Perceived and Actual Emotional Control among Youth: Are There Differential Relations with Anxiety and Aggression?

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Perceived and Actual Emotional Control among Youth:
Are There Differential Relations with Anxiety and Aggression?

A Dissertation

Submitted to the Graduate Faculty of the
University of New Orleans
in partial fulfillment of the
requirements for the degree of

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in
Applied Developmental Psychology

By

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ABSTRACT

The perception of and actual ability to control emotional responses during stressful, taxing situations are important to an individual’s well-being. Studies have shown that both low perceived control and a low actual ability for emotional control are related to internalizing and externalizing problems in youth. However, significant gaps in research exist in terms of testing theoretical predictions about how perceived and actual emotional control are associated with anxiety and aggressive behavior problems, particularly among adolescents. The first goal of this study was to examine two objective measures of actual control (i.e., vagal tone and vagal regulation) and their link with anxiety and aggressive behavior problems in youth ages 11-17 years. The second goal was to examine individual differences in youths’ ability to voluntarily control their heart rate and its association with youths’ perceived control and/or anxiety and aggressive behavior. The final goal was to expand upon Scott and Weems’ (2010) recent work by testing an adapted model of control using these two measures of actual emotional control.

Eighty youth (aged 11-17 years; 51% female; 37.5% African American) and their primary caregivers participated in this study. Youth completed a physiological assessment in which they watched a relaxing video, rested quietly, increased and decreased their heart rate, and performed a mildly challenging cognitive task while their heart rate, skin conductance and body temperature were measured. Youth and their caregivers also completed questionnaires measuring youths’ anxiety, aggression, and perceived control. The results indicated that resting vagal tone (i.e., high frequency – heart rate variability) was negatively associated with anxiety symptoms (and perceived anxiety control) in this adolescent sample but not aggression. Conversely, anxiety (child-reported) and aggression (parent-reported) were both associated with a maladaptive vagal
augmentation in response to a challenging cognitive task. The findings also suggested there were individual differences in youths’ heart rate control (but were better at increasing it) and that less change in increasing heart rate was related to more child-reported anxiety symptoms. However, the results did not provide support for differential of prediction of anxiety symptoms versus aggressive behavior problems between control profiles.

Keywords: anxiety, aggression, emotion regulation, actual control, perceived control, vagal tone
Perceived and Actual Emotional Control among Youth:
Are There Differential Relations with Anxiety and Aggression?

1. Introduction

The desire to control (or regulate) one’s emotions stretches far into the depths of human history and has often been discussed among philosophers, religious orders (e.g., Buddhism), and more recently medical and mental health professionals (e.g., Dingfelder, 2003; Ekman, Davidson, Ricard, & Wallace, 2005; Freud, 1923/1960; Gross, 1998a; Mennin, 2004, Nolan et al., 2005). For example, Indo-Tibetan Buddhists have instructed others how to control their emotions for thousands of years using methods quite similar to those employed in modern cognitive behavioral therapy (CBT) and emotion regulation training (e.g., improve emotional knowledge and awareness, breathing and relaxation exercises; Ekman et al., 2005; Mennin, 2004; Nolan et al., 2005). In addition, both early clinicians (e.g., Freud, 1923/1960) and modern researchers (Cole, Martin, & Dennis, 2004; Gross, 1998a; Thompson, 1994) have suggested that poor emotional control may play a significant role in the development and maintenance of emotional and behavioral problems.

Studies have provided evidence for the non-specific role of emotion dysregulation (actual control) across a number of internalizing (e.g., anxiety) and externalizing (e.g., aggressive behavior) problems in youth (i.e., children and adolescents), but have yet to clarify whether the specific indices (e.g., resting high frequency – heart rate variability [HF-HRV]) of emotional regulation are non-specific indictors of emotional dysregulation across these two types of psychopathology (Beauchaine, 2001; Ciarrochi & Scott, 2006; Pine et al., 1998). Previous theory and research has also suggested that one’s perception of control (perceived control) may be just
as important in determining an individual’s well-being (e.g., Bandura, 1977; 1982; Taylor & Brown, 1988; Weems, Silverman, Rapee, & Pina, 2003). For example, poor perceived control (e.g., self-efficacy) has been theorized to undermine one’s actual ability to control oneself or the environment (even when it is controllable) and has been shown to be associated with a number of negative outcomes in youth (e.g., anxiety-related symptoms; Bandura, 1977; 1982; Seligman, 1975; Weems et al., 2003).

Despite evidence that both actual and perceived control plays a role in youth’s emotional and behavioral problems, relatively little research has empirically assessed both actual and perceived control in the same study or examined whether or not unique patterns of individual actual and perceived control (i.e., control profiles) are differentially related to internalizing or externalizing problems in youth (Endler, Speer, Johnson, & Flett, 2001; Scott & Weems, 2010). Recent studies report discrepancies between one’s perception of and actual anxious responding (e.g., faster heart rate, blushing) with anxious young adults perceiving poor control over physiological responses when in actuality they experience no anxiety-related symptoms at the physiological level (Mauss, Wilhelm, Gross, 2004). Furthermore, other studies examining perceived control in relation to aggressive behavior have found misperceptions of heightened control among aggressive youth (Boxtel, Castro, & Goossens, 2004) suggesting anxiety and aggressive behavior may be uniquely associated with specific control-related beliefs (low vs. high perceived control, respectively).

A recent theoretical model of control may help disentangle the complex relationship between control and both internalizing and externalizing problems (Weems & Silverman, 2006; Scott & Weems, 2010; see Figure 1). In general, the model suggests that youth control profiles
can be identified based upon perceived control and an actual ability to control. Specifically, the model suggests that there are youth with both high actual and perceived control (i.e., upper right quadrant in Figure 1), youth with high actual control and low perceived control (i.e., upper left quadrant in Figure 1), youth with high perceived and low actual control (i.e., lower right quadrant in Figure 1) and youth with both low perceived and low actual control (i.e., lower left). Moreover, the model posits that these control profiles (Figure 1) are important individual differences that may help to identify various fight versus flight responses. For example, recent evidence suggests (Scott & Weems, 2010) a youth’s control profile can help identify those likely to experience anxiety, versus engage in aggressive behavior, versus experience both anxiety and aggression among school-aged children and adolescents (6-17 years). While findings were consistent with predictions in that control profiles (i.e., perceived and actual control) were differentially related to anxiety and aggressive behavior problems in youth, an important limitation of the study was the use of parent-reported measures of competence as an index of actual control, which may not represent youth’s actual ability to regulate anxious or aggressive emotional responses. Therefore, the aim of the proposed study is to expand upon Scott and Weems’s (2010) work and directly test the model using objective measures of actual emotional control (i.e., voluntary control over heart rate and heart rate variability).
The following sections set up the theoretical and empirical background for the present study. The first section, **The Construct of Control: Actual vs. Perceived Control**, provides a review of the broad construct of control, delineates actual from perceived control, and presents an in-depth outline of the Weems and Silverman (2006) theoretical model of control and how it might be related to anxiety versus aggression. The second section, **Emotion and Emotion Regulation: A Domain-Specific Example of Control**, reviews the theoretical underpinnings of emotion and emotion regulation (i.e., a specific domain of control), as well as provides an eclectic working definition of emotion regulation. The third section, **Actual Control: The**
Physiological Regulation of Emotion and Relations with Anxiety and Aggressive Behavior, describes the role of the autonomic nervous system in emotion regulation, provides rationale for using two different physiological markers as proxies of actual emotional control, and outlines research findings linking both markers with anxiety and aggressive behavior problems in youth (i.e., voluntary control over heart rate and high frequency – heart rate variability (HF-HRV)). The fourth section, Perceived Emotional Control: Differential Relations with Anxiety and Aggressive Behavior, briefly reviews two theories of control-related issues associated with anxiety and aggressive behavior and discusses how research suggests the disparate association between both poor and heightened perceived control and anxiety and aggressive behavior, respectively. The final section, Statement of the Problem, delineates the specific research questions and hypotheses for this study.

1.1 The Construct of Control: Actual vs. Perceived Control

Control-related beliefs and actual control over personal or environmental conditions have long been implicated in the development and maintenance of both internalizing and externalizing problems (Chorpita & Barlow, 1998; Seligman, 1975; Taylor & Brown, 1988; Weems & Silverman, 2006; Weisz, Sweeney, Proffitt, & Carr, 1993). However, there still seems to be a great deal of confusion in the literature as to the exact nature and underlying features of this construct (Skinner, 1996; Weems & Silverman, 2006). Skinner (1996) suggests one reason for this confusion is that ‘control’ is a umbrella term often used to illustrate various domain-specific facets of control (e.g., self-efficacy, locus of control, emotion regulation, etc.) and one should be sure to make clear distinctions between actual and perceived control.
1.1.1 Actual Control. An extensive review of the literature suggests that ‘control’ is a multifaceted construct generally used to exemplify how an individual modulates oneself or the environment across a wide-range of domains (Skinner, 1996; Seligman, 1975; Weems and Silverman, 2006; Weisz & Stipek, 1982). More specifically, actual (objective) control may first be viewed as an individual’s ability to produce a desirable (e.g., feeling of calm and relaxation) or avoid an undesirable (e.g., test anxiety) domain-specific outcome (e.g., physiological regulation; Skinner, 1996). Two hypothetical youth, Josh and Sarah help to illustrate different types of actual emotional control over the experience of anger and anxiety. In the first scenario, Josh has just teased and pushed around by a peer in school. Though Josh could easily become angry with the boy he instead begins to take steady, deep breaths in order to keep his own heart rate and breathing at an average pace and thus allowing for state of calmness (production of desirable outcome). In the second scenario, Sarah is about to take a difficult math exam for which she has been studying for weeks. As Sarah begins the test she begins to feel anxious and nervous but then decides to answer the easiest questions first, which will provide her with extra time to devote to much harder questions. Therefore, Sarah makes the exam more manageable and is able to avoid the debilitating panic symptoms (avoidance of an undesirable outcome).

The two examples illustrate that a youths’ actual ability to control an event or situation may be accomplished from either within the individual (e.g., physiological regulation) or within the context of a specific event (e.g., taking a test; Skinner, 1996). Thus, even though both Josh and Sarah demonstrated having actual emotional control (i.e., produced and avoided certain outcomes) over their respective circumstances, control was obtained using two distinct methods. That is, Josh was able to produce a state of calmness through physiological regulation of his own
emotional response by taking slow, deep breaths (individual). Conversely, Sarah actually changed the contextual nature of the test (environmental) to control her feelings of panic by not simply following the traditional format (answering each question in succession), but instead first answering the questions she had confidence were correct.

In a third scenario, Josh and Sarah could control their emotions simply by avoiding the situation all together whether it be physically removing them from it or suppressing emotional states during it. Though this specific type of control is not the focus of the present study, it does pose an important question as to whether this method of avoidant control would constitute a lack of actual control. For example, a number of studies have found that both children and adolescents do employ different forms of emotion regulation (e.g., avoidance and emotional suppression; Gullone, Hughes, King, & Tonge, 2010; Carthy, Horesh, Apter, & Gross, 2010), which may help eliminate the negative emotional experience at that specific moment. However, research also suggests that the long-term consequences of using such strategies is, in essence, an indication of poor emotional control because these strategies seem to produce more negative emotion and more future maladaptive emotional responses (e.g., feelings of anxiety and aggressive responses; Ebata & Moos, 1991).

Lastly and drawing from Weisz and Stipek’s (1982) two-factor model of perceived control, actual control is only possible when one’s ability to produce or avoid an outcome is (1) contingent upon his or her behavior and (2) there is actual competence to perform the actions necessary to produce or avoid an outcome (Abramson, Seligman, & Teasdale, 1978; Bandura, 1982; Seligman, 1975; Skinner, 1996). In regards to the first condition, Seligman and colleagues (Abramson et al., 1978; Seligman, 1975) have suggested that control is only possible when an
individual’s behavior produces a specific outcome (i.e., contingent upon one’s behavior). An excellent example of this contingency-based theory of control is derived from work on Seligman’s (1975) concept of learned helplessness. Specifically, he proposes that control is demonstrated when an individual’s ability to escape from an undesirable outcome (e.g., a noise) is dependent upon his or her own actions (e.g., pressing a lever) and loss of control is exhibited when no action or behavior of the individual will allow him or her to escape an undesirable outcome (Sherrod, Moore, & Underwood, 1979).

The second condition originates from Bandura’s (1977; 1982) self-efficacy theory in which he proposes that even if an outcome is contingent upon one’s actions, personal control is not possible when he or she is not capable of performing that action (i.e., lacks competence). Drawing back upon the hypothetical youth and more specifically the first scenario with Josh, it is possible for him not to know how to regulate physiological states (incompetence) even though he possessed the ability to regulate his own emotional response during the earlier anger-provoking situation (contingent). That is, Josh may have never learned or practiced how to take slow, deep breaths in order to produce states of calmness or relaxation during time of stress. Thus, in this latter example Josh may actually lack control over his emotional state, even though the ability to regulate his positive emotional responses is within his behavioral repertoire.

1.1.2 Perceived Control. Perceived control on the other hand refers to an individual’s ‘belief’ about having the means (contingency) and capability (competence) to produce or avoid an outcome (Bandura, 1977; 1982; Rotter, 1966; Skinner, 1996; Weisz & Stipek, 1982). Specifically, Weisz and Stipek (1982) propose contingency beliefs are based upon one’s perception of whether the ability to produce or avoid an outcome is dependent upon one’s own
actions (i.e., internal locus of control) or is derived from an external source (i.e., external locus of control; Rotter, 1966). For example, Sarah may have an internal locus of control and believe her ability to prevent panicking about the test is due to her implementing an effective test-taking strategy. However, she may just as easily have an external locus of control and believe that the exam being easier was not due to her test-taking strategy but instead was due to her teacher asking easy questions.

Weisz and colleagues (Weisz & Stipek, 1982; Weisz et al., 1993) further utilize Bandura’s (1982) model of self-efficacy to propose that competency beliefs are based upon one’s actual ability to engage in a behavior(s) in order to produce or avoid an outcome when such outcome is contingent upon that particular behavior (i.e., self-efficacy). Bandura (1977; 1982) also suggests that competency beliefs can provide the motivation to learn or master the necessary skills for accomplishing a number of tasks (e.g., emotion regulation). Again drawing upon our hypothetical youth and more specifically the second scenario involving Sarah, she is likely to be motivated to study and practice more math problems if she believes she is capable of performing well on her math exam. In fact, Bandura (1977; 1982) would most likely postulate that Sarah’s high self-efficacy would result in her mastering these math skills over time and thus increasing her actual competency for performing similar math problems in the future.

1.1.3 Application of Weems and Silverman’s (2006) Model of Control to Anxiety versus Aggression. Although theory and research has shown that both actual and perceived control is associated with emotional and behavioral problems, perceived control (e.g., self-efficacy and learned helplessness) has often been thought to play a more significant role in the maintenance of psychopathological problems (Bandura, 1977; 1982; Frazier, Keenan, Anders, Perera, Shallcross,
& Hintz, 2011; Rotter, 1966; Seligman, 1975). That is, previous theoretical models of control suggest the actual ability to control or regulate oneself or the environment is inconsequential in producing or avoiding specific outcomes if one holds poor perceptions of control (e.g., self-efficacy, learned helplessness). For example, Bandura (1977; 1982) postulated that poor self-efficacy alone (despite whether or not actual control is possible) can lead to poor performance across a number of domains (e.g., emotional control) because these negative cognitions may undermine one’s ability to control and may even interfere with learning how to actually control similar situations in the future.

Therefore, the majority of studies examining control-related issues have mainly focused on whether or not poor perceptions of control help predict emotional and behavioral problems and relatively little research has examined both actual and perceived control simultaneously (Skinner, 1996; Weems & Silverman, 2006). However, the reemergence of emotion regulation research over the past two decades has renewed interest in understanding how failure of actual emotional control may be related to emotional and behavioral problems. In fact, a number of studies suggest that emotion dysregulation may play a significant role in both internalizing and externalizing problems (e.g., Beauchaine, 2001; Martel et al., 2007). Furthermore, both theory and research suggests emotion regulation deficits may interact with specific cognitive bias to either promote or attenuate the risk of developing or maintaining specific internalizing and externalizing problems, such as anxiety and aggressive behavior (Weems, Zakem, Costa, Cannon, & Watts, 2005; Weems & Silverman, 2006).

The Weems and Silverman (2006) model of control emphasizes the need to consider both perceived and actual control and that individuals may differ in the correspondence between their
actual and perceived abilities. Such individual patterns of perceived and actual control (i.e.,
control profiles) may be differentially associated with anxiety and aggressive behavior problems
(Scott & Weems, 2010). Furthermore, a person’s perception of control may or may not always
correspond to one’s actual ability to control “internal” events (e.g., emotional response) or
“external” factors in the environment (frightening situation, event, or object). For example, a few
studies have now demonstrated that social phobic individuals may perceive lacking control over
specific anxious responding (e.g., perceive they will experience faster heart rate) during a
speech, though they actually seem quite capable of regulating anxious responses (e.g., no actual
experience of faster heart rate or blushing; Mauss, Wilhelm, & Gross, 2004).

The model presented in Figure 1 (above) more specifically postulates (Scott & Weems,
2010) that individuals with both high actual and perceived control over anxiety-related
symptoms (e.g., greater physiological response) should have the least anxiety and aggressive
behavior problems (i.e., upper right quadrant in Figure 1). Individuals who have a high actual
and low perceived control profile (i.e., upper left quadrant in Figure 1) should experience
relatively more anxiety symptoms than aggressive behavior due to their inaccurate perception of
poor control. That is, their perceived inability to effectively control stressful situations would
likely drive greater anxiety but their actual ability for emotional control would give them less
reason to act out aggressively. Conversely, individuals with low actual and high perceived
control profiles (i.e., lower right quadrant in Figure 1) should exhibit relatively more aggressive
behavior than anxiety symptoms. This is due to them being overconfident in their own abilities to
control various emotional responses (e.g., anxiety and aggression), during stressful times even
when in actuality they have little emotional control. Finally, individuals with both low actual and
perceived control profiles (i.e., lower left quadrant in Figure 1) should display high anxiety and aggressive behaviors due to their perceived inability to handle stressful situations and act out behaviorally due to an actual deficit for emotional control.

As noted, preliminary evidence for the Weems and Silverman (2006) model comes from a test amongst a group of 203 youth (6-17 years of age) in the New Orleans area (Scott & Weems, 2010). Youth were first grouped into the four control profiles as proposed in the Weems and Silverman (2006) model and was based upon standardized total scores of self-reported perceived control over anxiety and parent-reported competencies (e.g., daily activities, social, and academic). The results provided general support for the model in that each control profile significantly differed in terms of both self- and parent-reported anxiety symptoms and aggressive behavior, respectively. The study also supported more specific predictions derived from the model with those youth having a low perceived/high actual control profile reporting or being reported as having more anxiety symptoms than aggressive behavior and youth with a high perceived/low actual control profile reporting fewer anxiety symptoms than their parent’s reports of aggressive behavior.

Though the Scott and Weems (2010) study does present preliminary evidence for control profiles being uniquely associated with specific emotional and behavior problems, the study’s measure of actual control limits our ability to directly test the model. First, the objective measure of control was based upon parent reports of competencies that may be subject to parental biases (i.e., under- or over-reporting). Secondly, the measure assesses competencies ranging from social to academic skills but does not provide a direct measure how well the youth actually control (or regulate) emotional responses. Taking into consideration the main premise of the Scott and
Weems (2010) adaptation of the Weems and Silverman (2006) model of control is that patterns of perceived and actual control may be differentially related to internalizing and externalizing problems, it would be best to provide a measure of actual control that complements one’s perceived control.

One domain-specific example of control that may best capture individual differences in both actual and perceived control is emotion regulation, which refers to how an individual regulates emotional experiences within oneself and across a number of situations during one’s lifetime (Gross, 1998a). Emotion regulation provides a strong foundation for testing the Weems and Silverman (2006) model because it is theoretically relevant to discerning between one’s actual and perceived control over emotion-related responses (e.g., faster heart rate associated with anxiety). Furthermore, it adheres to the same underlying principles as control in that the elicitation or suppression of an emotional response (i.e., desired and undesired emotions) is contingent upon one’s own behavior and one must possess the actual capability to control his or her own emotional states. Therefore, the next section provides a concise but detailed description of a theoretical perspective on both emotion and emotion regulation, provides a working definition of emotion regulation, and briefly describes various ways youth regulate (or control) their emotions.

2. Emotion and Emotion Regulation: A Domain-Specific Example of Control

2.1 Emotion

What is emotion? This simple question has plagued philosophers and researchers for many of years and it seems the answer is more complex than the question itself (Frijda, 1986; Gross, 1998a; James, 1884; Ochsner & Gross, 2005; Zajonc, 1980; 1984). In fact, a standard
definition of emotion has yet to be developed within the literature, even though most theoretical perspectives agree upon several key features (Campos, Frankel, & Camras, 2004; Cole, et al., 2004; Gross, 1998a; Gross & Thompson, 2007). For example, a number of emotion theorists suggest emotions organize or prepare other internal regulatory systems (e.g., behavioral or cognitive) or external events (e.g., social interactions) for accomplishing a personal and meaningful goal (Campos et al., 2004; Gross & Thompson, 2007; Thompson, 1994). Therefore, we have chosen to adopt Gross and Thompson’s (2007) modal model of emotion because it (1) best provides a comprehensive overview of emotion as based upon differing theoretical perspectives (Campos et al., 2004; Cole et al., 2004; Thompson, 1994) and (2) is a solid foundation for establishing a working definition of emotion regulation.

Gross and Thompson’s (2007) modal model of emotion represents an integrated and expanded view of several emotion theories and incorporates general ideas that are presented within other theoretical models (Cole et al., 2004; Frijda, 1986; Gross, 1998a; James, 1884). Specifically, Gross and Thompson (2007) propose that most emotion theories agree upon three core features of emotion, which are:

1) An emotion occurs when an individual attends to an internal or external event that pertains to achieving a specific goal. The exact nature of one’s goal may vary widely from avoiding dangerous situations (personal and enduring goal) to performing well on a math test (self-efficacious and transient goal), but the meaning of each goal will produce a specific emotion. Therefore, as the meaning of a particular goal changes so does the emotion that arises in response to this goal.
2) An emotion arises within a flexible, multi-system structure and involves independent but also interactive changes among cognitive, behavioral, and affective (subjective and physiological) sub-systems of the individual. For example, anxiety may manifest itself within increased negative cognitions (e.g., “I have no control over passing this exam.”), avoidance behaviors (e.g., “I’m too sick to go to school”), and both physiological (e.g., faster heart rate) and subjective feelings of anxiety (e.g., “I feel nervous.”)

3) Emotions are malleable and may be modulated at any time or level of the individual during the emotion-eliciting process and vary across a number of situational domains.

These three core features provide the contextual basis for explicating the specific sequential mode of an emotional response as presented in Gross and Thompson’s (2007) modal model of emotion. A more detailed discussion of the model is presented below and illustrates how these features are interwoven into a time-dependent and integrative view of an emotional response.

The emotional process first begins when a person attends to a specific internal (e.g., memory) or external (e.g., father yelling) event (i.e., an emotional cue) that holds some meaning for him or her. This selective attention towards an emotional cue next evokes a cognitive appraisal or rapid assessment of the situation in regards to such things as familiarity, valence, and importance (Gross & Thompson, 2007). If this initial appraisal warrants further action from the individual, then it is likely to trigger specific changes (or responses) at one or more of the individual sub-systems (i.e., cognitive, behavioral, and affective [physiological and experiential]) and thus may result in an emotional response (e.g., anxiety, anger, sadness, etc.). However, Gross and Thompson (2007) are quick to note that even attention to or appraisal of an emotional
cue does not necessarily mean an emotional response will arise because emotions are malleable and may be regulated at any point during the emotion generative process.

2.2 Emotion Regulation

It is this last feature of the model that is most relevant because it relates to whether or not an individual is capable of engaging in emotional control. Similar to emotion, emotion regulation is a complex and obscure construct that has generated a number of theoretical perspectives trying to explain how and when emotions are regulated (Campos et al., 2004; Cole et al., 2004; Gross, 1998; James, 1884; Thompson, 1994). Unfortunately, the waters appear even more muddled in terms of defining emotion regulation and there is still a bit of disagreement as to what type of emotions are regulated, when emotions can be regulated, and how emotions are regulated. Thus, drawing from the work of several emotion regulation theories (e.g., Campos et al., 2004; Cole et al., 2004; Gross, 1998a; Thompson, 1994) an eclectic, working definition of emotion regulation that encompasses the full spectrum of actual emotional control is presented for this study. Emotion regulation may be defined as the automatic or voluntary modulation (i.e., enhance, decline, or maintain) of negative and positive emotions at any level of the individual (i.e., cognitive, behavioral, affective [subjective or physiological]) to achieve a personal and meaningful goal (Cole et al., 2004; Gross, 1998a; 1999; Thompson, 1994). Furthermore, emotion regulation may occur at any point during the emotional generative process and may involve modifications to the latency, intensity, rise time, magnitude, and duration of an emotional response (Campos et al., 2004; Gross, 1998a; Thompson, 1994). However, emotion regulation does not involve emotion being a regulator of other individual sub-systems (e.g., behavioral
regulation) because this process would be synonymous with what constitutes as an emotion (Fridja, 1986; Gross & Thompson, 2007).

