Published in final edited form as:

J Clin Child Adolesc Psychol. 2019 ; 48(1): 153–165. doi:10.1080/15374416.2016.1270828.

Emotion dysregulation across emotion systems in attention-deficit/hyperactivity disorder (ADHD)

Erica D. Musser¹ and Joel T. Nigg²

¹Florida International University, Department of Psychology, 11200 SW 8th St, Miami, FL 33199

²Oregon Health & Science University, Portland, OR 97239-3098

Abstract

Objective.—Children with attention-deficit/hyperactivity disorder (ADHD) display alterations in both emotion reactivity and regulation. One mechanism underlying such alternations may be reduced coherence among emotion systems (i.e., autonomic, facial affect). The present study sought to examine this.

Method.—100 children (50 with ADHD combined presentation), aged 7 to 11 years (62% male, 78% White), completed an emotion induction and suppression task. This task was coded for facial affect behavior across both negative and positive emotion eliciting task conditions.

Electrocardiogram and impedance cardiography data were acquired throughout the task. Time-linked coherence of facial affect behavior and autonomic reactivity and regulation were examined during the induction conditions using Hierarchical Linear Modeling.

Results.—While ADHD and typically developing children did not differ with respect to rates of facial affect behavior displayed (all $F < 2.09$, $p > .29$), the ADHD group exhibited reduced coherence between facial affect behavior and an index of parasympathetic functioning (i.e., respiratory sinus arrhythmia [RSA]; $\gamma_{10} = -0.03$, $SE = 0.02$, $t(138) = -1.96$, $p = 0.05$). In contrast, children in the control group displayed a significant, positive ($\gamma_{10} = 0.06$, $SE = 0.01$, $t(138) = 4.07$, $p < .001$) association between facial affect behavior and RSA.

Conclusions.—Children with ADHD may receive conflicting emotional signals at the levels of facial affective behavior and parasympathetic functioning when compared to typically developing youth. Weakened coherence among these emotion systems may be an underlying mechanism of emotion dysregulation in ADHD. Implications for etiology and treatment are discussed.

Keywords

ADHD; autonomic reactivity; emotion regulation; facial affect

There is increasing recognition of the importance of emotion reactivity and regulation in attention-deficit/hyperactivity disorder (ADHD). A recent meta-analysis of 77 studies, including over 32,000 youth, suggests that ADHD is uniquely associated with both elevated emotion reactivity (weighted ES $d = .95$) and emotion dysregulation (weighted ES $d = .80$), with effect sizes similar in magnitude to those observed in studies of ADHD and executive

dysfunction (weighted ES $d = .46-.69$ range; Graziano & Garcia, 2016). While the link between ADHD and emotion-related dysfunction is increasingly well-established, empirical investigations of mechanisms contributing to disruptions in emotion reactivity and regulation in ADHD are still relatively few. Identifying such mechanisms would potentially allow for the development of novel treatments, as well as improvements in treatment matching, given the heterogeneity observed in the disorder (Karlunas et al., 2014).

Identifying such mechanisms requires both care and specificity. Both emotion reactivity and regulation are multi-systemic, multi-dimensional constructs, which encapsulate a broad range of processes (Gross & Jazaieri, 2014; Rosen et al., 2013; Thompson, 2011). For example, recent work from our lab has demonstrated that ADHD is associated with both altered sympathetic-based emotion *reactivity* (i.e., indexed via cardiac pre-ejection period [PEP]; Karlunas et al., 2014; Musser et al., 2011) and altered parasympathetic-based emotion *regulation* (i.e., indexed via respiratory sinus arrhythmia [RSA]; Karlunas et al., 2014; Musser et al., 2011). Further, these alterations in autonomic functioning appear during both induction and suppression of both negative and positive emotions (Karlunas et al., 2014; Musser et al., 2011).

Crucially, however, this does not complete the picture. Emotion reactivity and regulation are also multi-dimensional in terms of the presence of multiple emotion response systems (e.g., facial affective behavior, subjective experience, and autonomic and central nervous system responding; Eckman 2016), which raises important questions about how these multiple systems work together or interact with one another. The functionalist theory of emotion proposes that *coherence*, or coordination among an individual's emotional response systems as the emotion unfolds over time, is a key mechanism involved in adaptive emotion reactivity and regulation (Lench, Bench, Darbor, & Moore, 2015; Livingston, Kahn, Berkman, 2015). The same theory proposes that weak coherence, or poor coordination across emotional response systems, increases the probability of experiencing conflicting emotional signals, and thus, maladaptive responding to emotional cues and emotion-related psychopathology (Ekman, 1992a,b; Lench et al., 2015; Livingston et al., 2015; Mauss, et al., 2006).

In support of the proposal that poor coherence underlies psychopathology, weak coherence of emotional systems has been reported in studies of adults with depression and schizophrenia (Kring et al., 1993; Sloan et al., 1994). Additionally, one prior study has been conducted with children with disruptive behavior disorders (i.e., conduct and oppositional defiant disorder; Marsh et al., 2008). Each of these disorders has also been characterized by disruptions in both emotion reactivity and regulation (Kring et al., 1993; Marsh et al., 2008; Sloan et al., 1994). Coherence is presumed to increase over time with development (Lench et al., 2015; Livingston et al., 2014). However, this has yet to be examined in the context of developmental psychopathology except for one preliminary, cross-sectional study (Marsh et al., 2008).

In the only prior study to examine emotion response coherence among children with disruptive behavior disorders, Marsh and colleagues (2008) examined time-linked coherence of sad facial expressions and autonomic reactivity in the context of an emotionally evocative

film-clip. This study included a sample of 31 boys with disruptive behavior disorders and 23 controls, aged 9–13 years (Marsh et al., 2008). Reduced coherence among sad facial affect behavior and autonomic reactivity across both the parasympathetic and sympathetic branches was observed. However, the small sample size did not allow for an examination of ADHD symptoms or diagnoses, specifically. Further, coherence among emotional systems was only examined in the context of negative (i.e., sad) emotion induction, leaving questions regarding whether emotion systems coherence may be disrupted in the context of positive or other emotional contexts unanswered.

Here, we seek to open this line of investigation with the first examination of emotion systems coherence in children with ADHD. Recent theory suggests that mechanisms underlying disruptions in emotion reactivity and regulation are likely shared across many domains of psychopathology (Gross & Jazaieri, 2014; Insel et al., 2010). Therefore, like depression, disruptive behavior disorders, and schizophrenia, we hypothesize that ADHD will be related to reduced coherence of emotion systems during emotionally evocative situations.

