0.0%	
Sad <sup>6</sup>	1.3%	0.0%	5.6%	

Note. Differing superscripts would indicate pairwise comparisons that were significant after a modified Bonferroni correction for multiple groups ( $\alpha=0.016$ ) for all Chi-square comparisons for the emotion rated categorical variables. However, no significant omnibus Chi-square differences were observed at any of the task conditions.

<sup>1</sup> Sup: Surprise coded from the modified Facial Action Coding System

<sup>2</sup> Hap: Happiness coded from the modified Facial Action Coding System

<sup>3</sup> Ang: Anger coded from the modified Facial Action Coding System

<sup>4</sup> Anx: Anxiety coded from the modified Facial Action Coding System

<sup>5</sup> Fri: Fear coded from the modified Facial Action Coding System

<sup>6</sup> Sad: Sad coded from the modified Facial Action Coding System

**Table 5.** Study 1: Facial Action Coding System ratings scores according to task condition for ADHD and control groups

Variable	Control (n=24)	ADHD		M.E. Cond <sup>1</sup>	M.E. Group <sup>2</sup>	Interact <sup>3</sup>
		ADHD only (n=20)	ADHD+CU (n=8)	F-value	F-value	F-value
<b>FACS<sup>4</sup> Frequency</b>						
Surprise				5.051*	0.008	0.775
Neg. ind. <sup>5</sup>	1.21 (2.11)	1.93 (2.84)	1.00 (1.07)			
Neg. sup. <sup>6</sup>	0.46 (1.10)	0.20 (0.56)	0.25 (0.71)			
Baseline <sup>7</sup>	1.21 (1.49)	0.60 (1.21)	1.25 (2.06)			
Pos. ind. <sup>8</sup>	0.26 (0.68)	0.40 (1.12)	1.0 (1.82)			
Pos. sup. <sup>9</sup>	0.21 (0.41)	0.40 (0.73)	0.38 (0.52)			
Happy				35.965*	1.475	0.513
Neg. ind.	2.88 (2.09)	3.13 (2.27)	3.25 (2.49)			
Neg. sup.	0.04 (0.20)	0.10 (1.55)	0.01 (0.01)			
Baseline	0.25 (0.61)	1.00 (2.10)	0.50 (0.76)			
Pos. ind.	3.92 (2.54)	4.54 (2.20)	5.00 (2.62)			
Pos. sup.	0.71 (1.04)	1.33 (1.18)	0.25 (0.71)			
Angry				4.288*	1.390	1.047
Neg. ind.	0.54 (0.88)	0.87 (1.77)	1.25 (2.19)			
Neg. sup.	0.21(0.66)	0.13 (0.52)	1.00 (2.14)			
Baseline	0.33 (0.56)	0.93 (1.58)	0.63 (1.76)			
Pos. ind.	0.01 (0.01)	0.07 (0.25)	0.38 (0.74)			
Pos. sup.	0.25 (0.85)	0.20 (0.77)	0.01 (0.01)			
Anxious				19.786*	1.078	1.931
Neg. ind.	3.25 (2.92)	7.50 (3.59)	3.93 (2.37)			
Neg. sup.	2.50(2.96)	1.75 (2.13)	1.00 (1.18)			
Baseline	3.38 (3.41)	4.38 (3.42)	4.33 (4.95)			
Pos. ind.	1.58 (1.28)	2.07 (1.58)	1.63 (1.74)			
Pos. sup.	1.67 (2.12)	2.07 (1.44)	1.75 (2.05)			
Fright				19.477*	0.113	0.347
Neg. ind.	1.67 (2.01)	1.60 (1.54)	1.12 (2.10)			
Neg. sup.	0.08(0.28)	0.01 (0.01)	0.01 (0.01)			
Baseline	0.13 (0.49)	0.37 (1.06)	0.07 (0.26)			
Pos. ind.	0.01 (0.01)	0.01 (0.01)	0.01 (0.01)			
Pos. sup.	0.01 (0.01)	0.01 (0.01)	0.01 (0.01)			
Sad				2.147	0.697	1.836
Neg. ind.	1.08 (1.06)	2.00 (1.93)	0.50 (0.84)			
Neg. sup.	0.33(0.56)	0.73 (0.88)	0.75 (0.88)			
Baseline	1.00 (1.93)	0.80 (1.08)	0.75 (1.16)			
Pos. ind.	0.67 (0.91)	1.13 (1.30)	0.63 (0.74)			
Pos. sup.	0.50 (0.88)	0.88 (1.36)	0.33 (0.61)			
Any Emotion				36.449*	1.499	1.197
Neg. ind.	10.63 (4.90)	16.13 (7.64)	11.93 (7.11)			
Neg. sup.	3.63 (3.97)	3.75 (3.28)	2.47 (2.44)			
Baseline	6.80 (4.71)	8.77 (5.59)	8.25 (8.25)			
Pos. ind.	6.33 (2.76)	7.73 (3.21)	7.63 (3.26)			
Pos. sup.	3.33 (2.71)	4.33 (2.26)	3.25 (2.76)			

Note. Repeated measures ANOVA was used to examine differences based on task condition (ME Cond),

group (ME Group), and the interaction of task condition by group (Interact). Significant differences were observed in each of the coded facial affective behaviors according to task condition, with the exception of sadness. A review of the means shows that differences were in the expected directions. No significant differences were observed in the main effect of group or the task condition\*group level interactions, suggesting the task manipulations were equally effective for each group.

<sup>1</sup> ME Cond: Main effect of condition (i.e., task type)

<sup>2</sup> ME Group: Main effect of group (i.e., ADHD or control)

<sup>3</sup> Interact: Interaction effect of condition\*group

<sup>4</sup> FACS: Facial Action Coding System

<sup>5</sup> Neg, ind.: Negative Induction task condition

<sup>6</sup> Negsup.: Negative Suppression task condition

<sup>7</sup> Baseline.: Neutral Baseline task condition

<sup>8</sup> Pos, ind.: Positive Induction task condition

<sup>9</sup> Pos. sup.: Postive Suppression task condition

**Table 6.** *Study 1: Respiratory sinus arrhythmia (RSA; ms<sup>2</sup>) and pre-ejection period (PEP; ms) by task epochs for ADHD and control groups*

Variable	Control (n=75)	ADHD	
		ADHD only (n=54)	ADHD+CU (n=21)
<b>Baseline physiology data</b>			
Rest baseline			
RSA	7.07 (0.86)	7.22 (0.93)	7.23 (0.83)
PEP	98.64 (8.07)	96.02 (8.27)	99.55 (6.91)
Picture baseline 1			
RSA	6.66 (0.86)	6.93 (0.93)	6.99 (0.83)
PEP	97.38 (5.58)	93.04 (6.24)	101.10 (5.14)
<b>Task physiology raw scores</b>			
Negative induction			
RSA	6.98 (0.80)	7.21 (0.67)	7.28 (0.67)
PEP	97.59 (7.49)	94.40 (8.02)	101.17 (5.73)
Negative suppression			
RSA	7.12 (0.75)	7.25 (0.75)	7.00 (0.66)
PEP	97.45 (7.59)	95.09 (7.71)	99.31 (5.44)
Positive induction			
RSA	6.61 (0.79)	7.09 (0.83)	6.98 (0.71)
PEP	98.18 (7.33)	94.21 (7.55)	98.04 (5.35)
Positive suppression			
RSA	6.90 (0.79)	7.23 (0.82)	7.14 (0.74)
PEP	98.18 (7.45)	95.51 (8.27)	98.34 (6.32)
<b>Task physiology change scores</b>			
Negative induction			
RSA	0.33 (0.48)	0.25 (0.28)	0.06 (0.33)
PEP	0.61 (4.82)	0.98 (4.22)	1.58 (4.96)
Negative suppression			
RSA	0.43 (0.52)	0.32 (0.58)	0.06 (0.48)
PEP	0.59 (4.59)	1.58 (4.62)	0.62 (4.34)
Positive induction			
RSA	-0.05 (1.12)	0.153 (0.33)	-0.05 (0.35)
PEP	0.88 (3.75)	1.51 (3.74)	-0.71 (4.55)
Positive suppression			
RSA	0.25 (0.58)	0.26 (0.58)	-0.06 (0.43)
PEP	1.16 (4.09)	1.68 (4.49)	-0.11 (3.86)

*Note.* Repeated measures ANOVA was used to examine group differences in Respiratory Sinus Arrhythmia (RSA) and cardiac Pre-ejection Period (PEP) reactivity scores across the emotion induction and suppression conditions. Group comparisons were conducted using mixed model ANOVA decomposed as recommended by Keppel (2011). Thus, simple effects for RSA and PEP were only tested when justified by the results of higher order effects, and no further corrections were needed (Keppel, 2011). Each row under each heading represents next time point in design. Time increasing down table for the repeated measures design.

**Table 7. Study 2: Descriptive and diagnostic statistics for ADHD and control groups**

Variable	Control	ADHD	p-Value	eta <sup>2</sup>
Partial-	(n=50)	(n=50)		
<b>Demographics</b>				
Age (months; mean, SD)	103.51 (15.04)	102.64 (14.69)	0.775	0.001
Gender (% male)	52.0%	72.0%	0.015*	0.057
Race (% White)	76.0%	80.0%	0.632	0.003
Fam. Income (\$K; mean, SD)	99.5 (27.52)	78.13 (24.90)	0.016*	0.060
% 2 parent homes	93.0%	80.0%	0.093	0.013
Stimulant Med. (% on med.)	2.0%	42.0%	< 0.001*	0.135
WISC-IV <sup>1</sup> FSIQ <sup>2</sup> (mean, SD)	111.33 (11.48)	108.54 (14.05)	0.162	0.024
<b>ADHD-RS- T-scores-Parent (mean, SD)</b>				
Hyperactive/Impulsive	43.65 (5.36)	71.61 (10.94)	< 0.001*	0.745
Inattentive	43.48 (6.05)	73.09 (12.40)	< 0.001*	0.719
Total	43.04 (5.34)	74.01 (11.10)	< 0.001*	0.779
Hyperactive Symptoms	0.22 (0.56)	5.63 (2.36)	< 0.001*	0.744
Inattentive Symptoms	0.12 (0.56)	6.12 (2.72)	< 0.001*	0.733
<b>ADHD-RS- T-scores-Teacher (mean, SD)</b>				
Hyperactive/Impulsive	41.14 (2.91)	56.96 (9.44)	< 0.001*	0.601
Inattentive	42.47 (2.40)	56.37 (9.68)	< 0.001*	0.533
Total	41.18 (2.46)	57.05 (8.74)	< 0.001*	0.642
Hyperactive Symptoms	0.07 (0.26)	3.81 (3.26)	< 0.001*	0.442
Inattentive Symptoms	0.07 (0.37)	4.4 (3.43)	< 0.001*	0.492
<b>Conner's 3rd-T-score-Parent (mean, SD)</b>				
Inattention	46.51 (6.17)	75.38 (10.02)	< 0.001*	0.764
Hyperactivity	46.68 (7.92)	78.87 (9.82)	< 0.001*	0.770
Learning Problems	45.90 (6.09)	63.15 (13.64)	< 0.001*	0.428
Executive Functioning	46.44 (6.06)	71.28 (11.48)	< 0.001*	0.668
Aggression	46.54 (3.52)	58.85 (14.54)	< 0.001*	0.285
Peer Relations	50.98 (10.55)	70.09 (16.99)	< 0.001*	0.321
<b>Conner's 3rd-Teacher (mean, SD)</b>				
Inattention	45.32 (5.01)	66.68 (9.70)	< 0.001*	0.678
Hyperactivity	47.05 (6.71)	70.37 (14.37)	< 0.001*	0.545
Learning Problems	43.95 (3.77)	58.32 (9.63)	< 0.001*	0.521
Aggression	47.07 (6.15)	58.63 (16.17)	< 0.001*	0.202
Peer Relations	48.82 (9.11)	67.71 (16.35)	< 0.001*	0.356
<b>SDQ<sup>3</sup> Subscales-Parent (mean, SD)</b>				
Emotional Symptoms	1.17 (1.43)	2.85 (2.51)	< 0.001*	0.156
Conduct Problems	0.40 (0.67)	2.78 (1.66)	< 0.001*	0.502
Hyperactivity	1.55 (1.61)	8.30 (1.69)	< 0.001*	0.804
Peer Problems	0.53 (0.86)	2.60 (2.04)	< 0.001*	0.331
Prosocial Behavior	9.03 (1.11)	7.35 (2.14)	< 0.001*	0.212
Total Difficulties	3.66 (2.71)	16.50 (5.76)	< 0.001*	0.695
Impact/Impairment score	0.02 (0.13)	2.85 (2.77)	< 0.001*	0.388

<b>SDQ<sup>3</sup> Subscales-Teacher (mean, SD)</b>				
Emotional Symptoms	0.62 (1.28)	1.78 (2.33)	< 0.001 <sup>*</sup>	0.094
Conduct Problems	0.15 (0.45)	1.98 (1.73)	< 0.001 <sup>*</sup>	
0.379				
Hyperactivity	1.33 (1.76)	7.13 (2.30)	< 0.001 <sup>*</sup>	0.722
Peer Problems	0.72 (1.15)	2.80 (2.14)	< 0.001 <sup>*</sup>	0.287
Prosocial Behavior	8.12 (2.32)	6.38 (2.17)	< 0.001 <sup>*</sup>	0.128
Total Difficulties	2.83 (2.93)	13.68 (5.56)	< 0.001	0.622
Impact/Impairment score	0.10 (0.45)	1.50 (1.50)	< 0.001 <sup>*</sup>	0.317
<b>Comorbid Disorders (%; K-SADS<sup>4,5</sup>)</b>				
Mood Disorder (lifetime)	0.0%	12.0%	0.104	0.018
Anxiety Disorder	26.0%	32.0%	0.651	0.004
Conduct Disorder (CD)	0.0%	4.0%	0.167	0.029
Oppositional Defiant Disorder (ODD)	0.0%	22.0%	< 0.001	0.181
<b>CD Symptoms</b> (mean, SD; K-SADS)	0.05 (0.21)	0.27 (0.59)	0.011 <sup>*</sup>	0.064
<b>ODD Symptoms</b> (mean, SD; K-SADS)	0.04 (0.20)	1.98 (2.21)	< 0.001	0.313
<b>Total Anxiety Sx</b> (mean, SD; K-SADS)	0.84 (1.46)	1.79 (2.76)	0.032 <sup>*</sup>	0.047

*Note.* Differing superscripts indicate pairwise comparisons that were significant after a modified Bonferroni correction for multiple groups ( $\alpha=0.025$ ) for continuous variables, including: age, family income, estimated full-scale IQ, SDQ parent and teacher sub-scales, and comorbid symptoms; and Chi-square comparisons for categorical variables, including: gender, race, parent marital status, child medication status, and comorbid disorders.

