Linking Social Anxiety and Autonomic Responses to Social Stress in Preadolescence

by

Alexander K. Kaeppler

A thesis submitted to the Graduate Faculty of
Auburn University
in partial fulfillment of the
requirements for the Degree of
Master of Science

Auburn, Alabama
August 6, 2016

Keywords: social anxiety, physiological responses, autonomic, preadolescence

Approved by

Stephen A. Erath, Chair, Associate Professor of Human Development and Family Studies
Mona El-Sheikh, Leonard Peterson Professor of Human Development and Family Studies
Ben Hinnant, Associate Professor of Human Development and Family Studies
Margaret Keiley, Professor of Human Development and Family Studies

Abstract

A substantial body of theoretical literature suggests that social anxiety may be related to abnormal or inflexible autonomic nervous system activity. In recent years, this notion has been corroborated by several empirical studies, though contrasting evidence exists. Modest and inconsistent associations between social anxiety and psychophysiology may reflect discordance across subjective and physiological dimensions of emotion, or limitations and differences in the methods or analyses used across studies. To address these limitations, the physiological responses (heart rate, HR; respiratory sinus arrhythmia, RSA; skin conductance level, SCL; and pre-ejection period, PEP) of 123 preadolescents (Mage = 12.03 years; 50% females; 42% ethnic minorities) were measured continuously during a lab protocol designed to simulate common peer evaluation experiences, and growth in each physiological variable was examined across the full lab protocol and during two unique stages of social stress. Preadolescents also provided reports of global social anxiety on a well-validated questionnaire as well as context-specific anxiety during the peer evaluation protocol. Latent growth model analyses indicated that context-specific anxiety was associated with more dynamic HR responses across the full lab protocol and during the second stage of social stress, as well as differences in growth for RSA during the first stage of social stress. Global social anxiety, however, was related to slightly reduced SCL reactivity to stress and blunted SCL recovery following social stress in all analyses. Findings may point to the need for theoretical models that consider the context in which physiological responses are measured, as well as potential differences in context-specific and global measures of social anxiety.

Acknowledgements

I would first and foremost like to thank Dr. Stephen Erath for his mentorship, guidance, support, and excellent feedback throughout the process of completing my thesis. I can say with great confidence that the quality of this document would have suffered dearly had I not been provided the opportunity to work with Dr. Erath. His tireless efforts, positive attitude, and enthusiasm for my work helped me to stay focused and upbeat in times of frustration or stagnation, and it was often his excitement that compelled me to continue working and helped me to see the merit in what I was doing. Dr. Erath encouraged me to pursue a thesis that would test my abilities as a researcher and provide exposure to new analytical techniques and complex psychophysiological material, and I am very grateful for all of the help and support that he provided along the way. I feel that a great deal of what I have learned and accomplished during my time as a Master's student is due to having worked with Dr. Erath, and I look forward to continuing to work with him throughout my career.

I would also like to thank each of the members on my committee: Dr. Mona El-Sheikh, Dr. Ben Hinnant, and Dr. Margaret Keiley. My committee was incredibly helpful in identifying areas of my thesis in need of revision and providing detailed feedback, as well as helping me to determine the best analytical approach for testing my hypotheses. Given her expertise in psychophysiology, Dr. El-Sheikh was very helpful in clarifying aspects of the psychophysiological literature that I struggled to understand, and I was very fortunate to be able to incorporate much of her previous work into my own study. Similarly, Dr. Hinnant's

insight and detailed questions helped me to think about the relationship between social anxiety and psychophysiology in ways that I had not previously considered, and I always left our discussions feeling as if I had a much better grasp on the literature. I'd also like to thank Dr. Hinnant and Dr. Keiley for helping me to better understand growth modeling as an analytic strategy, and for always being there to discuss and troubleshoot problems that I may have encountered while conducting analyses.

Finally, I would be remiss if I did not thank my amazing friends and family for their constant support throughout the first few years of graduate school. Though graduate school can be quite grueling at times, it can also be an incredibly fun and rewarding experience if you have great friends around you to help you along the way. Likewise, my entire family has been invaluable to my success in graduate school and our weekly phone calls or "skype sessions" have kept me going for the past two and a half years. I wish to thank my Mom and Dad, who know all too well the struggles of graduate school and are always there to offer advice or just listen when life is a little bit harder, as well each of my siblings for the much needed phone calls and for always making me laugh. Similarly, I want to extend my gratitude to all of my friends who I cannot thank enough for always being there for me, and who will undoubtedly understand that a sincere thank you is about all that I can afford as a graduate student.