At a higher order, emotion responses have been demonstrated to cluster into positive affect (e.g., happiness, excitement) or negative affect (e.g., sadness, anxiety; Lindquist, Satpute, Wager, & Weber, 2015). Other models suggest that emotional responses cluster into approach (e.g., happiness) or avoidance (e.g., fear) domains (e.g., Carver, 2006; Carver Johnson, & Joorman, 2009). Importantly, while positive affect is generally associated with approach, anger may be a negative approach-based emotion (Carver & Harmon-Jones, 2009). In the case of ADHD, theories have proposed dysfunction both the negative *and* positive valence domains, as well as some theories proposing dysfunction specifically in the negative approach/anger domain (for a review see Nigg, 2006). This claim has received some empirical support with findings of a diminished parasympathetic regulatory response to negative emotion induction and an exaggerated parasympathetic regulatory response to positive emotion induction among children with ADHD (Musser et al., 2011). Furthermore, recent work by Karalunas and colleagues (2015; in a partially overlapping sample) reveals that patterns of autonomic-linked emotion responding that are specific to either the negative or positive valence domains may help to explain some of the behavioral heterogeneity observed in ADHD. Thus, the present study examined emotional response coherence in the context of both negative and positive emotion induction.

In the present study, the following hypotheses were examined: 1) Children with ADHD will show weaker statistical correspondence (i.e., coherence) between emotion response systems including: autonomic reactivity and facial affective behavior, during both negative and positive emotion induction, when compared to typically developing youth. Thus, a 2-way interaction of Group (i.e., ADHD or control) * Facial Affect Behavior (i.e., negative and/or positive affect) was examined in predicting autonomic functioning in both the parasympathetic (i.e., RSA) and sympathetic (i.e., PEP) branches. 2) Statistical correspondence (i.e., coherence) between autonomic nervous system activity and facial affective behavior will be specific to task context (i.e., negative induction vs. positive induction) for typically developing youth (but not for children with ADHD). That is, it is expected that positive facial affect will be associated with autonomic functioning during

positive induction, while negative facial affect behavior will be associated with autonomic functioning during negative induction for typically developing youth. Thus, a 3-way interaction of Group (i.e., ADHD or control) * Condition Type (i.e., negative or positive induction) * Facial Affective Behavior (i.e., negative and/or positive facial affect) interaction was also examined in predicting autonomic functioning in both the parasympathetic (i.e., RSA) and sympathetic (i.e., PEP) branches.

METHOD

Participants

Participants were 100 children, aged 7 to 11 years (mean age = 8.59, SD = 1.24 years). 50 met DSM-5 (APA, 2013) criteria for ADHD combined presentation (ADHD) and 50 were typically developing comparison youth. Siblings were not included in this sample. Table 1 displays demographic and diagnostic characteristics. The age range was selected as this is the age of typical onset of ADHD, and to ensure confirmed onset before age 12 (APA, 2013).

Recruitment and Identification Procedures.—Families were recruited from the greater Portland, OR metropolitan area through commercial mailings to all households in the community with children in the target age range, as well as via public advertising. The local Institutional Review Board approved the study. Parents provided written informed consent, and children provided written assent. Parents and children were compensated for their time. All procedures conformed to the Ethical Principles of Psychologists and Code of Conduct (APA, 2010).

Families volunteering, for the study passed through a multi-gate screening process to establish eligibility and diagnostic group assignment following procedures identical to those described in detail elsewhere (Musser et al., 2011). At the first gate, basic rule outs were checked (below). At the second gate, parents completed a structured, diagnostic interview (Kiddie Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Epidemiologic Version [KSADS-E]; Puig-Antich, J., & Ryan, N., 1996), and parents and teachers completed standardized rating scales. Children completed an IQ screener and standardized rating scales. Additionally, clinical observers wrote detailed notes. Finally, a clinical diagnostic team (i.e., a board-certified psychiatrist and licensed clinical psychologist) independently reviewed all information to arrive at a diagnosis using DSM-IV and DSM-5 criteria (the children included here met criteria by both DSM-IV and DSM-5). Inter-rater agreement was acceptable with ($k > .70$ for all disorders with base rates $> 5\%$, including ADHD). Disagreement was resolved by conference.

A diagnosis of ADHD required that the child's symptoms had a cross-situational presentation, evidence of impairment, onset by age 7 (so children would meet both DSM-IV and DSM-5 criteria), 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. Further, children were required to have elevated teacher and parent ratings of at least $T > 65$ on at least one major sub-scale of the ADHD Rating Scale. To limit excessive false positives from the "or" algorithm, the teacher was able to contribute a maximum of two

additional symptoms (that is, at least four symptoms had to be identified on the KSADS-E); the “or” algorithm was invoked in only 4 cases. This procedure was modified from that used in the DSM-IV field trials and the MTA study (MTA Cooperative Group, 1999).

Exclusion Criteria.—Exclusion criteria included the uses of psychoactive medications that could not be washed out for the study as described below (i.e., long-acting and non-stimulant psychiatric medications, such as antidepressants), an estimated Full Scale IQ < 75, current major depressive episode, lifetime mania or psychosis, pervasive developmental disorder, or learning disability. Other disorders were free to vary.

Medication Washout.—Prior to completing the tasks, children taking stimulant medication (i.e., 40% of the ADHD group) completed a medication washout equivalent to a minimum of five half-lives (i.e., 24–48 hours, depending on preparation).

Emotion Induction (and Suppression) Task

Each child was recorded as they completed the Emotion Induction (and Suppression) Task (Musser et al., 2011). This involved watching four, 2-min film clips taken from “Homeward Bound”, a film about two dogs and a cat who are separated from their child owners (negative emotion eliciting; 1’25”–1’29” of the film; during which the animals are discouraged and one is injured) and reunited with their human family (positive emotion eliciting; 1’32”–1’36” of the film, during which the animals happily find, greet, and play with the as they are reunited family). Prior work from our team has demonstrated the validity of these conditions using the Self-Assessment Manikin valence and arousal scales (Bradley and Lang 1994) for each clip, and this work has demonstrated that ADHD and typically developing youth do not differ in their ratings of these clips (for validity data, see Musser et al., 2011).

In the induction condition, children were asked to facially mimic 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 asked 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. Thus, the same sequence of the four task conditions was presented to each child: (1) negative induction, (2) negative suppression, (3) positive induction, and (4) positive suppression. These conditions were not counterbalanced as: (a) it was important to end with positive emotion for human subjects’ welfare, (b) the film was a continuous story and changing the order would confound suppression with cognitive challenge to interpret the story, and (c) putting induction prior to suppression maximized the suppression challenge.