Although this integrative definition provides a general understanding of what emotion regulation entails in terms of what is regulated and when regulation occurs, it still does not specifically answer the question “How are emotions regulated?” Gross (1998b) proposes there are a multitude of ways an individual may regulate emotions from the time an emotional situation or event is presented (i.e., before an emotional response) to well after the emotion is generated or experienced. Specifically, he postulates that there are two broad time-dependent categories of emotion regulation, which he coined antecedent-focused and response-focused emotion regulation. Even though this study is more interested in assessing indices related to response-focused emotion regulation, we briefly described the former strategy to illustrate the relative difference between these two concepts.

Antecedent-focused emotion regulation refers to the change or modification of an emotion even before an emotional response occurs and may involve such strategies as: 1) situation selection, 2) situation modification, 3) attention deployment, or 4) cognitive change (Gross 1998b; Gross & Thompson, 2007). For example, Gross (1998b) suggests individuals often refocus their attention on a non-threatening stimuli (e.g., pretending everyone is naked) when giving speeches and others may use reappraisal (i.e., “This test will be easy.”) to modify the emotional experience (e.g., fear) before a specific emotion has been elicited. Similarly, some individuals may change or modify the contextual nature of the situation (i.e., situation modification), as the hypothetical “Sarah” did above with her math test, in order to avoid
experiencing undesired emotional experiences (e.g., panic) she focused on her competencies first.

In comparison, response-focused emotion regulation occurs after an emotional response has begun and may consist of controlled changes or modifications (e.g., diminish, prolong) in the response pattern of one or more of the cognitive, behavioral, or physiological sub-systems to exert control over the emotional experience (Gross, 1998b). This emotion regulation strategy may involve modification of negative cognitions often induced when feeling angry (e.g., modifying “She always screws up” to “Everyone makes mistakes”) in an attempt to ‘calm down’ or approaching a feared object or situation to reduce feelings of anxiety (e.g., Sarah taking her test even though she nervous and experiencing butterflies). Furthermore, Josh’s deep breathing is a perfect example of response-focused emotion regulation because it was employed once he experienced feelings of anxiety (e.g., faster heart rate) and reduced the heightened anxiety-related physiological response he experienced (i.e., lowered heart rate).

A specific type of response-focused emotion regulation that has received considerable attention as of late has been the physiological regulation of an emotional response (Appelhans & Luecken, 2006; Culbert, Kajander, & Reaney, 1996). One reason for increased interest in this particular emotion regulation strategy is that it is theoretically and practically a more objective and direct measure of an individual’s ability to regulate emotional states across a number of situations (e.g., resting, cognitive tasks; Porges, Doussard-Roosevelt, & Maiti, 1994; Thayer & Lane, 2000). Additionally, specific physiological markers of dysregulation (e.g., heart rate variability) have been shown to be consistently associated with poor attention and emotion regulatory processes, as well as youth’s emotional and behavioral problems (e.g., Butler,
Wilhelm, & Gross, 2006). Thus, the following section provides an overview of how emotion arises and is regulated (voluntarily and involuntarily) within the autonomic nervous system and how two physiological measures may serve as a proxy for actual emotional control.

3. Actual Control: The Physiological Regulation of Emotion and Relations with Anxiety and Aggressive Behavior

The physiological response to an emotional or stressful event (e.g., butterflies, heart pounding) is thought to arise from activation of the autonomic nervous system (ANS; Appelhans & Luecken, 2006; Porges et al., 1994; Thayer & Lane, 2000). The ANS is a complex, whole-body system that is composed of two distinct and often opposing branches called the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). The SNS and PNS branches innervate a number of bodily systems (e.g., the heart, larynx, lungs, etc.) and are theoretically responsible for both helping produce an emotional response (i.e., reactivity) and regulate it via controlled changes or modulation of emotion response parameters (i.e., latency, intensity, rise time, magnitude, and duration) through various afferent and efferent neurobiological connections (e.g., vagal-heart connection; Beauchaine, 2000; Porges et al., 1994; Thayer and Lane, 2000).

More specifically, the primary function of the ANS is to help an individual maintain bodily homeostasis during times of rest and both quickly and efficiently adapt to stressful and emotional situations that call for immediate action (Porges et al., 1994; Thayer and Lane, 2000). The SNS’s role in this process is to prepare the body for greater metabolic output and help organize multiple bodily sub-systems (e.g., cognitive, behavioral, neurobiological) in response to stressful, taxing situations (similar to an emotional response). The SNS achieves this goal mainly through efferent outputs from fight and flight neurological regions (e.g., amygdala) and has an
excitatory effect (i.e., reactivity) on such organs as the heart (e.g., increases one’s heart rate). In contrast, the PNS’s role is to regulate the amount of time and degree of control the SNS has over bodily sub-systems during stressful/emotional events and seems to promote both the growth and restorative functions of the body during times of rest (Porges et al., 1994; Thayer & Lane, 2000). The PNS also accomplishes these tasks via neurobiological connections but in comparison to the SNS has inhibitory effects (i.e., regulation) on these similar bodily systems (i.e., decreases one’s heart rate).

3.1 Changing One’s Heart Rate: Voluntary Emotional Control

Though both the SNS and PNS seem to play a crucial role in helping the body adjust to a number of internal and external events, the earliest focus of psychophysiologica research was based on the more general measures of physiological states that represent both SNS and PNS influence (e.g., increased heart rate; increased galvanic skin response [GSR]; Van Lang, Tulen, Kallen, Rosbergen, Dieleman, & Ferdinand, 2007). Reasoning for this long-term investment in more general physiological measurements is that they are practically easy to measure and seem to provide reliable and valid indices of maladaptive emotional responses (thus suggesting poor regulation) associated with internalizing and externalizing problems (Van Lang et al., 2007; Weems et al., 2005).

Additionally, the common theme across past theories linking physiological reactivity and psychopathology was that internalizing and externalizing problems are differentially related to an over-active or under-active physiological system associated with avoidant (flight) and approach (fight) behaviors, respectively (Porges et al., 1994). This theoretical perspective led to a number of studies from the 1970s to 1990s that were designed to specifically examine whether or not an
individual could voluntarily control his or her heart rate (i.e., cognitively increase or decrease heart rate) and subsequently use this information to treat under- or over-reactive physiological profiles (Culbert, Kajander, & Reaney, 1996; Gatchel, 1975; Schneider, Sabot, Herrmann, & Cousins, 1978; Sirota, Schwartz, & Shapiro, 1976).

Several conclusions can be drawn from this specific line of research. First, it seem there are individual differences in the ability to voluntarily control heart rate and that on average most adults are better at increasing than decreasing their heart rate (e.g., Schneider et al., 1978; Scott & Weems, 2012). However, past research suggests that the act of decreasing one’s heart rate is associated with less perceived threat of an aversive stimulus and thus further exploration of both increasing and decreasing heart rate is critical (Sirota et al., 1976). Secondly, perceptions of control (e.g., locus of control) may have a significant impact on an individual’s ability to change his or her physiological states (e.g. heart rate; Blankstein & Egner, 1977; Ray & Lamb, 1974; Schneider et al., 1978). More specifically, several of these studies reported that individuals who viewed themselves as having more personal control were better able to increase their heart rate and some even responded better to biofeedback sessions (Schneider et al., 1978). Thus from a theoretical perspective, individuals who perceive themselves as having greater personal control may be better able to voluntarily regulate physiological states, such as heart rate. Finally, learning how to control one’s heart rate using biofeedback techniques does seem to significantly improve physiological regulation and is associated with better overall adjustment such as less anxiety and anger problems (e.g., Culbert et al., 1996; Schneider et al., 1978; Sirota et al., 1976). That is, voluntary regulation of heart rate may also serve as proxy of emotional regulation and
individuals with greater physiological control may better regulate emotional reactions during stressful events.

Overall, it seems voluntary control of heart rate may serve as one index of an individual’s actual emotional control (Culbert et al., 1996; Scott and Weems, 2010). Though the ability to learn how to control one’s heart rate has been demonstrated across several studies in adults (Blankstein & Egner, 1977; Ray & Lamb, 1974; Schneider et al., 1978), individual differences in youths’ actual ability to voluntarily control heart rate without prior feedback is not as well-established. Given this may provide an actual marker of emotional dysregulation in youth that can be easily assessed in various settings (e.g., school, clinician’s office), it seemed of practical and empirical importance to establish whether there were individual differences in voluntary heart rate control among youth and whether it would be associated with perceived control and youths’ anxiety problems.

3.2 Resting Vagal Tone and Vagal Regulation: Involuntary Emotional Control

Though the evidence suggests that individuals may voluntarily control their emotions, it is important to note here that most emotion regulatory processes (e.g., controlling heart rate) are still thought to be under involuntary or nonconscious control systems (Gross, 1998a). In terms of physiologically-mediated emotional control, a number of theories and research findings suggest emotion regulation via the autonomic nervous system is automatic and that PNS-mediated dysregulation is probably more closely associated with emotional and behavioral problems (Appelhans & Luecken, 2006). Therefore, the examination of this specific physiological pattern (i.e., poor heart rate variability) may provide a better indicator of who is and is not actually capable of emotional control (Beauchaine, 2001; Porges et al., 1994; Thayer & Lane, 2000).
Taking into consideration the complexity of assessing and interpreting this index of autonomic emotional control, the rest of this section is devoted to explaining how one conceptualizes and measures heart rate variability (HRV) indices specific to emotion regulation (i.e., HF-HRV).

Though most studies have examined the unique link between SNS reactivity and psychological problems (e.g., heightened heart rate or skin conductance in response to stress and anxiety; Culbert et al., 1996; De Young, Kenardy, & Spence, 2007; Weems et al., 2005), the specific role of the PNS in both emotional dysregulation and psychopathology has gained more recent attention (Appelhans & Luecken, 2006). One reason for this shift towards PNS biomarkers (e.g., heart rate variability; HRV) is that they may better represent estimates of whether or not an individual is actually capable (or competent) of emotion regulation (e.g., Porges et al., 1994 and Thayer & Lane, 2000). Previous findings support this view in that the PNS has a much quicker effect on heart rate as compared to the SNS (.5 second vs. 1 second delay, respectively) and is able to modulate heart rate across all frequency domains of the cardiac cycle (Berntson et al., 1997). Additionally, several studies have provided evidence for a strong link between PNS biomarkers and both attentional and emotion regulating processes (Butler et al., 2006; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009; Wetzel, Quigley, Morell, Eves, & Backs, 2006).

Theoretically, the PNS has direct control over heart rate via the vagus nerve (i.e., tenth cranial nerve), which forms a negative feedback loop between several neurological structures thought responsible for top-down emotion regulation (e.g., nucleus ambiguus, medial prefrontal cortex) and the heart’s sinoartial (SA) node (i.e., pacemaker) (Oschner & Gross, 2005; Porges et al., 1994; Thayer & Lane, 2000). Porges (2007) further proposes the PNS regulates emotion via
triggering vagal withdrawal or induction (i.e., vagal tone) in response to a specific emotional response (e.g., anxiety or anger). For example, a normal anxious or fearful response to a scary situation (e.g., giving a speech) would most likely cause vagal withdrawal and result in a more sympathetic response (i.e., an increase in heart rate). In contrast, vagal induction may occur in response to feelings of anger or hostility thus reducing sympathetically-mediated heart rate response and helping one “keep a cool head” in spite of experiencing negative emotions.

One common method of estimating vagal tone or parasympathetic-mediated heart rate control is through assessing the degree of rhythmic fluctuation (i.e., increasing and decreasing) of normal beat-to-beat (or R-R) intervals in a cardiac cycle (e.g., 5 minutes) during inhalation and exhalation of the respiratory cycle (i.e., respiratory sinus arrhythmia [RSA]; Beauchaine, 2001; Berntson et al., 1997; Porges et al., 1994). Specifically, RSA is considered a measure of vagal tone that results from an individual’s heart rate increasing during inhalation due to reduced vagal efferent activity and decreasing during exhalation because of induced vagal efferent activity. Porges (2007) refers to this phenomenon as the “vagal brake” of emotion in that greater efferent vagal tone acts like a brake in helping slow one’s heart rate and releasing this brake (i.e., reduced efferent vagal tone) aids in increasing heart rate. He further postulates that the purpose of this “vagal brake” is to help the individual flexibly and rapidly respond to stressful and emotional tasks. Thus, a closer examination of RSA indices may provide a fair estimate of one’s ability (or lack thereof) to control emotional responses (Beauchaine, 2001; Porges, 2007; Thayer & Lane, 2000; 2009).

Though several statistical methods are used to assess autonomic vagal control (e.g., peak-to-valley; Porges et al., 1994), one of the more popular statistical techniques is the power spectral
analysis of the artifact-free inter-beat intervals as recorded using an electrocardiogram (EKG),
which has allowed researchers to partial out the effects of SNS and PNS on one’s heart rate
(Appelhans & Luecken, 2006; Berntson et al., 1997; Task Force of the European Society of
Cardiology and the North American Society of Pacing and Electrophysiology, 1996). This
frequency-based technique specifically produces three spectral components (very low frequency
[VLF], low frequency [LF], and high frequency [HF or RSA frequency band]) with the absolute
power or variance of heart rate (ms$^2$/Hz) being distributed within each frequency domain
(frequency is quantified using hertz [Hz] or cycles per second). Pharmacological blockade
studies suggest the HF-HRV (RSA frequency band) component provides a true estimate of PNS-
mediated heart rate control, while LF-HRV is often a mixed measure of both SNS- and PNS-
mediated variation (e.g., Cacioppo, Berntson, Binkley, Quigley, Uchino, & Fieldstone, 1994).
Furthermore, the use of frequency-based methods of RSA estimation as opposed to other
methods (e.g., time domain indices) in youth and adult samples has shown little differences in
measurement strategy and/or the relationship between specific HF-HRV patterns and emotional
and behavioral problems (e.g., anxiety, depression, aggressive behavior; Salomon, Matthews, &
Allen, 2000).

Theory and research suggests assessment of HF-HRV during times of rest (supine or
sitting) is a reliable and valid indicator of general PNS functioning and one’s ability to regulate
or control emotional responses during stressful and/or emotional events (Beauchaine, 2001;
Porges, 2007; Thayer & Lane, 2000). For example, pharmacological blockade studies have also
found that PNS-mediated control is more prevalent during rest (supine or sitting; Cacioppo et al.,
1994) and specific irregular HF-HRV patterns (i.e., low HF-HRV) are thought to be signs of an
inflexible and rigid PNS-mediated regulatory system (Porges, 2007; Thayer & Lane, 2000). Therefore, individual’s with low resting HF-HRV are likely to experience vagal dysregulation (loss of emotional control) in response to stressful situation and result in a collapse of both physiological regulation of emotion (e.g., more sympathetically-mediated control) and other neurobiological structures related to emotional control through disruption of negative feedback loops from the SA node (Thayer and Lane, 2000).

Additionally, more recent theory and research has also suggested that changes in HF-HRV in response to a challenging or stressful event as compared to baseline measures (i.e., vagal regulation; stress HR-HRV minus baseline HF-HRV) may serve as an alternative indicator of emotional dysfunction in youth (i.e., vagal regulation; Porges, 2007; Hinnant & El-Sheikh, 2009; Monk et al., 2001). Porges (2007) has suggested that greater vagal suppression is a flexible, adaptive response to a challenging or stressful event, which in turn promotes functional behavior (e.g., social engagement and focused attention) and may help one effectively prepare for fight (approach) or flight (avoidant) responses. In contrast, he proposes that individuals who suffer from such emotional and behavioral problems such as anxiety and aggression display a more maladaptive or dysregulated response to stress with considerably less or no vagal suppression (i.e., vagal augmentation; no change or an increase in HF-HRV from resting baseline). However, as will be discussed in this next subsection, support for this theory and data on the link between HF-HRV indices (resting and vagal regulation) and both anxiety symptoms and aggressive behavior problems in youth is mixed and so further research is needed to clarify the nature of resting HF-HRV and vagal regulation in terms of anxiety and aggressive behavior problems in youth.
3.2.1 Resting Vagal Tone: Relations with Anxiety and Aggressive Behavior. The examination of distinct autonomic markers (e.g., resting heart rate, skin conductance) for youth who suffer from internalizing and externalizing problems has been an ongoing process for a number of years (e.g., Culbert et al., 1996; De Young et al., 2007; Weems et al., 2005). Most researchers have focused their attention on more easily measured indices such as mean heart rate or skin conductance (at rest or in response to stress or fearful situations) and have provided some evidence to suggest a specific link between under- and over-control of physiological responses and externalizing and internalizing problems, respectively (e.g., De Young et al., 2007; Lorber, 2004; van Lang et al., 2007; Raine, Venables, & Mednick, 1997; Weems et al., 2005).

In contrast, a growing body of work examining physiological indices of emotion regulation (e.g., resting vagal tone) suggests youth with internalizing and externalizing problems may share an underlying and non-specific dysregulation of emotion (e.g., low resting vagal tone; El-Sheikh, Arsiwalla, Hinnant, & Erath, 2011; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Greaves-Lord et al., 2007; Mezzacappa et al., 1997; Pine et al., 1998). However, there are still inconsistencies in the literature that bring into question the non-specificity of vagal tone indices, specifically in regards to anxiety and aggressive behavior problems. This subsection is devoted to discussing these inconsistencies and the ways in which future research needs to further examine the link between vagal tone indices (at rest and during stressful events) and both anxiety and aggression during adolescence.

In terms of anxiety-related problems, several studies examining differences in resting vagal tone between youth with and without anxiety disorders have for the most part found lower resting vagal tone in anxiety disorder groups (Blom, Olsson, Serlachius, Ericson, & Ingvar,
found in a sample of 64 children and adolescents (8-18 years of age) that those youth with anxiety disorders ($n = 34$) had lower resting vagal tone as compared to an age- and gender-matched control group ($n = 30$; $813.39 \text{ ms}^2/\text{Hz} [SD = 998.28]$ vs. $2389.44 \text{ ms}^2/\text{Hz} [SD = 2919.42]$, respectively). In addition, Blom et al. (2010) also reported reduced resting vagal tone in a sample of adolescent females with an anxiety disorder ($n = 20$) as compared to a healthy control group ($n = 57$), but this finding was only significant for those girls who were currently taking a serotonin reuptake inhibitor ($5.22 \ln[\text{ms}^2/\text{Hz}]$ vs. $5.94 \ln[\text{ms}^2/\text{Hz}]$, respectively) and medication use explained 15.5% of variance in vagal tone. However, Monk et al. (2001) also reported finding a significant difference in resting vagal tone between medication-free anxiety-disordered youth ($n = 24$) and controls ($n = 12$) ranging in age from 9-18 years when controlling for age and gender as covariates ($2869.16 \text{ ms}^2/\text{Hz} [SD 3654.18]$ and $5877.63 [SD = 4179.15]$, respectively). Taken together these two findings suggest that researchers be aware that the use of medication may play a role in decreased vagal tone reported in anxious youth, but it is also possible that other factors (e.g., gender as a moderator not just covariate) may also explain group differences in resting vagal tone. That is, even though differences between anxiety disordered and control samples imply a linear association between levels of subjective anxiety, it is reasonable that group differences reported may be due to other differences in the samples used.

Importantly, it is not clear from the extant literature whether there is in fact a linear association between resting vagal tone and anxiety symptoms (not just general internalizing problems) with inconsistent findings being reported across a number of studies. For example, El-Sheikh and colleagues (El-Sheikh, Hargar, & Whitson, 2001; El-Sheikh & Whitson, 2006; El-
Sheikh et al., 2011; Wetter & El-Sheikh, 2012) have reported “no significant association”
between vagal tone and child-reported anxiety symptoms on the Revised Child Manifest Anxiety
Scale (RCMAS; Reynolds & Richmond, 1978; r’s ranging from +.02 to -.17, p > .05). These
findings were from four independent samples of children (n’s ranged from 57 to 251 and their
ages from 6-11 years) who came from two-parent homes and had no diagnosis of attention-
deficit hyperactivity disorder (ADHD). Mezzacappa et al. (1997) also reported “no significant
correlation” between child-reported anxiety levels (anxiety scores were derived from the “Social
Anxiety” scale of the Jesness Inventory of Adolescent Personality [Jesness, 1971]) and resting
vagal tone in a homogenous sample of 15 year-old boys (specific statistics were not reported for
the association between anxiety and resting vagal tone in this publication).

In contrast, other studies have reported finding a significant link between resting vagal
tone and anxiety problems in youth. For example, El-Sheikh et al. (2011) found in their second
study that there was a negative association between resting vagal tone and two separate measures
of anxiety problems (RCMAS[r = -.17, p < .05] and Trauma Symptoms Checklist for Children
[TSCC; Briere, 1996; r = -.17, p < .05]) among a sample of 219 second and third graders (Mage =
9.31, SD = .79) who also came from two-parent homes and had no ADHD diagnosis. Greaves-
Lord et al. (2007) also reported a significant association between resting vagal tone and anxiety
symptoms (Revised Child Anxiety and Depression Scale - Child Version [RCADS-C; Chorpita,
Yim, Moffitt, Umemoto, & Francis, 2000] for boys (β = -.15, p < .05 R² = 1.7 %), but not girls (β
= .05, p < .05 R² = .2 %) in large community sample of early adolescents (n = 2230; age 10-13
years). The pattern of findings suggests that studies should continue to examine the moderating
role of gender in this relationship.
In addition, only one study (Mezzacappa et al., 1997; 15 year-old boys-only sample) has examined the linear association between resting vagal tone and anxiety problems in older adolescents (i.e., 14-17 years of age). This is surprising given that both past theory and research (e.g., Casey, Jones, & Hare, 2008; Hollenstein, McNeely, Eastabrook, Mackey, & Flynn, 2011; Vasilev et al., 2009) suggests this is a critical developmental period with neurobiological structural asymmetry (mature limbic region but still underdeveloped pre-frontal cortical regions; Casey et al., 2008), a developmental shift to decreased RSA across adolescence (Hollenstein et al., 2011) and lower resting RSA being associated with emotional dysregulation (Hollenstein et al., 2011; Vasilev et al., 2009). Thus, given the inconsistency of findings across several studies, the possible moderating role of gender, and the limited knowledge of this association in older adolescents, it research is needed to further explore the linear relationship between vagal tone and anxiety using multiple measures of anxiety (child and caregiver reports) and utilizing adolescent samples.

In terms of aggressive behavior problems, the exact nature of the link between lower resting vagal tone and aggression across youth it is also still not quite clear given the limited studies specifically examining aggression (and not more externalizing problems in general) and using samples consisting of both boys and girls. However, there seems to be accumulating evidence to support the negative relationship between vagal tone and aggressive-related behavior, at least in boys (Gordis et al., 2010; Mezzacappa et al., 1996; 1997; Pine et al., 1998). For example, Mezzacappa et al. (1997) found resting vagal tone was negatively associated with antisocial behavior (Parameter estimate = -4.06, $p < .05$) in a sample of 15-year-old adolescent boys and Pine et al. (1998) reported lower resting vagal tone was associated with externalizing
problems \( r = -.38, p < .05 \) among 62 adolescent boys at risk for delinquency (7-11 years of age).

Similarly, the two studies that have examined the vagal tone and aggression link in samples consisting of both boys and girls have found that this negative relationship is only significant with boys and suggests gender is a moderating factor. For example, Gordis et al. (2010) reported in a sample of 362 youth (9-16 years of age; even when controlling for a history of maltreatment exposure) that resting vagal tone was associated with aggressive behavior problems but only for boys \( (\beta = -.21, p < .05; \text{girls: } \beta = -.11, p > .05) \). Beauchaine, Hong, and Marsh (2008) found a similar pattern among youth aged 8-12 whose parents reported they were diagnosed with oppositional defiant disorder and/or conduct disorder in that aggression (i.e., \( T \) score > 70 on Child Behavior Checklist Aggression subscale; Achenbach, 1991; Achenbach & Rescorla, 2001) was associated with change of resting vagal tone over three minutes but once again was only observed in boys (see Beauchaine et al., 2008 for visual depiction of change for boys and girls with low and high aggressive behavior). Taken altogether these findings provide initial evidence that the negative association between vagal tone and aggression in youth may be gender specific and that as with anxiety problems gender should be tested as a possible moderator of this relationship.