<sup>1</sup> WISC-IV: Wechsler Intelligence Scales for Children

<sup>2</sup> Full-Scale Intelligence Quotient (estimated)

<sup>3</sup> Strengths and Difficulties Questionnaire

<sup>4</sup> Kiddie Schedule of Affective Disorders and Schizophrenia

<sup>5</sup> 0% of the sample had autism, eating disorders, learning disorders, post-traumatic stress disorder, psychosis, or substance use disorders

**Table 8.** Study 2: Hierarchical linear model variables presented according to model tested

Outcome	Model 1		Model 2		Model 3	
	Approach <sup>1</sup>	Avoidance <sup>2</sup>	RSA <sup>3</sup>	PEP <sup>4</sup>	RSA <sup>3</sup>	PEP <sup>4</sup>
Intercept <sup>5</sup>	I.V. <sup>6</sup>	I.V.	I.V.	I.V.	I.V.	I.V.
Intercept*group <sup>7</sup>	I.V.	I.V.	I.V.	I.V.	I.V.	I.V.
Approach <sup>1</sup>	D.V. <sup>8</sup>	-	I.V.	I.V.	I.V.	I.V.
Approach*group <sup>9</sup>	-	-	I.V.	I.V.	I.V.	I.V.
Avoid <sup>2</sup>	-	D.V.	I.V.	I.V.	I.V.	I.V.
Avoid*group <sup>10</sup>	-	-	I.V.	I.V.	I.V.	I.V.
IndVSup <sup>11</sup>	-	-	I.V.	I.V.	-	-
IndVSup*group <sup>12</sup>	-	-	I.V.	I.V.	-	-
NegVPos <sup>13</sup>	-	-	-	-	I.V.	I.V.
NegVPos*group <sup>14</sup>	-	-	-	-	I.V.	I.V.
Appro*IndVSup <sup>15</sup>	-	-	I.V.	I.V.	-	-
Appro*IndVSup*group <sup>16</sup>	-	-	I.V.	I.V.	-	-
Avoid*IndVSup <sup>17</sup>	-	-	I.V.	I.V.	-	-
Avoid*IndVSup*group <sup>18</sup>	-	-	I.V.	I.V.	-	-
Appro*NegVPos <sup>19</sup>	-	-	-	-	I.V.	I.V.
Appro*NegVPos*group <sup>20</sup>	-	-	-	-	I.V.	I.V.
Avoid*NegVPos <sup>21</sup>	-	-	-	-	I.V.	I.V.
Avoid*NegVPos*group <sup>22</sup>	-	-	-	-	I.V.	I.V.
RSA <sup>3</sup>	-	-	D.V.	-	D.V.	-
PEP <sup>4</sup>	-	-	-	D.V.	-	D.V.

*Note.* Each of the proposed models examined according to which independent (IV) and dependent (DV) variables are being examined.

<sup>1</sup> Approach: Facial Action Coding System Approach Factor

<sup>2</sup> Avoidance (Avoid): Facial Action Coding System Avoidance Factor

<sup>3</sup> RSA: Respiratory Sinus Arrhythmia

<sup>4</sup> PEP: Cardiac Pre-ejection Period

<sup>5</sup> Intercept: The intercept of each of the individual models being examined

<sup>6</sup> I.V.: Represents that the specific variable being examined is an independent variable in the model being examined

<sup>7</sup> Intercept\*group: represents the overall main effect of group (i.e., ADHD versus control)

<sup>8</sup> D.V.: Represents the specific variable being examined is the dependent variable in the model being examined

<sup>9</sup> Approach\*group: Facial Action Coding System Approach Factor by Group (i.e., ADHD versus control) interaction

<sup>10</sup> Avoid\*group: Facial Action Coding System Avoidance Factor by Group (i.e., ADHD versus control) interaction

<sup>11</sup> IndVSup: Dummy coded induction versus suppression variable

<sup>12</sup> IndVSup\*group: Interaction of dummy coded induction versus suppression variable by Group (i.e., ADHD versus control)

<sup>13</sup> NegVPos: Dummy coded negative induction versus positive induction variable

<sup>14</sup> NegVPos\*group: Dummy coded negative induction versus positive induction variable by Group (i.e., ADHD versus control)

---

<sup>15</sup>Appro\*IndVSup: Facial Action Coding System Approach Factor by dummy coded induction versus suppression interaction

<sup>16</sup>Appro\*IndVSup\*group: Facial Action Coding System Approach Factor by dummy coded induction versus by Group (i.e., ADHD versus control)

<sup>17</sup>Avoid\*IndVSup: Facial Action Coding System Avoidance Factor by dummy coded induction versus suppression

<sup>18</sup>Avoid\*IndVSup\*group: Facial Action Coding System Avoidance Factor by dummy coded induction versus by Group (i.e., ADHD versus control)

<sup>19</sup>Appro\*NegVPos: Facial Action Coding System Approach Factor by dummy coded negative induction versus positive induction interaction

<sup>20</sup>Appro\*NegVPos\*group: Facial Action Coding System Approach Factor by dummy coded negative induction versus positive induction interaction by Group (i.e., ADHD versus control)

<sup>21</sup>Avoid\*NegVPos: Facial Action Coding System Avoidance Factor by dummy coded negative induction versus positive induction interaction

<sup>22</sup>Avoid\*NegVPos\*group: Facial Action Coding System Avoidance Factor by dummy coded negative induction versus positive induction interaction by Group (i.e., ADHD versus control)

**Table 9.** Study 2: Raw Facial Action Coding System ratings according to task condition by group

Variable	Control (n=50)	ADHD (n=50)	M.E. Cond <sup>1</sup>	M.E. Group <sup>2</sup>	Interact <sup>3</sup>
			F-value	F-value	F-value
Surprise			6.491*	0.413	1.124
Neg. ind. <sup>4</sup>	0.10 (0.84)	0.20 (0.36)			
Neg. sup. <sup>5</sup>	0.36 (0.89)	0.24 (0.62)			
Baseline <sup>6</sup>	0.05 (0.21)	0.08 (0.25)			
Pos. ind. <sup>7</sup>	0.28 (0.79)	0.34 (0.99)			
Pos. sup. <sup>8</sup>	0.29 (0.59)	0.56 (0.93)			
Happy			105.175*	0.021	1.527
Neg. ind.	2.97 (1.96)	2.37 (2.25)			
Neg. sup.	0.26 (0.69)	0.22 (0.96)			
Baseline	0.52 (0.88)	0.46 (1.32)			
Pos. ind.	4.52 (2.58)	4.78 (2.86)			
Pos. sup.	1.21 (1.68)	1.78 (1.98)			
Angry			5.742*	3.202	0.993
Neg. ind.	0.54 (0.86)	0.71 (1.52)			
Neg. sup.	0.17 (0.50)	0.46 (1.48)			
Baseline	0.40 (0.72)	0.90 (1.50)			
Pos. ind.	0.14 (0.51)	0.29 (0.84)			
Pos. sup.	0.21 (0.69)	0.24 (0.66)			
Anxious			6.717*	0.112	1.796
Neg. ind.	3.95 (3.29)	3.90 (3.67)			
Neg. sup.	4.85 (3.99)	3.29 (3.59)			
Baseline	5.65 (4.69)	6.34 (4.61)			
Pos. ind.	3.93 (4.35)	4.34 (4.58)			
Pos. sup.	4.67 (4.31)	4.17 (3.71)			
Fright			21.170*	0.309	0.273
Neg. ind.	0.05 (0.49)	0.20 (0.50)			
Neg. sup.	0.10 (0.55)	0.17 (1.09)			
Baseline	0.19 (0.85)	0.34 (1.09)			
Pos. ind.	0.01 (0.01)	0.01 (0.01)			
Pos. sup.	0.19 (1.20)	0.10 (0.63)			
Sad			3.657*	0.071	2.526
Neg. ind.	1.58 (1.70)	0.88 (1.50)			
Neg. sup.	0.90 (1.19)	1.71 (2.59)			
Baseline	0.60 (1.32)	0.68 (1.19)			
Pos. ind.	1.51 (1.81)	1.17 (1.99)			
Pos. sup.	1.10 (1.54)	0.98 (1.49)			
Any Emotion			16.094*	0.002	1.007
Neg. ind.	11.17 (5.33)	9.86 (6.78)			
Neg. sup.	6.63 (4.72)	6.10 (4.80)			
Baseline	8.41 (5.83)	9.41 (6.47)			
Pos. ind.	10.40 (5.52)	10.93 (5.16)			
Pos. sup.	7.68 (5.27)	7.83 (4.06)			

*Note.* Repeated measures ANOVA was used to examine differences based on task condition (ME Cond), group (ME Group), and the interaction of task condition by group (Interact). Significant differences were

observed in each of the coded facial affective behaviors according to task condition. A review of the means shows that differences were in the expected directions. No significant differences were observed in the main effect of group or the task condition\*group level interactions, suggesting the task manipulations were equally effective for each group.

<sup>1</sup> ME Cond: Main effect of condition (i.e., task type)

<sup>2</sup> ME Group: Main effect of group (i.e., ADHD or control)

<sup>3</sup> Interact: Interaction effect of condition\*group

<sup>4</sup> Neg, ind.: Negative Induction task condition

<sup>5</sup> Negsup.: Negative Suppression task condition

<sup>6</sup> Baseline.: Neutral Baseline task condition

<sup>7</sup> Pos, ind.: Positive Induction task condition

<sup>8</sup> Pos. sup.: Postive Suppression task condition

**Table 10.** *Study 2: Approach and Avoidance factor scores according to task conditions by group*

Variable	Control ( <i>n</i> =50)	ADHD ( <i>n</i> =50)
<b>FACS<sup>1</sup> Frequency</b>		
Approach		
Neg. ind. <sup>2</sup>	2.59 (1.54)	2.26 (2.36)
Neg. sup. <sup>3</sup>	0.31 (0.71)	0.48 (1.27)
Baseline <sup>4</sup>	0.78 (0.51)	0.68 (0.45)
Pos. ind. <sup>5</sup>	4.48 (1.95)	4.78 (2.35)
Pos. sup. <sup>6</sup>	1.05 (1.43)	1.51 (1.54)
<b>FACS<sup>1</sup> Frequency</b>		
Avoidance		
Neg. ind.	4.18 (3.16)	3.51 (2.96)
Neg. sup.	3.92 (2.79)	3.46 (2.81)
Baseline	0.52 (0.88)	0.46 (1.32)
Pos. ind.	1.84 (3.41)	1.96 (3.38)
Pos. sup.	2.17 (3.17)	1.72 (2.72)

**Note.** Group differences were not explored here, as they were examined using multilevel modeling in the primary analyses.

<sup>1</sup> FACS: Facial Action Coding System

<sup>2</sup> Neg. ind.: Negative Induction task condition

<sup>3</sup> Negsup.: Negative Suppression task condition

<sup>4</sup> Baseline.: Neutral Baseline task condition

<sup>5</sup> Pos. ind.: Positive Induction task condition

<sup>6</sup> Pos. sup.: Postive Suppression task condition

**Table 11.** Study 2: Respiratory sinus arrhythmia (RSA;  $ms^2$ ) and pre-ejection period (PEP; ms) by task epochs for ADHD and control groups

Variable	Control ( <i>n</i> =50)	ADHD ( <i>n</i> =50)
<b>Baseline physiology data</b>		
Rest baseline		
RSA	7.04 (1.13)	7.38 (1.15)
PEP	99.02 (10.14)	93.77 (10.04)
Picture baseline		
RSA	6.45 (1.09)	6.94 (1.33)
PEP	96.31 (9.33)	91.61 (9.84)
<b>Task physiology raw scores</b>		
Negative induction		
RSA	6.79 (1.09)	7.18 (1.24)
PEP	98.10 (10.40)	93.17 (10.49)
Negative suppression		
RSA	6.75 (1.22)	7.09 (1.28)
PEP	97.91 (9.93)	93.59 (10.10)
Positive induction		
RSA	6.53 (1.09)	6.91 (1.28)
PEP	98.93 (10.40)	93.71 (10.55)
Positive suppression		
RSA	6.68 (1.08)	7.26 (1.14)
PEP	98.18 (9.68)	93.58 (10.35)
<b>Task physiology change scores</b>		
Negative induction		
RSA	0.31 (0.63)	0.23 (0.59)
PEP	1.79 (4.99)	0.55 (7.05)
Negative suppression		
RSA	0.27 (0.52)	0.14 (0.66)
PEP	1.60 (5.29)	0.97 (7.53)
Positive induction		
RSA	0.08 (0.74)	-0.04 (0.71)
PEP	1.43 (5.64)	1.10 (9.27)
Positive suppression		
RSA	0.20 (0.63)	0.31 (0.52)
PEP	1.86 (6.22)	0.96 (7.77)

**Note.** Group differences in Respiratory Sinus Arrhythmia (RSA) and cardiac Pre-ejection Period (PEP) reactivity scores across the emotion induction and suppression conditions were not explored here, as they were examined using multilevel modeling in the primary analyses.

**Table 12.** Study 2: Hierarchical linear model results presented according to model tested

	Model 1		Model 2		Model 3	
Outcome	Approach <sup>1</sup>	Avoid <sup>2</sup>	RSA <sup>3</sup>	PEP <sup>4</sup>	RSA	PEP
Intercept <sup>5</sup>	0.64**	1.51**	6.87**	96.07**	6.82**	96.00**
Intercept*group <sup>6</sup>	-0.01	-0.09	0.20	-2.24*	0.3*	-2.07*
Approach	-	-	0.01	-0.13	0.02	-0.34
Approach*group <sup>7</sup>	-	-	0.01	0.03	-0.05	0.37
Avoid	-	-	0.02	0.09	0.01	0.11
Avoid*group <sup>8</sup>	-	-	-0.02	-0.21	-0.07*	0.18
IndVSup <sup>9</sup>	-	-	-0.07*	-0.22	-	-
IndVSup*group <sup>10</sup>	-	-	0.05	-0.71	-	-
NegVPos <sup>11</sup>	-	-	-	-	-0.17*	0.35
NegVPos*group <sup>12</sup>	-	-	-	-	0.03	-0.14
Appro*IndVSup <sup>13</sup>	-	-	0.03*	0.57*	-	-
Appro*IndVSup*group <sup>14</sup>	-	-	0.01	0.39	-	-
Avoid*IndVSup <sup>15</sup>	-	-	-0.03*	-0.45*	-	-
Avoid*IndVSup*group <sup>16</sup>	-	-	-0.01	0.23	-	-
Appro*NegVPos <sup>17</sup>	-	-	-	-	-0.05*	-0.53*
Appro*NegVPos*group <sup>18</sup>	-	-	-	-	-0.04*	0.39*
Avoid*NegVPos <sup>19</sup>	-	-	-	-	0.03	0.06
Avoid*NegVPos*group <sup>20</sup>	-	-	-	-	-0.01	0.18*

Note. Hierarchical Linear Modeling (HLM) was used to predict the outcome variables listed in each of the models above. The predictors included in each model have the relevant unstandardized beta weight for the predictor of interest.