Importantly, only the negative and positive emotion induction conditions were utilized in the present study, as prior work suggests emotional suppression is associated with reduced emotion response coherence across the domains of facial affect and autonomic responsivity (Gross & Levenson, 1993; Mauss et al., 2005). Further, the primary research question being addressed herein is related to emotional coherence during emotional experience rather than during the suppression of emotions.

Facial Action Coding System.—After data collection was complete, children's facial affect was coded from recordings using a simplified version of the Facial Action Coding System (FACS; Ekman, 1992b), for the negative and positive induction task segments, by two research assistants masked to condition and child group status. 40% of videos were coded for reliability (all ICCs > .85). For the purposes of this study, only six facial affective behavior frequency domains were coded (i.e., FACS was simplified to focus on only six emotions intended to reflect positive [happiness] and negative [anger, anxiety, fear, and sadness] valence, as well as surprise). The full Facial Action Coding System has been well validated with standard inter-rater reliabilities of $\alpha = .89$ (Gross & Levenson, 1993).

Physiological Recording

Overview.—Disposable silver/silver-chloride electrodes were placed in a standard 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 (2 minutes of rest and 2 minutes of neutral baseline) and each of the task epochs (2 minutes of negative induction and 2 minutes of positive induction). The R-R series was sampled at 1000 Hz. Heart rate (HR), inner-beat-interval (IBI), and respiration rate (RR) were derived using MindWare Heart Rate Variability and Impedance Cardiography v. 2.6 softwares completed by two raters with satisfactory inter-rater agreement ($k > .85$ for each epoch). No between-group differences were observed in the rate of artifacts (all $p > .50$).

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 and predicts vagal control during pharmacological blockade (Berntson, Cacioppo, & Quigley, 1993). RSA was derived using spectral analysis of the R-R time series (Berntson, et al., 1997; Wilhelm, Grossman, & Roth, 2005) and processed in 30 second epochs (i.e., a total of 4, 30-second epochs per 2-minute baseline and task condition), using MindWare Heart Rate Variability V. 2.6 (Mindware, 2008a). 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 (Z_0) ensuring that these signals remained within the analytical bandwidth. RSA means and standard deviations for each of the task conditions are reported by group in Table S1.

Cardiac pre-ejection period (PEP).—PEP, was derived from ECG and ICG in 30 second epochs (i.e., a total of 4, 30-second epochs per 2-minute baseline and task condition), using MindWare Impedance Cardiography V. 2.6 (MindWare, 2008b). This system allows 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). PEP means and standard deviations for each of the task conditions are reported by group in Table S1.

Physiological Baseline Conditions.—A resting baseline period of two minutes was presented before the Emotion Induction and Suppression Task. Prior to completing the resting baseline condition, the electrodes were placed on the child (as described above), and the child was given the opportunity to engage in a period of play for 5 minutes. For the resting baseline period, the child was seated in a quiet room with an experimenter seated on the other side of a cubicle-style barrier, able to see the child via a webcam but not viewable by the child. The child was instructed to be as still and quite as possible.

A neutral baseline period of approximately two minutes was presented between the negative and positive task conditions. The neutral baseline required the child to observe a set of ten neutral pictures from the International Affective Picture System (including pictures of beads, tires, clouds, mushrooms, a cup, dustpan, hydrant, lightbulb, pillar and spoon; Lang, Bradley, & Cuthbert, 1997) on a computer screen. Each picture was presented for 10 seconds. This baseline was used to account for physiological responses associated with orienting and attending to a stimulus (Jennings, van der Molen, & Somsen, 1998). The Self-Assessment Mankin valence and arousal scales were presented to children on the computer screen between each picture and responses were self-paced. Prior work from our team has demonstrated the validity of these pictures as neutral using the Self-Assessment Mankin valence and arousal scales (Bradley and Lang 1994; for validity data, see Musser et al., 2011).

Data Analysis Plan

Data reduction for primary analyses.—First, to reduce concerns related to model over-saturation and to enhance the likelihood that models would converge, only the facial affect behavior codes and autonomic indexes obtained during the first minute of each of the negative and positive induction conditions were utilized (i.e., 2 30-second epochs per task condition).

Second, prior empirical work has supported a bi-factor solution for emotion, including positive and negative valence (Carver & Scheier, 1990; Lindquist et al., 2015). As such, models were examined including frequencies of facial affective behavior domains of positive (happiness) and negative (anger, anxiety, sadness) valence. Frequency codes for fear and surprise were omitted from these models, given that both fear and surprise were coded relatively infrequently and there is evidence that surprise may load on either positive or negative valence depending on context (Carver, 2006). Positive and negative valence facial affective behavior were examined in a single model.

Additionally, to further probe the specificity of coherence between autonomic functioning and specific facial affective behaviors, the frequencies of each the six, specific facial affective behavior codes (i.e., anger, anxiety, fear, happiness, sadness, and surprise) were examined as predictors of autonomic functioning via separate models for each. For frequencies of each of the positive, negative, and specific coded facial affect behavior domains arranged by task condition and group see Table S2.

Primary analyses.: Hierarchical Linear Modeling (HLM) was used to analyze levels of coherence among indexes of emotion, including facial affective behavior (modified Facial Action Coding System ratings) and physiological measures (RSA and PEP, in separate models) to test the hypothesis that children with ADHD would show weaker statistical coherence among indexes of emotion, including autonomic reactivity and facial affect. All models were analyzed in HLM 6.0 (Raudenbush, Bryk, Cheong, & Congdon, 2004).

The outcome variables were ANS activity (i.e., RSA, PEP in separate models). At level one, three types of variables were included in the model simultaneously. First, facial affective behavior (i.e., positive and negative valence) was treated as a predictor of ANS activity (first for RSA, then PEP in a separate model). Second, the stimulus condition (i.e. negative and positive induction) was included as a dummy coded variable (Negative=-1, Positive=1). Third, the interaction term of the type of stimulus condition with the coded facial affective behavior rating was included at Level 1 to examine the prediction of autonomic nervous system activity (i.e., RSA, then PEP) by frequency of facial affective behavior by stimulus valence. Thus, the model examined statistical coherence during conditions of negative induction compared to positive induction.

At level 2, group effects were tested using a dummy coded time invariant variable (1=ADHD, 0=control). It was expected that the associations between autonomic nervous system activity (i.e., RSA and PEP) and facial affective behavior (i.e., positive and negative valence) 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., positive and negative valence, Level 1), which would represent a task-specific response pattern. For example, it was expected that positive-valence facial affective behavior would predict ANS activity under positive induction, while negative valence facial affective behavior would predict ANS activity under negative induction for the control group. Further, it was hypothesized that these associations would be diminished in the ADHD group.