Additionally, past theory and research suggests that aggression should be treated as a multidimensional construct with subtypes being based upon the forms (proactive versus reactive) and functions (overt versus relational) of aggression (Marsee & Frick, 2007; Vitaro, Gendreau, Tremblay, & Olingny, 2006). More specifically, the different subtypes of aggression have been shown to be uniquely and differentially related to various individual characteristics, such as
reactive forms of aggression being associated with anxiety-related symptoms and emotional
dysregulation. For example, Marsee (2008) found that anxiety was uniquely related to reactive
relational aggression and that this path was indirect via emotional dysregulation problems.
Scarpa, Haden, and Tanaka (2010) recently tested the link between resting HRV and proactive
versus reactive aggression in a sample of 62 youth (aged 6-13 years). The results were consistent
with past theoretical models of aggression in that there was a negative association between
HRV and reactive aggression ($\beta = -.47, p < .05$), but a positive association between HRV and
proactive aggression ($\beta = .43, p < .05$). These findings are consistent with past research (see
Frick & Morris, 2004 for review) that suggests reactive forms of aggression may be more closely
associated with physiological states of overarousal or emotional dysregulation, while proactive
aggression may be more associated with an emotionally ‘cold-tempered’ state or in this case
physiological underarousal. This study highlights the importance of examining the biological
underpinnings of the more specific subtypes of aggression and that lower resting vagal tone may
be a better indicator of emotional dysregulation associated with reactive aggression but not
proactive aggression. However, given that only one study has examined with the relationship
between vagal tone and the different forms of aggression, the specificity of resting vagal tone in
relation to specific types of aggression is still unknown and needs further investigation.

3.2.2 Vagal Regulation (Change in Vagal Tone from Rest to Stressor): Relations
with Anxiety and Aggressive Behavior. As previously discussed above in section 3.2, past
theory suggests that poor vagal regulation in response to a challenging or stressful event may be
a sign of inflexibility and rigidity of physiological responding to stress and may be a non-specific
indictor of emotional dysregulation in both internalizing and externalizing problems (Porges,
That is, youth with a wide range of emotional and behavioral problems may have a more maladaptive blunted or augmented vagal response (i.e., little change or increase in vagal tone from baseline to stressor) that prevents them adequately preparing for stressful events as opposed to the more adaptive and quick vagal withdrawal which prepares the individual to cope both emotionally and physically in merely seconds (Porges, 2007).

Several studies have begun to investigate the link between vagal regulation and both internalizing and externalizing problems in youth between 6 to 17 years of age and control for baseline measures of vagal tone (i.e., some studies only examine the correlation between youths’ problems and vagal tone during the stress task and not the change from resting baseline to stressor; see Graziano & Derefnko, 2013 for review). However, only three studies have specifically examined the association between vagal regulation and anxiety and no study to date has examined link between vagal regulation and aggression in youth when controlling for baseline measures of vagal tone. Thus, as with resting vagal tone, the relationship between vagal regulation and both anxiety and aggressive behavior problems is still quite unclear and further empirical investigation is needed.

In terms of the association between anxiety problems and vagal regulation, inconsistent findings are reported within the literature. In the first study, Monk et al. (2001) reported that anxiety-disordered youth (9-18 years of age) exhibited little change of vagal tone from baseline to CO² challenge (2869.16 ms²/Hz to 3226.81 ms²/Hz) relative to healthy controls (5877.63 ms²/Hz to 4632.5 ms²/Hz), but after respiration rates were entered as a covariate (to control for CO² effects) this result became non-significant.² Secondly, El-Sheikh et al. (2001) also did not find a concurrent linear association between vagal regulation and child-reported anxiety
symptoms using the RCMAS (no specific statistics were reported) among sample of 75 youth aged 8-12 years. However, the third study (Greaves-Lord et al., 2010) did find a longitudinal association between vagal regulation (obtained when children were 10-12 years of age) and child-reported anxiety symptoms using the RCADS-C that was moderated by gender in that low vagal reactivity (i.e., blunted vagal response to stress) predicted anxiety symptoms two years later in girls only (12-14 years of age).

Conversely, studies using more general measures of internalizing problems have for the most part consistently found that vagal regulation is associated with internalizing problems and, as with resting vagal tone, age and gender may possibly play a role in this relationship (e.g., Boyce et al., 2001; El-Sheikh & Whitson, 2006; Hinnant & El-Sheikh, 2009). For example, El-Sheikh and Whitson (2006) found that among 133 youth (aged 8-12 years) lower levels of vagal suppression in response to listening to a pre-recorded argument was prospectively associated with greater child-reported internalizing problems two years later but only for boys ($\beta = .34, p < .05$). Hinnant and El-Sheikh (2009) reported similar findings in that vagal augmentation predicted more parent-reported internalizing symptoms ($\beta = .22, p < .01$) two years later in another independent sample of 176 children (from third to fifth grade). However, Willhelm, Schuengel, and Koot (2009) reported no concurrent association ($r = -.09$ and $r = -.13, p > .05$) between vagal regulation and both internalizing in a sample of 99 youth aged 10-17 years. Though Willhelm et al.’ (2009) study was not longitudinal, as with El-Sheikh and colleagues (El-Sheikh and Whitson, 2006; Hinnant and El-Sheikh, 2009) the contrast in findings may be due to the age of the sample and it is possible that the strength of the association between vagal regulation and internalizing problems diminishes as youth move into adolescence.
In contrast, little research has specifically examined the link between vagal regulation and externalizing problems among youth 6 to 12 years of age and the findings are inconsistent in the literature (Boyce et al., 2001; El-Sheikh et al., 2001; Hinnant & El-Sheikh, 2009). For example, Boyce et al. (2001) found support for this association among a sample of 122 children (ages 6-7 years) in that there was a significant difference in vagal regulation between children with low and high externalizing problems (n = 66 vs. n = 17, respectively) with the high externalizing group showing less vagal suppression (χ² = 13.11, p < .05). Similarly, El-Sheikh et al. (2001) reported that greater vagal suppression was associated with less parent-reported externalizing problems (β = -79.55, p < .05) in 8-12 year-old youth (n = 75) and Hinnant & El-Sheikh (2009) found that lower resting vagal tone interacted with vagal regulation during stress (vagal augmentation) to predict more parent-reported externalizing symptoms two years after the initial physiological assessment (from third to fifth grade). However, a couple of other studies have failed to find a linear association between vagal regulation and externalizing problems in 6 to 12 year-old youth. For example, El-Sheikh and colleagues (El-Sheikh, 2001; El-Sheikh & Whitson, 2006) did not find a significant association between vagal regulation and externalizing problems (r = .11, p > .05 and r’s ranging from -.07 to .00 for parent and teacher report measures, p > .05) in two independent samples of youth ranging in age from 6 to 12 years-old.

Overall, there is some preliminary evidence to suggest that (1) resting vagal tone is negatively associated with both anxiety and overall aggressive behavior problems in youth (though age and gender may effect this association) and (2) youth with internalizing and externalizing in general youth may exhibit an inflexible and rigid regulatory response to stress (i.e., vagal augmentation; Boyce et al., 2001; El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006;
Hinnant & El-Sheikh, 2009). However, there appears to be a significant gap in the literature in that few studies have examined the more specific associations between resting vagal tone and vagal regulation and both anxiety symptoms (not just internalizing symptoms in general) and aggressive behavior problems (not just externalizing symptoms in general) across adolescence. Given this is a developmental period in which youth are theoretically more prone to experience emotional dysregulation due to structural neurobiological changes in emotional centers (Casey et al., 2008) and have been shown to have decreased vagal tone (Hollenstein et al., 2011), it seems examining resting vagal tone and vagal regulation in an adolescent sample may provide a clearer picture of biological underpinnings of youth who experience anxiety and aggressive behavior problems.

3.3 End Notes.

\(^1\) The non-significant difference of resting vagal tone found between the anxiety disordered youth and control group was non-significant after controlling for age, gender, and respiration rate. Monk et al. (2001) did report that the anxiety-disordered group had lower resting vagal tone when just controlling for age and gender as reported in the main text. More recent theory and research suggests that statistically controlling (i.e., using as a covariate) for respiration rates when examining resting vagal tone indices may inflate Type II error and eliminate “true” group differences (see Denver, Reed, & Porges, 2007).

\(^2\) Though controlling for respiration rates when examining HF-HRV may be statistically problematic (see Denver et al., 2007) during times of rest, stress tasks such as CO\(_2\) inhalation may affect respiration rates in such a way that group differences in change from baseline to CO\(^2\)
challenge may have been due to changes in respiratory rates and not “true” group differences in vagal tone change.

4. Perceived Emotional Control: Differential Relations with Anxiety and Aggressive Behavior

Several theoretical models and research findings have proposed that poor perceived control may also play a role in the development and maintenance of anxiety problems (Barlow, 2002; Chorpita & Barlow, 1998; Muris et al., 2001; Rapee, Craske, Brown, & Barlow, 1996; Weems et al., 2003; Weems & Silverman, 2006). For example, Chorpita and Barlow (1998) have suggested that anxiety problems stem from repeated-exposure to uncontrollable and/or unpredictable environments during early developmental periods. Theoretically, these early uncontrollable environments help instill poor perceived control over anxiety-provoking situations (i.e., maladaptive cognitive schemas), which are continuously paired with the activation of anxiety-related biological systems (e.g., amygdala, sympathetic nervous system). Thus, over time poor perceived control becomes a trigger for the activation of anxious symptoms (e.g., faster heart rate) and even events that are deemed controllable may lead to the experience of pathological anxiety (Barlow, 2004; Chorpita & Barlow, 1998).

A number of studies have investigated Chorpita and Barlow’s (1998) hypothesized relationship between perceived control and anxiety symptoms in adult samples using the Anxiety Control Questionnaire (ACQ; Rapee et al., 1996) which is a brief self-report measure of perceived emotional control over anxiety (e.g., Moore & Zebb, 1999). Rapee et al. (1996) more specifically tried to capture perceptions of control related to one’s belief about the degree of control over anxiety-related “internal” emotional reactions (e.g., fast heart rate) and “external”
events (e.g., fear-producing situation or object). The consistent finding across these studies is that low perceived control as measured on the ACQ is often associated with anxiety symptoms (e.g., physiological arousal) and other negative outcomes within adult community and inpatient samples (e.g., Lang & McNiel, 2006; Moore & Zebb, 1999).

A few studies have also begun to examine this unique relationship between perceived control over anxiety and anxiety symptoms in children and adolescents (ages 6-17 years; Frala, Leen-Feldner, Blumenthal, & Barreto, 2010; Muris, Mayer, den Adel, Roos, & van Wamelen, 2009; Muris, Schouten, Meesters, & Gijsbers, 2003) using a developmentally modified version of the ACQ (i.e., the Anxiety Control Questionnaire for Children [ACQ-C]; Weems et al., 2003). Similar to adult populations, ACQ-C scores (higher scores indicate more control) have been shown to be inversely related to both anxiety problems and anxiety-disordered status in both clinical and nonclinical youth (e.g., Weems et al., 2003; Muris et al., 2009). For example, Weems et al. (2003) found a lack of perceived control over anxiety was associated with increased self-reported anxiety symptoms and predicted anxiety disorder diagnostic status in a sample of 9-17 year-olds. Similar findings have been reported over time with poor perceived control (e.g., social interaction competency beliefs) being concurrently and prospectively (4-week follow-up) related to youth’s (age 10-14 years) self-reported anxiety problems (Muris et al, 2003).

In comparison to anxiety problems among youth, Baumeister, Smart, and Boden (1996) have theorized that externalizing problems, such as aggressive behavior, may be more closely associated with overconfidence or a heightened self-esteem (e.g., higher perceived control). Specifically, they propose that individuals who are more aggressive tend to view themselves
much more favorably (i.e., high self-appraisal) than others and when confronted with contradictory information would most likely retaliate in an aggressive and violent manner. In addition, Baumeister et al. (1996) suggest these individuals may seek out opportunities (i.e., less avoidant) to prove they are capable of performing such unattainable feats even though in reality they most likely would fail to live up to their own expectations. Thus, an aggressive youth may take courses beyond their intellectual capability in school and become aggressive when earning a failing grade on their first test.

Though relatively little research has directly examined the link between perceived control and aggression, there is some initial indirect evidence for an inverse relationship between control and aggressive behavior (e.g., Boxtel et al., 2004; Briggs, Tovar, & Corcoran, 1996; Heavey, Adelman, Nelson, & Smith, 1989; Scott & Weems, 2010). For example, Heavey et al. (1989) reported that children who had high perceived environmental control (e.g., decision making) and exhibited anger problems at school (i.e., possible lack of actual control) were rated by their parents as engaging in more disruptive behavior than youth with low perceived control. Similarly, Boxtel et al. (2004) found that in a sample of 179 Dutch school children (3rd and 4th graders) perceived social competence was positively correlated (r = .58) with a reputation for fighting among peer-rejected children. These findings are consistent with Baumeister et al.’s (1996) theorizing in that rejected children may be over confident (i.e., high self-esteem) in their abilities and when combined with contradictory evidence to these perceptions (i.e., unfavorable peer evaluations) aggressive behavior may arise (Boxtel et al., 2004).

The results of the above studies regarding high perceived control and aggressive behavior should be alarming when taking into consideration that one of the primary goals of CBT is to
help youth perceive more control over themselves and the environment (Kendall, 1994). Though this may be helpful for those youth who have already obtained the skills necessary for adequate emotion regulation (i.e., high actual/low perceived control profile), it may be detrimental for those youth who do not know how to regulate specific emotional responses (i.e., low actual/low perceived control profile). In fact, Suveg, Sood, Comer, and Kendall (2009) recently found that self-reported regulation of other emotions besides anxiety (e.g., anger) was still impaired following CBT. Thus, differentiating between actual and perceived control in youth may have both a theoretical and practical importance in helping better understand and predict the type of emotional and behavioral problems youth would experience during one’s lifetime.

5 Statement of the Problem

The implication of failing to control one’s emotions during emotional and stressful times has been a main concern of both clinicians and researchers for well over 120 years (e.g., Freud, 1923/1960; James, 1884; Gross, 1998a). Though previous theories (Beauchaine, 2001, Porges et al., 1994; Porges, 2007) suggest that an indices of one’s actual inability of emotional regulation (i.e., resting vagal tone or vagal regulation in response to stress) is non-specifically related to internalizing and externalizing problems in youth, a number of studies have challenged the specificity of these associations (particularly in terms of anxiety and aggression) and even more so the exact nature of these relationships (El-Sheikh et al., 2001; Gordis et al., 2010; Greaves-Lord et al., 2007; Mezzacappa et al., 1996; Scarpa, et al., 2010). In addition, most research to date have mainly examined the link between vagal tone indices (resting vagal tone and vagal regulation) and both anxiety and aggressive behavior problems in children and young adolescents (6 to 13 years of age). The lack of attention to older adolescents has left a major gap
in the literature that needs to be addressed empirically considering past theory and research suggests it is a developmental period of neurobiological change in emotional control centers and a lower vagal tone has been shown to be associated with emotional dysregulation across adolescence (Casey et al., 2008; Hollenstein et al., 2011; Vasilev et al., 2009).

One possible reason for these inconsistencies in the literature regarding the specificity of resting vagal tone and vagal regulation is that another factor may be partly responsible for the anxiety and aggression exhibited in youth. More specifically, a few studies have now suggested that not all individuals with internalizing problems lack the actual ability to control emotional responses, even though they report poor emotional reactivity to a stressful task (e.g., Mauss et al., 2004). Conversely, research has shown that heightened perceptions of control are related to aggressive behavior problems when control does not seem possible (Boxtel et al., 2004). Thus, examination of cognitive factors related to one’s perceptions of emotional control in conjunction with one’s actual ability for emotional control may help better predict youth’s specific emotional and behavioral problems.

The Weems and Silverman (2006) model of control suggests that empirical studies should examine both actual and perceived control and Scott and Weems (2010) adaptation suggests that patterns of actual and perceived control (i.e., control profiles) may be differentially related to internalizing versus externalizing problems. As noted, preliminary evidence for the predictions exists, but this evidence is limited by the measure of actual emotional control (Scott & Weems, 2010). Research suggests that recording physiological measures (e.g., heart rate) during times of rest and stress, as well as when voluntarily changing one’s heart rate would be a theoretically improved way to measure actual emotional control. Therefore, two physiological
markers of emotion regulation (i.e., resting vagal tone and voluntary control of heart rate) were used in the present study to objectively measure emotion-related physiological regulation and estimate one’s actual ability for actual emotional control (Porges, 2007; Thayer & Lane, 2000).

In summary, there were three broad goals for the study. The first goal was to test the linear association between both resting vagal tone and vagal regulation (resting vagal tone to cognitive challenge task vagal tone) and both anxiety and aggressive behavior problems in a community sample of youth ages 11-17. The second goal was to establish whether 1) there were individual differences in youths’ ability to voluntarily control their heart rate, 2) youth on average were actually able to increase and/or decrease their heart rate, and 3) this ability was related to perceptions of control and/or emotional and behavioral problems, such as anxiety and aggression. The final goal was to expand upon Scott and Weems (2010) recent work by testing the Weems and Silverman (2006) model of control using these two objective measures of actual emotional control.

### 5.1 Hypotheses

1) Youths’ actual physiological control (Video and Resting Baseline HF-HRV) non-specifically be associated with anxiety and aggressive behavior problems and perception of control (ACQ-C total scores) would be associated with anxiety problems. More specifically:

   a. Youths’ HF-HRV (Video and Resting Baseline) would be *negatively* associated with RCMAS Total Anxiety, RCADS-C Total Anxiety, RCADS-P Total Anxiety, PSC-C Total Aggression, and PSC-P Total Aggression.
b. Youths’ anxiety symptoms (i.e., RCADS-C and RCADS-P Total Anxiety) would predict a blunted response (little to no change in HF-HRV) or vagal augmentation (increase in HF-HRV) as opposed to vagal suppression (decrease in HF-HRV) from Video and Resting Baseline to the Mental Arithmetic Task.

c. Youths’ ACQ-C total scores would be negatively associated with RCMAS Total Anxiety, RCADS-C Total Anxiety, and RCADS-P Total Anxiety and positively associated with respective actual control measures (i.e., Video and Resting HF-HRV and HR Control Change Score).

2) There would be individual differences in one’s ability to change one’s heart rate in a respective direction (i.e., increase or decrease), but on average youth would be able to increase and decrease their heart rate during the HR Control Task. Theoretically, more anxiety and poorer perceptions of control should be associated with less ability to control heart rate.

3) Assessing adolescents actual control (HF-HRV [Resting Baseline] and HR Control Change Score [Overall]) and perceived control (ACQ-C Total Score) would result in the identification of the four control profiles outlined in the Weems and Silverman (2006) model and would therefore include the following groups: High actual/high perceived control profile (upper right quadrant in Figure 1), High actual/low perceived control profile (upper left quadrant in Figure 1), High perceived/low actual control profile (lower right quadrant in Figure 1), and Low actual/low perceived profile (lower left quadrant in Figure 1).
4) The four control profiles would significantly differ in levels of reported anxiety symptoms and aggressive behavior problems. More specifically:

a. Youth with a high perceived/high actual control profile would have the fewest reported anxiety symptoms and aggressive behavior problems as compared to the other three control profiles.

b. Youth with a high actual/low perceived control profile would have higher reported anxiety symptoms than those youth with a high perceived/low actual control profile and have more reported anxiety symptoms than aggressive behavior.

c. Youth with a low actual/high perceived control profile would have more reported aggressive behavior problems than those youth with a low perceived/high actual control profile and have more reported aggressive behavior than anxiety symptoms.

d. Youth with a low perceived/low control profile would have both high reported anxiety symptoms and aggressive behavior problems than those youth with both a high perceived/high actual control profile.

e. There would be no differences in reported anxiety symptoms and aggressive behavior problems for those youth with either a high actual/high perceived or a low actual/low perceived control profile.
6 Method

6.1 Participants

Ninety six caregiver-youth dyads (80 caregivers and 96 youth) from New Orleans and the surrounding area participated in this study from August 2011 to March 2013. Recruitment procedures entailed (1) distributing flyers (see attached) in local intermediate and secondary schools, (2) recruiting undergraduate students enrolled in psychology courses at the University of New Orleans to refer caregiver-adolescent dyads, and (3) posting an advertisement (same content as attached flyers) via the internet (i.e., Craigslist and Facebook). The flyers and advertisements specifically asked for help from caregiver-youth dyads in conducting a project on the emotional reactivity of both caregivers and teenagers. Families were notified that both the caregiver and youth would be compensated for approximately two and half hours of their time (i.e., total of $50: parent received $30 for each adolescent and each adolescent received $20). An examination of how families were recruited for this study showed that 50.0% ($n = 48$) were recruited via Craigslist, 15.6% ($n = 15$) heard about the project from a family member or friend, 7.3% ($n = 7$) heard about it in their psychology course, 4.2% ($n = 4$) responded to the flyer, and 7.3% ($n = 7$) of families were missing referral information.

No specific exclusion criteria were used in recruitment of families given the initial goal of obtaining community sample that represented the general population. However, based on a review of the current literature, exclusion criteria for analyses were applied for consistency with past research (e.g., Blom et al. 2010). First, two parents did report that their children had history of a pervasive developmental disorder (one with Aspergers and one with Autism) and thus were excluded from further analysis. Second, of the 94 remaining youth, caregivers also reported that
27% ($n = 25$) of youth were currently taking stimulants (e.g., Vyvanse, Adderall, Concerta; $n = 8$), serotonin reuptake inhibitors (SSRIs; e.g., Celexa; $n = 1$), cold/allergy/asthma-related medicine (e.g., singular, albuterol pump $n = 5$), birth control ($n = 3$), antibiotics ($n = 2$), anti-seizure ($n = 1$), anti-psychotic ($n = 1$), and natural supplement (i.e., Biotin; $n = 1$). In addition, two caregivers reported that their child was taking multiple types medications (e.g., stimulant, SSRI, allergy medicine) and another caregiver did not report the specific medication(s) being used.

Given past research suggests that use of psychotropic drugs such as antipsychotics, anti-seizure, stimulants, and SSRIs may affect HF-HRV indices (e.g., Blom et al. 2010), youth currently taking these medications and the one child for which the parent did not report the specific medication currently used were excluded from further analyses ($n = 14$). The final sample thus consisted of 80 socioeconomic and ethnically diverse group of youth (68 child-caregiver dyads) aged 11-17 years ($\text{Mean}_{\text{age}} = 13.88$, $\text{SD}_{\text{age}} = 1.95$) with fifty-one percent being female. Caregivers reported youth’s ethnicities as 37.5% African American ($n = 30$), 33.8% Euro-American ($n = 27$), 23.8% other/mixed ethnic background ($n = 19$) and 5% Hispanic ($n = 4$). The median family income ($n_{\text{family}} = 67$) was between $20,000 and $49,999 a year.\(^1\)

6.2 Measures

6.2.1 Demographic Information

Caregivers were asked to provide personal information regarding their children such as age, gender, ethnicity, and family income. In addition, they were asked to provide information regarding any known medications their child was currently be administered (i.e., what type of medication and last time of administration).
6.2.2 Actual Control. Actual control was measured in four different ways for this study (three physiological indicators and one parent-reported measure). The three physiological indices were computed from a protocol that is specifically described under the Procedures section. The first physiological index was the *Mean Heart Rate Control Change Score (HRCS; Voluntary Control of Heart Rate)* which represented youths’ overall ability (magnitude) to voluntarily change (or control) their mean heart rate in a respective direction across a number of trials (i.e., increase heart rate when asked to increase or decrease heart rate when asked to decrease).

The second physiological index was the *High Frequency – Heart Rate Variability (HF-HRV; Involuntary Parasympathetic-mediated Regulation of Heart Rate)* which drawing from numerous past studies (Appelhans & Luecken, 2006; Berntson et al., 1997; Butler et al., 2006; Vasilev et al., 2009; Wetzel et al., 2006) was calculated using “normal” IBIs extracted from the EKG signal (more specific details on this process is described in the Procedures section). The HF-HRV for the Resting Baseline (five-minute segment) was used as the main index of actual emotional control to test the adapted Weems and Silverman (2006) model.

The third physiological index was *Vagal Regulation* or change in HF-HRV from the Video or Resting Baseline conditions to the Mental Arithmetic Task. The HF-HRV from the first three minutes of the Video Baseline, Resting Baseline, and Mental Arithmetic Task were used in multilevel modeling to derive a repeated measures index of Vagal Regulation in youth (this method of analysis is described in the Results section). This method allowed for an alternative measure of actual emotional control and to test specific hypotheses related to vagal regulation and youths’ emotional and behavioral problems.