“-“ indicates that the specific variable of interest was not included in the model of interest

\*\*indicates significance at the p<0.01 level

\*indicates significance at the p<0.05 level

<sup>1</sup> Approach: Facial Action Coding System Approach Factor

<sup>2</sup> Avoidance (Avoid): Facial Action Coding System Avoidance Factor

<sup>3</sup> RSA: Respiratory Sinus Arrhythmia

<sup>4</sup> PEP: Cardiac Pre-ejection Period

<sup>5</sup> Intercept: The intercept of each of the individual models being examined

<sup>6</sup> Intercept\*group: represents the overall main effect of group (i.e., ADHD versus control)

<sup>7</sup> Approach\*group: Facial Action Coding System Approach Factor by Group (i.e., ADHD versus control) interaction

<sup>8</sup> Avoid\*group: Facial Action Coding System Avoidance Factor by Group (i.e., ADHD versus control) interaction

<sup>9</sup> IndVSup: Dummy coded induction versus suppression variable

<sup>10</sup> IndVSup\*group: Interaction of dummy coded induction versus suppression variable by Group (i.e., ADHD versus control)

<sup>11</sup> NegVPos: Dummy coded negative induction versus positive induction variable

<sup>12</sup> NegVPos\*group: Dummy coded negative induction versus positive induction variable by Group (i.e., ADHD versus control)

<sup>13</sup> Appro\*IndVSup: Facial Action Coding System Approach Factor by dummy coded induction versus suppression interaction

<sup>14</sup> Appro\*IndVSup\*group: Facial Action Coding System Approach Factor by dummy coded induction versus by Group (i.e., ADHD versus control)

<sup>15</sup> Avoid\*IndVSup: Facial Action Coding System Avoidance Factor by dummy coded induction versus

suppression

<sup>16</sup>Avoid\*IndVSup\*group: Facial Action Coding System Avoidance Factor by dummy coded induction versus by Group (i.e., ADHD versus control)

<sup>17</sup>Appro\*NegVPos: Facial Action Coding System Approach Factor by dummy coded negative induction versus positive induction interaction

<sup>18</sup>Appro\*NegVPos\*group: Facial Action Coding System Approach Factor by dummy coded negative induction versus positive induction interaction by Group (i.e., ADHD versus control)

<sup>19</sup>Avoid\*NegVPos: Facial Action Coding System Avoidance Factor by dummy coded negative induction versus positive induction interaction

<sup>20</sup>Avoid\*NegVPos\*group: Facial Action Coding System Avoidance Factor by dummy coded negative induction versus positive induction interaction by Group (i.e., ADHD versus control)

**Table 13. Study 3: Descriptive and diagnostic statistics for ADHD and control groups**

Variable	Control	ADHD	p-Value	eta <sup>2</sup>
Partial-	(n=50)	(n=50)		
<b>Demographics</b>				
Age (months; mean, SD)	97.14 (7.03)	98.59 (9.47)	0.385	0.008
Gender (% male)	56.0%	70.0%	< 0.001*	0.124
Race (% White)	88.0%	82.0%	0.557	0.007
Fam. Income (\$K; mean, SD)	89.25 (25.71)	80.00 (26.90)	0.334	0.010
% 2 parent homes	86.0%	82.0%	0.846	0.003
Stimulant Med. (% on med.)	0.0%	45.0%	< 0.001*	0.178
WISC-IV <sup>1</sup> FSIQ <sup>2</sup> (mean, SD)	110.87 (12.18)	108.92 (13.60)	0.154	0.014
<b>ADHD-RS- T-scores-Parent (mean, SD)</b>				
Hyperactive/Impulsive	45.39 (7.85)	72.46 (14.33)	< 0.001*	0.586
Inattentive	45.30 (7.78)	74.26 (12.11)	< 0.001*	0.676
Total	44.99 (7.98)	75.05 (12.80)	< 0.001*	0.671
Hyperactive Symptoms	0.32 (0.87)	5.46 (2.54)	< 0.001*	0.655
Inattentive Symptoms	0.26 (0.72)	6.21 (2.57)	< 0.001*	0.721
<b>ADHD-RS- T-scores-Teacher (mean, SD)</b>				
Hyperactive/Impulsive	43.19 (2.73)	58.83 (9.12)	< 0.001*	0.596
Inattentive	41.93 (2.64)	56.07 (7.52)	< 0.001*	0.633
Total	42.05 (2.46)	57.95 (7.68)	< 0.001*	0.682
Hyperactive Symptoms	0.07 (0.25)	4.58 (3.16)	< 0.001*	0.526
Inattentive Symptoms	0.07 (0.32)	4.5 (3.04)	< 0.001*	0.536
<b>Conner's 3rd-T-score-Parent (mean, SD)</b>				
Inattention	47.22 (6.32)	74.36 (10.57)	< 0.001*	0.716
Hyperactivity	46.02 (6.98)	77.85 (10.58)	< 0.001*	0.765
Learning Problems	46.18 (6.98)	60.89 (11.93)	< 0.001*	0.370
Executive Functioning	46.58 (7.10)	70.40 (10.11)	< 0.001*	0.657
Aggression	46.10 (3.43)	59.45 (13.84)	< 0.001*	0.315
Peer Relations	50.98 (10.55)	70.09 (16.99)	< 0.001*	0.321
<b>Conner's 3rd-Teacher (mean, SD)</b>				
Inattention	44.80 (3.88)	65.56 (9.38)	< 0.001*	0.679
Hyperactivity	47.06 (6.48)	73.80 (15.11)	< 0.001*	0.574
Learning Problems	44.55 (4.10)	55.96 (8.06)	< 0.001*	0.447
Aggression	48.27 (8.24)	62.56 (16.87)	< 0.001*	0.227
Peer Relations	48.84 (7.53)	69.55 (15.39)	< 0.001*	0.427
<b>SDQ<sup>3</sup> Subscales-Parent (mean, SD)</b>				
Emotional Symptoms	1.00 (0.93)	2.17 (1.95)	< 0.001*	0.131
Conduct Problems	0.36 (0.69)	2.71 (1.92)	< 0.001*	0.406
Hyperactivity	1.92 (1.88)	8.33 (1.78)	< 0.001*	0.758
Peer Problems	0.78 (1.18)	2.63 (1.91)	< 0.001*	0.258
Prosocial Behavior	9.34 (1.00)	6.94 (2.18)	< 0.001*	0.342
Total Difficulties	4.06 (3.09)	15.83 (5.24)	< 0.001*	0.658
Impact/Impairment score	0.02 (0.14)	3.06 (2.44)	< 0.001*	0.450

<b>SDQ<sup>3</sup> Subscales-Teacher (mean, SD)</b>				
Emotional Symptoms	0.56 (1.13)	1.83 (1.98)	< 0.001 <sup>*</sup>	0.139
Conduct Problems	0.22 (0.62)	2.38 (2.28)	< 0.001 <sup>*</sup>	
<b>0.303</b>				
Hyperactivity	1.20 (1.58)	7.42 (2.27)	< 0.001 <sup>*</sup>	0.722
Peer Problems	0.50 (0.84)	2.67 (2.17)	< 0.001 <sup>*</sup>	0.311
Prosocial Behavior	8.62 (1.50)	5.58 (2.51)	< 0.001 <sup>*</sup>	0.358
Total Difficulties	2.48 (2.37)	14.29 (5.93)	< 0.001	0.639
Impact/Impairment score	0.06 (0.42)	1.75 (1.45)	< 0.001 <sup>*</sup>	0.393
<b>Comorbid Disorders (%; K-SADS<sup>4,5</sup>)</b>				
Mood Disorder (lifetime)	2.0%	10.0%	0.254	0.028
Anxiety Disorder	18.0%	32.0%	0.165	0.026
Conduct Disorder (CD)	0.0%	2.0%	0.237	0.010
Oppositional Defiant Disorder (ODD)	0.0%	32.0%	< 0.001	0.235
<b>CD Symptoms</b> (mean, SD; K-SADS)	0.02 (0.14)	0.21 (0.49)	0.009 <sup>*</sup>	0.067
<b>ODD Symptoms</b> (mean, SD; K-SADS)	0.10 (0.42)	1.98 (2.08)	< 0.001	0.286
<b>Total Anxiety Sx</b> (mean, SD; K-SADS)	0.70 (1.51)	2.38 (4.33)	0.011 <sup>*</sup>	0.064

*Note.* Differing superscripts indicate pairwise comparisons that were significant after a modified Bonferroni correction for multiple groups ( $\alpha=0.025$ ) for continuous variables, including: age, family income, estimated full-scale IQ, SDQ parent and teacher sub-scales, and comorbid symptoms; and Chi-square comparisons for categorical variables, including: gender, race, parent marital status, child medication status, and comorbid disorders.

<sup>1</sup> WISC-IV: Wechsler Intelligence Scales for Children

<sup>2</sup> Full-Scale Intelligence Quotient (estimated)

<sup>3</sup> Strengths and Difficulties Questionnaire

<sup>4</sup> Kiddie Schedule of Affective Disorders and Schizophrenia

<sup>5</sup> 0% of the sample had autism, eating disorders, learning disorders, post-traumatic stress disorder, psychosis, or substance use disorders

**Table 14.** Study 3: Differences in Parent-Child Interaction System (PARCHISY) codes according to group

Variable	Control (n=50)	ADHD (n=50)	F-value
<b>PARCHISY--Parent Scores</b>			
Etch-A-Sketch			
Positive content	4.80 (1.26)	4.26 (1.14)	5.045 (0.049)*
Negative content	1.66 (0.96)	2.08 (1.35)	3.204 (0.032)
Positive affect	3.72 (1.28)	3.20 (1.21)	4.361(0.043)*
Negative affect	1.44 (0.93)	1.76 (1.17)	2.292 (0.023)
Responsiveness	4.84 (1.27)	4.60 (0.99)	1.110 (0.010)
On-task Behavior	6.38 (1.05)	6.50 (0.79)	0.421 (0.004)
Verbalizations	4.28 (0.81)	3.98 (0.80)	3.497 (0.034)
Reciprocity	4.30 (1.41)	3.54 (1.37)	7.412 (0.070)*
Conflict	1.28 (0.73)	1.52 (0.81)	2.410 (0.024)
Cooperation	4.48 (1.51)	4.02 (1.15)	2.921 (0.029)
Factor-Sensitivity	18.42 (5.03)	16.42 (3.90)	4.92 (0.045)*
Factor-Intrusiveness	4.38 (2.22)	5.36 (3.10)	3.03 (0.028)
Maze			
Positive content	3.88 (1.24)	3.50 (1.04)	2.769 (0.027)
Negative content	1.84 (1.29)	2.20 (1.28)	1.951 (0.020)
Positive affect	4.54 (1.19)	3.66 (1.27)	12.68(0.115)*
Negative affect	1.64 (1.06)	2.16 (1.20)	5.25 (0.051)*
Responsiveness	4.50 (1.15)	4.06 (0.94)	4.42 (0.043)*
On-task Behavior	6.68 (0.55)	6.68 (0.51)	0.001 (0.001)
Verbalizations	4.08 (0.75)	3.84 (0.68)	2.80 (0.028)
Reciprocity	4.02 (1.30)	3.28 (1.08)	9.511 (0.088)*
Conflict	1.28 (0.73)	1.68 (0.94)	6.23 (0.060)*
Cooperation	3.32 (1.25)	2.74 (0.99)	6.62 (0.063)*
Factor-Sensitivity	15.72 (4.12)	13.58 (3.28)	8.23 (0.081)*
Factor-Intrusiveness	23.78 (2.96)	21.62 (3.75)	10.185 (0.110)*
<b>PARCHISY—Child Scores</b>			
Etch-A-Sketch			
Positive affect	3.19 (1.12)	3.17 (0.92)	0.005 (0.001)
Negative affect	1.79 (0.94)	1.71 (1.03)	0.150 (0.002)
Responsiveness	5.49 (0.83)	5.34 (0.99)	0.547 (0.007)
On-task Behavior	6.26 (0.93)	6.00 (0.95)	2.612 (0.026)
Compliance	2.02 (0.96)	2.00 (1.27)	0.009 (0.001)
Autonomy	4.23 (1.23)	3.98 (1.31)	0.857 (0.010)
Activity	3.16 (0.75)	3.71 (1.17)	6.51 (0.074)*
Verbalizations	4.07 (0.78)	4.35 (1.03)	1.908 (0.023)
Factor-Agreeableness	-3.62 (1.45)	-3.75 (1.02)	0.045 (0.001)
Factor-Engagement	6.75 (1.81)	6.89 (0.89)	0.057 (0.001)
Maze			
Positive affect	3.95 (1.34)	3.83 (1.14)	0.208 (0.003)
Negative affect	2.16 (1.16)	2.27 (0.92)	0.213 (0.003)
Responsiveness	5.65 (0.72)	5.27 (0.81)	5.28 (0.060)*

On-task Behavior	5.49 (1.28)	5.12 (1.38)	1.592 (0.019)
Compliance	1.81 (0.76)	1.95 (0.89)	0.575 (0.007)
Autonomy	3.49 (1.12)	3.15 (1.04)	2.10 (0.025)
Activity	3.02 (0.64)	3.41 (0.84)	5.87 (0.067)
Verbalizations	4.23 (0.81)	4.17 (0.77)	0.128 (0.002)
Factor- <i>Agreeableness</i>	-5.62 (2.76)	-4.87 (2.78)	1.530 (0.019)
Factor- <i>Engagement</i>	6.51 (1.26)	6.56 (1.07)	0.037 (0.001)

---

*Note.* ANOVA results for group comparisons of Parent Child Interaction System (PARCHISY) coded parent and child behavior

\*indicates significance at the  $p < 0.05$  level

**Table 15.** Study 3: Differences in parent expressed emotion codes from the Five Minute Speech Sample according to group

Variable	Control (n=50)	ADHD (n=50)	$\chi^2$
Expressed Emotion Subgroup			38.030*
Low	30.0% <sup>a</sup>	8.0% <sup>b</sup>	
b <sup>1</sup> /Crit <sup>2</sup>	2.0% <sup>a</sup>	14% <sup>b</sup>	
b/EOI <sup>3</sup>	44.0% <sup>a</sup>	6.0% <sup>b</sup>	
b/Crit b/EOI	8.0% <sup>a</sup>	20.0% <sup>a</sup>	
Crit	4.0% <sup>a</sup>	16.0% <sup>b</sup>	
EOI	8.0% <sup>a</sup>	14.0% <sup>a</sup>	
Crit b/EOI	2.0% <sup>a</sup>	16.0% <sup>b</sup>	
EOI b/Crit	0.0% <sup>a</sup>	4.0% <sup>a</sup>	
Crit/EOI	2.0% <sup>a</sup>	2.0% <sup>a</sup>	
Expressed Emotion Final Rating			14.929*
Low	84.0% <sup>a</sup>	46.0% <sup>b</sup>	
High	16.0% <sup>a</sup>	54.0% <sup>b</sup>	

*Note.* Differing superscripts indicate pairwise comparisons that were significant after a modified Bonferroni correction for multiple groups ( $\alpha=0.025$ ) for Chi-square comparisons.

<sup>1</sup> b/: borderline rating of the associated dimension (i.e., Crit or EOI)

<sup>2</sup> Crit: Criticism rating from expressed emotion coding of the Five Minute Speech Sample

<sup>3</sup> EOI: Emotional Over-involvement rating from expressed emotion coding of the Five Minute Speech Sample

**Table 16.** Study 3: Child respiratory sinus arrhythmia (RSA;  $ms^2$ ) and pre-ejection period (PEP; ms) by task epoch for ADHD and control groups

Variable	Control ( <i>n</i> =50)	ADHD ( <i>n</i> =50)	F-val
<b>Baseline physiology data</b>			
Rest baseline			
RSA	7.41 (1.16)	7.52 (1.30)	0.109 (0.002)
PEP	99.61 (12.04)	95.19 (10.10)	6.993 (0.108)*
Picture baseline			
RSA	6.62 (1.22)	6.78 (1.01)	0.472 (0.005)
PEP	99.82 (10.90)	94.88 (9.32)	5.930 (0.057)*
<b>Task physiology raw scores</b>			
Etch-a-sketch			
RSA	6.39 (1.00)	6.57 (0.94)	0.824 (0.008)
PEP	100.41 (10.18)	95.04 (14.49)	3.847 (0.039)*
Marble Maze			
RSA	6.33 (0.92)	6.53 (0.79)	1.424 (0.014)
PEP	101.28 (9.21)	95.85 (11.52)	6.794 (0.065)*
<b>Task physiology change scores</b>			
Etch-A-Sketch			
RSA	0.23 (0.81)	0.20 (0.61)	0.043 (0.001)
PEP	0.22 (6.26)	0.35 (12.62)	0.004 (0.001)
Marble Maze			
RSA	0.29 (0.82)	0.24 (0.72)	0.109 (0.001)
PEP	1.46 (5.85)	0.97 (10.44)	0.086 (0.001)
Etch-to-Maze			
RSA	0.06 (0.48)	0.04 (0.58)	0.039 (0.001)
PEP	1.24 (3.73)	0.60 (5.86)	0.437 (0.004)

*Note.* Repeated measures ANOVA was used to examine group differences in Respiratory Sinus Arrhythmia (RSA) and cardiac Pre-ejection Period (PEP) reactivity scores across the emotion induction and suppression conditions. Group comparisons were conducted using mixed model ANOVA decomposed as recommended by Keppel (2011). Thus, simple effects for RSA and PEP were only tested when justified by the results of higher order effects, and no further corrections were needed (Keppel, 2011). Each row under each heading represents next time point in design. Time increasing down table for the repeated measures design.