Thus, the full equations for the bi-factor (i.e., positive/negative) models were as follows:

Level-1:

$$\begin{aligned} \text{ANS}_{ij} = & \beta_{0j} + \beta_{1j}(\text{TIME}_{ij}) + \beta_{2j}(\text{POSITIVE FACIAL AFFECT}_{ij}) + \\ & \beta_{3j}(\text{NEGATIVE FACIAL AFFECT}_{ij}) + \beta_{4j}(\text{TASK CONDITION}_{ij}) + \\ & \beta_{5j}(\text{POSITIVE FACIAL AFFECT} * \text{TASK CONDITION}_{ij}) + \beta_{6j}(\text{NEGATIVE} \\ & \text{FACIAL AFFECT} * \text{TASK CONDITION}_{ij}) + r_{ij} \end{aligned}$$

Level-2:

$$\begin{aligned} \beta_{0j} = & \gamma_{00} + \gamma_{01}(\text{GROUP}_j) + u_{0j} \\ \beta_{1j} = & \gamma_{10} + \gamma_{11}(\text{GROUP}_j) \\ \beta_{2j} = & \gamma_{20} + \gamma_{21}(\text{GROUP}_j) \\ \beta_{3j} = & \gamma_{30} + \gamma_{31}(\text{GROUP}_j) \\ \beta_{4j} = & \gamma_{40} + \gamma_{41}(\text{GROUP}_j) \end{aligned}$$

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

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

where ANS_{ij} represents RSA (and PEP in a separate model), *POSITIVE FACIAL AFFECT*_{ij} represents the frequency of *positive* facial affective behavior, *NEGATIVE FACIAL AFFECT*_{ij} represents the frequency of *negative* facial affective behavior, *TASK CONDITION*_j is a dummy coded variable representing whether the stimulus condition involved negative = -1 or positive = 1 emotional stimuli, *POSITIVE FACIAL AFFECT* $1 * TASK CONDITION_{ij}$ represents the interaction of *positive* facial affective behavior frequency and task condition (i.e., negative vs. positive induction), *NEGATIVE FACIAL AFFECT* $* TASK CONDITION_{ij}$ represents the interaction of *negative* facial affective behavior frequency and task condition (i.e., negative vs. positive induction). Finally, $GROUP_j$ is a dummy coded variable representing ADHD = 1 or control = -1 group status. Thus, a total of 2 primary models were examined, including a RSA as an outcome of interest in the first model and PEP as an outcome of interest in the second model.

Finally, modified models for each of the six specific facial affective behaviors (utilizing only a single SPECIFIC FACIAL AFFECT FREQUENCY and SPECIFIC FACIAL AFFECT FREQUENCY * TASK CONDITION term) were examined for both RSA and PEP.

RESULTS

Preliminary Analyses

Descriptive and diagnostic overview of sample.—Descriptive and diagnostic statistics are reported for by group in Table 1. Groups did not differ reliably with respect to age, race, IQ, or presence of either anxiety or conduct disorder diagnosis. Groups differed in gender ratio (more boys in the ADHD than control group), family income (families of ADHD children earning less than control children). Additionally, 40% of the ADHD sample was prescribed a stimulant medication and 22% of the ADHD sample met criteria for ODD diagnosis. Importantly, the inclusion of age, anxiety or conduct disorder diagnosis, gender, income, IQ, medication status, or race did not alter the primary results. As such, for simplicity and to reduce model oversaturation, all models are presented without these potential covariates. However, ODD diagnosis emerged as a relevant covariate such that when included, patterns of significance were altered. As such, results are presented with the inclusion of ODD diagnosis as a Level 2, time-invariant covariate.

Primary Analyses

To test the primary hypotheses that 1) children with ADHD would show weaker statistical correspondence (i.e., coherence) among indexes of positive emotion (i.e., autonomic reactivity and facial affective behavior) during positive induction and 2) children with ADHD would show weaker statistical correspondence among indexes of negative emotion during negative induction, when compared to controls, two separate (i.e., one for RSA and one for PEP) two-level models were constructed.

Predicting RSA from ADHD, positive and negative facial affective behavior, and task condition.—Full model details are provided in Table 2. Only examinations of the hypotheses are noted in the text. Specifically, the 3-way interactions predicting RSA from *Positive* facial affective behavior*task condition*group and *Negative* facial affective behavior*task condition*group were examined. As noted in the hypotheses, *Positive* facial affect was of interest during the positive emotion induction condition and *Negative* facial affect was of interest during the negative emotion induction condition.

With respect to *Positive* facial affective behavior, the 3-way interaction of group by Positive facial affective behavior by task condition (i.e., positive vs. negative induction) was significant ($\gamma_{5f} = -0.03$, $SE = 0.01$; $t(288) = -2.15$, $p = .03$)¹. However, when ODD diagnosis was included in the model as a Level 2, time-invariant covariate, this 3-way interaction became non-significant ($\gamma_{5f} = -0.02$, $SE = 0.01$; $t(282) = -1.56$, $p = .11$). None of the other main effects or 2-way or 3-way interactions involving Positive facial affect behavior were significant (all $t(282) < 1.70$, all $p > 0.10$). Thus, Positive facial affect behavior was not considered further in this model.

A different pattern emerged for *Negative* facial affective behavior. In contrast to the primary hypothesis, the 3-way interaction of group by Negative facial affect behavior by task condition was nonsignificant ($\gamma_{6f} = -0.01$, $SE = 0.01$; $t(282) = -0.94$, $p = .35$). However, in support of the weakened coherence hypothesis, a significant 2-way group by Negative facial affect behavior interaction ($\gamma_{41} = -0.05$, $SE = 0.01$; $t(282) = -3.81$, $p < .001$) emerged, suggesting that coherence between Negative facial affect behavior and RSA was moderated by group status (when collapsed across negative and positive induction task conditions). Specifically, the association between Negative facial affect behavior and RSA for controls was positive ($\gamma_{10} = 0.06$, $SE = 0.01$, $t(138) = 4.07$, $p < .001$), while the association between Negative facial affect behavior and RSA for the ADHD group was negative and failed to reach significance ($\gamma_{10} = -0.03$, $SE = 0.02$, $t(138) = -1.96$, $p = 0.05$; Figure 1). Thus, it appears that ADHD children have reduced coherence between RSA and Negative facial affect compared to typically developing youth, irrespective of the nature of the emotional context.