Achenbach & Rescorla, 2001) as a parent-reported index of actual control it was also used in the
present study. The competence section of the CBCL specifically asks parents about their youths’
(ages 6-18) competence in day-to-day activities, academic, and social situations with higher
scores representing more overall competence. The CBCL is a widely used measure showing
good reliability and validity estimates (Achenbach, 1991; Achenbach & Rescorla, 2001) and
more specifically the competence subscale has been shown to be significantly correlated with
other measures of youths’ social skills (Frankel & Myatt, 1994). The total CBCL competence
raw score across all three domains (activities, academic, and social) was also used in this study.

6.2.3 Perceived Control. Perceived control was also measured in two different ways. The first
measure was a child-report of perceived control over anxiety and was measured using the
Anxiety Control Questionnaire for Children (ACQ-C; Weems et al., 2003), which is a 30-item
developmentally modified version of the ACQ (Rapee et al., 1996). The ACQ-C was designed to
assess youths’ control beliefs over anxiety-related “external” threats (e.g., fear-producing
objects, events, and situations) and/or “internal” emotional or bodily reactions (e.g., flushed
face). The items on the ACQ-C were changed to be developmentally appropriate and include
such questions as “I can usually stop my anxiety from being seen by other people” and “I can
usually stop thinking about things that make me nervous or afraid if I try.” Each item is rated by
the participant on a 6-point Likert-type scale consisting of: 0 (Strongly Disagree), 1 (Moderately
Disagree), 2 (Slightly Disagree), 3 (Slightly Agree), 4 (Moderately Agree) or 5 (Strongly Agree).
The ACQ-C has shown to have both good reliability and validity with internal consistency
estimates ranging .92 to .94 in two independent samples and has been shown to have convergent
validity with the NSLOC (r = -.22) and RCMAS (r = -.47; Weems et al., 2003). The internal
consistency estimate of the ACQ-C total score in the present study was excellent (Cronbach’s α
Higher scores represent more perceived control over anxiety-related events and the ACQ-C total score was used as the primary measure of perceived control in this study.

The second measure of perceived control was the child-reported *Post HR Control Evaluation (PHRC; see Appendix A)* which was specifically designed for this study in an effort to assess youths’ HR control beliefs of increasing, decreasing, and overall increasing and decreasing their heart rate during the HR Control Task (protocol described in the Procedures section). Given the two measures of actual control (HRCS and HF-HRV) are physiological in nature, it seemed quite reasonable that perceptions of control over youths’ physiological states may correspond better with the physiological measures of actual control. The PHRC is a visual analogue scale (VAS) that is commonly and widely used throughout pain research (Wewers & Lowe, 1990) and specifically asked youth to make a vertical mark on a 100 mm line with specific anchors at both ends of the line. The PHRC included three separate questions that asked youth how well he or she thought he or she 1) increased, 2) decreased, or 3) overall increased and decreased their heart rate with “very poorly” (0 mm) and “very well” (100 mm) being the two anchors. Higher scores indicated more perceived control over controlling their heart rate in the respective direction (increasing or decreasing) and as a whole (overall increasing and decreasing). The HR Control Beliefs (Overall) score was used as the main index of perceived control to test the study’s main hypotheses regarding the Scott and Weems (2010) adaptation of the Weems and Silverman (2006) model of control.

6.2.4 Internalizing and Anxiety Symptoms. Child-reported internalizing and anxiety symptoms were measured using the *Revised Child Anxiety and Depression Scale (RCADS-C; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000)*. The RCADS-C is a modified version of the SCAS
(Spence, 1997) developed specifically to assess anxiety and depression symptoms in children and adolescents (age 8-18 years) as based on DSM-IV (APA, 1994) criteria. The RCADS is comprised of 47 items and ask youth to rate how often each statement applies to them using a 4-point Likert type scale: 0 (Never), 1 (Sometimes), 2 (Often), and 3 (Always). In an effort to replicate and expand upon Scott & Weems (2010) past research, the present study utilized both the RCADS-C total internalizing (anxiety and depression) and anxiety subscale scores. Scott and Weems (2010) recently reported excellent internal consistency of the RCADS-C total internalizing (anxiety/depression) score in a sample of youth (age 6-17 years; $\alpha = .94$). In addition, Weems and Costa (2005) demonstrated convergent validity of the RCADS-C for adolescents (age 12-17 years) with the total anxiety score correlating significantly with the RCMAS total score ($r = .81$). The internal consistency estimates for the total anxiety and total internalizing score were excellent in the present study (Cronbach’s $\alpha = .93$ and $.94$, respectively).

Additionally, the Revised Child Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978) was also given because it is a more generalized measure of anxious emotion and has been shown to have differential relations with HF-HRV as compared to the RCADS-C. The RCMAS is a commonly used 37-item measure that assesses youth’s general level of manifest anxiety (28 items) and includes a 9-item lie scale. The measure was constructed using a “Yes”/”No” format with youth indicating whether they believe each item is true or not true of oneself. The RCMAS has been utilized in a number of studies as a general measure of anxiety and has been found to have moderate to good reliability and validity estimates (e.g., Reynolds & Richmond, 2000; Varela & Biggs, 2006). For example, both Reynolds and Richmond (2000) reported Cronbach’s alphas ranging .78 to .85 in a European American and African American sample of 10-13 year-
olds ($n = 4,952$) and Varela and Biggs (2006) found Cronbach’s alphas similarly ranging from .75 to .89 among a sample of European Americans, Mexican Americans, and Mexicans ages 10-14 years. In addition, Muris, Merckelbach, Ollendick, King, and Bogie (2002) reported moderate to excellent convergent validity between the RCMAS and other established measures of anxiety ($r = .88$, $p < .05$; State Trait Anxiety Inventory for Children [STAIC]; Spielberger, 1973) and fear ($r = .63$, $p < .05$; Fear Survey Schedule for Children-Revised [FSSCR]; Ollendick, 1983) in a sample of 521 adolescents (12-18 years of age). The internal consistency for this study was consistent with past research (Reynolds & Richmond, 2000; Varela & Biggs, 2006) and was good with a Cronbach’s alpha = .85. Higher total scores indicate greater manifested anxiety and the RCMAS total anxiety score (minus the lie scale items) was utilized as a more general measure of youths’ anxiety in this study.

Parent-reported anxiety symptoms were measured using the Revised Child Anxiety and Depression Scale – Parent Version (RCADS-P; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000; Weems, Costa, Watts, Taylor, & Cannon, 2007), which was designed to gain the parents’ perspective on the frequency of DSM-IV criteria (APA, 1994) symptoms related to anxiety disorders (excluding Posttraumatic Stress Disorder and Specific Phobias) and depression as exhibited by their children. The RCADS-P is practically identical to the child version (except “I” was changed to “my child”) and asks caregivers to rate how often each anxiety and depression symptom is true of the child on a rating scale consisting of: 1 (Never), 2 (Sometimes), 3 (Often), and 4 (Always). The RCADS-P has been shown to be a reliable and valid measure of children’s anxiety and depressive symptoms with excellent internal consistency for total anxiety and total internalizing score (Cronbach’s $\alpha = .91$ and .93, respectively) being recently reported (Ebesutani
et al., 2011) and modest to good convergent validity with the RCADS – Child Version (Weems & Costa, 2005). Higher scores represent greater frequency of anxiety and depression symptoms as observed by the caregiver and the RCADS-P Total Anxiety score was used the present study. Internal consistency for the RCADS-P Total Anxiety in this sample was excellent (Cronbach’s $\alpha = .90$) and consistent with past research (e.g., Ebessutani et al., 2011).

**6.2.5 Aggressive Behavior.** Child-reported aggression was evaluated with the 40-item *Peer Conflict Scale – Youth Self-Report (PCS-C; Marsee & Frick, 2007)* which assesses the four subtypes of aggression (i.e., reactive relational, reactive overt, proactive relational and proactive overt) in children and adolescents. Youth were asked to rate how true each statement describes him or herself on a 4-point rating scale that consists of 0 (Not at all true), 1 (Somewhat true), 2 (Very true), and 3 (Definitely true). Some example items presented to youth on the PCS-C were: “When I am teased, I will hurt someone or break something” (reactive overt); “When others make me mad, I write mean notes about them and pass the notes around” (reactive relational); “I threaten others to get what I want” (proactive overt); and “To get what I want, I try to steal other’s friends from them” (proactive relational). Higher scores indicate more self-reported aggression and for this study the PCS-C total aggression score was used as a global measure of the child’s aggressive behavior and the PCS-C aggressive subtype scores (reactive relational, reactive overt, proactive relational and proactive overt) were used to provide a more specific index of aggression.

Internal consistency estimates for the four subscales (coefficient alphas = .76 to .90) suggest the PCS is a reliable measure for non-clinical and clinical samples (Marsee & Frick, 2007; Marsee et al., 2011). In the more recent study of 882 youth (high school students, detained
youth, residential youth), the PCS showed good factor structure validity and the four subscales were uniquely associated with other measures of delinquency and externalizing problems (Marsee et al., 2011). The total aggression score had an excellent reliability estimate in this study ($\alpha = .92$) and the acceptable to good estimates for the four subtypes were similar to past reports [reactive relational ($\alpha = .81$), reactive overt ($\alpha = .81$), proactive relational ($\alpha = .84$) and proactive overt ($\alpha = .73$)].

Parent-reported aggression was assessed using the 40-item *Peer Conflict Scale – Parent Version* (PCS-P; Marsee & Frick, 2007), which is intended to assess the parents’ view of their child’s aggressive behavior as related to the four subtypes of aggression (i.e., reactive relational, reactive overt, proactive relational and proactive overt). The PCS-P wording is identical to the child version (except “I” was changed to “My child”) and parents were asked to rate how true each statement described their child on a 4-point Likert-type scale that includes: 0 (Not at all true), 1 (Somewhat true), 2 (Very true), and 3 (Definitely true). The internal consistency was excellent for the PCS-P total aggression score (Cronbach’s $\alpha = .94$) and acceptable to good for each of the PCS-P four subtypes of aggression in this study [reactive relational ($\alpha = .88$), reactive overt ($\alpha = .88$), proactive relational ($\alpha = .78$) and proactive overt ($\alpha = .81$)]. As with the child version of the PCS, higher scores on the PCS-P represent more parent-reported youth aggression. In addition, the PCS-P total aggression score was used to provide an overall index of youth’s aggressive behavior and the PCS-P aggressive subtype scores (reactive relational, reactive overt, proactive relational and proactive overt) were used to provide a more specific index of youth’s aggressive behavior in this study.

Alternatively, the *Child Behavior Checklist* (CBCL; Achenbach, 1991; Achenbach & Rescorla, 2001) was also given to provide an alternative measure of parent-reported aggressive
behavior and to allow a replication of previous findings (Scott & Weems, 2010). The CBCL is an 113-item measure used to assess a child or adolescent’s (ages 6-18) behavioral and social problems as reported by the caregiver. The CBCL provides scores for internalizing and externalizing symptoms as well as youth’s total competence across several domains (e.g., social competence). The caregiver is asked to rate the frequency of their child’s behaviors on a rating scale consisting of 0 (Not at all), 1 (Sometimes), and 2 (Always) with higher scores representing more problems or competence. The CBCL is a widely used measure showing good reliability and validity estimates with coefficient alphas ranging from .89 to .96 for the internalizing, externalizing, and total scales (Achenbach, 1991; Achenbach & Rescorla, 2001). Total scores for the CBCL aggressive behavior subscale were used as an alternative measure of aggressive behavior and higher scores indicated more parent-reported aggressive behavior. The internal consistency for the aggressive behavior subscale was good to excellent for this study ($\alpha = .89$).

6.2.6 Other Physiological Measures. Mean heart rate, skin conductance, and external body temperature from the Video Baseline, Resting Baseline, and Mental Arithmetic Task (procedural protocol for data collection and extraction for each task is described below in the Procedures section) were collected as alternative measures of other regulatory systems (e.g., sympathetic nervous system). These measures were specifically used in this study as a manipulation check of youths’ physiological stress reactivity to the Mental Arithmetic Task.

6.3 Procedure

Caregiver-youth dyads interested in participating in the study contacted the research lab via the phone number printed on a flyer and/or advertisement. A member of the research staff provided the caregiver with an overview of the project and gathered further contact (e.g., name, phone number, etc.) and screening information. For those caregiver-youth dyads who were still interested in participating in the study, the research staff scheduled an appointment for them to
come into the lab at a time of their convenience. Each dyad was instructed (if all possible) not to eat, drink (water was acceptable), or smoke cigarettes one hour before coming to the research lab.

Upon arrival, a trained graduate assistant (GA) explained to both the parent and child the purpose of the study and had them sign written informed consent and youth assent forms, respectively. In addition, the GA gave both the caregiver and child an opportunity to ask further questions and answered such questions before proceeding with the study. Once informed consent and assent was obtained, both the GA and a trained research assistant (RA) escorted the dyad to the control room and the GA explained that he/she would be sitting in this room during the physiological assessment period and providing instructions through a microphone. This was intended to ease the novelty of the situation and attempt to lessen any apprehension the child or caregiver might have about the location of the GA and RA. Then the RA escorted the caregiver and child to the physiological assessment room and placed the physiological sensors on the child (the caregiver was present for this procedure).

Physiological measurements (i.e., heart rate, respiration, skin conductance, and temperature) were collected and stored on a Dell Studio XPS, Intel ® Core™, 2.67GHz, 3GB RAM using Biograph Infiniti software and the accompanying hardware competent, ProComp Infiniti encoder (Meyers, 2010). The Biograph Infiniti software was run using a Microsoft Windows 7, 32-bit operating system and output was automatically stored within a designated file (using only the child’s unique id number) on the computer. Sensors connected to both the ProComp Infiniti encoder were attached to youth via specially designed cables and fiber optic wiring, respectively. First, the electrocardiogram (EKG) sensors (3) were attached using UniGel
electrodes (pre-gelled) and were placed on the right (1) and left (1) abdomens (below the rib cage) and at the top of the sternum (1). Secondly, the respiration band will be strapped around the chest (outside the shirt) when the participant fully expands his/her abdomen upon request from the experimenter. Lastly, the blood volume pulse (BVP) sensor was placed on the participant’s middle finger, the galvanic skin response (GSR) sensors on the index and ring fingers, and the temperature sensor on the baby finger of child’s non-dominant hand. The RA then instructed the child to sit in a comfortable chair and face the computer monitor, while the caregiver was taken to another quiet room to complete the parent-report measures.

The RA next shut the door to the physiological room and entered the control room (adjacent to the physiological room) where the GA began running a scripted physiological protocol using the Biograph Infiniti software. The GA checked the physiological signals for poor connections and other possible artifacts before proceeding with the physiological assessment. If a problem was discovered before (or during) the assessment, the RA was asked to adjust the sensor(s) per the GA’s instructions. The physiological assessment protocol lasted approximately 60 minutes and was video and audio recorded (and monitored live) via a webcam attached to the ceiling behind the child.

The first phase of the physiological assessment (i.e., Video Baseline) consisted of the experimenter asking the child to relax and watch a short film clip (i.e., Coral Sea Dreaming Film Clip). This five-minute film clip displayed coral reef and undersea fish and was extrapolated from the DVD version of Coral Sea Dreaming. The purpose of this relaxation-based film clip was to help youth acclimate to the testing environment, while keeping their attention focused on the monitor and recording Video Baseline physiological data. This method of producing more
relaxed and stable physiological states has shown good reliability and validity (as compared to resting baseline) in young adults (Piferi, Kline, Younger, & Lawler, 2000), but to our knowledge this was the first time it has been utilized with youth. If problems with the signal were detected, the experimenter asked a research assistant (RA) to readjust sensor placement on the child immediately following this phase.

The next phase consisted of the experimenter asking the child to relax and breathe normally for five minutes (i.e., Resting Baseline) while physiological data was recorded. The third phase (i.e., HR Control Task; drawing from past studies such as Schneider et al., 1978; Sirota et al., 1976, and Scott & Weems, 2012) consisted of two blocks of one-minute trials (3 trials per block) in which the experimenter instructed the participant to increase (speed up) his heart rate when the text “INCREASE” (Block 1) appeared on the screen or decrease (slow down) his heart rate when the text “DECREASE” (Block 2) appeared screen. In addition, the child was told that short passages (presented for 30 seconds) would appear on the screen (passages were at a 4th grade reading level and ranged from 100 – 110 words) and he was to read each passage silently to himself (the child was also instructed to start over if he finished reading the passage). A two-minute resting baseline (no reading) was obtained between Blocks 1 and 2 in order to reestablish a baseline heart rate before the child proceeded to Block 2 trials. The overall purpose of this design was to give the participant an opportunity to return to baseline levels of physiological response and reduce carry-over effects from the prior increase trials. Once the youth had completed the final 30-second baseline, the RA walked into the physiological room and handed the youth the Post HR Control Evaluation form (see Appendix A). The experimenter then provided instructions for the youth on how to compete the Post HR Control Evaluation
form, while the RA closed the door and stood out in the hallway. This part of the study lasted for approximately 3-5 minutes and upon completion the RA was asked by the youth to retrieve the Post HR Control Evaluation form.

The fourth phase the child participated in a modified non-vocal and developmentally appropriate version of the serial 7’s task (i.e., Mental Arithmetic Task; Stroud et al., 2009). This phase consisted of the child being asked to subtract a specific number (13 if child’s age was 11-14 years; 17 if child’s age was 15-17 years) from 500 and continue to subtract that number from each subsequent answer. The task lasted exactly three minutes and five seconds (only the first three minutes of data were extracted and analyzed) and children were asked to type their answers into a key-pad placed directly in front of them on the desk. The purpose of this nonverbal procedure was to minimize fluctuations in EKG and respiration signals due to speaking that may influence the HF-HRV index. A manipulation check of stress reactivity using paired sample t-tests for the physiological indicators of stress indicated a significant increase in mean heart rate (beats per minute) from the Video Baseline (3 minute; $M_{\text{heart rate}} = 80.41, SD = 11.32$) and Resting Baseline (3 minute; $M_{\text{heart rate}} = 80.85, SD = 11.01$) to the Mental Arithmetic Task [$M_{\text{heart rate}} = 83.09, SD = 11.21; t(79) = 3.74$, $p < .001$ and $t(79) = 3.40$, $p = .001$, respectively]. Similarly, there was a significant increase from the Video Baseline (3 minute; $M_{\text{skin conductance}} = 1.85, SD = 1.65$) and Resting Baseline (3 minute; $M_{\text{skin conductance}} = 1.77, SD = 1.60$) to the Mental Arithmetic Task [$M_{\text{skin conductance}} = 3.55, SD = 2.30; t(78) = 14.30$, $p < .001$, $t(78) = 14.28$, $p < .001$, respectively]. However, the only significant difference in temperature was from the Resting Baseline (3 minute; $M_{\text{temperature}} = 89.96, SD = 5.56$) to the Mental Arithmetic Task [$M_{\text{temperature}} = 87.08, SD = 4.54; t(79) = -4.78$, $p < .001$] and youths’ temperature from Video Baseline (3
minute; $M_{temperature}= 87.69, SD = 5.93$) to Mental Arithmetic Task did not significantly change $[t(79) = .90, p = .37]$.

In the final and fifth phase, youth watched 10 video vignettes (10-20 seconds in length) depicting ambiguous situations youth may have with their peers in between a 10 second resting baseline before the vignette began and a 30 seconds recovery period after the vignette ended. The youth then were asked to orally answer six questions after each video vignette that assessed their level of hostile attribution in response to each video vignette. The physiological measurements obtained during this final phase of the physiological assessment were not used in this study.

After the physiological protocol was completed, the RA led the child to a quiet room and administered the youth questionnaire packet. Upon completion of both the physiological assessment and questionnaires the caregiver-youth dyad were provided with a debriefing form and asked if they had questions or concerns. The GA then thanked for their time and provided $50 for their participation.

**6.3.1 Physiological Data Extraction.** EKG data from the Video Baseline, Resting Baseline, HR Control Task and Mental Arithmetic Task were analyzed using the Kubios HRV 2.1 software (Tarvainen, Niskanen, Lipponen, Ranta-aho, & Karjalainen, 2009). The data were first manually and visually inspected by the first author and trained RAs for artifacts (e.g., misidentified, missed, or extra heart beats) in the time series. Next, an automatic artifact detection algorithm (medium correction setting) was used to detect interbeat-intervals that were .25 seconds above or below the mean inter-beat intervals for the entire time series. If the automatic detection results were consistent with manual inspection findings, than automatic correction of inter-beat intervals artifacts was applied to the data (manual correction was implemented for discrepancies). This
method of artifact correction allowed for a standardized procedure of normalizing the inter-beat intervals data (i.e., without under- or over-correction) and individuals with 10% or more corrupted data were excluded from further analyses. The artifact-free inter-beat intervals were then resampled at .025 Hz and the entire time series was detrended using a second-order polynomial (Porges et al., 1994) in an effort to remove non-stationary data. The inter-beat intervals were then finally subjected to a power spectral analysis using a Fast Fourier Transform algorithm for the two baseline (Video and Resting Baseline) and Mental Arithmetic Task conditions, which produced absolute power (or variance) distributions for low frequency HRV (.04 to .15 Hz) and high frequency HRV (.15 to .40 Hz) consistent with reported standards for spectral analysis (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Respiration frequency was estimated using the Kubios 2.1 software and was based on an EKG derived respiration algorithm that specifically derives the beat-to-beat R-wave peak amplitudes and the Fast Fourier Transform spectra for this R-peak amplitude time series. The respiratory frequency is obtained as the frequency corresponding to the most significant peak in the Fast Fourier Transform spectra. This specific method of estimating respiration frequency is now common, with evidence supporting its reliability and validity for providing respiration frequencies (Mazzanti, Lamaberti, & de Bie; 2003; Zhao, Reisman, & Findley; 1994).  

Examination of the EKG derived respiration frequencies of this sample indicated that youths’ respiration frequencies fell within the standard respiratory or high frequency band (i.e., RSA; .15 to .40 Hz; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996) and thus no adjustment of bandwidth was needed.
Mean heart rate for the each baseline and increase/decrease trial was also calculated and refers to the mean number of “normal” inter-beat intervals per minute (i.e., beats per minute) derived from the EKG signal during each epoch (e.g., 1-minute baseline or increase/decrease trial). Mean heart rate helped determine the amount and direction of mean heart rate change for each youth from each increase and decrease baseline to each increase and decrease trial (i.e., [mean heart rate for increase trial – mean heart rate at increase baseline] + [mean heart rate at decrease baseline – mean heart rate for decrease trial] for all trials). Summation of the difference scores for each epoch yielded the HR Control Change Score (Overall). In addition, a difference score was also calculated separately for the increase and decrease trial epochs and yielded the HR Control Change Score (Increase) and HR Control (Decrease).

6.4 End Notes.

1All children whose siblings participated in the study were included in the analyses given that no family or parent level variables were specifically used in this study. That is, it is reasonable that both child reports about their own behavior and parent reports about two or more children in the same family could be considered independent observations. In addition, the small number of families (or clusters) with two or more children was relatively small (n = 12) as compared to the families with one child (n = 56).

7. Results

7.1 Preliminary Examination of Study Variables

Preliminary examination of the data indicated that all missing data was missing at random and mean imputation was implemented for scale scores that had no more 10% of missing data. In addition, one youth was unable to complete the HR Control Task and thus was missing
physiological and self-report data related to HR Control (i.e., all HR Control Change and HR Control Belief scores). Further examination of the distributions and scatter plots between the main study variables identified four univariate outliers for the RCADS-C \((n = 1)\) PCS-C Total Aggression \((n = 1)\), PCS-P Total Aggression \((n = 1)\), HF-HRV (Video Baseline [5 minute segments]; \(n = 2)\), HF-HRV (Video Baseline [3 minute segment]; \(n = 1)\), HF-HRV (Resting Baseline [3 minute and 5 minute segments]; \(n = 1)\), Skin Conductance (Video and Resting Baseline [3 minute]; \(n = 1)\). Pairwise deletion of cases was used to handle univariate outliers given that different variables were used in each analysis and each case had usable data for other analyses. Evaluation of multivariate outliers using multiple regression techniques (i.e., calculating Mahalanobis distances for each case based the study variables used to test the main hypotheses) found no evidence of multivariate outliers \(\chi^2(10) = 29.59, p > .001\).

The means, standard deviations, range, and skewness of the study variables after exclusion of outliers for the child-reported measures, parent-reported measures, and child physiological measures are summarized in Tables 1, 2, and 3, respectively.