**Table 17.** Study 3: Correlations among psychophysiological, parenting expressed emotion, parenting behavior, child symptoms, and child behavior

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
RSA <sup>1</sup>	1	.113	.013	.138	-.175	.015	-.114	.151	.196	.178	.106	.164	.314**	0.075	.082
PEP <sup>2</sup>		1	-.186	-.196	-.137	-.012	.034	-.097	-.192	-.147	.064	-.054	-.001	-.064	-.184
EE <sup>3</sup>			1	.709**	.546**	-.102	-.050	.357**	.342**	.359**	.061	.017	.296**	.086	.244*
Criticism <sup>4</sup>				1	-.133	-.140	-.079	.370**	.364**	.380**	-.111	.019	.283*	.233*	.076
EOI <sup>5</sup>					1	.098	.144	-.014	-.021	-.021	.077	.106	-.072	-.052	.187
Par Sensitivity <sup>6</sup>						1	.617**	-.203*	-.196	-.205*	-.169	.176	-.178	-.050	-.150
Par Intrusive <sup>7</sup>							1	.215*	.245*	.238*	-.337**	-.143	-.076	-.210*	.024
ADHD-RS HI <sup>8</sup>								1	.869**	.966**	.161	-.077	.486**	.223*	.263**
ADHD-RS Inatten <sup>9</sup>									1	.967**	.064	.050	.443**	.152	.253*
ADHD-RS Total <sup>10</sup>										1	.114	-.013	.479**	.190	.265**
Child Agree <sup>11</sup>											1	.007	.163	-.123	.062
Child Engage <sup>12</sup>												1	.102	.047	-.128
ODD Sx <sup>13</sup>													1	.241*	.271**
CD Sx <sup>14</sup>														1	.025
Anxiety Sx <sup>15</sup>															1

Note.

\*\* Correlation is significant at the 0.01 level (2-tailed).

\* Correlation is significant at the 0.05 level (2-tailed)

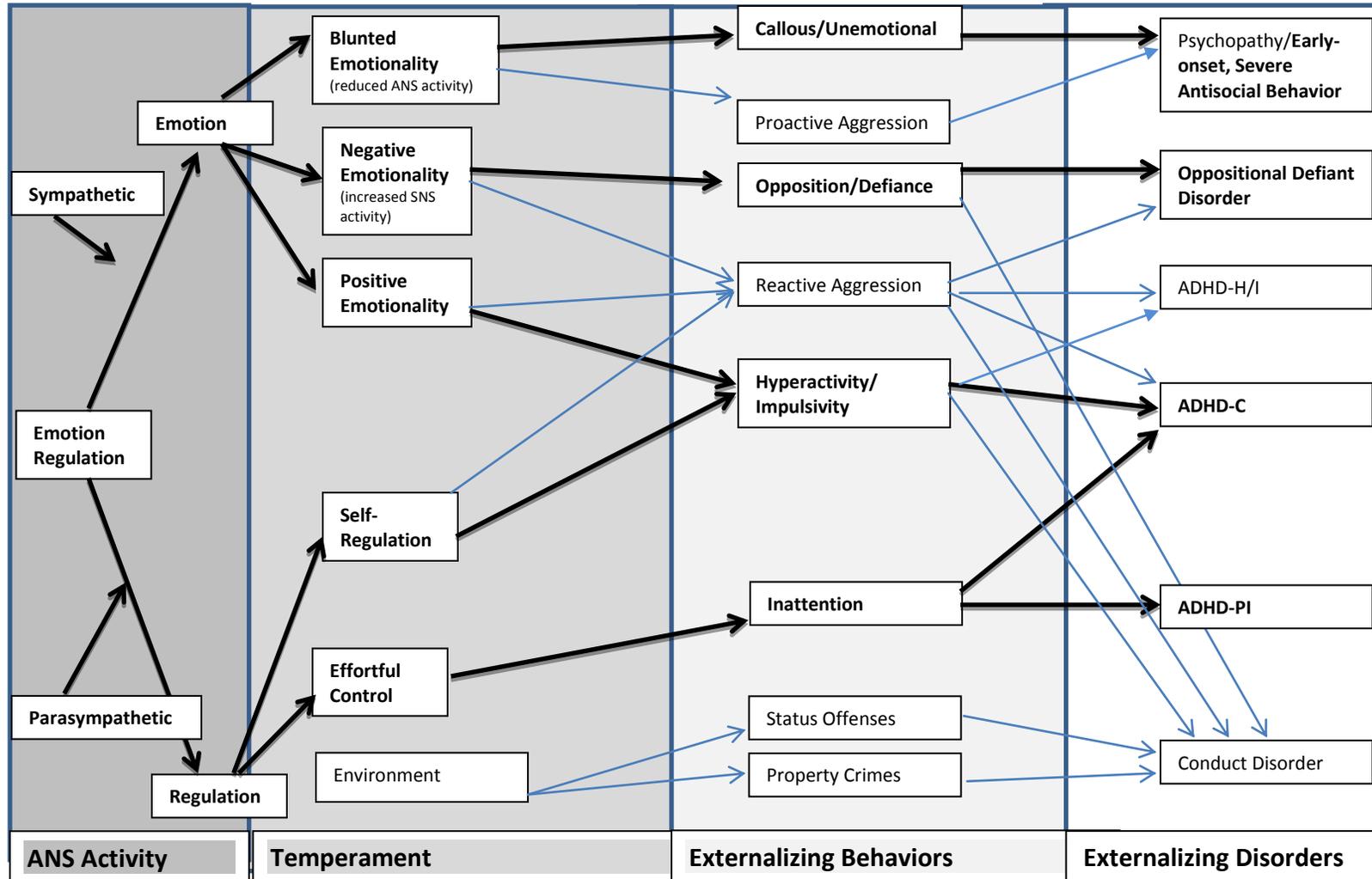
<sup>1</sup>RSA: Child Respiratory Sinus Arrhythmia—index of parasympathetic nervous system activity

- <sup>2</sup>PEP: Child cardiac Pre-ejection Period—index of sympathetic nervous system activity
- <sup>3</sup>EE: Parent Expressed Emotion—coded as low or high from the Five Minute Speech Sample
- <sup>4</sup>Criticism: Parent Criticism—sub-rating coded as low, borderline, or high from the Five Minute Speech Sample
- <sup>5</sup>EOI: Parent Emotional Over-involvement—sub-rating coded as low, borderline, or high from the Five Minute Speech Sample
- <sup>6</sup>Par Sensitivity: Parental Sensitivity—factor from ratings of the Parent Child Interaction System (PARCHISY)
- <sup>7</sup>Par Intrusive: Parental Intrusiveness—factor from ratings of the Parent Child Interaction System (PARCHISY)
- <sup>8</sup>ADHD-RS HI: Child hyperactive/impulsive symptoms from the ADHD Rating Scale
- <sup>9</sup>ADHD-RS Inatten: Child inattentive symptoms from the ADHD Rating Scale
- <sup>10</sup>ADHD-RS Total: Child total symptoms from the ADHD Rating Scale
- <sup>11</sup>Child Agree: Child Agreeableness—factor from ratings of the Parent Child Interaction System (PARCHISY)
- <sup>12</sup>Child Engage: Child Engagement—factor from ratings of the Parent Child Interaction System (PARCHISY)
- <sup>13</sup>ODD Sx: Child oppositional defiant disorder symptoms taken from the Kiddie Schedule of Affective Disorders and Schizophrenia (K-SADS)
- <sup>14</sup>CD Sx: Child conduct disorder symptoms taken from the K-SADS
- <sup>15</sup>Anxiety Sx: Child anxiety symptoms taken from the K-SADS

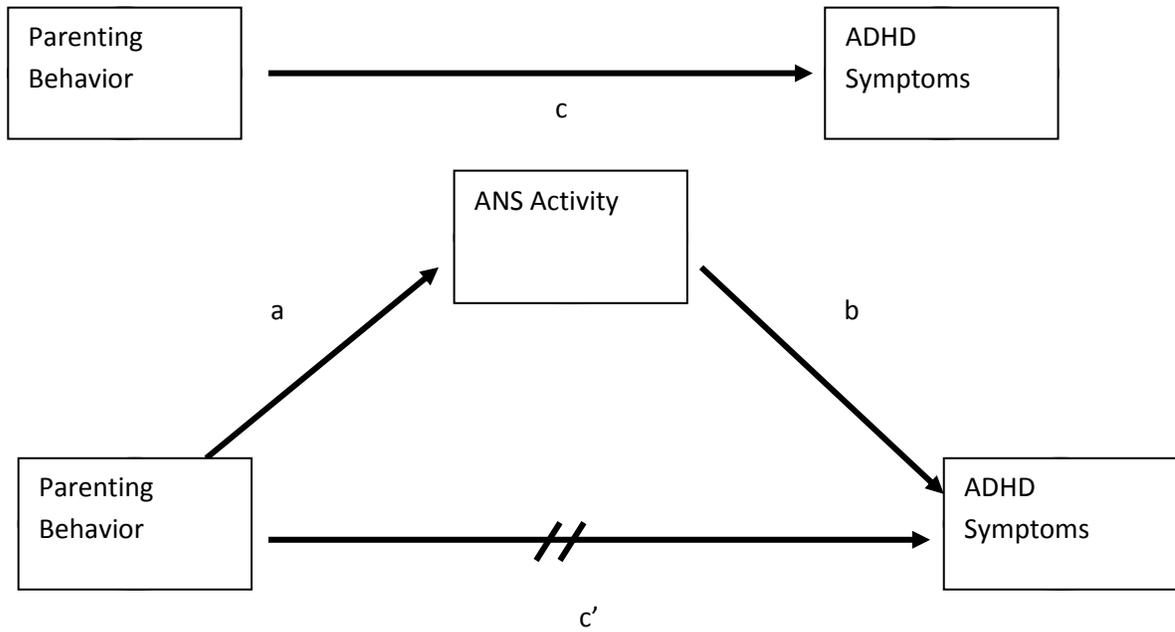
APPENDIX B

FIGURES

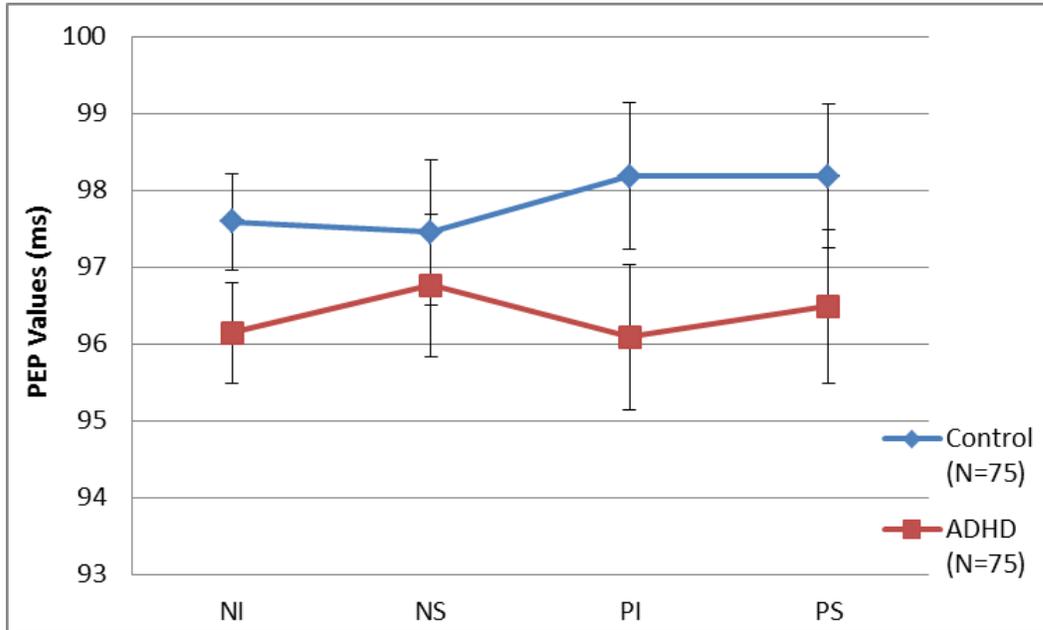
**Figure 1.** Unifying theoretical model of the roles of ANS, emotion, emotion regulation, temperament, parenting, and behavior in the development of externalizing pathology



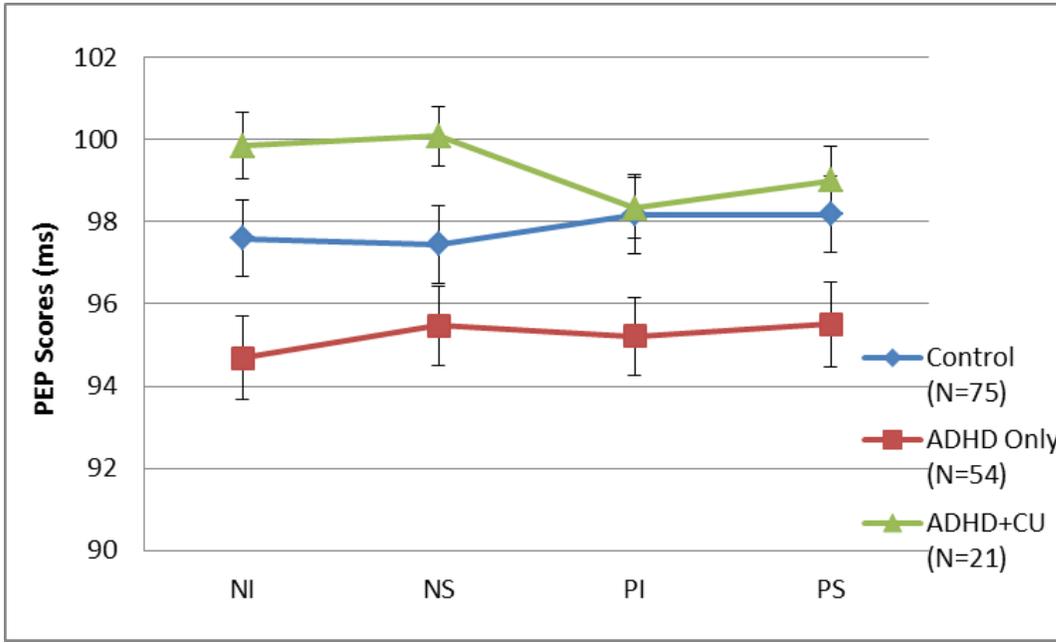
**Figure 2.** Descriptive model examining the mediating role of autonomic nervous system activity in the association between parenting behavior and ADHD symptoms.