Predicting PEP from ADHD, positive and negative facial affective behavior, and task condition.—Full model details are provided in Table 2. With PEP as the outcome variable of interest, there was a main effect of the ODD covariate ($\gamma_{02} = -4.29$, $SE = 1.97$; $t(99) = -2.18$, $p = .03$; see Table 2). An examination of PEP grand means according to ODD diagnostic status reveals that children without a diagnosis of ODD ($M = 96.60$, $SD = 10.07$) had significantly greater PEP across task conditions compared to children with ODD ($M = 91.23$, $SD = 8.81$; $F(1, 99) = 3.18$, $p = .04$). However, for PEP, none of the other main effects or 2-way or 3-way interactions (including those related to ADHD diagnoses) were significant predictors (all $t < 1.5$, $p > .11$; see Table 2).

Individual Emotion Specificity Checks

To examine whether children with ADHD show weaker statistical correspondence among indexes of each of the 6 specific emotions based on task condition, similar two-level models to those described above were constructed (with RSA and PEP in separate models).

However, in these models, each of the six facial affect behaviors coded (i.e., anger, anxiety, fear, happiness, sadness, and surprise) were examined separately.

Predicting RSA from ADHD, specific facial affective behavior, and task condition.—Full model details are provided in Table 2 for each of the models examined (excluding the model for fear, as this model failed to converge). Of each of the models examined, only the model including anger resulted in a significant 3-way interaction of group by facial affect behavior by task condition predicting RSA ($\gamma_{41} = -0.13$, $SE = 0.06$; $t(288) = -2.16$, $p = .03$; see Table 2). In decomposing the 3-way interaction according to group, for controls the 2-way interaction of Angry facial affect behavior by task condition was significant ($\gamma_{41} = 0.06$, $SE = 0.01$, $t(138) = 4.07$, $p < .001$), while the 2-way interaction failed to reach significance for the ADHD group ($\gamma_{41} = -0.12$, $SE = 0.18$, $t(138) = -0.65$, $p = .51$). Further decomposition of the significant 2-way interaction between Angry facial affect behavior by task condition for controls according to task condition revealed that Angry facial affect behavior was significantly associated with RSA in the negative task induction condition ($\gamma_{21} = 0.49$, $SE = 0.11$, $t(48) = 4.41$, $p < .001$), but not the positive induction task condition ($\gamma_{21} = 0.05$, $SE = 0.05$, $t(48) = 0.92$, $p = .363$).

Additionally, there was also a significant 2-way interaction of Anxious facial affective behavior by group ($\gamma_{31} = -0.04$, $SE = 0.01$; $t(288) = -2.97$, $p = .003$; see Table 2). Decomposition by group revealed, the association between Anxious facial affect and RSA for controls was positive ($\gamma_{20} = 0.05$, $SE = 0.02$, $t(149) = 2.81$, $p = .006$), while the association between Negative facial affect behavior and RSA for the ADHD group was negative and failed to reach significance ($\gamma_{20} = -0.03$, $SE = 0.02$, $t(149) = -1.89$, $p = .06$).

Each of the other main effects, 2-way, and 3-way interactions predicting RSA across each of the models examining specific facial affective facial behaviors were non-significant (all $t < 1.6$, $p > .10$; see Table 2).

Predicting PEP from ADHD, specific facial affective behavior, and task condition.—Again, full model details are provided in Table 2. For each of the models examining the 5 facial affect behaviors with PEP as the outcome variable of interest, none of the main effects, 2-way or 3-way interactions involving ADHD reached significance (all $t < 1.5$, $p > .10$; see Table 2).

DISCUSSION

This study examined the coherence of facial affect behavior and autonomic indices of emotion reactivity and regulation in children with and without ADHD. The two-factor structure of emotion valence (i.e., positive vs. negative valence) used here reflects that described in previous literature (Carver & Scheier, 1990; Lindquist et al., 2015).

The primary hypothesis was that children with ADHD would show weaker coherence among indexes of emotion reactivity and regulation, including autonomic reactivity and facial affect behavior, when compared to typically developing children, during induction of both negative and positive emotions. Specifically, it was hypothesized that reduced

coherence between positive facial affect behavior and ANS activity (both RSA and PEP) would be more salient in the positive induction condition, while reduced coherence between negative facial affect behavior and ANS activity would be more salient in the negative induction condition (for both RSA and PEP). This double dissociation was largely the case, in the parasympathetic domains, but with some important caveats.

Specifically, during the positive emotion induction condition, facial expressions of positive emotion were associated with PNS withdrawal (i.e., decreased RSA) for typically developing children. However, the opposite pattern emerged for children with ADHD, such that positive emotions were associated with increased PNS activity (i.e., increased RSA). This is congruent with theories that have suggested children with ADHD may misinterpret positive emotions and treat them as something aversive, which needs to be regulated (Braaten et al., 2000; Cohen & Strayer, 1996; Izard et al., 2001). Additionally, these results are supportive of several theories of the roles of temperament in ADHD have suggested that disruptions in the positive emotion domain may be particularly salient to ADHD (Martel, 2009; Nigg, 2006). Importantly, however, the inclusion of ODD as a covariate in this model, this pattern no longer held.

The examination of negative facial affect revealed an important caveat. In contrast to our hypothesis, there was no specificity with respect to task condition, but rather a positive association between negative facial affect and parasympathetic functioning was observed across task conditions for the control group, but not the ADHD group. Thus, in children with ADHD, coherence between facial affect behavior and parasympathetic functioning was diminished across *both* positive and negative induction conditions. 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 negative affective behavior suggests that physiological dysregulation may play a central role in the inappropriate negative affect displayed by some children with ADHD. That is, the diminished association between physiological emotion response and negative affect behavior may help to explain why some children with ADHD are prone to displays of inappropriate negative affect and/or irritability. With decoupled physiological and behavioral responses to affective challenges, children with ADHD may receive conflicting information regarding their emotional experience, resulting in inappropriate displays of negative affect and reduced ability to respond appropriately to the emotion (Fabes et al, 1994).

When individual facial affect behaviors were examined, reduced coherence between facial affect and parasympathetic functioning among children with ADHD appeared to be specific to anger and anxiety. This is of interest, given prior work demonstrating the important role that both anger/frustration/irritability (Shaw, Stringaris, Nigg, & Leibenluft, 2014; Sullivan et al., 2015) and anxiety (Krone & Newcorn, 2015; Pliszka, 2000) play in ADHD.

In contrast to our hypotheses, neither facial affect behavior nor ADHD appeared to be significantly associated with the index of sympathetic functioning (i.e., PEP). However, this may be in line with prior literature which has linked PEP to beta-adrenal functioning and response to reward specifically. Thus, it may be that the task conditions utilized here were insufficient to engage these processes.

These primary findings of reduced coherence between parasympathetic functioning and facial affect 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 positive and negative facial affective behavior. Additionally, 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. Results also were not explained by child gender, the use of stimulant medications, or the presence of other disorders, including CD, ODD, or anxiety.