Table 1. Means, Standard Deviations, Ranges, and Skew for Child Report Measures

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>M</th>
<th>SD</th>
<th>Min - Max</th>
<th>Skew</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACQ-C Total Score</td>
<td>80</td>
<td>73.63</td>
<td>19.49</td>
<td>21 – 120</td>
<td>-.08</td>
</tr>
<tr>
<td>HR Control Beliefs (Overall)</td>
<td>79</td>
<td>60.97</td>
<td>19.15</td>
<td>14 – 97</td>
<td>.08</td>
</tr>
<tr>
<td>HR Control Beliefs (Increase)</td>
<td>79</td>
<td>55.15</td>
<td>22.16</td>
<td>10 - 96</td>
<td>-.13</td>
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<tr>
<td>HR Control Beliefs (Decrease)</td>
<td>79</td>
<td>56.91</td>
<td>24.62</td>
<td>7 – 100</td>
<td>-.34</td>
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<tr>
<td>RCMAS Total Anxiety</td>
<td>80</td>
<td>10.97</td>
<td>5.83</td>
<td>1 – 23</td>
<td>.15</td>
</tr>
<tr>
<td>RCADS-C Total Anxiety</td>
<td>79</td>
<td>61.88</td>
<td>14.53</td>
<td>38 – 104</td>
<td>.76</td>
</tr>
<tr>
<td>RCADS-C Total Internalizing</td>
<td>80</td>
<td>79.02</td>
<td>19.20</td>
<td>48 – 137.93</td>
<td>.80</td>
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Table 1 (continued)

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
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<th>SD</th>
<th>Min - Max</th>
<th>Skew</th>
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<tbody>
<tr>
<td>PCS-C Total Aggression</td>
<td>79</td>
<td>10.28</td>
<td>10.44</td>
<td>0 – 49.23</td>
<td>1.74</td>
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<tr>
<td>- PCS-C Reactive Overt</td>
<td>80</td>
<td>4.38</td>
<td>4.49</td>
<td>0 – 21</td>
<td>1.57</td>
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<tr>
<td>- PCS-C Reactive Relational</td>
<td>80</td>
<td>2.99</td>
<td>3.63</td>
<td>0 – 19</td>
<td>1.96</td>
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<tr>
<td>- PCS-C Proactive Overt</td>
<td>80</td>
<td>1.63</td>
<td>2.68</td>
<td>0 – 13.33</td>
<td>2.68</td>
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<tr>
<td>- PCS-C Proactive Relational</td>
<td>80</td>
<td>2.03</td>
<td>3.16</td>
<td>0 – 18</td>
<td>2.67</td>
</tr>
</tbody>
</table>

Note: ACQ-C = Anxiety Control Questionnaire for Children; HR = Heart Rate; RCMAS = Revised Child Manifest Anxiety Scale; RCADS-C = Revised Child Anxiety and Depression Scale – Child Version; PCS-C = Peer Conflict Scale – Child Version.

Table 2. Means, Standard Deviations, Ranges, and Skew for Parent Report Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
<th>M</th>
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<tbody>
<tr>
<td>RCADS-P Total Anxiety</td>
<td>80</td>
<td>51.99</td>
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<td>38 – 92</td>
<td>1.54</td>
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<tr>
<td>PCS-P Total Aggression</td>
<td>79</td>
<td>6.00</td>
<td>7.11</td>
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<td>- PCS-P Reactive Overt</td>
<td>80</td>
<td>2.48</td>
<td>3.86</td>
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<td>2.71</td>
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<tr>
<td>- PCS-P Reactive Relational</td>
<td>80</td>
<td>1.84</td>
<td>3.40</td>
<td>0 – 24</td>
<td>3.99</td>
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<tr>
<td>- PCS-P Proactive Overt</td>
<td>80</td>
<td>.71</td>
<td>1.86</td>
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<tr>
<td>- PCS-P Proactive Relational</td>
<td>80</td>
<td>1.84</td>
<td>2.84</td>
<td>0 – 18</td>
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<td>CBCL Aggressive Behavior</td>
<td>80</td>
<td>6.19</td>
<td>6.02</td>
<td>0 – 24</td>
<td>1.22</td>
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Note: RCADS-P = Revised Child Anxiety and Depression Scale – Parent Version; PCS-P = Peer Conflict Scale – Parent Version; CBCL = Child Behavior Checklist.

Table 3. Means, Standard Deviations, Ranges, and Skew for Physiological Measures

<table>
<thead>
<tr>
<th>Measure</th>
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<tr>
<td>Mean HR (5 Minute Video Baseline)</td>
<td>80</td>
<td>80.76</td>
<td>11.23</td>
<td>53.36 – 109.48</td>
<td>.23</td>
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<tr>
<td>HF-HRV (5 Minute Video Baseline)</td>
<td>78</td>
<td>1146.18</td>
<td>1117.13</td>
<td>66.27 – 5637.63</td>
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<tr>
<td>Mean HR (5 Minute Resting Baseline)</td>
<td>80</td>
<td>81.08</td>
<td>11.05</td>
<td>55.75 – 108.87</td>
<td>.20</td>
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Table 3 (continued)

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<tr>
<th></th>
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<tr>
<td>HF-HRV (5 Minute Resting Baseline)</td>
<td>79</td>
<td>1072.41</td>
<td>989.79</td>
<td>70.45 – 4988.26</td>
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<tr>
<td>Mean HR (3 Minute Video Baseline)</td>
<td>80</td>
<td>80.41</td>
<td>11.32</td>
<td>53.66 – 109.65</td>
<td>.27</td>
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<tr>
<td>HF-HRV (3 Minute Video Baseline)</td>
<td>79</td>
<td>1232.33</td>
<td>1230.04</td>
<td>66.46 – 5919.05</td>
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<td>Mean HR (3 Minute Resting Baseline)</td>
<td>80</td>
<td>80.85</td>
<td>11.01</td>
<td>56.37 – 108.38</td>
<td>.20</td>
</tr>
<tr>
<td>HF-HRV (3 Minute Resting Baseline)</td>
<td>79</td>
<td>1089.80</td>
<td>1028.52</td>
<td>69.75 – 5975.44</td>
<td>2.29</td>
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<tr>
<td>Mean HR (3 Minute Mental Arithmetic Task)</td>
<td>80</td>
<td>83.09</td>
<td>11.21</td>
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<td>.01</td>
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<td>HF-HRV (3 Minute Mental Arithmetic Task)</td>
<td>80</td>
<td>1187.75</td>
<td>1170.47</td>
<td>83.83 – 5135.20</td>
<td>1.89</td>
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<td>HR Control Change Score (Overall)</td>
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<td>9.79</td>
<td>13.79</td>
<td>-20.42 – 48.56</td>
<td>.25</td>
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<td>HR Control Change Score (Increase)</td>
<td>79</td>
<td>8.76</td>
<td>13.38</td>
<td>-16.91 – 53.35</td>
<td>.74</td>
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<td>HR Control Change Score (Decrease)</td>
<td>79</td>
<td>1.03</td>
<td>8.51</td>
<td>-23.21 – 21.87</td>
<td>-.50</td>
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<tr>
<td>Skin Conductance ( 3 Minute Video Baseline)</td>
<td>79</td>
<td>1.85</td>
<td>1.65</td>
<td>.02 – 9.36</td>
<td>2.41</td>
</tr>
<tr>
<td>Temperature (3 Minute Video Baseline)</td>
<td>80</td>
<td>87.69</td>
<td>5.93</td>
<td>71.79 – 96.35</td>
<td>-.71</td>
</tr>
<tr>
<td>Skin Conductance ( 3 Minute Resting Baseline)</td>
<td>79</td>
<td>1.77</td>
<td>1.60</td>
<td>.02 – 8.70</td>
<td>2.17</td>
</tr>
<tr>
<td>Temperature (3 Minute Resting Baseline)</td>
<td>80</td>
<td>89.96</td>
<td>5.56</td>
<td>71.84 – 96.92</td>
<td>-1.22</td>
</tr>
<tr>
<td>Skin Conductance (3 Minute Mental Arithmetic Task)</td>
<td>79</td>
<td>3.55</td>
<td>2.30</td>
<td>.02 – 13.03</td>
<td>1.76</td>
</tr>
<tr>
<td>Temperature (3 Minute Mental Arithmetic Task)</td>
<td>80</td>
<td>88.08</td>
<td>4.54</td>
<td>72.30 – 96.28</td>
<td>-.85</td>
</tr>
</tbody>
</table>

Note: HR = Heart Rate (beats per minute); HF-HRV (high frequency – heart rate variability; ms^2/Hz).

As shown above in Tables 1 and 2, the RCADS-C and RCADS-P scores (Total Internalizing and Total Anxiety) were slightly positively skewed. The PCS-C and PCS-P scores (Total Aggression and each of the four subtypes) were more positively skewed, but this skewness is consistent with past research using this measure (Crapanzano, Frick, & Terranova, 2010) and the variables were not transformed. Skin conductance indices for the Video Baseline (3 minute) and Resting Baseline (3 minute) were also positively skewed, but this is expected given most individuals probably are not as aroused during a normal resting state. However, the
HF-HRV indices for the Video Baseline (3 minute and 5 minute), Resting Baseline (3 minute and 5 minute), and Mental Arithmetic Task were severely positively skewed in this sample. The scores were log-transformed using base 10 [log(10)] in an effort to produce a more normal distribution and is a standard practice with HF-HRV indices in the literature (see e.g., Monk et al., 2001).

Examination of differences in the physiological indices (i.e., mean heart rate and HF-HRV) for the Video Baseline (3 minute and 5 minute) and Resting Baseline (3 minute and 5 minute) using paired sample t-tests indicated that there was no significant difference between the two baseline conditions in mean heart rate or HF-HRV (p > .05). Furthermore, intercorrelation matrices for the child- and parent-reported measures and physiological measures are appended to this document (see Appendices B and C, respectively).

7.2 Hypothesis 1A: Low HF-HRV (Resting Baseline) Would Be Associated with More Child- and Parent-reported Anxiety Symptoms and Aggression.

As shown in Tables 4 and 5, both Video Baseline (5 minute) and Resting Baseline (5 minute) HF-HRV was negatively associated with the RCMAS Total Anxiety, RCADS-C Total Anxiety, RCADS-P Total Anxiety, and PCS-P Proactive Overt Aggression, but not with PCS-C Total Aggression or PCS-P Total Aggression. Test of age (continuous) and gender as possible moderators of the associations using Soper’s (2006) Interaction program indicated no significant age or gender by symptoms interactions (p > .05). Furthermore, there was no significant association between the Video Baseline (5 minute) or Resting Baseline (5 minute) and either the PCS-C or PCS-P Total Reactive Aggression when controlling for PCS-C or PCS-P Total Proactive Aggression or vice versa (p > .05).
Additionally, HF-HRV during the Mental Arithmetic Task was not associated with any child- and parent-reported measures. However, the HR Control Change Score (Increase) was negatively associated with RCMAS Total Anxiety, RCADS-C and RCADS-P Total Anxiety, RCADS-C Total Internalizing, PCS-C Reactive Overt and PCS-C Proactive Overt Aggression. Once again test of age (continuous) and gender as possible moderators of the associations using Soper’s (2006) Interaction program indicated no significant age or gender by symptoms interactions ($p > .05$).

Table 4. Pearson Correlations between Child-Reported Measures and Physiological Control

<table>
<thead>
<tr>
<th></th>
<th>HF-HRV (VB)</th>
<th>HF-HRV (RB)</th>
<th>HF-HRV (MT)</th>
<th>HRCH (OV)</th>
<th>HRCH (IN)</th>
<th>HRCH (DC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCMAS Anx</td>
<td>-.28**</td>
<td>-.25**</td>
<td>-.10</td>
<td>-.16</td>
<td>-.25**</td>
<td>.15</td>
</tr>
<tr>
<td>RCADS-C Anx</td>
<td>-.29**</td>
<td>-.23*</td>
<td>-.11</td>
<td>-.17</td>
<td>-.29***</td>
<td>.18</td>
</tr>
<tr>
<td>RCADS-C Int</td>
<td>-.28**</td>
<td>-.22*</td>
<td>-.11</td>
<td>-.18</td>
<td>-.33***</td>
<td>.22*</td>
</tr>
<tr>
<td>PCS-C Aggr</td>
<td>.03</td>
<td>-.02</td>
<td>.06</td>
<td>-.11</td>
<td>-.18</td>
<td>.11</td>
</tr>
<tr>
<td>PCS-C RO</td>
<td>.08</td>
<td>.04</td>
<td>-.08</td>
<td>-.19</td>
<td>-.25**</td>
<td>.09</td>
</tr>
<tr>
<td>PCS-C RR</td>
<td>.03</td>
<td>-.02</td>
<td>.04</td>
<td>-.11</td>
<td>-.11</td>
<td>.00</td>
</tr>
<tr>
<td>PCS-C PO</td>
<td>.05</td>
<td>.00</td>
<td>-.09</td>
<td>-.10</td>
<td>-.25**</td>
<td>.06</td>
</tr>
<tr>
<td>PCS-C PR</td>
<td>.03</td>
<td>-.03</td>
<td>-.01</td>
<td>-.18</td>
<td>-.15</td>
<td>-.05</td>
</tr>
</tbody>
</table>

Note: ***$p < .001$; **$p < .05$; *$p = .05$; □ Spearman’s Rho was also statistically significant; □□ Spearman’s Rho was not significant; HF-HRV = Log-transformed High Frequency Heart Rate Variability; VB = Video Baseline (5 minute); RB = Resting Baseline (5 minute); MT = Mental Arithmetic Task; HRCH = Heart Rate Change; OV = Overall; IN = Increase; DC = Decrease; RCMAS = Revised Child Manifest Anxiety Scale; RCADS-C = Revised Child Anxiety and Depression Scale – Child Version; PCS-C = Peer Conflict Scale – Child Version; Anx = Anxiety; Int = Internalizing; Aggr = Aggression; RO = Reactive Overt; RR = Reactive Relational; PO = Proactive Overt; PR = Proactive Relational; n = 79 for correlations with the RCADS-C Total Anxiety, PCS-C Total Aggression, Resting Baseline (5 minute) HF-HRV, HR Control Change Score (Overall, Increase, and Decrease);
n = 78 for correlations using the Video Baseline HF-HRV (5 minute); Pearson correlations using log-transformed (base 10) PCS-C Total Aggression, PCS-C RO, PCS-C RR, PCS-C PO, and PCS-C PR produced identical results.

Table 5. Pearson Correlations between Parent-Reported/Combined Measures and HR Indices

<table>
<thead>
<tr>
<th></th>
<th>HF-HRV (VB)</th>
<th>HF-HRV (RB)</th>
<th>HF-HRV (MT)</th>
<th>HRCH (OV)</th>
<th>HRCH (IN)</th>
<th>HRCH (DC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCADS-P Anx</td>
<td>-.30***</td>
<td>-.32***</td>
<td>-.18</td>
<td>-.16</td>
<td>-.07</td>
<td>-.16</td>
</tr>
<tr>
<td>PCS-P Aggr</td>
<td>-.02</td>
<td>-.07</td>
<td>.04</td>
<td>.11</td>
<td>.08</td>
<td>.05</td>
</tr>
<tr>
<td>PCS-P RO</td>
<td>-.02</td>
<td>.02</td>
<td>.03</td>
<td>-.02</td>
<td>-.02</td>
<td>-.01</td>
</tr>
<tr>
<td>PCS-P RR</td>
<td>-.02</td>
<td>-.04</td>
<td>-.07</td>
<td>-.09</td>
<td>-.07</td>
<td>-.04</td>
</tr>
<tr>
<td>PCS-P PO</td>
<td>-.08</td>
<td>.14</td>
<td>-.05</td>
<td>-.03</td>
<td>-.02</td>
<td>-.01</td>
</tr>
<tr>
<td>PCS-P PR</td>
<td>-.05</td>
<td>-.05</td>
<td>-.07</td>
<td>.01</td>
<td>.01</td>
<td>.02</td>
</tr>
<tr>
<td>CBCL Comp</td>
<td>.12</td>
<td>.10</td>
<td>.05</td>
<td>-.08</td>
<td>-.08</td>
<td>-.01</td>
</tr>
<tr>
<td>CBCL Aggr</td>
<td>.05</td>
<td>-.00</td>
<td>.13</td>
<td>-.04</td>
<td>-.05</td>
<td>-.01</td>
</tr>
<tr>
<td>RCADS-CP Anx</td>
<td>-.35***</td>
<td>-.29**</td>
<td>-.13</td>
<td>-.15</td>
<td>-.27**</td>
<td>.17</td>
</tr>
<tr>
<td>PCS-CP Aggr</td>
<td>.06</td>
<td>.00</td>
<td>.05</td>
<td>.00</td>
<td>-.09</td>
<td>.15</td>
</tr>
<tr>
<td>PCS-CP RO</td>
<td>.12</td>
<td>.11</td>
<td>.08</td>
<td>-.13</td>
<td>-.19</td>
<td>.08</td>
</tr>
<tr>
<td>PCS-CP RR</td>
<td>.03</td>
<td>.00</td>
<td>-.02</td>
<td>-.11</td>
<td>-.12</td>
<td>.00</td>
</tr>
<tr>
<td>PCS-CP PO</td>
<td>-.02</td>
<td>-.08</td>
<td>-.15</td>
<td>-.19</td>
<td>-.23□□</td>
<td>.05</td>
</tr>
<tr>
<td>PCS-CP PR</td>
<td>.00</td>
<td>-.01</td>
<td>-.09</td>
<td>-.12</td>
<td>-.10</td>
<td>-.02</td>
</tr>
</tbody>
</table>

Note: ***p < .001; **p < .05; (2-tailed); □□ Spearman’s Rho was not significant; HF-HRV = Log-transformed High Frequency Heart Rate Variability; VB = Video Baseline (5 minute); RB = Resting Baseline (5 minute); MT = Mental Arithmetic Task; HRCH = Heart Rate Change; OV = Overall; IN = Increase; DC = Decrease; RCADS-P = Revised Child Anxiety and Depression Scale – Parent Version; PCS-P = Peer Conflict Scale – Parent Version; CBCL = Child Behavior Checklist; RCADS-CP = Revised Child Anxiety and Depression Scale – Combined Child and Parent; PCS-CP = Peer Conflict Scale – Combined Child and Parent; Anx = Anxiety; Aggr = Aggression; Comp = Competence; RO = Reactive Overt; RR = Reactive Relational; PO = Proactive Overt; PR = Proactive Relational; n = 79 for correlations with the PCS-P Total Aggression, Resting Baseline (5 minute) HF-HRV, and HR
Control Change Score (Overall, Increase, and Decrease); \( n = 78 \) for correlations with the Video Baseline (5 minute) HF-HRV and CBCL Total Competence; Pearson correlations using log-transformed (base 10) PCS-P Total Aggression, PCS-P RO, PCS-P RR, PCS-P PO, and PCS-P PR produced identical results.

**7.3 Hypothesis 1B: Vagal Regulation would be associated with Child- and Parent-reported Anxiety Symptoms and Aggressive Behavior Problems.**

Multilevel modeling was conducted using the software program HLM 7.0 (Raudenbush, Bryk, Cheong, Congdon, & Toit, 2011; see also Bryk & Raudenbush, 1987; Bryk & Raudenbush, 1992) in order to examine individual differences in HF-HRV change (random effects at level-1; repeated observations within the individual) from the two baseline physiological recordings (Video and Resting Baseline) to the Mental Arithmetic Task and to determine whether child- and parent-reported anxiety symptoms or aggressive behavior (random effects at level-2; individual characteristics) predicted this change. In an effort to construct reliable and valid intercepts and slopes from the Video and Resting Baseline to the Mental Arithmetic Task, the Video Baseline (3 minute) and Resting Baseline (3 minute) HF-HRV indices were used to directly correspond with the three-minute HF-HRV derived from the Mental Arithmetic Task.

The outcome variable was the HF-HRV index scores for the Video Baseline (3 minute) or Resting Baseline (3 minute) and Mental Arithmetic Task conditions. Time (coded 0 = Video Baseline [3 minute] or Resting Baseline [3 minute], 1 = Mental Arithmetic Task) was entered as the level-1 predictor and age (grand-mean centered), gender (coded 0 = boy and 1 = girl), and the RCADS-C Total Anxiety, RCADS-P Total Anxiety, PCS-C Total Aggression, or PCS-P Total Aggression were entered continuous predictors at level-2 (random intercepts and slopes of Time were predicted from these variables) All continuous predictors at level-2 were grand-mean
centered to reduce multicollinearity (Tabachnick & Fidell, 2007). There were four models tested for the Video Baseline (3 minute) and Resting Baseline (3 minute) using the RCADS-C Total Anxiety, RCADS-P Total Anxiety, PCS-C Total Aggression, or PCS-P Total Aggression as a predictor in each separate model (all together eight models were tested).

Overall, the results indicated that there were no cross-level age or gender effects on the change of HF-HRV from Video Baseline (3 minute) or Resting Baseline (3 minute) to the Mental Arithmetic Task when entered simultaneously with the RCADS-C Total Anxiety, RCADS-P Total Anxiety, PCS-C Total Aggression, or PCS-P Total Aggression in each model. However, the results presented in Figure 2 did reveal a significant effect of RCADS-C Total Anxiety on the change of HF-HRV from the Video Baseline (3 minute) to the Mental Arithmetic Task [coefficient = 0.005, $t(75) = 2.07$, $p < .05$]. More specifically, it showed an increase in HF-HRV for those youth +1 standard deviation above the mean on RCADS-C Total Anxiety (vagal augmentation) and a decrease in HF-HRV for those youth with -1 standard deviation below the mean on the RCADS-C Total Anxiety (vagal suppression). Conversely, the results indicated there was no significant effect of RCADS-C Total Anxiety on the change in HF-HRV from Resting Baseline (3 minute) to the Mental Arithmetic Task [coefficient = 0.003, $t(75) = 1.84$, $p = .07$] or of RCADS-P Total Anxiety on the change in HF-HRV from Video Baseline or Resting Baseline to the Mental Arithmetic Task [coefficient = 0.004, $t(76) = 1.11$, $p = .27$ and coefficient = 0.002, $t(76) = .63$, $p = .53$, respectively].
Figure 2. HF-HRV Change from Video Baseline (3 minute) to Mental Arithmetic Task for Low Anxious (-1 SD below the Mean), Average Anxious (Mean), and High Anxious (+1 SD above the Mean) Youth as Based on RCADS-C Total Anxiety

Additionally, the results also indicated a significant effect of RCMAS Total Anxiety on the change from both Video Baseline (3 minute) and Resting Baseline (3 minute) to the Mental Arithmetic Task [coefficient = 0.01, t(76) = 2.22, p < .05; coefficient = 0.01, t(76) = 2.27, p < .05]. As shown in Figures 3 and 4, the slope once again for youth who were +1 standard deviation above the mean on RCMAS-C Total Anxiety showed an increase in HF-HRV for those youth +1 standard deviation above the mean on RMCAS Total Anxiety (vagal augmentation) and a decrease in HF-HRV for those youth with -1 standard deviation below the mean on the RCMAS Total Anxiety (vagal suppression). In contrast, the RCADS-P Total Anxiety had no
effect on the change in HF-HRV from the Video Baseline (3 minute) or Resting Baseline (3 minute) to the Mental Arithmetic Task [coefficient = 0.004, t(76) = 1.11, \( p = .27 \); coefficient = 0.002, t(76) = .63, \( p = .53 \), respectively].

Figure 3. HF-HRV Change from Video Baseline (3 minute) to Mental Arithmetic Task for Low Anxious (-1 SD below the Mean), Average Anxious (Mean), and High Anxious (+1 SD above the Mean) Youth as Based on RCMAS Total Anxiety
In terms of using the PCS-C or PCS-P as level-2 predictors, the results indicated there was no significant effect of PCS-C Total Aggression on HF-HRV change from the Video Baseline (3 minute) and Resting Baseline (3 minute) to Mental Arithmetic Task [coefficient = 0.003, \( t(75) = .76, \ p = .45 \); coefficient = 0.003, \( t(75) = .65, \ p = .52 \)] or for the PCS-P Total Aggression from the Video Baseline (3 minute) to Mental Arithmetic Task [coefficient = 0.004, \( t(75) = .66, \ p = .51 \)]. However, the results did reveal a significant effect of PCS-P Total
Aggression on HF-HRV change from the Resting Baseline to Mental Arithmetic Task

[coefficient = 0.01, t(75) = 2.45, p < .05] and is shown below in Figure 5.

Figure 5. HF-HRV Change from Resting Baseline (3 minute) to Mental Arithmetic Task for Low Aggressive (-1 SD below the Mean), Average Aggressive (Mean), and High Aggressive (+1 SD above the Mean) Based on PCS-P Total Aggression

7.4 Hypothesis 1C: Youths’ ACQ-C Total Scores would be Negatively Associated with Child- and Parent-reported Anxiety Symptoms and Positively Associated with Respective Actual Control Measures (Resting HF-HRV and HR Control Change Score).