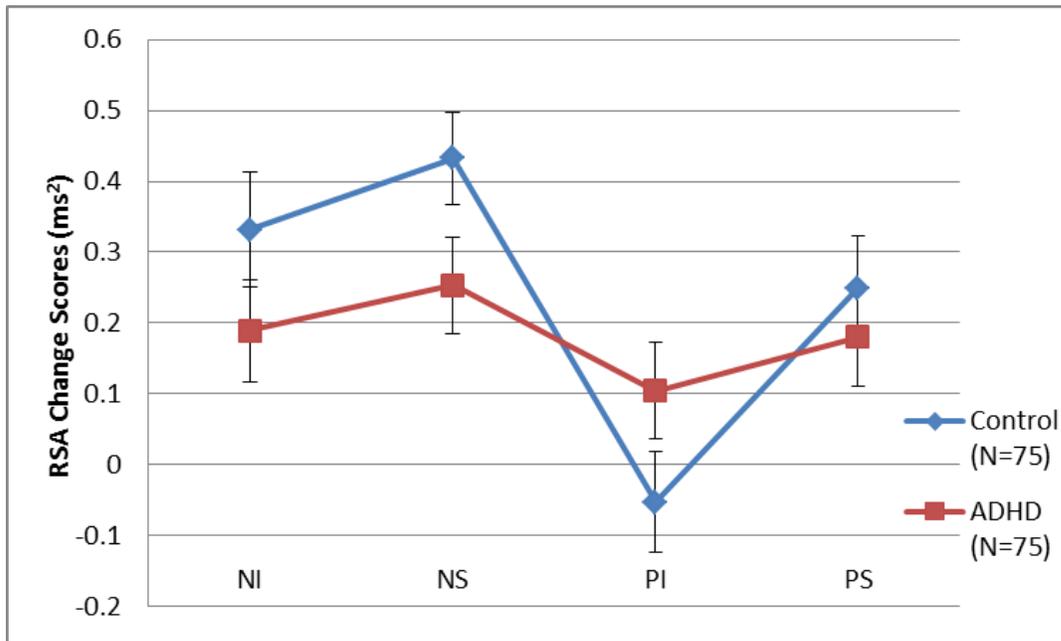
**Figure 3.** Study 1: Mean cardiac preejection period (PEP) raw scores for each of the task epochs: negative induction (NI), negative suppression (NS), positive induction (PI), and positive suppression (PS) for control and full ADHD (non-divided) groups.



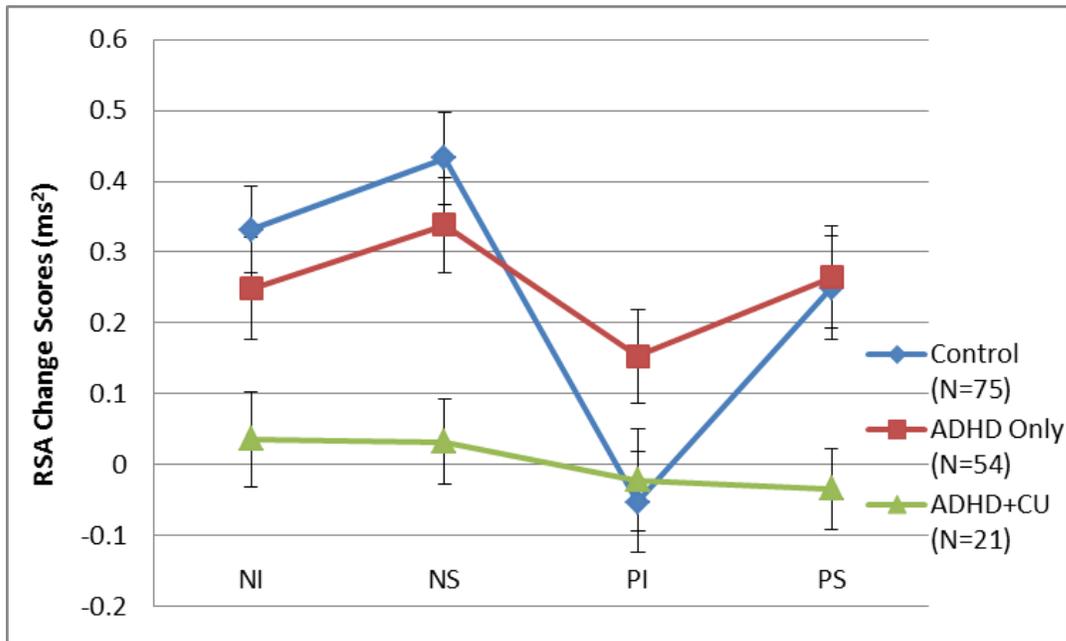
**Figure 4.** Study 1: Mean cardiac prejection period (PEP) raw scores for each of the task epochs: negative induction (NI), negative suppression (NS), positive induction (PI), and positive suppression (PS) for control, ADHD, and ADHD+CU groups.



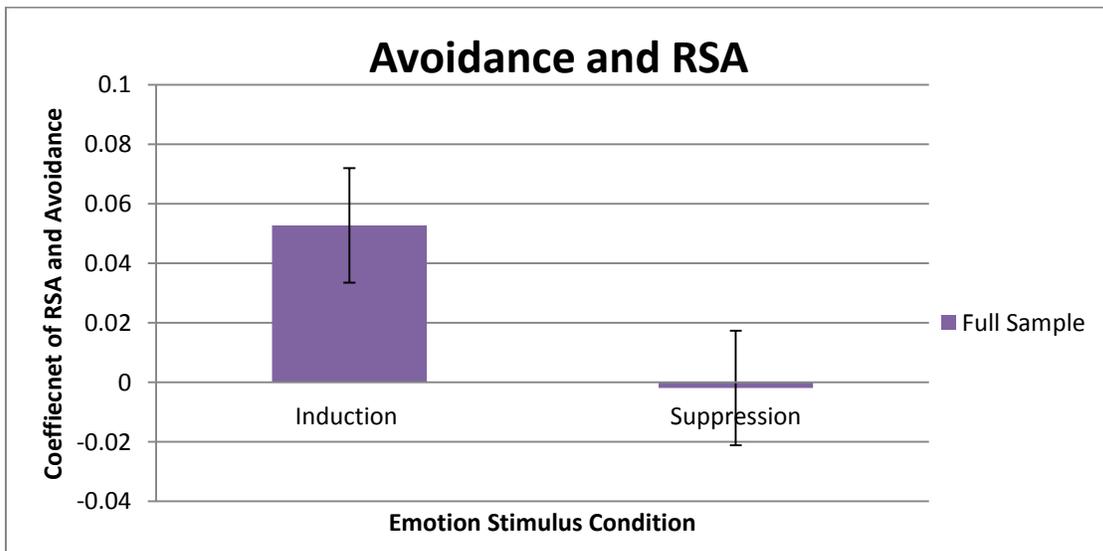
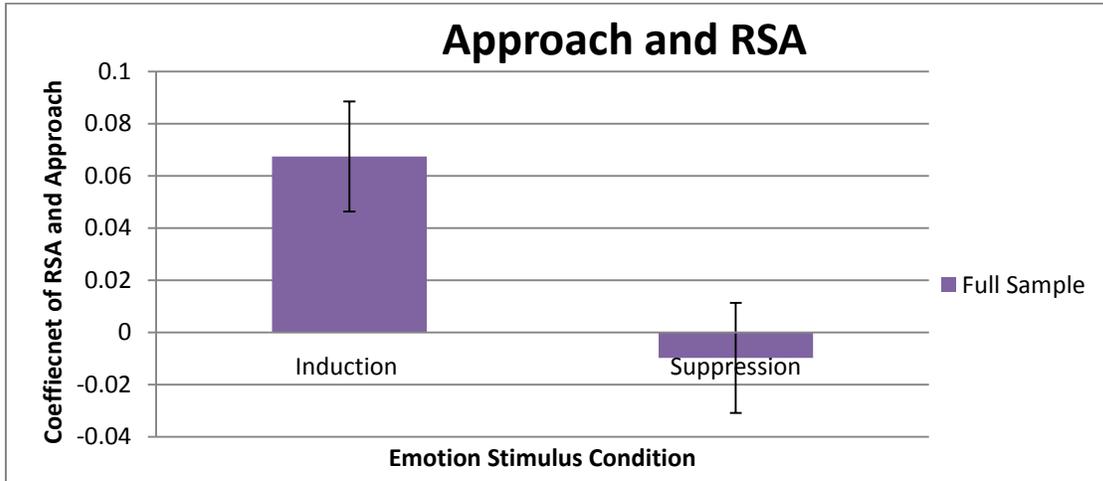
**Figure 5.** Study 1: Mean respiratory sinus arrhythmia (RSA) change scores from baseline to each of the task epochs: negative induction (NI), negative suppression (NS), positive induction (PI), and positive suppression (PS) for control and full ADHD (non-divided) groups.



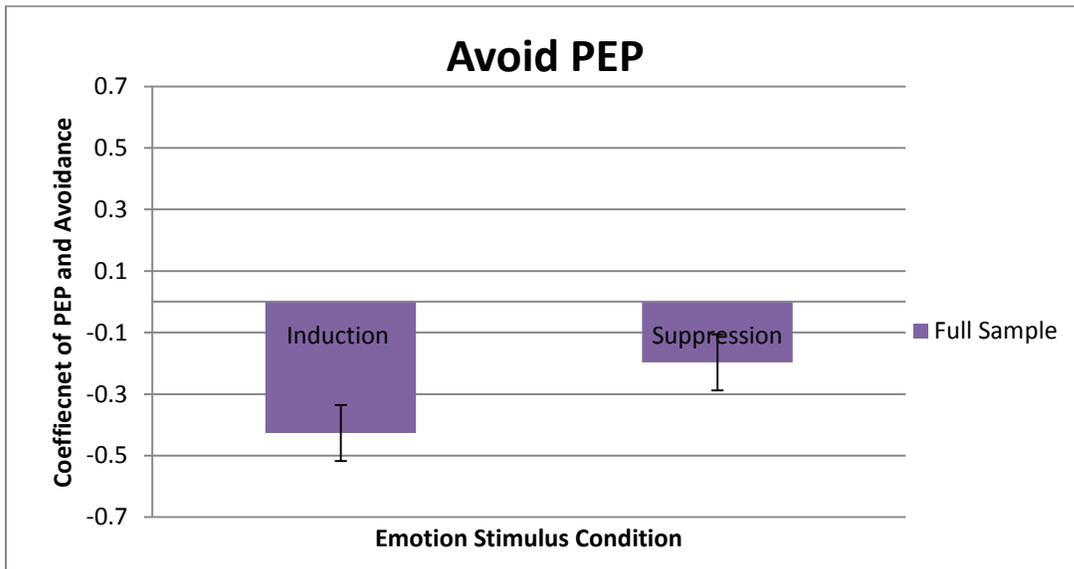
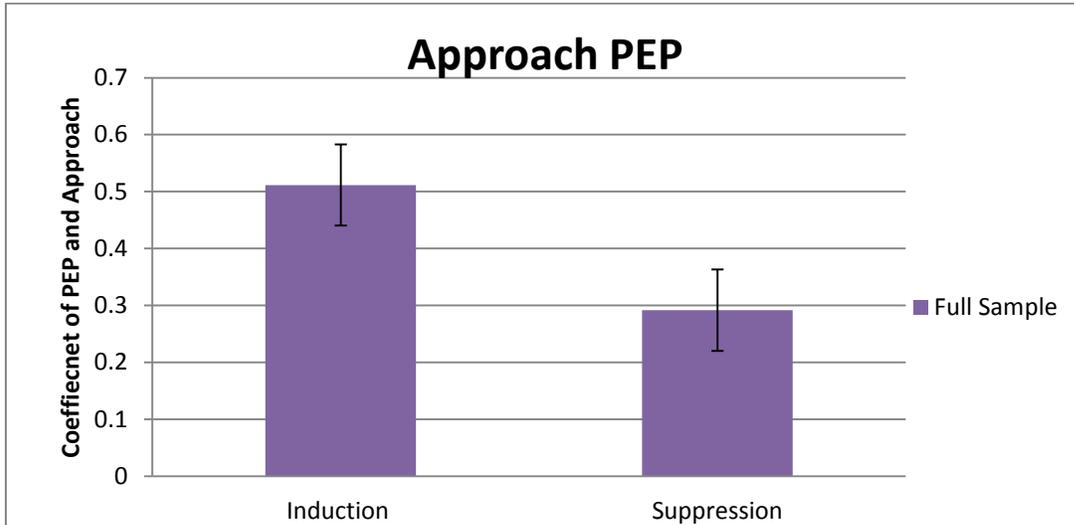
**Figure 6.** Study 1: Mean respiratory sinus arrhythmia (RSA) change scores from baseline to each of the task epochs: negative induction (NI), negative suppression (NS), positive induction (PI), and positive suppression (PS) for control, ADHD, and ADHD+CU groups.



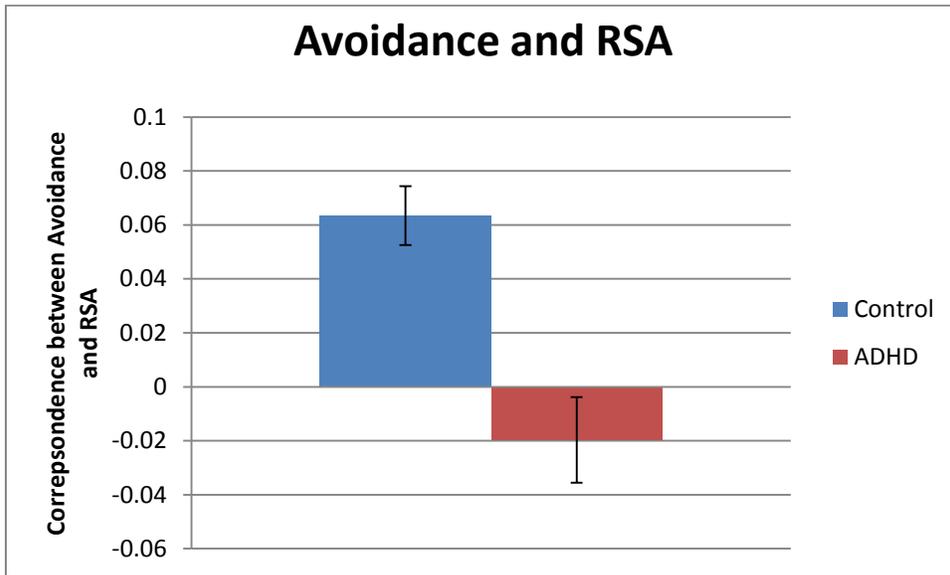
**Figure 7a and b.** Study 2: HLM results of correspondence (standardized beta weights) between facial affective behavior type (*Approach* and *Avoidance*) and respiratory sinus arrhythmia (RSA) during induction compared to during suppression.



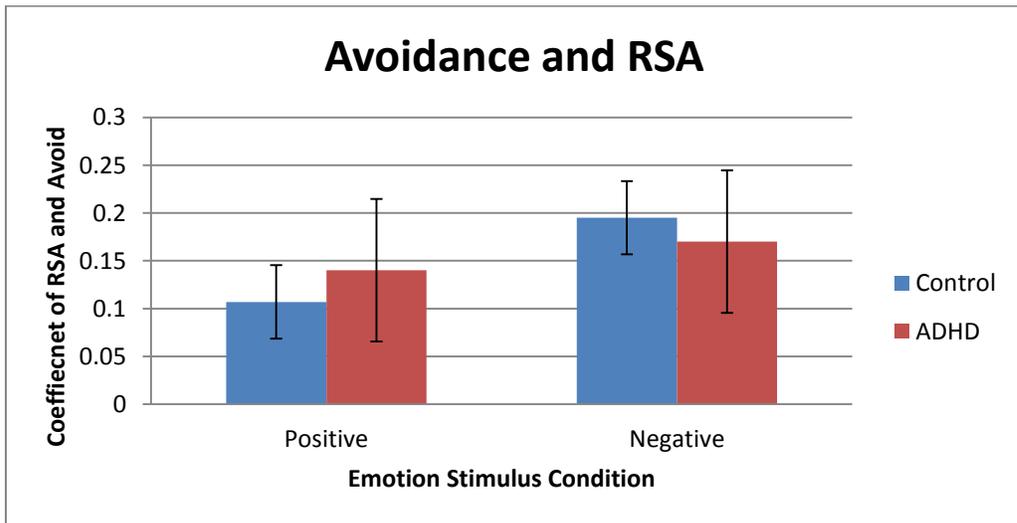
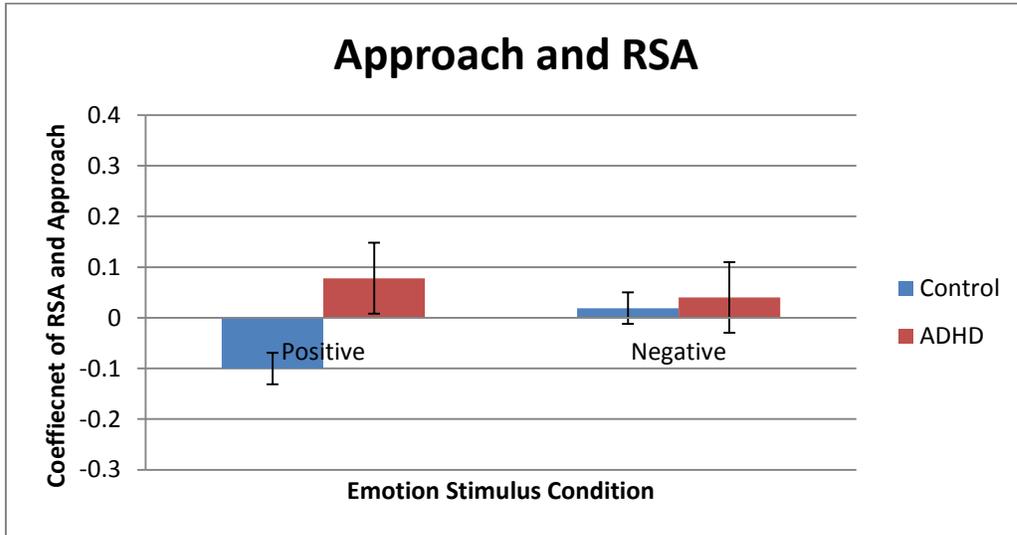
**Figure 8 a and b.** Study 2: HLM results of correspondence (standardized beta weights) between facial affective behavior type (*Approach* and *Avoidance*) and cardiac pre-ejection period (PEP) during induction compared to during suppression.