The results of this study should be considered in the context of several possible limitations and future directions. First, this sample was selected to specifically examine questions related to emotion reactivity and regulation in children with ADHD. While the current study did rule out explanatory power of ODD, CD, and anxiety disorders in the primary hypotheses of interest, the sample size and sample composition did not allow for statistical power to fully examine whether these associations are specific to ADHD. Further, the sample was too small to examine additional moderating factors, such as gender. Second, 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. Third, the stimuli used to elicit “negative” and “positive” 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. Stimuli designed to elicit more “pure” and specific emotions may yield stronger results (Levenson, 1992).

In conclusion, our findings are in line with the primary assumption of the functionalist theory of emotion, which suggests that coherence across emotion systems enhances adaptiveness. Here, youth with ADHD displayed a more poorly coordinated emotion systems. These findings are consistent with hypotheses that ADHD is a disorder that involves alterations of emotion reactivity and regulation in addition to difficulties in cognition and behavior.

The findings are clinically relevant in that they demonstrate the importance of teaching children with ADHD skills in the areas of emotion in addition to behavioral and cognitive coping. Specifically, if these results are replicated, it may be beneficial to work clinically with children with ADHD to develop the skills to better interpret the variety of emotional signals they receive or to develop skills to better integrate responses across emotional systems.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

FUNDING

National Institute of Mental Health (R01 MH059105, R01 MH086654)

Reference

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- American Psychiatric Association. (2010). *Ethical Principles of Psychologists and Code of Conduct*. Washington, DC: Author.
- Beauchaine TP (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. [PubMed: 11393643]
- Beauchaine TP, Gatzke-Kopp L, & Mead HK (2007). Polyvagal Theory and developmental psychopathology: emotion dysregulation and conduct problems from preschool to adolescence. *Biol Psychol*, 74(2), 174–184. [PubMed: 17045726]
- Berntson GG, Bigger JT, Jr., Eckberg DL, Grossman P, Kaufmann PG, Malik M, et al. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology*, 34(6), 623–648. [PubMed: 9401419]
- Berntson GG, Cacioppo JT, & Quigley KS (1993). Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology*, 30(2), 183–196. [PubMed: 8434081]
- Berntson GG, Lozano DL, Chen YJ, & Cacioppo JT (2004). Where to Q in PEP. *Psychophysiology*, 41(2), 333–337. [PubMed: 15032999]
- Braaten EB & Rosen LA (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. [PubMed: 10780132]
- Bradley MM, & Lang PJ (1994). Measuring emotion: the Self-Assessment Manikin and the Semantic Differential. *J Behav Ther Exp Psychiatry*, 25(1), 49–59. [PubMed: 7962581]
- Carver CS (2006). Approach, avoidance, and the self-regulation of affect and action. *Motivation and Emotion*, 30(2), 105–110.
- Carver CS, & Harmon-Jones E (2009). Anger is an approach-related affect: evidence and implications. *Psychological Bulletin*, 135(2), 183. [PubMed: 19254075]
- Carver CS, Johnson SL, Joorman J (2009). Two-mode models of self-regulation as a tool for conceptualizing effects of the serotonin system in normal behavior and diverse disorders. *Current Directions in Psychological Science*, 18, 195–199. [PubMed: 20161026]
- Carver CS, & Scheier MF (1990). Origins and functions of positive and negative affect: a control-process view. *Psychological Review*, 97(1), 19.
- Cohen D & Strayer J (1996). Empathy in conduct-disordered and comparison youth. *Developmental Psychology*, 32(6), 988–998.
- Ekman P (1992a). An argument 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. [PubMed: 1348139]
- Ekman P (2016). What scientists who study emotion agree about. *Perspectives on Psychological Science*, 11(1), 31–34. [PubMed: 26817724]
- Fabes RA, 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. [PubMed: 7859549]
- Graziano P, & Garcia A (2016). Attention-deficit/hyperactivity disorder and children's emotion dysregulation: A meta-analysis. *Clinical Psychology Review*, 46, 106–123. [PubMed: 27180913]
- Gross JJ, & Jazaieri H (2014). Emotion, emotion regulation, and psychopathology an affective science perspective. *Clinical Psychological Science*, 2(4), 387–401.
- Gross JJ, & Levenson RW (1993). Emotional suppression: physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology*, 64(6), 970. [PubMed: 8326473]

- Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, ... & Wang P (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, 167(7), 748–751. [PubMed: 20595427]
- 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. [PubMed: 11294223]
- Jennings JR, van der Molen MW, & Somsen RJ (1998). Changes in heart beat timing: reactivity, resetting, or perturbation? *Biol Psychol*, 47(3), 227–241. [PubMed: 9564451]
- Karalunas SL, Fair D, Musser ED, Aykes K, Iyer SP, & Nigg JT (2014). Subtyping attention-deficit/hyperactivity disorder using temperament dimensions: toward biologically based nosologic criteria. *JAMA psychiatry*, 71(9), 1015–1024. [PubMed: 25006969]
- Kring AM, Smith DA, & Neale JM (1993). Individual differences in dispositional expressiveness: The development and validation of the Emotional Expressivity Scale. *Journal of Personality and Social Psychology*, 66, 934–949.
- Krone B, & Newcorn JH (2015). Comorbidity of ADHD and anxiety disorders. *Attention-Deficit Hyperactivity Disorder in Adults and Children*, 98.
- Lang PJ, Bradley MM, & Cuthbert BN (1997). International affective picture system (IAPS): Technical manual and affective ratings.
- Lench HC, Bench SW, Darbor KE, & Moore M (2015). A functionalist manifesto: goal-related emotions from an evolutionary perspective. *Emotion Review*, 7(1), 90–98.
- Levenson RW (1992). Autonomic nervous system differences among emotions. *Psychological Science*, 3 (1), 23–27.
- Lindquist KA, Satpute AB, Wager TD, Weber J, & Barrett LF (2015). The brain basis of positive and negative affect: evidence from a meta-analysis of the human neuroimaging literature. *Cerebral Cortex*, bhv001.
- Livingston JL, Kahn LE, & Berkman ET (2015). Motus moderari: A neuroscience-informed model for self-regulation of emotion and motivation. In *Handbook of Biobehavioral Approaches to Self-Regulation* (pp. 189–207). Springer New York.
- MTA Cooperative Group (1999). Moderators and mediators of treatment response for children with attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry*, 56, 1088–1096 [PubMed: 10591284]
- Marsh P, Beauchaine TP, & Williams B (2008). Dissociation of sad facial expressions and autonomic nervous system responding in boys with disruptive behavior disorders. *Psychophysiology*, 45(1), 100–110. [PubMed: 17868261]
- Martel MM (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. [PubMed: 19508495]
- Mauss IB, Evers C, Wilhelm FH, & Gross JJ (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. [PubMed: 16702153]
- Mauss IB, Levenson RW, McCarter L, Wilhelm FH, & Gross JJ (2005). The Tie That Binds? Coherence Among Emotion Experience, Behavior, and Physiology. *Emotion*, 5(2), 175–190. [PubMed: 15982083]
- 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
- Musser ED, Backs RW, Schmitt CF, Ablow JC, Measelle JR, and Nigg JT (2011). Emotion regulation via the autonomic nervous system in children with attention-deficit/hyperactivity disorder (ADHD). *Journal of Abnormal Child Psychology*, 39, 841–852. [PubMed: 21394506]
- Nigg JT (2006). Temperament and developmental psychopathology. *J Child Psychol Psychiatry*, 47(3–4), 395–422. [PubMed: 16492265]
- Pliszka SR (2000). Patterns of psychiatric comorbidity with attention-deficit/hyperactivity disorder. *Child and Adolescent Psychiatric Clinics of North America*, 9(3), 525–540. [PubMed: 10944655]