The ACQ-C total score for this sample was significantly associated with the RCMAS anxiety score (r = .28, p < .05) and positively associated with HF-HRV in the Video Baseline (5 minute), Resting Baseline (5 minute) and Mental Arithmetic Task (Table 7). However, the ACQ-
C score was not related to any other child- or parent-reported measures (see Appendix B). In addition, HR Control Beliefs (Increase and Overall) were both positively associated with youths' HR Control Change Scores (Overall and Increase), but youths' HR Control Beliefs (Decrease) was not related to any HR Control Change Scores (Overall, Increase, or Decrease) as shown below in Table 6.

Table 6. Pearson Correlations between Perceived Control Measures and Actual (Physiological) Control Indices

<table>
<thead>
<tr>
<th></th>
<th>HF-HRV (VB)</th>
<th>HF-HRV (RB)</th>
<th>HF-HRV (MT)</th>
<th>HRCH (OV)</th>
<th>HRCH (IN)</th>
<th>HRCH (DC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACQ-C</td>
<td>.27**</td>
<td>.25**</td>
<td>.22*</td>
<td>.02</td>
<td>.05</td>
<td>-.05</td>
</tr>
<tr>
<td>HRCB (OV)</td>
<td>.07</td>
<td>.09</td>
<td>.06</td>
<td>.23**</td>
<td>.27**</td>
<td>-.05</td>
</tr>
<tr>
<td>HRCB (IN)</td>
<td>.09</td>
<td>.17</td>
<td>.06</td>
<td>.25*</td>
<td>.26**</td>
<td>-.01</td>
</tr>
<tr>
<td>HRCB (DC)</td>
<td>-.06</td>
<td>-.09</td>
<td>-.04</td>
<td>.20</td>
<td>.21</td>
<td>.00</td>
</tr>
</tbody>
</table>

Note: ***p < .001; **p < .05; *p = .05 (2-tailed); HF-HRV = Log-transformed High Frequency Heart Rate Variability; VB = Video Baseline (5 minute); RB = Resting Baseline (5 minute); MT = Mental Arithmetic Task; HRCH = Heart Rate Change; OV = Overall; IN = Increase; DC = Decrease; ACQ-C = Anxiety Control Questionnaire for Children; HRCB = Heart Rate Control Beliefs; n = 79 for correlations with the Resting HF-HRV (5 minute), HR Control Change Score (Overall, Increase, and Decrease) and HR Control Beliefs (Overall, Increase, and Decrease); n = 78 for correlations using the Video Baseline HF-HRV (5 minute).

7.5 Hypothesis 2: There would be Individual Differences in Voluntary HR Control and Youth on Average Would Increase and Decrease Their HR during the HR Control Task.

The means and standard deviations for each baseline and trial condition within Block 1 and Block 2 for 79 youth (one youth repeatedly coughed during the HR Control task and was unable to finish) are presented in Table 7. Two separate repeated measures MANOVAs were
conducted to examine whether there was mean HR change on average across baseline and trials for each block (i.e., Increase Trials in Block 1 and Decrease Trials in Block 2).

Table 7. Means and Standard Deviations for Baseline and Trial Conditions within Block 1 (Increase Trials) and Block 2 (Decrease Trials) of the HR Control Task

<table>
<thead>
<tr>
<th>Block 1 (Increase Trials)</th>
<th>M</th>
<th>SD</th>
<th>Block 2 (Decrease Trials)</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase Baseline 1</td>
<td>78.80</td>
<td>11.02</td>
<td>Decrease Baseline 1</td>
<td>80.76</td>
<td>10.35</td>
</tr>
<tr>
<td>Increase Trial 1</td>
<td>82.21</td>
<td>11.84</td>
<td>Decrease Trial 1</td>
<td>79.89</td>
<td>10.23</td>
</tr>
<tr>
<td>Increase Baseline 2</td>
<td>80.96</td>
<td>10.96</td>
<td>Decrease Baseline 2</td>
<td>80.81</td>
<td>9.84</td>
</tr>
<tr>
<td>Increase Trial 2</td>
<td>84.04</td>
<td>12.27</td>
<td>Decrease Trial 2</td>
<td>80.82</td>
<td>10.13</td>
</tr>
<tr>
<td>Increase Baseline 3</td>
<td>83.46</td>
<td>10.87</td>
<td>Decrease Baseline 3</td>
<td>81.36</td>
<td>10.68</td>
</tr>
<tr>
<td>Increase Trial 3</td>
<td>85.74</td>
<td>12.61</td>
<td>Decrease Trial 3</td>
<td>81.19</td>
<td>9.70</td>
</tr>
<tr>
<td>Increase Baseline 4</td>
<td>82.82</td>
<td>10.70</td>
<td>Decrease Baseline 4</td>
<td>80.91</td>
<td>10.71</td>
</tr>
</tbody>
</table>

The results of the two repeated-measures MANOVAs are presented in Figure 6 and indicated that there was a significant change across baseline and trial conditions for Block 1 (Increase Trials; \(F(6, 73) = 11.36, p < .001, \text{partial } \eta^2 = .483\)). However, there was no significant change across baseline and trial conditions for Block 2 (Decrease Trials; \(F(6, 73) = \))
1.92, \( p = .09 \), partial \( \eta^2 = .136 \)). Follow-up analyses using paired-sample tests (one-tailed, \( p < .05 \)) for Block 1 (Increase Trials) indicated that there was a significant increase in mean HR from Increase Baseline 1 to Increase Trial 1 [mean difference = 3.41; \( t(78) = 5.69, p < .001 \)], Increase Baseline 2 to Increase Trial 2 [mean difference = 3.07; \( t(78) = 5.27, p < .001 \)], Baseline 3 to Increase Trial 3 [mean difference = 2.28; \( t(78) = 3.84, p < .001 \)] and a significant decrease in mean HR from Increase Trial 1 to Increase Baseline 2 [mean difference = -1.24; \( t(78) = -2.43, p < .001 \)] and from Increase Trial 3 to Increase Baseline 4 [mean difference = -2.92; \( t(78) = -4.51, p < .001 \)].

Figure 6. Mean Heart Rate across Baseline and Trials for Block 1 (Increase Trials) and Block 2 (Decrease Trials)
7.6 Hypothesis 3: Measures of Perceived (ACQ-C) and Actual Control (Resting HF-HRV or HR Control Change Score) Would Identify the Four Control Profiles Outlined in the Adapted Weems and Silverman (2006) Model of Control.

To determine whether patterns of perceived control over anxiety and actual emotional control would cluster into the hypothesized four distinct control profiles, two separate two-step cluster analyses using SPSS 21.0 were conducted. The standardized Resting Baseline (5 minute) HF-HRV index and the ACQ-C total scores were the variables used in the first set of cluster analyses and the standardized and HR Control Change Scores and ACQ-C total scores were used second set of cluster analyses (i.e., mean = 0 and standard deviation = 1). This two-step procedure is designed to automatically identify relatively homogeneous groups of cases using a log-likelihood distance criterion and Schwarz’s Bayesian Criterion (BIC) for examining the best fitting model.

The first step involved a sequential pre-clustering approach in which a modified cluster feature (CF) tree was constructed and contained a number of sub-clusters (i.e., leaf entries) produced from cases in the data set and using a specified distance criterion (i.e., the distance of each case to specific sub-cluster determined whether it was absorbed within that sub-cluster or became its own sub-cluster; see Zhang, Ramakrishnon, & Livny, 1996). This process continued until all cases were classified within a sub-cluster or a pre-specified maximum size of the CF tree was reached (the following maximum values were used: number of nodes = 585, number of branches per node = 8, and number of tree levels = 3). If the maximum size values were exceeded, then a new smaller CF tree was constructed using the old CF as an initial base model and an increased threshold distance criterion. An optional “outlier handling” procedure
was also implemented for the both analyses in an effort to deal with cases that did not fit well within a sub-cluster. The process entailed identifying sub-clusters in which the numbers of cases in them were less than 25% of the size of the largest sub-cluster. Outlier sub-clusters (and subsequent cases) were excluded from step 2.

The second step involved a two-stage hierarchical clustering procedure in which sub-clusters formed in step 1 were used as input data (instead of individual cases from the original data set). The hierarchical clustering began with an algorithm that formed a starting cluster derived from the step 1 sub-cluster data set and was compared each other sub-cluster. The sub-cluster with the smallest distance from the starting cluster was then merged with it and this process was repeated until all sub-clusters were merged into one cluster (i.e., the reverse process of building step 1’s CF tree). To determine the best fitting cluster solution the algorithm first calculated an initial BIC estimate for each cluster solution (e.g., 5, 4, 3, etc.) using the ratio of change in the BIC from the first merging relative to each successive merging. During the second stage the algorithm refined the BIC initial estimate by identifying the largest relative increase in distance between the two closest clusters (i.e., ratio of distance measures) from each hierarchical clustering stage (Satish & Bharadhwaj, 2010). The program defines the most optimal cluster model as the one with ratio of distance change that is 1.15 larger than the model with second largest ratio of distance change or if neither model achieves this value than the model with the largest number of clusters is the most optimal cluster solution.

Further evaluation of each final cluster model was conducted via examination of two goodness of fit measures which included: 1) the pattern of change for BIC estimates in each cluster solution (an increase in BIC from the previous model suggests poorer fit) and 2) the
Silhouette coefficient of cluster separation (distance of cases from next closest cluster) and cohesion (distance from center of its cluster) which produces an average value ranging from -1 (poor model) to 1 (excellent model; Kaufman & Rousseeuw, 1990). In addition, SPSS 21.0 produces a predictor importance value for each variable entered into the model ranging from 0 (poor) to 1 (excellent). This index was used to determine whether the two variables were contributing relatively the same in predicting cluster membership. Finally, the replicability of the best fitting cluster model was examined by drawing four random 75% sub-samples from the data (Satish & Bharadhwaj, 2010). Given that the two-step cluster analysis also allows for a set number of clusters to be identified (similar to k-means clustering), the hypothesized four control profiles were manually produced as a comparison for the automated model(s) that had more or less than four groups (e.g., 3 or 5 groups).

7.6.1 Resting Baseline (5 minute) HF-HRV and ACQ-C Total Score Cluster Groups

The two-step cluster analysis using the standardized Resting Baseline (5 minute) HF-HRV index and ACQ-C total scores for $n = 79$ (one case was missing the Resting Baseline [5 minute] HF-HRV index) automatically produced a three-cluster solution that was not consistent with control model presented in Figure 1 (see p. 6). The standardized means and standard deviations for each of the three cluster groups are presented in Table 8 and included the following groups:

1. A **accurate low control** group ($n = 27$): These youth had patterns of low perceived control and actual control.

2. A **over efficacious** group ($n = 26$): These youth had patterns of high perceived control and actual control.
3. An **accurate high control** group (n = 26): These youth had patterns of high perceived and actual control.

Examination of the goodness of fit indices suggested that the three cluster solution was a fair to good fitting model with a silhouette coefficient of 0.5 (Kaufman & Rousseeuw, 1990) and a BIC that decreased from a two-cluster solution (113.71) to a three-cluster solution (110.16) but then increased for a four-cluster solution (126.57; see Figure 7). Further evaluation of the predictor importance index indicated that the Resting Baseline (5 minute) HR-HRV (1.00) was a relatively more important predictor of cluster memberships than the ACQ-C total score (.89).

To examine group differences in Resting Baseline (5 minute) HR-HRV and ACQ-C total scores between the three cluster groups, a MANOVA was also conducted with the Resting Baseline (5 minute) HR-HRV and ACQ-C total scores as the dependent variables and the four cluster solution as the independent variable. The results presented in Table 9 indicated a significant difference of Resting Baseline (5 minute) HR-HRV and ACQ-C total scores between the three clusters and post-hoc probing using Bonferonni corrected pairwise comparisons revealed that there was no significant difference between high perceived and high actual control group and the high perceived and low actual control group on the ACQ-C scores and the high perceived/low actual control group and low perceived and low actual control group. The replicability of the three-cluster solution was tested by drawing four random 75% sub-samples from the data set and indicated that this model was the not a good fit for each random sub-sample with all four analyses producing only a two-cluster model. Given the relatively fair fit of this model and inconsistent replicability using 75% sub-samples, no further analyses using this specific three-cluster model were conducted.
Table 8. Means, Standard Deviations, and Cluster Differences for the ACQ-C Total Scores and Resting Baseline (5 minute) HF-HRV

<table>
<thead>
<tr>
<th></th>
<th>HP/LA (n = 26)</th>
<th>LP/LA (n = 27)</th>
<th>HP/HA (n = 26)</th>
<th>Cluster Effect</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACQ-C</td>
<td>.60 (.55)</td>
<td>-.90 (.63)</td>
<td>.73 (.84)</td>
<td>F(2, 76) = 47.43***</td>
<td>.56</td>
</tr>
<tr>
<td>HF-HRV (RB)</td>
<td>-.66 (.47)</td>
<td>-0.41 (.86)</td>
<td>1.07 (.51)</td>
<td>F(2, 76) = 56.81***</td>
<td>.60</td>
</tr>
</tbody>
</table>

Note: ***p < .001; ACQ-C = Anxiety Control Questionnaire for Children; HF-HRV (RB) = Resting Baseline (5 minute) High Frequency – Heart Rate Variability; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control; Dissimilar superscripts indicate significant differences for pairwise comparisons using Bonferroni corrections.
A four-cluster model was also manually derived using the two-step cluster procedures with both the Resting Baseline (5 minute) HF-HRV and ACQ-C total scores entered as standardized variables. The results indicated a model that was mostly consistent with the control model presented in Figure 1 (see p. 6). The standardized means and standard deviations for each of the four groups are presented in Table 9 and included the following groups:

1. A *over efficacious* group (n = 24): These youth had patterns of moderate perceived control and low actual control.

2. An *accurate high control* group (n = 24): These youth had patterns of high perceived and actual control.
3. An *under efficacious* group (n = 21): These youth had patterns of low perceived control and high actual control.

4. An *accurate low control* group (n = 10): These youth had patterns of low perceived and actual control.

Examination of the goodness of fit index suggested that the four-cluster solution was a fair to good fitting model with a silhouette coefficient of 0.5 (Kaufman & Rousseeuw, 1990). Further evaluation of the predictor importance index indicated that Resting Baseline (5 minute) HR-HRV score was relatively more important in predicting cluster membership than the ACQ-C total score (1.00 vs. 0.62, respectively).

To examine group differences in Resting Baseline (5 minute) HR-HRV and ACQ-C total scores between the four cluster groups, a MANOVA was conducted with the Resting Baseline (5 minute) HR-HRV and ACQ-C total scores as the dependent variables and the four cluster solution as the independent variable. The results presented in Table 9 indicated a significant difference of Resting Baseline (5 minute) HR-HRV and ACQ-C total scores between the four clusters and post-hoc probing using Bonferroni corrected pairwise comparisons revealed no significant differences between the high perceived and high actual control group and low perceived and high actual control group or between the low perceived and low control group and the low perceived and high actual control group on ACQ-C total scores.

Cross-tabulation with a four group model derived from a mean-split of the standardized Resting Baseline (5 minute) HR-HRV and ACQ-C total scores (i.e., mean = 0 and standard deviation = 1) also suggested that there was a significant difference in group membership [$\chi^2 (9) = 163.05, p < .001$] and that only 52.6% (n = 10) of the low perceived and low actual control group from the two-step cluster procedure derived four group model were assigned to that same
group in the mean-split model (see Table 10). Given the slightly poorer fit of the two-step cluster procedure four group model and its lack of agreement with the mean-split low perceived and low actual control group (as used in Scott & Weems, 2010), it seemed that the standardized mean-split groups may better represent control profiles in this sample and it would be more reasonable to test the study’s main hypotheses using the standardized mean-split control profiles.
Table 9. Means, Standard Deviations, and Measure Differences for Clusters Formed Using the ACQ-C and HF-HRV (Resting Baseline)

<table>
<thead>
<tr>
<th>Cluster</th>
<th>ACQ-C</th>
<th>HF-HRV (RB)</th>
<th>Cluster Effect</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP/LA (n = 24)</td>
<td>.66 (.53)¹</td>
<td>-.67 (.43)¹</td>
<td>Wilkes Lambda F (6, 148) = 53.79***</td>
<td>.599</td>
</tr>
<tr>
<td>HP/HA (n = 24)</td>
<td>.83 (.78)²</td>
<td>1.08 (.53)²</td>
<td>F(3, 75) = 37.27***</td>
<td>.599</td>
</tr>
<tr>
<td>LP/HA (n = 21)</td>
<td>-.74 (.50)³</td>
<td>.13 (.44)³</td>
<td>F(3, 75) = 81.05***</td>
<td>.764</td>
</tr>
<tr>
<td>LP/LA (n = 10)</td>
<td>-1.00 (.83)³</td>
<td>-1.43 (.66)⁴</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: ***p < .001; ACQ-C = Anxiety Control Questionnaire for Children; HF-HRV (RB) = Resting Baseline (5 minute) High Frequency – Heart Rate Variability; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control; Dissimilar superscripts indicate significant differences for pairwise comparisons.
Table 10. Cross-tabulation of Two-Step Procedure Forced Cluster Groups by Control Profile Groups Formed using Mean-Split of Standardized Resting Baseline (5 minute) HF-HRV and ACQ-C Total Scores.

<table>
<thead>
<tr>
<th>Mean-Split Groups</th>
<th>LP/LA (n = 10)</th>
<th>LP/HA (n = 21)</th>
<th>HP/LA (n = 24)</th>
<th>HP/HA (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LP/LA (n = 19)</td>
<td>n = 10 (52.6%)</td>
<td>n = 9 (47.4%)</td>
<td>n = 0</td>
<td>n = 0</td>
</tr>
<tr>
<td>LP/HA (n = 13)</td>
<td>n = 0</td>
<td>n = 11 (84.6%)</td>
<td>n = 0</td>
<td>n = 2 (15.4%)</td>
</tr>
<tr>
<td>HP/LA (n = 25)</td>
<td>n = 0</td>
<td>n = 1 (4%)</td>
<td>n = 24 (96%)</td>
<td>n = 0</td>
</tr>
<tr>
<td>HP/HA (n = 22)</td>
<td>n = 0</td>
<td>n = 0</td>
<td>n = 0</td>
<td>n = 22 (100%)</td>
</tr>
</tbody>
</table>

Note: Model $\chi^2 (9) = 163.05, p < .001$; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control.
7.6.2 ACQ-C and HR Control Change Score Cluster Groups

The two-step cluster analysis using standardized ACQ-C and HR Control Change Scores for 95 youth (one case was missing HR Control change score) also produced a three-cluster solution that was not consistent with the control model presented in Figure 1 (see p. 6). The standardized means and standard deviations for each cluster are presented in Table 11 and included the following groups:

1. A *under efficacious* group (n = 20): These youth had patterns of low perceived control and high actual control.

2. A *moderately over efficacious* group (n = 43): These youth had patterns of moderate perceived control and low actual control.

3. An *accurate high control* group (n = 16): These youth had patterns of high perceived and actual control.

Examination of the goodness of fit indices suggested that the 3-cluster solution was a fair to good fitting model with a silhouette coefficient of 0.5 (Kaufman & Rousseeuw, 1990) and a BIC that decreased from a 2 cluster solution (119.18) to 3 cluster solution (117.70) but then increased for the 4 cluster solution (124.29; see Figure 8). Further evaluation of the predictor importance index indicated that the both the HR Control Change Score (Overall) and the ACQ-C were relatively important (1.00) in predicting group membership.

To examine group differences in HR Control Change Scores and ACQ-C total scores between the three cluster groups, a MANOVA was also conducted with the HR Control Change Scores and ACQ-C total scores as the dependent variables and the four cluster solution as the independent variable. The results presented in Table 11 indicated a significant difference of Resting Baseline (5 minute) HR-HRV and ACQ-C total scores between the three clusters and
post-hoc probing using Bonferonni corrected pairwise comparisons revealed that there was no significant difference between high perceived and high actual control group and the low perceived and high actual control group on the HR Control Change Scores. Furthermore, the replicability of the three-cluster solution was tested by drawing four random 75% sub-samples from the data set and indicated that this model was the not a good fit for each random sub-sample with all four analyses producing only a two-cluster model. Given the relatively fair fit of this model and inconsistent replicability using 75% sub-samples, no further analyses using this specific three-cluster model were conducted.
Table 11. Means, Standard Deviations, and Measure Differences for Clusters Formed Using the ACQ-C and HR Control Change Score

<table>
<thead>
<tr>
<th></th>
<th>LP/HA (n = 20)</th>
<th>Mod. HP/LA (n = 43)</th>
<th>HP/HA (n = 16)</th>
<th>Cluster Effect</th>
<th>Partial η²</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACQ-C Total Score</td>
<td>-1.02 (.68)¹</td>
<td>.33 (.68)²</td>
<td>1.06 (.76)³</td>
<td>F(2, 76) = 43.20**</td>
<td>.532</td>
</tr>
<tr>
<td>HR Control Change Score (Overall)</td>
<td>.68 (.91)¹</td>
<td>-.62 (.65)²</td>
<td>1.05 (.55)³</td>
<td>F(2, 76) = 43.07**</td>
<td>.531</td>
</tr>
</tbody>
</table>

Note: **p < .01; ACQ-C = Anxiety Control Questionnaire for Children; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control; Dissimilar superscripts indicate significant differences for pairwise comparisons.
A four-cluster model was also manually derived using the two-step cluster procedures with both the HR Control Change Scores and ACQ-C total scores entered as standardized variables. The results indicated a model that was mostly consistent with four hypothesized control profiles presented in Figure 1 (see p. 6). The standardized means and standard deviations for each group are presented in Table 12 and included the following groups:

1. A *moderately accurate low control group* (n = 29): The youth had patterns of moderately low perceived control and low actual control.

2. An *under efficacious* group (n = 19): These youth had patterns of low perceived control and high actual control.
3. An accurate high control group (n = 16): These youth had patterns of high perceived and actual control.

4. A over efficacious group (n = 15): These youth had patterns of moderate perceived control and low actual control.

Examination of the goodness of fit index suggested that the four-cluster solution was also a fair fitting model with a silhouette coefficient of 0.5 (Kaufman & Rousseeuw, 1990). However, as compared to the three-cluster model, the predictor importance index indicated that the ACQ-C and HR Control measures were relatively equal in importance of predicting cluster membership (0.96 and 1.00, respectively) for the four-cluster model. To further examine group differences in HR Control Change Scores and ACQ-C total scores between the four cluster groups, a MANOVA was also conducted with the HR Control Change Scores and ACQ-C total scores as the dependent variables and the four cluster solution as the independent variable. The results presented in Table 12 indicated a significant difference of HR Control Change Score and ACQ-C total scores between the four clusters and post-hoc probing using Bonferonni corrected pairwise comparisons revealed that there was no significant difference between the high perceived/high actual control group and high perceived and low actual control group on ACQ-C total scores and between high perceived and high actual control group and the low perceived/high control group on the HR Control Change Scores.

Cross-tabulation with a four group model derived from a mean split of the standardized HR Control Change Scores and ACQ-C total scores (i.e., mean = 0 and standard deviation = 1) suggested that there was a significant difference in group membership \( \chi^2 (9) = 131.45, p < .001 \) and that only 65.2% (n = 15) and 66.7% (n = 16) of youth from the high perceived and low actual control group and high perceived and high actual control group, respectively, were
assigned to the same group using the mean-split model (see Table 13). Taking into consideration that the two step cluster procedure model was a fair fitting model and its poor agreement of group membership for two of the four control profiles created using standardized mean-splits (as used in Scott & Weems, 2010), it once again seemed most reasonable that the mean-split groups may best represent distinct control profiles in this particular sample.
Table 12. Means, Standard Deviations, and Measure Differences for Clusters Formed Using the ACQ-C and HR Control Change Score

<table>
<thead>
<tr>
<th>Cluster</th>
<th>ACQ-C Total Score</th>
<th>HR Control Change Score (Overall)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mod. LP/LA (n = 29)</td>
<td>-.07 (.49)$^1$</td>
<td>-.41 (.55)$^1$</td>
</tr>
<tr>
<td>LP/HA (n = 19)</td>
<td>-1.00 (.70)$^2$</td>
<td>.76 (.86)$^2$</td>
</tr>
<tr>
<td>HP/HA (n = 16)</td>
<td>1.06 (.76)$^3$</td>
<td>1.05 (.55)$^2$</td>
</tr>
<tr>
<td>HP/LA (n = 15)</td>
<td>.98 (.56)$^3$</td>
<td>-1.03 (.59)$^3$</td>
</tr>
</tbody>
</table>

Cluster Effect: $F(4, 148) = 47.58^{**}$

Partial $\eta^2$: .637

Wilkes Lambda $F(3, 75) = 43.95^{**}$

Partial $\eta^2$: .607

Note: **$p < .001$; ACQ-C = Anxiety Control Questionnaire for Children; Mod. = Moderate; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control; Dissimilar superscripts indicate significant differences for pairwise comparisons.
Table 13. Cross-tabulation of Two-Step Procedure Forced Cluster Groups by Control Profile Groups Formed using Mean-Split of Standardized HR Control Change Scores and ACQ-C Total Scores.