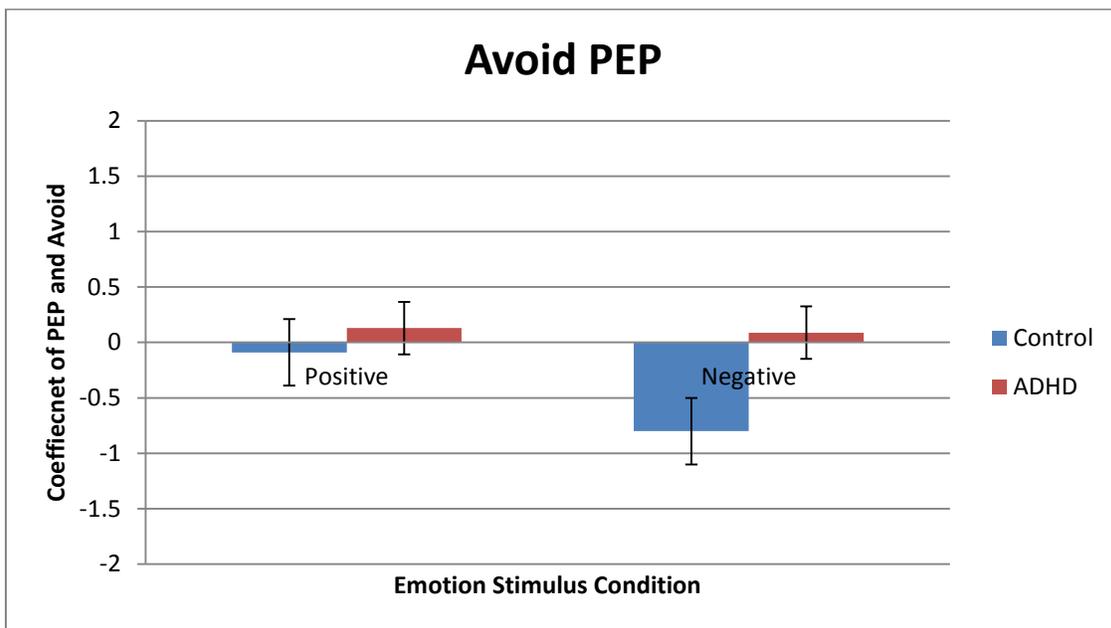
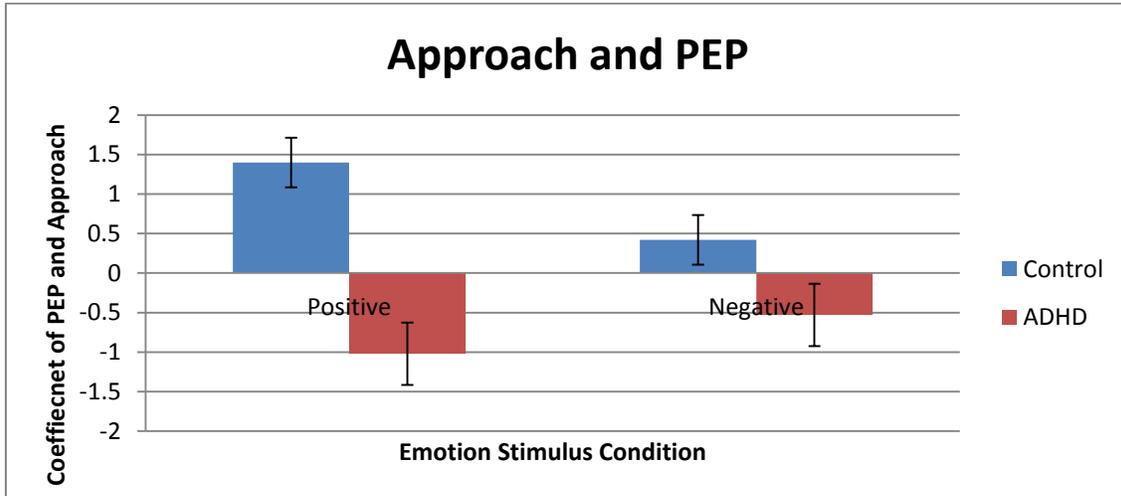
**Figure 9.** Study 2: HLM results of correspondence (standardized beta weights) between *Avoidance* facial affective behavior type and respiratory sinus arrhythmia (RSA) across induction conditions (negative and positive collapsed).



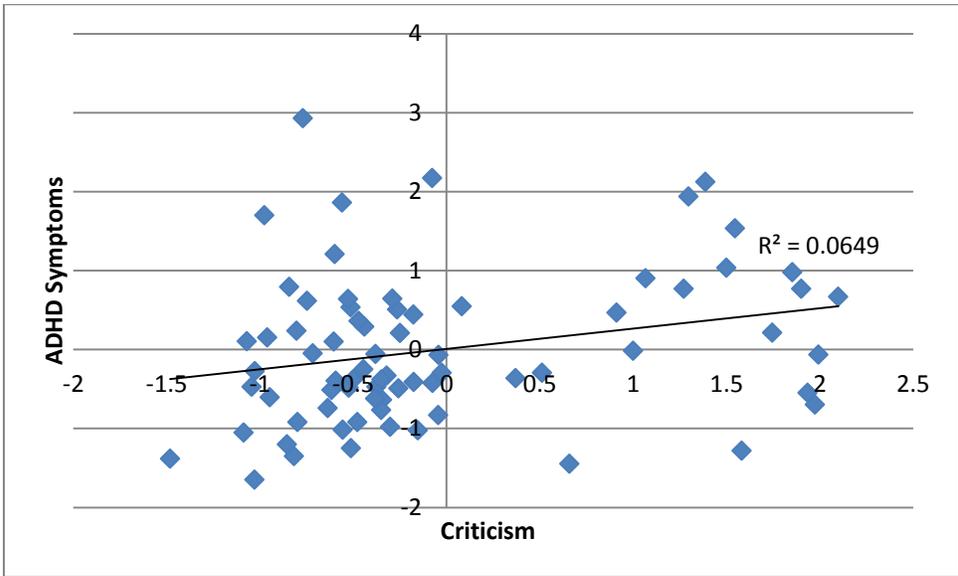
**Figure 10 a and b.** Study 2: HLM results of correspondence (standardized beta weights) between facial affective behavior type (*Approach* and *Avoidance*) and respiratory sinus arrhythmia (RSA) during positive induction compared to negative induction.



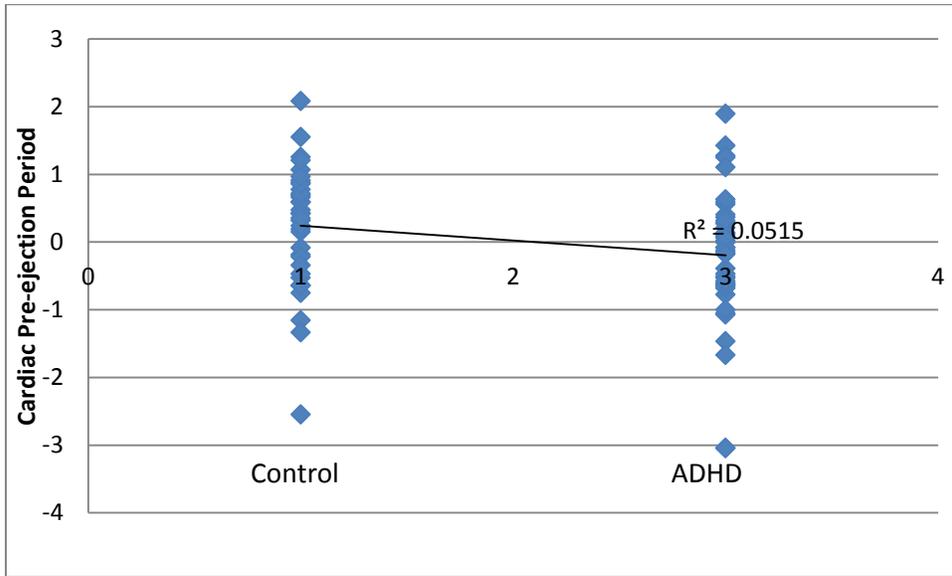
**Figure 11 a and b.** Study 2: HLM results of correspondence (standardized beta weights) between facial affective behavior type (*Approach* and *Avoidance*) and cardiac pre-ejection period (PEP) during positive induction compared to negative induction.



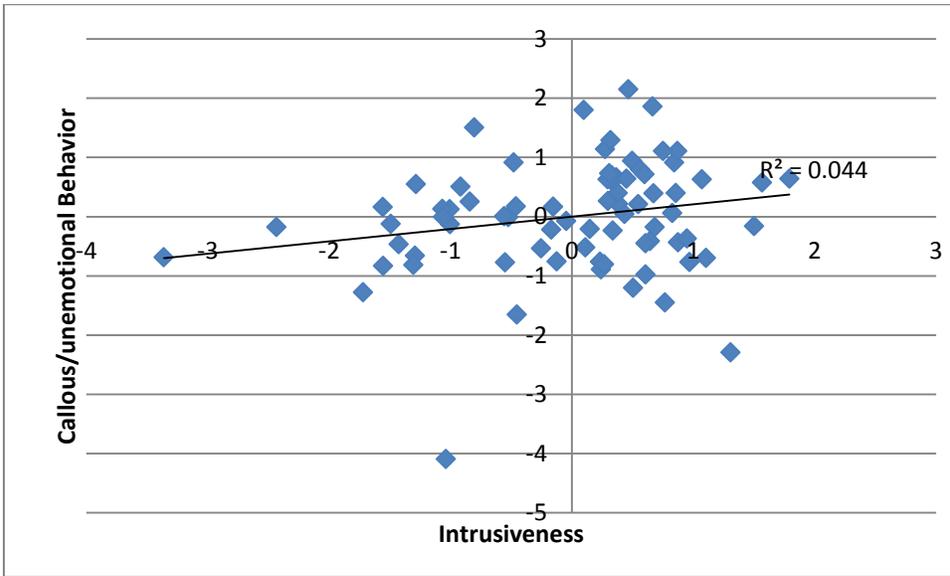
**Figure 12.** Study 3: Regression of parental expressed criticism on child attention-deficit/hyperactivity disorder symptoms



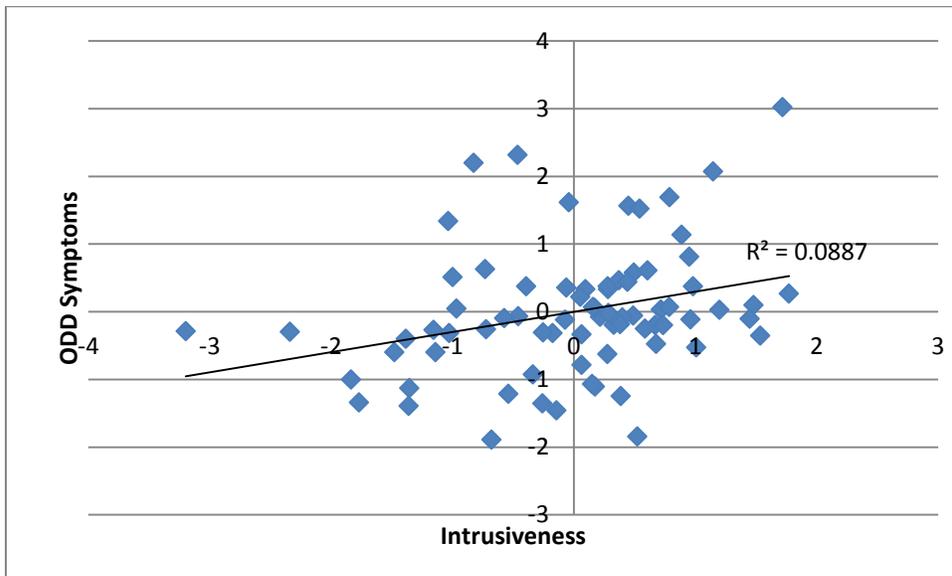
**Figure 13.** Study 3: Regression of child cardiac pre-ejection period (PEP) on child attention-deficit/hyperactivity disorder diagnosis



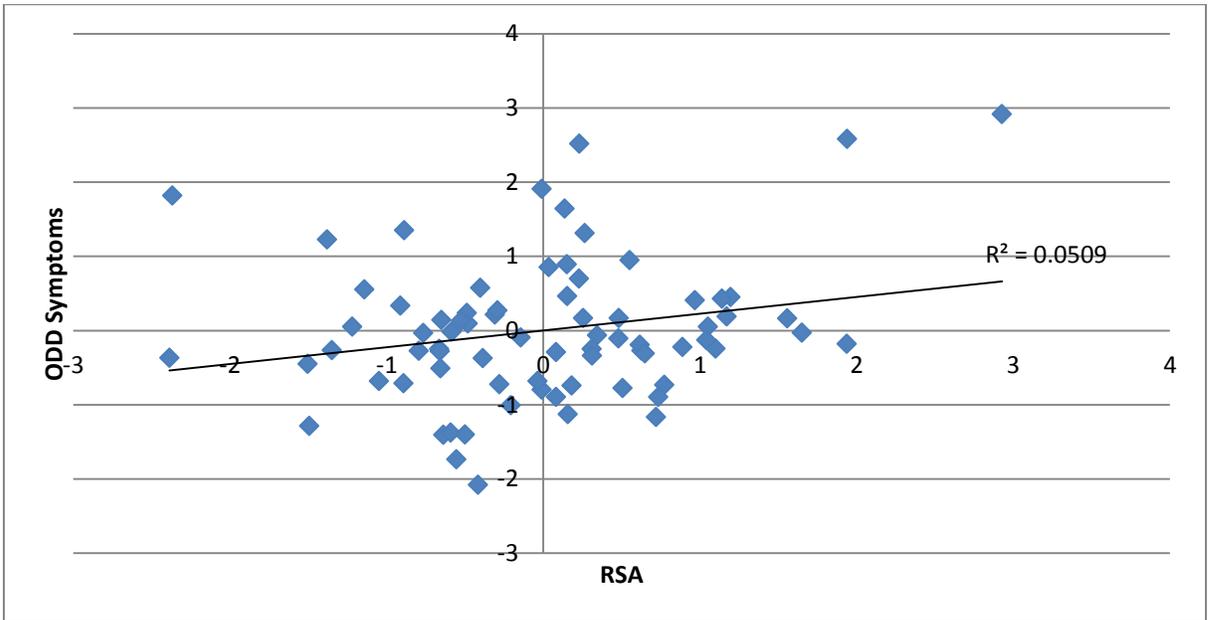
**Figure 14.** Study 3: Regression of parental intrusiveness on child callous/unemotional behavior symptoms