- Porges SW (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42, 123–146.
- Porges SW (2007). The polyvagal perspective. *Biological Psychology*, 74, 116–143. [PubMed: 17049418]
- Puig-Antich J, & Ryan N (1996). *Kiddie Schedule for Affective Disorders and Schizophrenia*. Pittsburgh, PA: Western Psychiatric Institute.
- Raudenbush Bryk, Cheong, & Congdon (2004) *HLM 6.0 software* Scientific Software International, Inc., Skokie, IL
- Rosen PJ, Epstein JN, & Van Orden G (2013). I know it when I quantify it: ecological momentary assessment and recurrence quantification analysis of emotion dysregulation in children with ADHD. *ADHD Attention Deficit and Hyperactivity Disorders*, 5(3), 283–294. [PubMed: 23338519]
- Shaw P, Stringaris A, Nigg J, & Leibenluft E (2014). Emotion dysregulation in attention deficit hyperactivity disorder. *American Journal of Psychiatry* 171: 276–293 [PubMed: 24480998]
- Sloan RP, Shapiro PA, Bigger JT, Jr., Bagiella E, Steinman RC, & Gorman JM (1994). Cardiac autonomic control and hostility in healthy subjects. *Am J Cardiol*, 74(3), 298–300. [PubMed: 8037145]
- Sullivan EL, Holton KF, Nousen EK, Barling AN, Sullivan CA, Propper CB, & Nigg JT (2015). Early identification of ADHD risk via infant temperament and emotion regulation: a pilot study. *Journal of Child Psychology and Psychiatry*, 56(9), 949–957. [PubMed: 25968589]
- Thompson RA (2011). Emotion and emotion regulation: Two sides of the developing coin. *Emotion Review*, 3(1), 53–61.
- Wilhelm FH, Grossman P, & Roth WT (2005). Assessment of heart rate variability during alterations in stress: complex demodulation vs. spectral analysis. *Biomed Sci Instrum*, 41, 346–351. [PubMed: 15850130]

Figure 1. HLM results of correspondence (standardized beta weights) between *Negative* facial affective behavior type and respiratory sinus arrhythmia (RSA) across induction conditions (negative and positive collapsed).

Table 1.

Descriptive and diagnostic statistics for ADHD and control groups

Variable	Control (<i>n</i> =50)	ADHD (<i>n</i> =50)	p-Value	Partial- eta ²
Demographics				
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
Stimulant Med. (% on med.)	0.0%	40.0%	< 0.001*	0.135
WISC-IV ¹ FSIQ ² (mean, SD)	111.33 (11.48)	108.54 (14.05)	0.162	0.024
ADHD-RS- T-scores-Parent³ (mean, SD)				
Hyperactive/Impulsive	43.65 (5.36) ⁷	1.61 (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
Comorbid Disorders (%; K-SADS^{4,5})				
Anxiety Disorder	26.0%	32.0%	0.651	0.004
Conduct Disorder (CD)	0.0%	4.0%	0.167	0.029
Oppositional Defiant Dis (ODD)	0.0%	22.0%	< 0.001*	0.181

Note. For continuous variables, including: age, family income, estimated full-scale IQ, ADHD-RS parent-rated sub-scales; and Chi-square comparisons for categorical variables, including: gender, race, child medication status, and comorbid disorders.

¹WISC-IV: Wechsler Intelligence Scales for Children

²Full-Scale Intelligence Quotient (estimated)

³Attention-deficit/hyperactivity disorder rating scale, t-scores

⁴Kiddie Schedule of Affective Disorders and Schizophrenia

⁵0% of the sample had autism, eating disorders, learning disorders, mood, post-traumatic stress disorder, psychosis, or substance use disorders

Hierarchical linear models of group, facial affective behaviors, and task condition predicting autonomic response

Table 2.

Primary Analyses		Secondary Analyses				
	Positive (1) and Negative (2) ¹ (γ , SE)	Anger ² (γ , SE)	Anxious ³ (γ , SE)	Happy ⁴ (γ , SE)	Sad ⁵ (γ , SE)	Surprise ⁶ (γ , SE)
Outcome: Respiratory Sinus Arrhythmia Intercept ⁷	6.03, 0.38 **	6.22, 0.46 **	6.04, 0.38 **	6.34, 0.36 **	6.19, 0.39 **	6.06, 0.46 **
Intercept *ADHD ⁸	0.17, 0.20	0.06, 0.20	0.12, 0.20	0.01, 0.19	0.13, 0.19	0.04, 0.20
Intercept *ODD ⁹	0.38, 0.23	0.45, 0.34	0.45, 0.26	0.44, 0.23	0.38, 0.25	0.65, 0.34
Time ¹⁰	0.05, 0.13	-0.02, 0.14	0.04, 0.13	-0.08, 0.12	-0.02, 0.13	-0.01, 0.14
Time *ADHD	-0.03, 0.07	0.01, 0.06	-0.01, 0.07	0.04, 0.07	-0.01, 0.07	0.02, 0.06
Time *ODD	0.01, 0.09	-0.02, 0.11	-0.01, 0.09	-0.01, 0.08	0.01, 0.09	-0.06, 0.11
Task Condition ¹¹	-0.43, 0.24	-0.09, 0.16	-0.17, 0.18	-0.17, 0.20	-0.08, 0.16	-0.11, 0.17
Task Condition *ADHD	0.07, 0.11	-0.05, 0.07	-0.05, 0.09	-0.06, 0.09	-0.01, 0.07	-0.05, 0.07
Task Condition *ODD	1.13, 0.20	0.06, 0.12	0.08, 0.12	0.18, 0.17	0.01, 0.11	0.11, 0.12
Facial Affect 1 ¹²	0.06, 0.04	0.51, 0.17 *	0.10, 0.03 **	0.05, 0.04	0.07, 0.09	-0.54, 0.23
Facial Affect 1 *ADHD	-0.01, 0.02	-0.15, 0.06 *	-0.05, 0.01 *	-0.01, 0.02	-0.04, 0.03	0.05, 0.05
Facial Affect 1 *ODD	-0.02, 0.03	-0.04, 0.12	<0.01, 0.02	-0.02, 0.04	<0.01, 0.09	0.22, 0.22
Facial Affect 2 ¹³	0.10, 0.03 **	--	--	--	--	--
Facial Affect 2 *ADHD	-0.05, 0.01 **	--	--	--	--	--
Facial Affect 2 *ODD	<0.01, 0.01	--	--	--	--	--
Facial Affect 1 *Task Condition	0.07, 0.04	0.26, 0.18 #	0.03, 0.03	0.05, 0.04	-0.07, 0.07	-0.28, 0.22
Facial Affect 1 *Task Condition *ADHD	-0.02, 0.01 #	-0.13, 0.06 *	<0.01, 0.02	-0.01, 0.01	-0.04, 0.04	-0.04, 0.05
Facial Affect 1 *Task Condition *ODD	-0.03, 0.03	0.12, 0.12	-0.01, 0.01	-0.02, 0.04	0.12, 0.09	0.31, 0.20
Facial Affect 2 *Task Condition	0.03, 0.02	--	--	--	--	--
Facial Affect 2 *Task Condition *ADHD	-0.01, 0.01	--	--	--	--	--