Table of Contents

List of Tables

List of Figures

List of Abbreviations

ANS	Autonomic nervous system
BAS	Behavioral activation system
BIS	Behavioral inhibition system
EDA	Electrodermal activity
FFFS	Fight, flight, or freeze system
HR	Heart rate
HRV	Heart rate variability
HSA	High Social Anxiety
LSA	Low Social Anxiety
MANOVA	Multivariate analysis of variance
PEP	Pre-ejection period
PNS	Parasympathetic nervous system
RSA	Respiratory sinus arrhythmia
SA	Social anxiety
SCL	Skin conductance level
SNS	Sympathetic nervous system
TSST-C	Trier Social Stress Test for Children

I. INTRODUCTION

Social anxiety refers to feelings of distress and intense discomfort in social contexts due to fears of rejection and negative evaluations by others (Beidel & Turner, 2007). In the vast majority of circumstances, the experience of social anxiety is mild and short-lived, often confined to transitional periods or isolated events, such as instances of public speaking. However, for some individuals, feelings of social anxiety may be quite severe and may persist across multiple social contexts. In its most severe form, an individual's social anxiety may reach clinical levels, leading to the diagnosis of Social Phobia. These intense and enduring feelings of anxiety often undermine social and educational adjustment (Beidel & Turner, 2007), and forecast other disorders such as depression (Schneier, Johnson, Horning, Liebowitz, & Weissman, 1992; Stein, Tancer, Gelernter, Vittone, & Uhde, 1990) and substance abuse (Kushner, Sher, & Beitman, 1990; Schneier, Johnson, Horning, Liebowitz, & Weissman, 1992).

Feelings of social anxiety commonly increase around the transition to adolescence, as demonstrated by self-reports of increased social worries and anxiety (Somerville, 2013; Westenberg, Gullone, Bokhorst, Heyne, & King, 2007), stronger physiological responses to social stress (Stroud et al., 2009; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010; van den Bos, de Rooij, Miers, Bokhorst, & Westenberg, 2014), and higher rates of social phobia diagnoses (Beidel & Turner, 2007) in early adolescence compared to childhood. Perhaps not surprisingly, this increase in social anxiety coincides with the transition into a developmental period characterized by high levels of peer interaction and potential scrutiny by others. During this time children increasingly evaluate themselves in comparison to their peers (Costanzo, Miller-Johnson, & Wencel, 1995; Parker, Rubin, Price, & DeRosier, 1995), and the growing importance of peer relationships heightens concerns of negative peer interactions. Even in

normative samples, elevated levels of social anxiety in preadolescence and early adolescence have been associated with significant impairment in academic, social, and emotional functioning (Kingery, Erdley, Marshall, Whitaker, & Reuter, 2010). In particular, young adolescents with social anxiety may report lower friendship quality (La Greca & Harrison, 2005; Vernberg, Abwender, Ewell, & Beery, 1992), lower peer acceptance (Erath, Flanagan, & Bierman, 2007; Greco & Morris, 2005; La Greca & Lopez 1998; La Greca & Stone, 1993), and more frequent experiences of peer victimization, which, in turn, may exacerbate feelings of social anxiety (Erath, Tu, & El-Sheikh, 2012).

Although the social and psychological correlates of social anxiety are well-established, until recently the psychophysiology of social anxiety in particular has been relatively understudied. Central to the manifestation of social anxiety are fears of negative evaluations in conjunction with intense feelings of physiological arousal (Beidel & Turner, 2007), which may shape the behavioral responses of socially anxious individuals. Those who experience intense physiological arousal in social situations may allocate attentional resources to internal cues of threat at the expense of social interaction, resulting in insensitive social behavior (Rapee & Heimberg, 1997; Vasey, El-Hag, & Daleiden, 1996). Additionally, high levels of baseline or anticipatory arousal may preclude adaptive physiological reactivity that can promote heightened engagement and attention, thus undermining social competence in challenging situations (Erath & Tu, 2014). Failure to recover to baseline levels after a challenging situation may also increase the likelihood of subsequent avoidance, as failure to recover at a physiological level may make social situations particularly aversive. This, in turn, may create a cyclical pattern in which socially anxious individuals either seek to avoid similar future interactions, thereby isolating themselves from others, or receive the negative feedback they fear as a result of awkward or

insensitive behavior in social situations (Rubin & Burgess, 2001). Thus, a better understanding of the psychophysiology of socially anxious individuals is critical to the advancement of both etiological and intervention models of social anxiety. The present study aims to contribute to this understanding by examining associations between preadolescents' social anxiety and autonomic nervous system activity before, during, and after peer evaluation challenges.

In recent years, research has shifted from an emphasis on the cognitive and behavioral components of social anxiety towards a more comprehensive model, including the psychophysiology of socially anxious individuals. Cognitive models posit that social anxiety is characterized by abnormal cognitions manifested by a tendency to view others as inherently negative in their evaluations, to attach tremendous importance to being viewed positively by others, and to misinterpret both internal and external cues (Rapee & Heimberg, 1997). Such models implicitly minimize the influence of potential differences in physiological functioning, instead emphasizing the role that distorted cognitions play in the development and maintenance of social anxiety. For example, a number of researchers have identified a biased perception of bodily symptoms in individuals with social anxiety (Anderson & Hope, 2009; Schmitz, Blechert, Kramer, Asbrand, & Tuschen-Caffier, 2012; Mauss, Wilhelm, & Gross, 2003; Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014), which suggests that socially anxious individuals may simply be more aware of physiological changes experienced during social-evaluative situations. Though these models are indeed informative and offer fascinating insight into the cognitive components of social anxiety, they pay little attention to physiological arousal; yet physiological arousal (e.g., increased heart rate, sweating, blushing) is considered central to the experience and behavioral expression of social anxiety (Beidel & Turner, 2007). Furthermore, theoretical models that focus primarily on cognitive distortions associated with social anxiety may overlook aspects

of physiological arousal that are critical to intervention efforts and to a more complete understanding social anxiety and corresponding social behavior.