<table>
<thead>
<tr>
<th>Mean-Split Groups</th>
<th>LP/LA (n = 29)</th>
<th>LP/HA (n = 19)</th>
<th>HP/LA (n = 15)</th>
<th>HP/HA (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LP/LA (n = 15)</td>
<td>n = 12 (80%)</td>
<td>n = 3 (20%)</td>
<td>n = 0</td>
<td>n = 0</td>
</tr>
<tr>
<td>LP/HA (n = 17)</td>
<td>n = 1 (5.9%)</td>
<td>n = 16 (94.1%)</td>
<td>n = 0</td>
<td>n = 0</td>
</tr>
<tr>
<td>HP/LA (n = 23)</td>
<td>n = 8 (34.8%)</td>
<td>n = 0</td>
<td>n = 15 (65.2%)</td>
<td>n = 0</td>
</tr>
<tr>
<td>HP/HA (n = 24)</td>
<td>n = 8 (33.3%)</td>
<td>n = 0</td>
<td>n = 0</td>
<td>n = 16 (66.7%)</td>
</tr>
</tbody>
</table>

Note: Model $\chi^2 (9) = 131.45, p < .001$; LP = Low Perceived Control; HP = High Perceived Control; LA = Low Actual Control; HA = High Actual Control.
7.7 Hypothesis 4: Control Profiles would be Differentially Related to Anxiety and Aggression.

A series of 2 (symptoms: Anxiety versus Aggressive Behavior) x 4 (control profile group) mixed (i.e., within and between subject effects) analysis of variance (ANOVA) tests were conducted to examine between and within control profile differences of youth’s anxiety and aggressive behavior problems (i.e., is there a significant symptom by control profile interaction). The child- and parent-reported measures of anxiety and aggression were standardized (i.e., mean of 0 and standard deviation of 1) in order to make a direct comparison of between and within effects.

In addition, profile analyses using a combined score derived from the RCADS-C Total Anxiety and RCADS-P Total Anxiety scale and the PCS-C Total Aggression and PCS-P Total Aggression were conducted. The combined score was calculated by taking the highest score from the child and parent report and higher scores represented more anxiety or aggressive behavior. For example, if a child scored a 72 on the RCADS-C Total Anxiety scale and a 90 on the RCADS-P Total Anxiety scale then the final RCADS-Combined Child/Parent Total Anxiety score for that child would be 90. This method of creating outcome variables based on multi-informant data is used in research (Aucoin, Frick, & Bodin, 2006) to obtain a more accurate measurement of youth’s emotional and behavioral problems.

Control profiles were constructed using mean splits of total standardized scores on the Resting Baseline (5 minute) HF-HRV or HR Control Change Scores (i.e., actual control) and the ACQ-C total scores (i.e., perceived control). The four groups formed using this method corresponded to each of the four quadrants presented in Figure 1(median splits produced the same groups). Total scores were converted to standardized scores to provide each measure with a
mean of zero and a standard deviation of one. Standardized scores above and below the mean on Resting Baseline (5 minute) HF-HRV or HR Control Change Scores and the ACQ-C Total Scores were designated as having high or low actual control and high or low perceived control, respectively. A series of Pearson chi-square and one-way ANOVAs revealed that the control profiles for the Resting Baseline (5 minute) HF-HRV or HR Control Change Scores and ACQ-C total scores did not differ ($p > .05$) on age, gender, and ethnicity (Euro-American vs. Minority Ethnic Groups).

### 7.7.1 Resting Baseline (5 minute) HF-HRV and ACQ-C Total Score Profiles.

The results of the profile analysis for the Resting Baseline (5 minute) HF-HRV and ACQ-C total score groups indicated a non-significant interaction for the child-reported measures (RCMAS Total Anxiety and PCS-C Total Aggression [$F(3, 75) = 1.27, p = .29, \text{partial } \eta^2 = .048$], RCADS-C Anxiety and PCS-C Total Aggression [$F(3, 74) = 2.02, p = .12, \text{partial } \eta^2 = .076$]. However, the results did indicate a significant symptom (anxiety, aggression) by control profile interaction for the parent-reported measures (RCADS-P Total Anxiety and PCS-P Total Aggression [$F(3, 75) = 2.88, p < .05, \text{partial } \eta^2 = .010$]) and is presented in Figure 9. Follow-up single degree of freedom contrasts for each control profile were conducted to decompose the within-subjects component of the interaction. The results indicated that there was a significant difference between the RCADS-P Total Anxiety and PCS-P Total Aggression scores within the high perceived and low actual control profile and that there were more reported RCADS-P Total Anxiety symptoms [$F(1) = 5.44, p < .05$; see Table 14]. To decompose the between subjects effect, two separate one-way ANOVAs were conducted with the control profiles as the independent variable and the RCADS-P Total Anxiety and PSC-P Total Aggression scores as the dependent variable for each analysis. The non-z scores were used in these analyses in the interest
of reporting the full scale scores and the results are summarized in Table 14 (results were identical when using the z-scores). The results of the one-way ANOVAs indicated a significant difference between the control profiles in RCADS-P Total Anxiety symptoms \[F(3) = 2.84, p = .05\], but further examination of this finding using independent sample t-tests revealed no significant differences between the control profiles.

Figure 9. Symptoms (RCADS-P Total Anxiety and PCS-P Total Aggression) by Control Profiles (ACQ-C Total Score x Resting Baseline [5 minute] HF-HRV)
Table 14. Mean and Standard Deviations for RCADS-P Total Anxiety and PCS-P Total Aggression Symptoms by Resting Baseline (5 minute) HF-HRV and ACQ-C Total Score Control Profiles

<table>
<thead>
<tr>
<th>Measure</th>
<th>Full Sample</th>
<th>LP/LA</th>
<th>LP/HA</th>
<th>HP/LA</th>
<th>HP/HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCADS-P Total Anxiety</td>
<td>51.99 (10.04)</td>
<td>52.16 (10.52)(^\dagger)</td>
<td>49.36 (7.06)(^\dagger)</td>
<td>56.28 (12.03)(^\dagger)</td>
<td>48.65 (7.06)(^\dagger)</td>
</tr>
<tr>
<td>PCS-P Total Aggression</td>
<td>6.00 (7.01)</td>
<td>9.83 (9.95)(^\dagger)</td>
<td>6.14 (7.55)(^\dagger)</td>
<td>4.56 (4.64)(^\dagger)</td>
<td>4.41 (5.45)(^\dagger)</td>
</tr>
</tbody>
</table>

Note: Dissimilar superscripts indicate significant differences of independent-sample t-tests. RCADS-P = Revised Children’s Anxiety and Depression Scales – Parent Version; PCS-P = Peer Conflict Scale – Parent Version; LP/LA = Low Perceived/Low Actual; LP/HA = Low Perceived/High Actual; HP/LA = High Perceived/Low Actual; HP/HA = High Perceived/High Actual.
The results also revealed a significant symptom (anxiety, aggression) by control profile interaction for the combined child and parent reported measures (RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression [$F(3, 73) = 3.14, p < .05, \eta^2 = .114$]) and is presented in Figure 10. Once again follow-up single degree of freedom contrasts for each control profile were conducted to decompose the within-subjects component of the interaction. The results indicated that there was a significant difference between RCADS- Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression within the high perceived and low actual control profile and that there were more reported combined RCADS- Combined Child/Parent Total Anxiety symptoms [$F(1) = 2.43, p < .05$; see Table 15]. To decompose the between subjects effect, two separate one-way ANOVAs were conducted with the control profiles as the independent variable and the RCADS- Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression scores as the dependent variable for each analysis. The non-z scores were used in these analyses in the interest of reporting the full scale scores and the results are summarized in Table 15 (results were identical when using the z-scores). The results indicated a significant difference between the control profiles in RCADS-Combined Child/Parent Total Anxiety symptoms [$F(3) = 3.80, p = .05$]. Independent sample t-tests revealed that the there was a significant difference between the low perceived and high actual control profile and the high perceived and low actual control profile with more RCADS-Combined Child/Parent Total Anxiety symptoms being reported for the high perceived and low actual control profile [$t(37) = 2.79, p < .05$]. Similarly, the results also indicated a significant difference between the high perceived and low control profile and the high perceived and high actual control profile with more RCADS-Combined Child/Parent Total
Anxiety symptoms being reported for the high perceived and low actual control profile \([t(44) = 2.63, p < .05]\).

![Graph showing symptoms (RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression) by Control Profiles (ACQ-C Total Score x Resting Baseline [5 minute] HF-HRV)](image)

Figure 10. Symptoms (RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression) by Control Profiles (ACQ-C Total Score x Resting Baseline [5 minute] HF-HRV)
Table 15. Mean and Standard Deviations for RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression Symptoms by Resting Baseline (5 minute) HF-HRV and ACQ-C Total Score Control Profiles

<table>
<thead>
<tr>
<th>Measure</th>
<th>Full Sample</th>
<th>LP/LA</th>
<th>LP/HA</th>
<th>HP/LA</th>
<th>HP/HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCADS-Combined Total Anxiety</td>
<td>64.57 (14.22)</td>
<td>64.11 (14.89)\textsuperscript{1,2}</td>
<td>60.21 (10.90)\textsuperscript{1}</td>
<td>71.04 (12.01)\textsuperscript{2}</td>
<td>60.21 (15.91)\textsuperscript{1}</td>
</tr>
<tr>
<td>PCS-Combined Total Aggression</td>
<td>13.02 (10.27)</td>
<td>13.39 (10.52)\textsuperscript{1}</td>
<td>13.86 (8.93)\textsuperscript{1}</td>
<td>12.04 (7.57)\textsuperscript{1}</td>
<td>13.30 (13.77)\textsuperscript{1}</td>
</tr>
</tbody>
</table>

Note: Dissimilar superscripts indicate significant differences of independent-sample t-tests; RCADS-Combined = Revised Children’s Anxiety and Depression Scales – Combined Child/Parent Score; PCS = Peer Conflict Scale – Combined Child/Parent Score; LP/LA = Low Perceived/Low Actual; LP/HA = Low Perceived/High Actual; HP/LA = High Perceived/Low Actual; HP/HA = High Perceived/High Actual.
7.7.2 HR Control Change Score and ACQ-C Total Score Profiles. The results of the profile analyses for the ACQ-C and HR Control Change Score groups indicated no significant symptom (anxiety, aggression) by control profile interaction for the child-reported measures (RCMAS and PCS-C Total Aggression [$F(3, 74) = .24, p = .87$, partial $\eta^2 = .009$] and RCADS-C and PCS-C Total Aggression [$F(3, 73) = .50, p = .68$, partial $\eta^2 = .015$]) and combined child and parent reported measures (RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression [$F(3, 72) = 2.30, p = .08$, partial $\eta^2 = .087$]). However, once again there was a significant symptom (anxiety, aggression) by control profile interaction for the parent-reported measures (RCADS-P Total Anxiety and PCS-P Total Aggression [$F(3, 74) = 4.17, p < .05$, partial $\eta^2 = .145$]) and is presented in Figure 11. Follow-up single degree of freedom contrasts for each control profile were conducted to decompose the within-subjects component of the interaction. The results indicated that there was a significant difference between the RCADS-P Total Anxiety and PCS-P Total Aggression scores within the low perceived and high actual control profile and that there were more reported PCS-P Total Aggression symptoms [$F(1) = 8.71 p < .05$; see Table 16]. To decompose the between subjects effect, two separate one-way ANOVAs were conducted with the control profiles as the independent variable and the RCADS-P Total Anxiety and PSC-P Total Aggression scores as the dependent variable for each analysis. The non-z scores were used in these analyses in the interest of reporting the full scale scores and the results are summarized in Table 16 (results were identical when using the z-scores). The results of the one-way ANOVAs indicated a significant difference between the control profiles in PCS-P Total Aggression symptoms [$F(3) = 3.32, p < .05$]. Independent sample t-tests revealed that the there was a significant difference between the low perceived and high actual control profile and the high perceived and low actual control
profile with more PCS-P Total Aggression symptoms being reported for the low perceived and high actual control profile \( t(38) = 2.16, p < .05 \). Similarly, the results also indicated a significant difference between the high perceived and low control profile and the high perceived and high actual control profile with more PCS-P Total Aggression symptoms being reported for the high perceived and low actual control profile \( t(39) = 2.76, p < .05 \).

Figure 11. Symptoms (RCADS-P Total Anxiety and PCS-P Total Aggression) by Control Profiles (ACQ-C Total Score x HR Control Change Score)
Table 16. Mean and Standard Deviations for RCADS-Combined Total Anxiety and PCS-Combined Total Aggression Symptoms by Resting Baseline (5 minute) HF-HRV and ACQ-C Total Score Control Profiles

<table>
<thead>
<tr>
<th>Measure</th>
<th>Full Sample</th>
<th>LP/LA</th>
<th>LP/HA</th>
<th>HP/LA</th>
<th>HP/HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCADS-Combined Total Anxiety</td>
<td>51.99 (10.10)</td>
<td>53.600 (10.56)(^1)</td>
<td>48.59 (7.66)(^1)</td>
<td>51.36 (7.87)(^1)</td>
<td>54.00 (12.78)(^1)</td>
</tr>
<tr>
<td>PCS-Combined Total Aggression</td>
<td>5.88 (7.08)</td>
<td>5.07 (6.80)(^1,-,2)</td>
<td>10.41 (10.20)(^2)</td>
<td>4.91 (5.85)(^1)</td>
<td>4.08 (4.07)(^1)</td>
</tr>
</tbody>
</table>

Note: Dissimilar superscripts indicate significant differences of independent-sample t-tests; RCADS-Combined = Revised Children’s Anxiety and Depression Scales – Combined Child/Parent Score; PCS = Peer Conflict Scale – Combined Child/Parent Score; LP/LA = Low Perceived/Low Actual; LP/HA = Low Perceived/High Actual; HP/LA = High Perceived/Low Actual; HP/HA = High Perceived/High Actual.
7.8 Supplemental Analyses to Test Main Hypotheses

7.8.1 Test of the Model Using Continuous Measures of Actual and Perceived Control. Multilevel modeling using HLM 7.0 (Bryk & Raudenbush, 1992) was also used as an alternative test of the main hypotheses. An advantage of using HLM was that the test of the critical interaction could be implemented using continuous measures of perceived (i.e., ACQ-C scores) and actual control (Resting Baseline [5 minute] HF-HRV and HR Control Change Score) as level-2 random effect predictors (i.e., main effects, two-way interaction terms). In addition, it provided an excellent platform to examine whether age or gender moderated a cross-level interaction between the two-way perceived and actual control interaction term at level-2 (i.e., ACQ-C Total Score by Resting Baseline [5 minute] HF-HRV and HR Control Change Score) and the fixed effect repeated measures indicator variable at level-1. Therefore, a three-way interaction was included in the model (i.e., Age or Gender by ACQ-C Total Score by Resting Baseline [5 minute] HF-HRV index or HR Control Change Score) to test age and gender as possible moderators of the perceived and actual control interaction.

The z-scores for both the anxiety and aggression measures were entered as a continuous outcome variable at level-1 with the repeated measure indicator (i.e., Measure) as a level-1 predictor (0 = anxiety, 1 = aggression). The main effect predictors of age (gender [coded 0 = boy and 1 = girl] and ethnicity in the other models), ACQ-C Total Scores, Resting Baseline (5 minute) HF-HRV or HR Control Change Score, all possible two-way interactions, and the three-way interaction were entered as predictors at level-2 and all continuous variables were grand-mean centered. Random intercepts and slopes of the level-1 repeated measure indicator was predicted in each model. The results of these analyses produced an identical non-significant two-way (i.e., ACQ-C Total Score by Resting Baseline [5 minute] HF-HRV and HR Control
Change Score) and three-way interactions (i.e., Age or Gender by ACQ-C Total Score by Resting Baseline [5 minute] HF-HRV index or HR Control Change Score) for each outcome variables ($p > .05$) as found using the repeated measure MANOVAs in SPSS 21.0.

7.8.2 Test of the Model Using the Four Subtypes of Aggression. The dissertation committee recommended conducting profile analyses using each of the four types of aggression scores for the PCS-C and PCS-P (i.e., Reactive Relational, Reactive Overt, Proactive Relational, and Proactive Overt). Profile analyses for the Resting Baseline (5 minute) HF-HRV by ACQ-C Total Scores control profiles did not result in a significant interaction for each of the four subtypes when tested with the RCMAS Total Anxiety and for three of the four tests using the RCADS-C Total Anxiety. A summary of the final model for each analysis is presented in Appendix D. However, there was a significant symptom (anxiety, aggression) by control profile interaction for the RCADS-C Total Anxiety and PCS-C Proactive Relational Aggression [$F(3, 75) = 2.88, p < .05$, partial $\eta^2 = .104$].

Follow-up single degree of freedom contrasts within each control profile were conducted to decompose the within-subjects component of the interaction. The results indicated that there was a significant difference in symptoms within the high perceived and high actual control profile [$F(1) = 4.17, p < .05$] with more reported PCS-C Proactive Relational Aggression ($M = .37, SD = 1.54$) than RCADS-C Total Anxiety ($M = -.16, SD = 1.19$). To decompose the between subjects effect, two separate one-way analyses of variance (ANOVAs) were conducted with the control profiles as the independent variable and the non-z scores for the RCADS-C Total Anxiety and PSC-C Proactive Relational Aggression scores as the dependent variables for each analysis as to be comparable to the main analyses (z scores produced identical results). The results indicated no significant differences among the control profiles in RCADS-C Total Anxiety.
Anxiety \( F(3, 75) = 1.11, p = .35 \) or PCS-C Proactive Relational Aggression \( F(3, 75) = 1.40, p = .30 \).

The profile analyses using the RCADS-P Total Anxiety scale also resulted in three significant interactions when using the PCS-P Reactive Overt Aggression \( F(3, 76) = 3.10, p < .05 \), PCS-P Proactive Relational Aggression \( F(3, 76) = 2.87, p < .05 \), partial \( \eta^2 = .109 \), and PCS-P Proactive Overt Aggression \( F(3, 76) = 3.63, p < .05 \), partial \( \eta^2 = .125 \).

Follow-up single degree of freedom contrasts within each control profile were conducted to decompose the within-subjects component of each interaction. The results indicated that there was a significant difference within the high perceived and low actual control profile \( F(1) = 6.46, p < .05 \) with more RCADS-P Total Anxiety \( M = .41, SD = 1.21 \) being reported than PCS-P Reactive Overt and Proactive Overt Aggression \( M = -.31, SD = .57, M = -.29, SD = .26 \), respectively. In addition, there was a significant difference within the low perceived and low actual control profile \( F(1) = 6.60, p < .05 \) with more PCS-P Reactive Overt and Proactive Overt Aggression \( M = .36, SD = 1.47, M = .61, SD = 1.70 \), respectively than RCADS-P Total Anxiety \( M = .00, SD = 1.05 \).

To decompose the between subjects effects, separate one-way analyses of variance (ANOVAs) were conducted with the control profiles as the independent variable and the non-z scores for the PSC-P Reactive Overt Aggression, PSC-P Proactive Relational Aggression, or PSC-P Proactive Overt Aggression scores as the dependent variables for each analysis (z scores produced identical results). The results indicated there were significant differences between control profiles in RCADS-P Total Anxiety \( F(3, 76) = 2.84, p < .05 \), Proactive Relational Aggression \( F(3, 76) = 3.53, p < .05 \) and Proactive Overt Aggression \( F(3, 76) = 4.19, p < .05 \). Post hoc probing using independent sample t-tests indicated that those youth with a high
perceived and low actual control profile were reported as having more RCADS-P Total Anxiety ($M = 56.28, SD = 12.03$) than youth with the high perceived and high actual control profile ($M = 48.65, SD = 7.06, t[45] = 2.60, p < .05$). In addition, those youth with a low perceived and low actual control profile were reported as exhibiting more PCS-P Proactive Relational Aggression ($M = 3.58, SD = 4.41$) than youth with a high perceived and low actual control profile ($M = 1.36, SD = 2.00, t[42] = 2.24, p < .05$) and high perceived and high actual control profile ($M = 1.05, SD = 1.40, t[39] = 2.55, p < .05$). Youth with the low perceived and low actual control profile also were reported as exhibiting more PCS-P Proactive Overt Aggression ($M = .43, SD = .94$) than youth in the high perceived and low actual control profile ($M = .20, SD = .50, t[42] = 2.61, p < .05$) and high perceived and high actual control profile ($M = .41, SD = .96, t[39] = 2.09, p < .05$).

Profile analyses using each of the four types of aggression scores for the PCS-C and PCS-P (i.e., Reactive Relational, Reactive Overt, Proactive Relational, and Proactive Overt) did not produce significant interactions for the HR Control Change Score by ACQ-C Total Score control profiles when examined with the RCMAS, RCADS-C Total Anxiety, and RCADS-P Total Anxiety ($p > .05$). A summary of the final model for each analysis is presented in Appendix E.

7.8.3 Test of the Model Using HR Control Beliefs (Overall) and HR Control Change Score. Additionally, the ACQ-C may not capture youths’ perceived control over physiological states as used to measure actual control in this study (i.e., resting HF-HRV) and thus the model was further tested using control profiles derived from HR Control Beliefs (Overall) and HR Control Change Score. The results of the profile analyses were similar to the previous findings with no-significant interaction for the child-reported measures [RCMAS/PCS-C Total
Aggression \[ F(3, 74) = .19, p = .90, \text{partial } \eta^2 = .008 \], RCADS-C/PCS-C Total Aggression \[ F(3, 73) = .02, p = .99, \text{partial } \eta^2 = .001 \], parent-reported measures (RCADS-P/PCS-P Total Aggression \[ F(3, 74) = .31, p = .82, \text{partial } \eta^2 = .012 \]) and combined child/parent reported measures (RCADS-Combined/PCS-Combined Total Aggression \[ F(3, 72) = .08, p = .97; \text{partial } \eta^2 = .003 \]).

7.8.4 Replication of Scott and Weems (2010). We also attempted to replicate the previous findings from Scott and Weems (2010) using the standardized z-scores for the CBCL competence scores (actual control) and ACQ-C (perceived control) to construct the control profiles and the RCADS-C total internalizing score and CBCL aggressive behavior subscale as the dependent variables. There was not a significant control profile by symptom (anxiety symptoms and aggression) interaction \[ F(3, 76) = 1.15, p = .34, \text{partial } \eta^2 = .043 \].

7.9 Exploratory Analyses

Multilevel modeling using HLM 7.0 (Raudenbush, Bryk, Cheong, Congdon, & Toit, 2011; see also Bryk & Raudenbush, 1987; Bryk & Raudenbush, 1992) was conducted to further examine individual differences in HR change (random effects at level-1; repeated observations within the individual) across Block 1 (Increase) and Block 2 (Decrease) trials of the HR Control Task and to determine whether child- and parent-reported anxiety symptoms (fixed effects at level-2; individual characteristics) predicted this specific change. More recent theory and research (Laird & Weems, 2011; Laird & De Los Reyes, 2013) in the area of informant discrepancies suggests that the use of difference scores (i.e., HR Control Change Scores) may not provide a valid test for predicting child outcomes and thus alternative methods of testing differences between multiple measures may be beneficial.
The outcome variable was the mean HR for each baseline and trial within each block. Time (coded 0 = Baseline 1, 1 = Increase or Decrease Trial 1, 2 = Baseline 2, 3 = Increase or Decrease 2, 4 = Baseline 3, 5 = Increase or Decrease 3, 6 = Baseline 4) was entered as the level-1 predictor and age, gender, and the RCADS-C or RCADS-P Total Anxiety were entered as a continuous predictors at level-2 (random intercepts and slopes of Time were predicted from these variables). The results indicated a significant effect of RCADS-C Total Anxiety on mean HR change across Block 1 [Increase Trials; coefficient = 0.016, t(75) = 2.47, p < .05]. As shown in Figure 12, youth with -1 standard deviation below the mean slightly increased their mean HR during Block 1 (Increase Trials), but youth with +1 standard deviation above the mean on RCADS-C Total Anxiety had a steeper increase in mean HR across Block 1 (Increase Trials).
Figure 12. Mean HR Change across the HR Control Task for Block 1 (Increase Trials) for Low Anxious (-1 SD below the Mean), Average Anxious (Mean), and High Anxious (+1 SD above the Mean) Youth as Based on RCADS-C Total Anxiety

Similarly, the results also indicated a significant effect of RCADS-P on mean HR change across Block 2 [Decrease Trials; coefficient = 0.014, t(76) = 2.21, p < .05]. As shown in Figure 13, youth with -1 standard deviation below the mean had a slightly increased mean HR during Block 2 (Decrease Trials), but youth with +1 standard deviation above the mean on RCADS-C Total Anxiety had a steeper increase in mean HR across Block 2 (Decrease Trials). Conversely, all other effects were non-significant for RCADS-C and Block 2 [Decrease Trials; coefficient =
0.003, $t(75) = .62, p = .54$] and RCADS-P and Block 1 [Increase Trials; coefficient $= 0.003, t(76)$ $= .33, p = .75$]

Figure 13. Mean HR Change across the HR Control Task for Block 2 (Decrease Trials) for Low Anxious (-1 SD below the Mean), Average Anxious (Mean), and High Anxious (+1 SD above the Mean) Youth as Based on RCADS-P Total Anxiety

7.10 End Notes.

1 The model was also tested using individual slopes of HF-HRV change from Video and Resting Baseline (3 minute) to Mental Arithmetic Task as the measure of actual control. The results did not reveal a significant control profile by symptom (anxiety symptoms, aggressive
behavior) interaction for the Video or Resting Baseline slopes when the dependent variables were the RCMAS Total Anxiety and PCS-C Total Aggression \[F (3, 74 ) = .29, p > .05, \text{partial } \eta^2 = .01\], RCADS-C Total Anxiety and PCS-C Total Aggression \[F (3, 73) = .40, p > .05, \text{partial } \eta^2 = .02\], RCADS-P Total Anxiety and PCS-P Total Aggression \[F (3, 74) = 2.65, p > .05, \text{partial } \eta^2 = .10\], RCADS-Combined Child/Parent Total Anxiety and PCS-Combined Child/Parent Total Aggression \[F (3, 72) = 1.11, p > .05, \text{partial } \eta^2 = .04\].