	Primary Analyses		Secondary Analyses				
		Positive (1) and Negative (2) ¹ (γ , SE)	Anger ² (γ , SE)	Anxious ³ (γ , SE)	Happy ⁴ (γ , SE)	Sad ⁵ (γ , SE)	Surprise ⁶ (γ , SE)
Facial Affect 2 * Task Condition * ODD		<0.01, 0.02	--	--	--	--	--
Outcome: Cardiac Pre-ejection Period							
Intercept		104.27, 3.47 **	103.95, 4.13 **	102.95, 3.17 **	103.37, 3.29 **	104.71, 3.25 **	102.25, 4.18 **
Intercept * ADHD		-1.17, 1.70	-1.43, 1.84	-1.43, 1.63	-0.56, 1.63	-1.61, 1.64	-1.06, 1.84
Intercept * ODD		-4.29, 1.97 *	-4.31, 3.04	-3.39, 1.82	-4.82, 1.97 *	-4.20, 1.99 *	-3.59, 3.06
Time		-1.11, 1.06	-1.08, 1.29	-0.66, 0.98	-0.75, 1.01	-1.59, 0.98	-0.41, 1.29
Time * ADHD		-0.25, 0.51	-0.11, 0.58	-0.11, 0.48	-0.50, 0.47	-0.06, 0.48	-0.27, 0.57
Time * ODD		1.03, 0.72	1.06, 0.95	0.69, 0.69	1.25, 0.69	1.28, 0.73	0.82, 0.94
Task Condition		-0.80, 2.48	0.71, 1.47	0.60, 1.58	-0.34, 2.06	1.61, 1.24	-0.10, 1.50
Task Condition * ADHD		-0.18, 0.91	0.23, 0.66	0.28, 0.66	-0.03, 0.79	0.26, 0.67	0.53, 0.66
Task Condition * ODD		1.11, 1.93	-0.95, 1.07	-0.71, 0.80	0.02, 1.60	-1.56, 0.88	-0.90, 1.07
Facial Affect 1		0.34, 0.54	-1.10, 1.56	0.14, 0.32	0.14, 0.60	-2.75, 0.90 *	1.20, 2.07
Facial Affect 1 * ADHD		0.24, 0.15	0.87, 0.53	-0.02, 0.15	0.35, 0.20	0.28, 0.27	-0.61, 0.49
Facial Affect 1 * ODD		-0.61, 0.42	-1.51, 1.06	-0.03, 0.22	-0.61, 0.41	2.07, 0.70 *	0.70, 1.93
Facial Affect 2		-0.04, 0.33	--	--	--	--	--
Facial Affect 2 * ADHD		0.01, 0.13	--	--	--	--	--
Facial Affect 2 * ODD		-0.04, 0.23	--	--	--	--	--
Facial Affect 1 * Task Condition		0.48, 0.46	-0.86, 1.58	0.14, 0.29	0.29, 0.50	0.25, 0.71	1.05, 1.93
Facial Affect 1 * Task Condition * ADHD		0.12, 0.19	0.82, 0.53	0.04, 0.12	0.21, 0.23	-0.09, 0.28	-0.66, 0.47
Facial Affect 1 * Task Condition * ODD		-0.50, 0.42	-1.31, 1.07	-0.22, 0.11 *	-0.44, 0.48	-0.29, 0.58	0.77, 1.77
Facial Affect 2 * Task Condition		0.06, 0.25	-	--	--	--	--
Facial Affect 2 * Task Condition * ADHD		0.05, 0.10	-	--	--	--	--
Facial Affect 2 * Task Condition * ODD		-0.23, 0.17	-	--	--	--	--

Note. Hierarchical Linear Modeling (HLM) was used to predict the outcome variables (i.e., RSA and PEP; in separate models) listed above. The predictors included in each model include the unstandardized beta weight for the predictor of interest.

** indicates significance at the $p < 0.01$ level
* indicates significance at the $p < 0.05$ level

- ¹ Positive (1) and Negative (2): Models including facial affect behavior frequency domains of both positive (i.e., Facial Affect 1) and negative affect (i.e., Facial Affect 2)
- ² Angry: Models including facial affect behavior frequency domain of anger
- ³ Anxious: Models including facial affect behavior frequency domain of anxious
- ⁴ Happy: Models including facial affect behavior frequency domain of happy
- ⁵ Sad: Models including facial affect behavior frequency domain of sad
- ⁶ Surprise: Models including facial affect behavior frequency domain of surprise
- ⁷ Intercept: The intercept of each of the individual models being examined
- ⁸ ADHD: Attention-deficit hyperactivity disorder group status
- ⁹ ODD: Oppositional defiant disorder group status
- ¹⁰ Time: 30 second epoch of task time
- ¹¹ Task Condition: Negative induction versus positive induction condition
- ¹² Facial Affect 1 *: First (or only) facial action coding system frequency
- ¹³ Facial Affect 2: Second (not included in specific emotion models) facial action coding system frequency