**Figure 15.** Study 3: Regression of parental intrusiveness on child oppositional defiant disorder symptoms



**Figure 16.** Study 3: Regression of child respiratory sinus arrhythmia (RSA) on child oppositional defiant disorder symptoms



## REFERENCES CITED

- Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs: General and Applied*, 80(7), 1-37.
- Achenbach, T. M., Conners, C. K., Quay, H. C., Verhulst, F. C., Howell, C. T. (1989). Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *Journal of Abnormal Child Psychology*, 17(3), 299-323.
- Anderson, C. A., Hinshaw, S. P., Simmel, C. (1994). Mother-child interactions in ADHD and comparison boys: Relationships with overt and covert externalizing behavior. *Journal of Abnormal Child Psychology*, 22(2), 247-265.
- Barkley, R. A. (1997). ADHD and the nature of self-control. New York, NY: Guilford Press.
- Barkley, R. A., & Fischer, M. (2010). The unique contribution of emotional impulsiveness to impairment in major life activities in hyperactive children as adults. *J Am Acad Child Adolesc Psychiatry*, 49(5), 503-513.
- Baron, R. M. & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychology research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51(6), 1173-1182.
- Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. *Dev Psychopathol*, 13(2), 183-214.
- Beauchaine, T. P., Katkin, E. S., Strassberg, Z., & Snarr, J. (2001). Disinhibitory psychopathology in male adolescents: Discriminating conduct disorder from attention-deficit/hyperactivity disorder through concurrent assessment of multiple autonomic states. *Journal of Abnormal Psychology*, 110(4), 610-624.
- Beauchaine, T. P. (2003). Taxometrics and developmental psychopathology. *Development and Psychopathology*, 15(3), 501-527.
- Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal Theory and developmental psychopathology: emotion dysregulation and conduct problems from preschool to adolescence. *Biol Psychol*, 74(2), 174-184.
- Beauchaine, T. P., Hinshaw, S.P., Pang, K. L. (2010). Comorbidity of attention-deficit/hyperactivity disorder and early-onset conduct disorder: Biological, environmental, and developmental mechanisms. *Clinical Psychology Science and Practice*, 17(4), 327-336.

- Bentler, P. M., & Chou, C. (1987). Practical issues in structural modeling. *Sociological Methods & Research*, 68, 78-117.
- Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology*, 34(6), 623-648.
- Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology*, 30(2), 183-196.
- Berntson, G. G., Lozano, D. L., Chen, Y. J., & Cacioppo, J. T. (2004). Where to Q in PEP. *Psychophysiology*, 41(2), 333-337.
- Blascovich, J., Mendes, W. B., Hunter, S. B., Salomon, K. (1999). Social “facilitation” as challenge and threat. *Journal of Personality and Social Psychology*, 77 (1), 68-77.
- Blascovich, J. & Mendes, W. B. (2008). Challenge and threat appraisals: The role of affective cues. *Handbook on Approach and Avoidance Motivation*. A.J. Elliot (Ed.), 59-82.
- Boyce, W. T., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., & Kupfer, D. J. (2001). Autonomic reactivity and psychopathology in middle childhood. *Br J Psychiatry*, 179, 144-150.
- Braaten, E.B. & Rosen, L. A. (2000). Self-regulation of affect in attention-deficit/hyperactivity disorder (ADHD) and non-ADHD boys: Differences in empathetic responding. *Journal of Consulting and Clinical Psychology*, 68(2), 313-321.
- Bradley, M. M., & Lang, P. J. (1994). Measuring emotion: the Self-Assessment Manikin and the Semantic Differential. *J Behav Ther Exp Psychiatry*, 25(1), 49-59.
- Bryk, A.S., & Raudenbush, S.W. (1992). *Hierarchical Linear Models in Social and Behavioral Research: Applications and Data Analysis Methods* (First Edition). Newbury Park, CA: Sage Publications.
- Burleson, M. H., Poehlmann, K. M., Hawkey, L. C., Ernst, J. M., Berntson, G. G., Malarkey, W. B., et al. (2003). Neuroendocrine and cardiovascular reactivity to stress in mid-aged and older women: long-term temporal consistency of individual differences. *Psychophysiology*, 40(3), 358-369.
- Burgess, K. B., Rubin, K. H., Dwyer, K. M. Hastings, P. D. (2003). Predicting preschoolers’ externalizing behavior from toddler temperament, conflict, and maternal negativity. *Developmental Psychology*, 39 (1), 164-176.

- Burt, S. A. and Mikolajewski, A. J. (2008), Preliminary evidence that specific candidate genes are associated with adolescent-onset antisocial behavior. *Aggr. Behav.*, 34: 437–445.
- Burt, S. A. (2012). How do we optimally conceptualize the heterogeneity within antisocial behavior? An argument for aggressive versus non-aggressive behavioral dimensions. *Clinical Psychology Review*, 32(4), 263-279.
- Cacioppo, J. T. (1994). Social neuroscience: autonomic, neuroendocrine, and immune responses to stress. *Psychophysiology*, 31(2), 113-128.
- Cadesky, E. B., Mota, V. L., & Schachar, R. J. (2000). Beyond words: how do children with ADHD and/or conduct problems process nonverbal information about affect? *J Am Acad Child Adolesc Psychiatry*, 39(9), 1160-1167.
- Calkins, S. D. (1997). Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. *Dev Psychobiol*, 31(2), 125-135.
- Calkins, S. D., Smith, C. L., Gill, K. L. and Johnson, M. C. (1998), Maternal Interactive Style Across Contexts: Relations to Emotional, Behavioral and Physiological Regulation During Toddlerhood. *Social Development*, 7: 350–369.
- Calkins, S.D. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development*, 72 (1), 1-21
- Calkins, S. D. and Keane, S. P. (2004), Cardiac vagal regulation across the preschool period: Stability, continuity, and implications for childhood adjustment. *Dev. Psychobiol.*, 45: 101–112.
- Calkins, S. D. & Keane, S. P. (2006). The relation of maternal behavior and attachment security to toddler's emotions and emotion regulation. *Research in Human Development*, 3(1):21-31
- Campbell, S. B., March, C. L., Pierce, E. W., Ewing, L. J., Szumowski, E. K. (1991). Hard-to-manage preschool boys: Family context and the stability of externalizing behavior. *Journal of Abnormal Child Psychology*, 19 (3), 301-318.
- Campbell, S. B., Shaw, D. S., Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology*, 12 (2) , 467-488.
- Canli, T., Zhao, Z., Desmond, J.E., Kange, E., Gross, J., Gabrieli, J.D.E. (2001). An fMRI study of personality influences on brain reactivity to emotional stimuli. *Behavioral Neuroscience*, 115(1), 33-42.

- Canli, T., Amin, Z., Haas, B., Omura, K., Constable, R.T. (2004). A double dissociation between mood states and personality traits in the anterior cingulate. *Behavioral Neuroscience*, 118(5), 897-904.
- Castelloe, J. M., & O'Brien, R. G. (2000). Power and sample size determination for linear models. SUGI Proceedings. Cary, NC: SAS Institute Inc..
- Chronis-Tuscano, A., Molina, B. S. G., Pelham, W. E., Applegate, B., Dahlke, A., Overmyer, M., & Lahey, B. B. (2010). Very early predictors of adolescent depression and suicide attempts in children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, 67(10), 1044-1051.
- Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112, 155-159.
- Cohen, D. & Strayer, J. (1996). Empathy in conduct-disordered and comparison youth. *Developmental Psychology*, 32(6), 988-998.
- Cole, P.M., Martin, S.E., & Dennis, T.A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, 75(2), 317-333.
- Conners, K. C. (2008). Conners 3rd Edition Manual. New York, NY: Multi-Health Systems, Inc.
- Cottrell, N.B., Wack, D.L., Sekerak, G.J., Rittle, R.H. (1968). Social facilitation of dominant responses by the presence of an audience and the mere presence of others. *Journal of Personality and Social Psychology*, 9(3), 245-250.
- Crowell, S. E., Beauchaine, T. P., Gatzke-Kopp, L., Sylvers, P., Mead, H., & Chipman-Chacon, J. (2006). Autonomic Correlates of Attention-Deficit/Hyperactivity Disorder and Oppositional Defiant Disorder in Preschool Children. *Journal of Abnormal Psychology*, 115(1), 174-178.
- Dadds MR., Fraser J., Frost A., Hawes DJ. Disentangling the underlying dimensions of psychopathy and conduct problems in childhood: A community study. *Journal of Consulting and Clinical Psychology*, 73(3): 400-410.
- Deater-Deckard, K. (2000). Parenting and child behavioral adjustment in early childhood: A quantitative genetic approach to studying family processes. *Child Development*, 71, 468-484.
- Deater-Deckard, K., & O'Connor, T. G. (2000). Parent-child mutuality in early childhood: Two behavioral genetic studies. *Developmental Psychology*, 36(5), 561-570.

- Deater-Deckard, K., & Petrill, S. A. (2004). Parent child dyadica mutuality and behaviors problems: An investigation of gene-environment processes. *Journal of Child Psychology and Psychiatry*, 45, 1171-1179.
- Deater-Deckard, K., Pylas, M., & Petrill, S. A. (1997). The parent-child interaction system (PARCHISY). London: Institute of Psychiatry.
- Derryberry, D. & Rothbart, M.K. (1997). Reactive and effortful processes in the organization of temperament. *Development and Psychopathology*, 9(4), 633-652.
- DuPaul, G. J., Power, T. J., Anastopoulos, A. D., & Reid, R. (1998). ADHD Rating Scale-IV: Checklists, Norms, & Clinical Interpretation.
- Eisenberg, N., Guthrie, I. K., Fabes, R. A., Reiser, M., Murphy, B. C., Holgren, R., et al. (1997). The relations of regulation and emotionality to resiliency and competent social functioning in elementary school children. *Child Dev*, 68(2), 295-311.
- Eisenberg, N., Cumberland, A., Spinrad, T., Fabes, R., Shepard, S.A., Reiser, M., Murphy, B.C., Losoya, S.H. & Guthrie, I.K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development*, 72, 1112-1134.
- Eisenberg, N., Champion, C., & Ma, Y. (2004). Emotion-related regulation: An emerging construction. *Merril-Palmer Quarterly*, 50, 236.
- Eisenberg, N. & Spinrad, T.L. (2005). Emotion-related regulation: Sharpening the definition. *Child Development*, 75, 334-339.
- Ekman, P. (1992a). An arguement for basic emotions. *Cognition and Emotion*, 6, 169-200.
- Ekman, P. (1992b). Facial expressions of emotion: an old controversy and new findings. *Philos Trans R Soc Lond B Biol Sci*, 335(1273), 63-69.
- El-Sheikh, M., Kouros, C. D., Erath, E., Cummings, E. M., Keller, P., & Staton, L. (2009). Marital conflict and children's externalizing behavior: Pathways involving interactions between parasympathetic and sympathetic nervous system activity. *Monographs of the Society of Research on Child Development*, 74(1), vii-79.
- Elia, J., Ambrosini, P., & Berrettini, W. (2008). ADHD characteristics: I. Concurrent co-morbidity patterns in children & adolescents. *Child Adolesc Psychiatry Ment Health*, 2(1), 15.
- Fabes, R. A. (1994). Physiological, emotional, and behavioral correlates of gender segregation. *New Dir Child Dev*(65), 19-34.

- Fabes, R. A., Eisenberg, N., Karbon, M., Troyer, D., & Switzer, G. (1994). The relations of children's emotion regulation to their vicarious emotional responses and comforting behaviors. *Child Dev*, 65(6), 1678-1693.
- Fan, X. (2003). Two approaches for correcting correlation attenuation caused by measurement error: Implications for research practice. *Educational and Psychological Measurement*, 63, 915.
- Fang, H. (2006). A Monte Carlo study of power analysis of hierarchical linear modeling and repeated measures approaches to longitudinal data analysis. Dissertation, Department of Educational Studies, Ohio University.
- Faraone, S.V., Biederman, J., & Monuteaux, M.C. (2000). Attention-deficit disorder and conduct disorder in girls: Evidence for a familial subtype. *Biological Psychiatry*, 48, 21 – 29.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G\*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences (Version 39): *Behavior Research Methods*.
- Fivush, R. (2000). Methodological challenges in the study of emotional socialization. *Psychological Inquiry*, 9 (4). 281-283.
- Frick, P.J. & Ellis, M. (1999). Callous-unemotional traits and subtypes of conduct disorder. *Clinical Child and Family Psychology Review*, 2, 149-168.
- Frick, P.J., Cornell, A.H., Barry, C.T., Bodin, S.D., & Dane, H.E. (2003). Callous-unemotional traits and conduct problems in the prediction of conduct problem severity, aggression, and self-report of delinquency. *Journal of Abnormal Child Psychology*, 31, 457-470.
- Frick, P.J. & Morris, A.S. (2004). Temperament and developmental pathways to conduct problems. *Journal of Clinical Child and Adolescent Psychology*, 33, 54-68.
- Frick, P.J. & Nigg, J.T. (2012). Current issues in the diagnosis of attention-deficit hyperactivity disorder, oppositional defiant disorder, and conduct disorder. *Annual Review of Clinical Psychology*.
- Frick, P.J., & Viding, E.M. (2009). Antisocial behavior from a developmental psychopathology perspective. *Development and Psychopathology*, 21, 1111-1131.
- Frick, P.J. & White, S.F. (2008). Research review: The importance of callous-unemotional traits for developmental modes of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry*, 49, 359-375.