Separate tests of age and gender as possible moderators by entering interaction terms (e.g., Age [centered] by RCADS-C Total Anxiety [centered] and Gender by PCS-C Total Aggression [centered]) at level-2 did not result in any significant interactions \((p > .05)\).

8. Discussion

The present study made a number of incremental contributions to knowledge about the link between autonomic dysregulation and both anxiety and aggressive behavior problems across adolescence and further advanced the literature in regards to specific relations between perceived and actual emotional control. Discussion of the study’s findings is presented in the following subsections in the order in which the hypotheses were tested in the Results section. This is followed by a subsection on the limitations of the study and finally a brief summary of overall conclusions and future directions for continuing research in this specific area.

8.1 Hypothesis 1A: Low HF-HRV (Resting Baseline) Would Be Associated with More Child- and Parent-reported Anxiety Symptoms and Aggression.

The present study was the first to specifically examine the link between resting HF-HRV and both anxiety symptoms and aggressive behavior adolescent sample of youth aged 11-17 years. As predicted, a negative linear association was found between lower HF-HRV during baseline conditions (Video Baseline and Resting Baseline) and youths’ anxiety self- and
caregiver-reported symptoms. The findings are consistent with neurobiological theories of emotion regulation (Beauchaine et al., 2001; Porges et al., 1994; Porges, 2007) and past research that has shown an association in older children and young adolescents (8-13 years of age; Greaves-Lord et al., 2007; El-Sheikh et al., 2011, Study 2). Though El-Sheikh and colleagues (El-Sheikh et al., 2001; El-Sheikh & Whitson, 2006; El-Sheikh et al., 2011, Study 1; Wetter & El-Sheikh, 2012) failed to show this relationship in other studies, it is possible differences in the age range of their samples (6-12 years of age) compared to this study may account for the differences. Specifically, the linear association between reported anxiety symptoms and HRV may emerge in adolescents (who are theoretically experiencing dramatic shifts in emotion regulation; Casey et al., 2008).

In contrast, the findings did not show that low resting HF-HRV was associated with more aggressive behavior in this sample of youth. Moreover gender did not moderate this relationship as would be expected based on past research (Beauchaine et al., 2008; Gordis et al., 2010). Furthermore, resting HF-HRV was not related to any of the four specific subtypes of aggression and is inconsistent with Scarpa et al.’s (2010) finding that HRV was negatively associated with reactive aggression and positively related to proactive aggression. It is possible that low resting HF-HRV produces more of an effect in those youth who greater aggression-related problems. That is, the low base rate of reported aggression in this community sample of youth may have limited the associations. Future research might benefit from exploring the linear association among youth with severe aggression problems as compared to a community sample of youth.

Overall, finding that HF-HRV was negatively related to youths’ anxiety problems adds an important piece in understanding the physiological basis of anxious ‘flight’ responses in adolescents and its link to subjective reported anxiety symptoms. Theoretically, the findings are
in line with Porges’s polyvagal theory (Porges et al., 1994; Porges, 2007) and suggest that lower resting HF-HRV may be a good index of emotional dysregulation associated with anxious emotion in adolescence. More specifically, adolescence is developmental period associated with rapid, constant changes in family life, social networks, and academics (i.e., novelty) and lower resting HF-HRV may a signal a biological vulnerability to experience heightened and poorly regulated anxiety that may place some youth at greater risk for developing anxiety-related disorders (Casey et al., 2008).

Empirically, the present findings provide important implications for future research that uses resting vagal tone as an objective index of emotional dysregulation. Though the results of this study do not preclude directionality or causal inference of the relationship between resting HF-HRV and anxiety problems in youth, they do raise a series of questions about the specific origin of this biological vulnerability to pathological anxiety and what specific factors (e.g., genetic and environmental) may play a major role in the development of a dysregulated ‘flight’ response. Some research to date that has examined this exact question in younger children (e.g., pre-school; Scheeringa, Zeanah, Myers, & Putnman, 2004) has found evidence to support a similar relationship between anxiety-related symptoms (e.g., posttraumatic stress symptoms) derived from specific environmental exposure (e.g., exposure to trauma) and low resting vagal tone.

However, it seems the more important question is exactly when does this biological dysfunction first develop and how does development of other areas (e.g., cognitive, behavioral) across childhood influence or are influenced by this biological vulnerability. That is, at what period of development is a child most likely to begin exhibiting lower resting HF-HRV and when does it become linked with poor regulation of emotional states (e.g., prenatal vs. postnatal;
early vs. late childhood)? Furthermore, does puberty mark a sensitive period in which external factors (such as trauma exposure) may have a greater impact on the biological makeup of defensive response systems such as fight versus flight? Though this study does not address these issues the findings do stress the need to for future research to explore the exact nature of low resting vagal tone and how it is longitudinally related with clinical levels of anxiety symptoms.

8.2 Hypothesis 1B: Vagal Regulation would be associated with Child- and Parent-reported Anxiety Symptoms and Aggressive Behavior Problems.

This study is one of the first to report an association between vagal regulation (HF-HRV change from baseline to stressor) and both anxiety and aggressive behavior problems in an adolescent sample. In terms of anxiety symptoms, the results provided further evidence that greater anxiety symptoms are associated with an increase in HF-HRV (vagal augmentation) from baseline measures (Video Baseline and Resting Baseline) to the Mental Arithmetic Task, while less reported anxiety symptoms were associated with an overall decrease in HF-HRV from both baseline to stressor (vagal suppression). These findings are consistent with past research (Greaves-Lord et al., 2007) and the present study appears to be the first to report an association between poor vagal regulation and subjective reported anxiety symptoms when including older adolescents.

In addition, this study is the first to find an association between vagal regulation and a specific measure of aggression (i.e., the PCS-P) in an adolescent sample of youth (aged 11-17 years). More specifically, the findings (see Figure 6) suggests that more caregiver-reported total aggression (PCS-P Total Aggression) was associated with vagal augmentation (increased HF-HRV) in response to the Mental Arithmetic Task, while less caregiver-reported total aggression was associated with vagal suppression (decreased HF-HRV). This finding is consistent with
previous research that has shown more general externalizing problems are related to vagal augmentation in relatively younger samples of youth using parent reports but not child reports (6-12 years of age; Boyce et al., 2001; El-Sheikh et al., 2001; Hinnant & El-Sheikh, 2009).

However, these results are not consistent with Willemen et al.’s (2009) findings of “no association” between vagal regulation and parent-reported externalizing problems in a similar sample of adolescents ($n = 99$; 10-17 years of age). Two possible reasons for this discrepancy may be related to methodological differences. First, most studies to date (including Willemen et al., 2009) have focused on externalizing problems which in general may not explain variance in HF-HRV change specifically associated with aggressive behavior problems and thus lead to null findings. Secondly, Willemen et al. (2009) recorded physiological measurements in the home (more specifically the child’s own bed) as opposed to the lab in the present study, which may resulted in higher resting HF-HRV responses not typically experienced outside the home for those with emotional and behavioral problems (i.e., physiological states may be in a less defensive posture in an environment perceived as safe) and thus little change in or even a decrease in HF-HRV from baseline to stressor.

This study also makes a potential methodological contribution in this area as well. More specifically, the association between vagal regulation and child-reported anxiety symptoms (i.e., RCADS-C Total Anxiety) was found when using HF-HRV derived from the Video Baseline condition but not the Resting Baseline condition. It is plausible that the Video Baseline condition may have produced a less variable index of youth’s HF-HRV for use in examining change to a stressful cognitive task (in this case the Mental Arithmetic Task), due to it 1) helping standardize the physiological state of the individuals (Piferi et al., 2000) and 2) obtain a baseline measure of HF-HRV that may activate similar attentional processes as those produced in a mildly
challenging cognitive task. That is, all youth who participated in the study watched the same film clip and in contrast to the Resting Baseline with no stimuli, youths’ attention may have turned to the atypical nature of the situation (sit blankly for 5 minutes) where thinking or worrying about different things may have ensued.

Taken all together the finding that anxiety and aggression were both associated with poor vagal regulation during stress provides further support for Porges’s (2007) polyvagal theory in that maladaptive functioning across two different problem domains (anxiety and aggression) were associated with greater vagal augmentation as opposed to the more adaptive vagal suppression in response to stress. Of even more theoretical importance is that these findings point towards vagal regulation as a plausible non-specific index of emotion dysregulation across anxiety and aggressive behavior problems in youth. In addition, these findings also highlight the need to further examine differences in HF-HRV between traditional resting baselines and more attention-focused baselines (i.e., relaxing video) in youth and how changes in HF-HRV from both baselines to various stress tasks is effected by a randomized design (see Piferi et al., 2000).

A closer examination of the data does suggest however an interestingly alternative explanation for the current findings in that resting vagal tone (the intercept) may be driving the association between vagal augmentation and both anxiety symptoms and aggressive behavior (the slope). Laird and colleagues (e.g., Laird & Weems, 2011; Laird & De Los Reyes, 2013) have proposed that difference scores derived from two observations may be inherently flawed statistically and that the relationship between one of the two components and a second variable may be the reason for significant findings. In terms of the present study, observed variables tend to regress towards the mean on repeated occasions and thus one would expect those youth with higher intercepts at baseline to have more negative slope (vagal suppression) in response to the
stress task and those youth with lower intercepts to have a more positive slope (vagal augmentation). Therefore, the true difference between those youth with higher and lower anxious or aggressive behavior problems would be their higher and lower resting baseline HF-HRV, respectively, as opposed to a change in HF-HRV. Future studies will thus need to further examine this alternate theoretical perspective in terms of resting vagal tone and vagal regulation.

8.3 Hypothesis 1C: Youths’ ACQ-C Total Scores would be Negatively Associated with Child- and Parent-reported Anxiety Symptoms and Positively Associated with Respective Actual Control Measures (Video and Resting HF-HRV and HR Control Change Score).

As predicted, the ACQ-C Total Score was associated with subjective child-reported anxiety symptoms (i.e., the RCMAS), but not as strongly as previously reported in the literature (Weems et al., 2003). However, the ACQ-C was not significantly associated with any of the other measure child- or caregiver-reported measures of anxiety symptoms (i.e., RCADS-C and RCADS-P). One possible reason for this inconsistency with previous literature is that the ACQ-C did not perform as well in terms of effect sizes with anxiety as would be expected from previous studies (e.g., Muris et al., 2003; Weems et al., 2003). An alternative explanation is that these past studies have typically administered the ACQ-C as part of a semi-structured interview which may have led to a more valid assessment of youth’s anxiety control beliefs as compared to the present study. That is, youth in this study completed the ACQ-C on their own with help only given from a trained RA when youth asked for it and some youth may not have fully comprehended all questions though RAs did not report any questions or problems with understanding ACQ-C items.

The present study also makes a further contribution to the emotion regulation literature in that greater perceived control over anxiety as measured on the ACQ-C was found to be related to
higher HF-HRV across the Video Baseline and Resting Baseline. This finding provides initial empirical evidence that youths’ perceptions of anxious emotional control are associated with index of actual emotional control. Theoretically, this finding suggests that perceived control over anxiety may have an influence on one’s actual ability to regulate anxious emotion (Weems & Silverman, 2006). However, the correlational nature of these findings prevent one from making such directional and causal inferences and future research will need further examine Scott and Weems (2010) adaptation of the Weems & Silverman (2006) model before concluding that perceptions of control lead to poor regulation of anxious emotion through later childhood and adolescence.

8.4 Hypothesis 2: There would be Individual Differences in Voluntary HR Control and Youth on Average would Significantly Increase and Decrease Their HR during the HR Control Task.

In terms of the HR Control Task, the results of this study demonstrated for the first time, in youth, that there are individual differences in youths’ ability to voluntarily control their heart rate (without external biofeedback). Additionally, youth were on average able increase their heart rate when instructed, but just as some studies have reported with adult samples (Gatchel, 1975; Schneider et al., 1978; Sirota et al., 1976), youth had greater difficulty decreasing their heart rate throughout the task. In fact, youth were only able on average to decrease their heart rate between the Baseline 1 and Decrease Trial 1 (see Figure 6 above) and exhibited no change on subsequent trials.

One possible explanation is that the youths’ lack of ability to decrease their heart rate was due to a “floor effect” and that youth had little room for decreasing their heart rate after the initial trial. In fact, a closer inspection of Figure 6 illustrates this probable “floor effect” in that
youths’ mean heart rate after Decrease Trial 1 remained practically the same as the mean heart rate in Baseline 1. Though no studies in children or adults were found to have examined “floor effects” per se in regards to mean heart rate, one can speculate that youths’ physiological states during the decrease trial condition were close to a biologically predetermined mean heart rate (i.e., like a thermostat set to 75°F in a house) that is difficult to surpass. A better test of control as related to decreasing heart rate may be youths’ ability to perform this task immediately following a more stressful task that increases heart rate preceding the decrease trial. Future research is needed elucidate this alternative method of heart rate control in youth and adult samples.

Additionally, exploratory analyses revealed that there was a positive association between youths’ HR Control Change Score (Overall and Increase) and the corresponding HR Control Beliefs (Overall and Increase) assessed following task completion. This finding suggests that youth were somewhat accurate determining how well they did overall increasing/decreasing their heart rate and more specifically increasing their heart rate. However, youths’ HR Control Beliefs (Decrease) were not related to HR Control Change Score (Decrease) suggesting that were not as accurate at determining how well they could actually decrease their heart rate. One possible reason for this finding is that youth on average may not have a good reference of whether or not their heart rate decreased, especially given the little decrease produced overall in this sample (see Figure 6; e.g., Blankstein & Egner, 1977). That is, some youth may not be as good as others at noticing actual changes in heart rate over time (i.e., cardiac awareness) and may be easier to notice increased beating of the heart as opposed to the subtle changes associated with slowing beats of the heart.
Though no a priori hypotheses were derived for the association between HR Control Change Scores and anxiety symptoms in youth, theoretically, one would expect youth with greater anxiety symptoms to exhibit diminished voluntary control over heart rate (Weems & Silverman, 2006). In line with this theoretical perspective, the findings suggested that greater anxiety symptoms were associated with less change during the increase trials. However, multilevel modeling suggested there was also a steeper positive slope for youth reporting more anxiety symptoms as compared to youth reporting less anxiety symptoms across the increase trials. One possible explanation for this discrepancy in findings is that the task as a whole was more stressful for those youth with greater anxiety symptoms and thus voluntary HR control was exacerbated by an already ‘flight’ response that involuntarily increased their heart rate (Weems & Silverman, 2006). Overall, the findings discussed in this subsection provide further support for Weems and Silverman’s (2006) theoretical perspective in that perceptions of control may not always correspond to one’s actual ability to control and that lack of actual voluntary control may help to augment an already overaroused and taxed physiological response system. Therefore, as suggested by Weems and Silverman (2006) it is important that future research continue to distinguish between perceived and actual control when examining their roles in youths’ anxiety problems.

8.5 Hypothesis 3: Measures of Perceived (ACQ-C) and Actual Control (Resting HF-HRV or HR Control Change Score) would Identify the Four Control Profiles Outlined in the Adapted Scott & Weems (2010) Model of Control.

The results of the automated cluster analyses using the ACQ-C and either the Resting Baseline HF-HRV (5 minute) or HR Control Change Score (Overall) did not produce a good fitting model that could identify homogeneous control profiles as predicted in the Weems
Silverman (2006) model of control. The best model it did produce was a three-cluster solution that could not be replicated when drawing 75% random samples from the current sample. However, the forced 4-group cluster solution appeared to be a better fitting model and seemed to identify homogeneous control profiles as predicted and between-group differences in ACQ-C and Resting Baseline HF-HRV (5 minute) or HR Control Change Score (Overall) were consistent with predictions in Weems and Silverman 2006. Nevertheless, closer inspection of the forced four-cluster’s group assignment as compared to the mean-split derived control profiles suggested that a large proportion of youth were removed from the low perceived and low actual control profile when using the Resting Baseline HF-HRV and from the high perceived and low actual control group when using the HR Control Change Score (Overall). This finding suggests future research may fair better to construct their control profiles using the mean-split method provided more homogenous groups were formed based on actual and perceived control measures.

8.6 Hypothesis 4: Control Profiles would be Differentially Related to Anxiety and Aggression.

The findings overall did not provide support for Scott and Weems (2010) adaptation of Weems & Silverman model of control for differentiating anxiety and aggression in that the profile analyses produced only three significant findings and these were largely in opposite directions of the models predictions. More specifically, the results suggested that there were both between- and within-group differences in caregiver-reported and combined child and caregiver reported anxiety and aggressive behavior among the control profiles constructed from the Resting Baseline HF-HRV (5 minute) and the HR Control Change Score. Follow-up analyses however revealed unexpected findings in that the high perceived and low actual control profile
had more reported anxiety than aggression and that the low perceived and high actual control profile had more reported aggression than anxiety, respectively.

One possible reason for these non-significant and unexpected findings as mentioned above is that the primary measure of perceived control (ACQ-C) did not perform as well in terms of effect sizes with anxiety as it had in several previous studies (e.g., Muris et al., 2003; Weems et al., 2003). Another plausible explanation is that resting HF-HRV may not be the best index of emotional dysregulation (or lack of control) for youth experiencing externalizing or aggressive behavior problems. As discussed in section 8.1, resting HF-HRV was not related to total aggressive behavior across child- and caregiver-reported measures of aggression and thus is probably not the best indicator of poor actual emotional control for this particular sample. However, when slopes derived from HF-HRV change between the Resting Baseline (5 minute) and Mental Arithmetic Task (which appears to be a physiological marker of dysregulation of at least parent-reported aggression) did not change the findings. Future research may need to find a better way to quantify vagal regulation as opposed using individual slopes or examine other possible non-specific indicators of actual emotion dysregulation as it relates to both anxiety and aggression.

8.7 Limitations.

Though the results of present study provided several important contributions to the anxiety, aggression, and emotion regulation literature, it was not without its limitations. One limitation is that the cross-sectional design of the study precludes directional or causal interpretations of the findings. For example, it is impossible to rule out that the experience of chronic anxiety symptoms over the youths’ lifetime may have resulted in the low resting HF-HRV or the blunted/augmented vagal response to stressful events. Another limitation is that the
of the study exclusion of those youth who were currently taking medications to treat various medical and mental health conditions from analyses may have lessened the generalizability of the findings to those youth who suffer from more severe physical, emotion, and behavioral problems in the community. However, most of the findings were practically identical when including these youth in the analyses and suggests that the current findings would most likely generalize to other community samples.

In addition, exclusion of those youth who were currently taking medication at the time of participation also reduced the sample size. Though a priori power analysis suggested that the current sample size \( n = 80 \) was large enough to detect the main hypotheses of this study based upon previous research (Scott & Weems, 2010), it may not have been large enough to detect the small between- and within-group effect sizes of the more complicated models (e.g., profile analysis using repeated-measured MANOVAs) reported in the present study. Furthermore, it may help also explain why the ACQ-C did not help much in identifying those youth with anxiety and aggressive behavior problems. Thus, given the small effects found in this study, future research will need to ensure to test this specific model using larger samples in an effort to detect similar small effects.

**8.8 Conclusions.**

The present study makes several novel contributions throughout several areas in the literature (e.g., anxiety, aggression, emotion regulation) and provides a better understanding of the links between autonomic functions and psychopathology in older youth. First and foremost, the present study provides evidence for the specific role of autonomic dysregulation in both anxiety and aggressive behavior problems in late childhood and adolescence. More specifically, an overall interpretation of the findings suggests that resting HF-HRV may be more of a specific
index of poor emotional control associated with anxiety-related problems among youth than with aggressive behavior problems. However, further research is needed to elucidate the specificity and importance of resting HF-HRV given the low base rate of aggression reported in this community sample and the consistency of an association between resting HF-HRV and aggression being shown across several other studies.

Secondly, this study adds to a scant literature base on vagal regulation and anxiety problems by providing more support for the link between poor vagal regulation (i.e., vagal augmentation) and anxiety-related problems across a broad age range of older children and adolescents (11-17 years). In addition, the present study is the first to examine and report a link between poor vagal regulation (i.e., vagal augmentation) and specific aggressive behavior problems (not externalizing problems in general) in youth. This finding is important in the fact it suggests vagal regulation may be a non-specific indictor of emotional dysregulation for both anxiety and aggressive behavior problems. Future research will need to further examine this association in an effort to delineate whether or not vagal regulation can serve as a global measure of emotional dysregulation.
References


Scott, B.G., & Weems, C.F. (2012). *Anxiety control beliefs and voluntary control of heart rate: Actual control is still relevant for anxiety problems!* Unpublished manuscript.


http://dx.doi.org/10.1111/j.1469-7610.2008.01982.x


Appendix A. Post Heart Rate Control Evaluation

1. How well do you think you increased your heart rate during the task? 

   Very Poorly      Very Well

2. How well do you think you decreased your heart rate during the task? 

   Very Poorly      Very Well

3. How well do you think you did overall in terms of increasing and decreasing your heart rate during the task? 

   Very Poorly      Very Well
Appendix B. Intercorrelations of Child- and Parent-Reported Measures

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**Combined Child/Parent Reports**

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Note: **p < .01; *p < .05; ACQ-C = Anxiety Control Questionnaire for Children; RCMAS = Revised Child Manifest Anxiety Scale; RCADS = Revised Child Anxiety and Depression Scale; PCS = Peer Conflict Scale; CBCL = Child Behavior Checklist.
Appendix C. Intercorrelations of Age, Gender, and Physiological Measures

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Note: **p < .01; *p < .05; HR = Heart Rate, HF-HRV = Log-transformed High Frequency Heart Rate Variability.
Appendix D. Summary of Interaction Terms for Repeated Measures MANOVAs with the Resting Baseline (5 minute) by ACQ-C Total Score

Control Profiles and Four Subtypes of Aggression

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Note: *$p < .05$; RCADS = Revised Child Anxiety and Depression Scale; PCS = Peer Conflict Scale; RCMAS = Revised Child Manifest Anxiety Scale.
Appendix E. Summary of Interaction Terms for Repeated Measures MANOVAs with the HR Control Change Score by ACQ-C Total Score

Control Profiles and the Four Subtypes of Aggression

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Note: *$p < .05$; RCADS = Revised Child Anxiety and Depression Scale; PCS = Peer Conflict Scale; RCMAS = Revised Child Manifest Anxiety Scale.
Vita

The author was born in Brownwood, TX and received his primary and secondary education in the Bangs Independent School District. He obtained his Bachelor of Arts degree in psychology from Texas Tech University in 2005 and Master of Arts degree in psychology (clinical track) from Stephen F. Austin State University in 2008. He joined the University of New Orleans psychology graduate program to pursue a Ph.D. in Applied Developmental Psychology. He worked with Dr. Carl F. Weems in the Youth and Family Stress, Phobia, and Anxiety Research Laboratory from 2008 to 2013 and served as lab manager from 2010 to 2013.