- Frijda, N.H. (2008). The psychologists' point of view. In M. Lewis, J.M. Haviland-Jones, L. Feldman Barrett (Eds.), *Handbook of Emotions* 3rd Edition. New York: Guilford Press.
- Gatzke, L.M., & Raine, A. (2001). Treatment and prevention implications of antisocial personality disorder. *Current Psychiatric Reports*, 2, 1535-1645.
- Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biol Psychiatry*, 63(6), 577-586.
- Goodman, R. (2001). Psychometric properties of the strengths and difficulties questionnaire (SDQ). *Journal of the American Academy of Child & Adolescent Psychiatry*, 40, 1337-1345.
- Gross, & Levenson. (1993). Emotional suppression: physiology, self-report, and expressive behavior. *J Pers Soc Psychol*, 64(6), 970-986.
- Gross, & Levenson. (1997). Hiding feelings: the acute effects of inhibiting negative and positive emotion. *J Abnorm Psychol*, 106(1), 95-103.
- Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J Pers Soc Psychol*, 74(1), 224-237.
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J Pers Soc Psychol*, 85(2), 348-362.
- Hayano, J., Sakakibara, Y., Yamada, A., Yamada, M., Mukai, S., Fujinami, T., et al. (1991). Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. *Am J Cardiol*, 67(2), 199-204.
- Hayes, A. F., & Matthes, J. (2009). Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. *Behavior Research Methods*, 41, 924-936.
- Herpertz, S. C., Mueller, B., Qunaibi, M., Lichterfeld, C., Konrad, K., & Herpertz-Dahlmann, B. (2005). Response to emotional stimuli in boys with conduct disorder. *Am J Psychiatry*, 162(6), 1100-1107.
- Hibbs, E. D., Hamburger, S. D., Lenane, M., Rapoport, J. L., Kruesi, M. J. P., Keysor, C. S. and Goldstein, M. J. (1991), Determinants of Expressed Emotion in Families of Disturbed and Normal Children. *Journal of Child Psychology and Psychiatry*, 32: 757-770.
- Hinshaw, S. P., Zupan, B., Simmel, C., Nigg, J. T., & Melnick, S. M. (1997). Peer status in boys with and without attention-deficit hyperactivity disorder: Prediction from

- overt and covert antisocial behavior, social isolation, and authoritative parenting beliefs. *Child Development*, 68, 880-896.
- Hinshaw, S. P. 2010. Behavior Problems of Childhood and Adolescence. *Corsini Encyclopedia of Psychology*. 1–3.
- Hutcherson, C., Goldin, P., Ochsner, K., Gabrieli, J., Barrett, L., & Gross, J. J. (2005). Attention and emotion: Does rating emotion alter neural responses to amusing and sad films? *NeuroImage*, 27(3), 656-668.
- Izard, C., Fine, S., Schultz, D., Mostow, A., Ackerman, B., Youngstrom, E. (2001). Emotion knowledge as a predictor of social behavior and academic competence in children at risk. *Psychological Science*, 12 (1), 18-23.
- Jennings, J. R., van der Molen, M. W., & Somsen, R. J. (1998). Changes in heart beat timing: reactivity, resetting, or perturbation? *Biol Psychol*, 47(3), 227-241.
- Johnston, C. & Mash, L. (2001). Effects of medication, behavioral, and combined treatments on parents' and children's attributions for the behavior of children with attention-deficit hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, 69(1), 67-76.
- Kagan, J., & Snidman, N. (1991). Temperamental factors in human development. *Am Psychol*, 46(8), 856-862.
- Kagan, J. (1997). Temperament and the Reactions to Unfamiliarity. *Child Development*, 68: 139–143.
- Kelsey, R. M., Ornduff, S. R., McCann, C. M., & Reiff, S. (2001). Psychophysiological characteristics of narcissism during active and passive coping. *Psychophysiology*, 38(2), 292-303.
- Kendall, P.C. & Chu, B.C. (2000). Retrospective self-reports of therapist flexibility in a manual-based treatment for youth with anxiety disorders. *Journal of Clinical Child Psychology*, 29 (2), 209-220.
- Kerig, P. K., Cowan, P. A., Cowan, C. P. (1993). Marital quality and gender differences in parent-child interaction. *Developmental Psychology*, 29(6), 931-939.
- Kimonis, E.R., Frick, P.J., Skeem, J.L., Marsee, M.A., Cruise, K., Munoz, L.C., Aucoin, K.J., Morris, A.S. (2008). Assessing callous-unemotional traits in adolescent offenders: Validation of the inventory of callous-unemotional traits, *International Journal of Law and Psychiatry*, 31, 241-252.
- Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry*, 158, 848-856.

- Kring, A. M., Smith, D. A., & Neale, J. M. (1993). Individual differences in dispositional expressiveness: The development and validation of the Emotional Expressivity Scale. *Journal of Personality and Social Psychology*, 66, 934-949.
- Lahey, B.B. & Loeber, R. (1994). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review*, 13 (4), 19-340.
- Lahey, B. B., Applegate, B., Barkley, R. A., Garfinkel, B. (1994). DSM-IV field trials for oppositional defiant disorder and conduct disorder in children and adolescents, *The American Journal of Psychiatry*, 151(8), 1163-1171.
- Lahey, B.B., Goodman, S.H., Waldman, I.D., Bird, H., Canino, G., Jensen, P., Regier, D., Leaf, P.J., Gordon, R., Applegate, B. (1999). Relation of age of onset to the type and severity of child and adolescent conduct problems. *Journal of Abnormal Child Psychology*, 27 (4), 247-260.
- Lang, N. D. J., Tulen, J. H. M., Kallen, V. L., Rosbergen, B., Dieleman, G., & Ferdinand, R. F. (2006). Autonomic reactivity in clinically referred children attention-deficit/hyperactivity disorder versus anxiety disorder. *European Child & Adolescent Psychiatry*, 16(2), 71-78.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). International affective picture system (IAPS): Technical manual and affective ratings.
- Levenson, R.W. (1992). Autonomic nervous system differences among emotions. *Psychological Science*, 3 (1). 23-27.
- Loeber, R., Burke, J.D., Lahey, B.B., Winters, A., Zera, M. (2000). Oppositional defiant and conduct disorder: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39 (12), 1468–1484.
- MTA Cooperative Group (1999). Moderators and mediators of treatment response for children with attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry*, 56, 1088–1096
- Maedgen, J. W., & Carlson, C. L. (2000). Social functioning and emotional regulation in the attention deficit hyperactivity disorder subtypes. *J Clin Child Psychol*, 29(1), 30-42.
- Magana, A.B., Goldstein, M.J., Karno, M., Miklowitz, D.J., Jenkins, J., Falloon, I.R.H. (1986). A brief method for assessing expressed emotion in relatives of psychiatric patients. *Psychiatry Research*, 17(3), 203–212.
- Marsh, P., Beauchaine, T. P., & Williams, B. (2008). Dissociation of sad facial expressions and autonomic nervous system responding in boys with disruptive behavior disorders. *Psychophysiology*, 45(1), 100-110.

- Marsh, A.A., Finger, E.C., Mitchell, D.G.V., Reid, M.E., Sims, C., Kosson, D.S., Towbin, K.E., Leibenluft, E., Pine, D.S., & Blair, R.J.R. (2009). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry*, 165, 712-720.
- Marshall, V. G., Longwell, L., Goldstein, M. J. and Swanson, J. M. (1990). Family factors associated with aggressive symptomatology in boys with attention deficit hyper activity disorder: A research note. *Journal of Child Psychology and Psychiatry*, 31, 629–636.
- Martel, M. M. (2009). Research Review: A new perspective on attention-deficit/hyperactivity disorder: emotion dysregulation and trait models. *Journal of Child Psychology and Psychiatry*, 50(9), 1042-1051.
- Martel, M. M., & Nigg, J. T. (2006). Child ADHD and personality/temperament traits of reactive and effortful control, resiliency, and emotionality. *J Child Psychol Psychiatry*, 47(11), 1175-1183.
- Mauss, I. B., Evers, C., Wilhelm, F. H., & Gross, J. J. (2006). How to bite your tongue without blowing your top: implicit evaluation of emotion regulation predicts affective responding to anger provocation. *Pers Soc Psychol Bull*, 32(5), 589-602.
- Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The Tie That Binds? Coherence Among Emotion Experience, Behavior, and Physiology. *Emotion*, 5(2), 175-190.
- Mead, H. K., Beauchaine, T. P., Brenner, S. L., Crowell, S. E., Gatzke-Kopp, L., & Marsh, P. (2004). Autonomic response patterns to reward and negative mood induction among children with conduct disorder, depression, and both psychiatric conditions. Paper presented at the Annual Meeting of the Society for Psychophysiological Research, Santa Fe, NM.
- Melnick, S. M., & Hinshaw, S. P. (2000). Emotion regulation and parenting in AD/HD and comparison boys: linkages with social behaviors and peer preference. *J Abnorm Child Psychol*, 28(1), 73-86.
- Mendes, W.B., Major, B., McCoy, S., Blascovich, J. (2008). How attributional ambiguity shapes physiological and emotional responses to social rejection and acceptance. *Journal of Personality and Social Psychology*, 94(2), 278-291.
- Mezzacappa, E., Tremblay, R. E., Kindlon, D., Saul, J. P., Arseneault, L., Seguin, J., et al. (1997). Anxiety, antisocial behavior, and heart rate regulation in adolescent males. *J Child Psychol Psychiatry*, 38(4), 457-469.

- MindWare. (2008a). Mind Ware Heart Rate Variability V 2.6 System. Gahanna, OH: MindWare Technologies.
- MindWare. (2008b). Mind Ware Impedance Cardiography V 2.6 System. Gahanna, OH: MindWare Technologies.
- Moffitt, T.E., Lynam, D.R., Caspi, A., Wikstrom, P.H., Loeber, R., Novak, S. (2000). The interaction between impulsivity and neighborhood context on offending: Effects of impulsivity are stronger in poorer neighborhoods., *Journal of Abnormal Psychology*, 109, 563-574.
- Moran, P., Ford, T., Butler, G., Goodman, R. (2008). Callous and unemotional traits in children and adolescents living in Great Britain, *British Journal of Psychiatry*, 192, 65-66.
- Mullineaux, P. Y., Deater-Deckard, K., Petrill, S. A. and Thompson, L. A. (2009), Parenting and child behaviour problems: a longitudinal analysis of non-shared environment. *Inf. Child Develop.*, 18: 133–148.
- Murray, L., Fiori-Cowley, A., Hooper, R., & Cooper, P. (1999). The impact of postnatal depression and associated adversity on early mother-infant interactions and later infant outcomes. *Child Development*, 67, 2512-2526.
- Murray, L., Hipwell, A., & Hooper, R. (2000). The cognitive development of 5-year-old children of postnatally depressed mothers. *Journal of Child Psychology and Psychiatry*, 37, 927-935.
- Murray, L., Stanley, C., Hooper, R., & King, F. (2002). The role of infant factors in postnatal depression and mother-infant interactions. *Developmental Medicine and Child Neurology*, 38, 109-119.
- Musser, E.D., Backs, R.W., Schmitt, C.F., Ablow, J.C., Measelle, J.R., and Nigg, J.T. (2010). Emotion regulation via the autonomic nervous system in children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Abnormal Child Psychology*, 39, 841-852.
- Musser, E. D., Ablow, J. C. and Measelle, J. R. (2012), Predicting maternal sensitivity: The roles of postnatal depressive symptoms and parasympathetic dysregulation. *Infant Ment. Health J.*
- Nikolas, M.A. & Burt, S.A. (2010). Genetic and environmental influences on ADHD symptom dimensions of inattention and hyperactivity: A meta-analysis. *Journal of Abnormal Psychology*, 119(1), 1-17.
- Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychol Bull*, 127(5), 571-598.

- Nigg, J.T. & Casey, B.J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychopathology*, 17, 785-806.
- Nigg, J.T., Willcutt, E.G., Doyle, A.E., Sonuga-Barke, E.J.S. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biological Psychiatry*, 57, 1224-1230.
- Nigg, J. T. (2006). Temperament and developmental psychopathology. *J Child Psychol Psychiatry*, 47(3-4), 395-422.
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *J Child Psychol Psychiatry*, 37(1), 51-87.
- Peris, T. S. and Baker, B. L. (2000), Applications of the Expressed Emotion Construct to Young Children with Externalizing Behavior: Stability and Prediction over Time. *Journal of Child Psychology and Psychiatry*, 41: 457–462.
- Peris, T. S. and Hinshaw, S. P. (2003), Family dynamics and preadolescent girls with ADHD: the relationship between expressed emotion, ADHD symptomatology, and comorbid disruptive behavior. *Journal of Child Psychology and Psychiatry*, 44: 1177–1190.
- Porges, S.W., Doussard-Roosevelt, J.A. & Maiti, A.K. (1994). Vagal Tone and the Physiological Regulation of Emotion. *Monographs of the Society for Research in Child Development*, 59(2/3) 167-186.
- Porges, S.W. (1995). Cardiac vagal tone: A physiological index of stress. *Neuroscience & Biobehavioral Reviews*, 19(2), 225-233.
- Porges, S.W., Doussard-Roosevelt, J.A., & Greenspan, S.I. (1996). Infant regulation of the vagal “brake” predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology*, 29, 697-712.
- Porges, S.W. (1999). Emotion: an evolutionary by-product of the neural regulation of the autonomic nervous system. IN *The integrated Neurobiology of Affiliation*.(1st ed.) Carter, C.S., Lederhendler, I.I. & Kirkpatrick, B. MIT Press.
- Porges, S.W. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42, 123-146.
- Porges, S.W. (2007). The polyvagal perspective. *Biological Psychology*, 74, 116-143.
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers*, 36, 717-731.

- Preacher, K. J., Zhang, Z., & Zyphur, M. J. (2011). Alternative methods for assessing mediation in multilevel data: The advantages of multilevel SEM. *Structural Equation Modeling*, 18, 161-182.
- Puig-Antich, J., & Ryan, N. (1996). *Kiddie Schedule for Affective Disorders and Schizophrenia*. Pittsburgh, PA: Western Psychiatric Institute.
- Raine, A. (Ed.). (1996). *Autonomic nervous system activity and violence*. Hillsdale, NY: Erlbaum.
- Raine, A., Venables, R., & Mednick, S. (1997). Low resting heart rate at age three years predisposes to aggression at age 11 years: Evidence from the Mauritius Child Health Project. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36, 1457-1464.
- Raine, A. (2001). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry*, 43, 417-434.
- Raine, A. (2002). The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry*, 43, 417-434.
- Raudenbush, Bryk, Cheong, & Congdon (2004) HLM 6.0 software. Scientific Software International, Inc., Skokie, IL
- Rothbart, M.K.( 1998). Temperament. *Handbook of Child Psychology*.
- Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The children's behavior questionnaire. *Child Development*, 72(5), 1394-1408.
- Rothbart, M.K. & Sheese, B.E. (2007). Temperament and emotion regulation. In J.J. Gross (Ed.), *Handbook of Emotion Regulation*. New York: Guilford Press.
- Sloan, R. P., Shapiro, P. A., Bigger, J. T., Jr., Bagiella, E., Steinman, R. C., & Gorman, J. M. (1994). Cardiac autonomic control and hostility in healthy subjects. *Am J Cardiol*, 74(3), 298-300.
- Sonuga-Barke , E.J.S. (2002). Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. *Behavioural Brain Research*, 130( 1–2), 29–36.
- Starrels, M.E. (1994). Gender differences in parent-child relations. *Journal of Family Issues*, 15 (1), 148-165.
- Suess, P. E., Porges, S. W., & Plude, D. J. (1994). Cardiac vagal tone and sustained attention in school-age children. *Psychophysiology*, 31(1), 17-22.

- Viding E., Blair RJR., Moffitt TE., Plomin R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry*. 46(6): 592-597.
- Viding E., Jones AP., Frick PJ., Moffitt TE., Plomin R. (2008). Heritability of antisocial behavior at 9: Do callous-unemotional traits matter? *Developmental Science*. 11(1): 17-22.
- Vostanis, P. & Nicholls, J. (1992). Expressed emotion in parents of non-referred children aged 6 to 11 years from two school populations: a pilot study. *Child: Care, Health and Development*, 18, 249–257.
- Walcott, C. M., & Landau, S. (2004). The relation between disinhibition and emotion regulation in boys with attention deficit hyperactivity disorder. *J Clin Child Adolesc Psychol*, 33(4), 772-782.
- Wechsler, D. (2003). Wechsler Intelligence Scale for Children, Fourth Edition: Administration and Scoring Manual. San Antonio, TX: Psychological Corporation.
- Wechsler, D. (2005). Wechsler Individual Achievement Test-2nd Edition: Examiner's Manual. San Antonio, TX: Psychological Corporation.
- Wehmeier, P. M., Schacht, A., & Barkley, R. A. (2010). Social and emotional impairment in children and adolescents with ADHD and the impact on quality of life. *J Adolesc Health*, 46(3), 209-217.
- Wender, P.H.( 1972). Attention deficit disorder in adults. *Arch Gen Psychiatry*. 38(4), 449-456.
- Wilhelm, F. H., Grossman, P., & Roth, W. T. (2005). Assessment of heart rate variability during alterations in stress: complex demodulation vs. spectral analysis. *Biomed Sci Instrum*, 41, 346-351.
- Zahn-Waxler, C., Cole, P.M., Welsh, J.D., Fox, N.A. ( 1995). Psychophysiological correlates of empathy and prosocial behaviors in preschool children with behavior problems. *Development and Psychopathology*, 7, 27-48.