Evidence for physiological correlates of social anxiety has been largely inconsistent and studies to date have typically focused on adult populations, thereby limiting our understanding of the role of psychophysiology in the development of social anxiety in childhood and adolescence. Some psychophysiological studies suggest that socially anxious adults experience chronic hyperarousal as evidenced by elevated heart rates and blood pressure at rest and during stress tasks (Beidel, Turner, & Dancu, 1985; Eckman & Shean, 1997; Gerlach, Mourlane, & Rist, 2004; Gerlach, Wilhelm, Gruber, & Roth, 2001; Kramer et al., 2012; Matthews, Manuch, & Saab, 1986), however, other studies have failed to produce similar results (Anderson & Hope, 2009; Beidel, 1991; Edelmann & Baker, 2002; Hofmann, Newmann, Ehlers, & Roth, 1995; Miers, Blote, Sumter et al., 2011).

Inconsistencies in the literature on social anxiety and psychophysiology parallel the broader literature on emotion response coherence, in which self-reports of emotion are either not associated or only modestly associated with physiological measures (Evers et al., 2014; Hollenstein & Lanteigne, 2014). Modest and inconsistent associations between social anxiety and psychophysiology may reflect discordance across dimensions of emotion, or limitations in methods or analyses. For example, several researchers have identified potential methodological limitations such as small sample sizes (and thus limited power), large age ranges, and the differing nature of the anxiety-provoking tasks used across studies. Additionally, some have argued that the physiological measures used in previous studies (e.g., heart rate or heart rate variability) are too general and do not capture the specific underlying factors that contribute to overall autonomic nervous system activity (Schmitz, Kramer, Tuschen-Caffier, Heinrichs, &

Blechert, 2011). Indeed, research on social anxiety has recently shifted towards a focus on the various components of the autonomic nervous system (ANS) in an attempt to clarify some of these inconsistencies. By measuring both sympathetic and parasympathetic nervous system activity, researchers are able to examine the different factors that contribute to overall autonomic functioning. Thus, through more nuanced psychophysiological studies, researchers may begin to identify precisely how social anxiety is related to distinct components of the ANS.

Polyvagal theory describes the function of the ANS as related to emotions and behaviors, and provides insight into the potential associations between social anxiety and the parasympathetic and sympathetic nervous systems (Porges, 2007). Porges (2007) posits that the parasympathetic nervous system (PNS) serves as a "brake" (via the vagus nerve) that decelerates heart rate and facilitates calmness, attentional focus, and social engagement under normal circumstances. The PNS-vagal influence on attention and social communication can be explained by the evolutionary integration of neural networks that regulate the vagus nerve (which regulates cardiovascular activity) with neural networks that regulate the muscles of the face and head. These muscles are implicated in listening, speaking, and facial expressions, and together these neural networks comprise the Social Engagement System or "face-heart connection" (Porges, 2007; Thayer & Lane, 2000). Similarly, the calming function of the parasympathetic nervous system can be explained by the inhibitory influence of the vagus nerve on the cardiac pacemaker of the heart. In the context of challenging environmental demands, decreased parasympathetic input - or "vagal withdrawal" - allows for sustained attention and increased engagement. However, when this response is insufficient, further activation of the sympathetic nervous system (SNS) may occur, which facilitates mobilization or inhibition, and, at the extreme, fight or flight responses (Beauchaine, 2001; Porges, 2007).

Respiratory sinus arrhythmia (RSA), which reflects high frequency heart rate variations during the respiratory cycle, is an index of vagal input to the heart, and is commonly used as a measure of parasympathetic activity (Porges, 2007). Increases in RSA reflect "vagal augmentation," or an increase in parasympathetic influence on the heart. In contrast, decreases in RSA are associated with decreased parasympathetic influence, or "vagal withdrawal," typically accompanied by an increase in heart rate. Similarly, skin-conductance level (SCL) is a well-validated index of the behavioral inhibition (BIS) dimension of the SNS (Gray, 1987), which inhibits behavior in the face of possible threat or punishment (Beauchaine, 2001). High SCL may reflect anxious arousal, whereas low SCL may reflect fearlessness or impulsivity in challenging or threatening circumstances. However, moderate-to-high SCL may also reflect engagement and inhibitory control efforts (Sheppes, Catran, & Meiran, 2009). SNS activity can also be measured via pre-ejection period (PEP), a commonly-used index of the behavioral activation (BAS) dimension of the SNS (Gray, 1987), which is sensitive to approach/reward situations (Beauchaine, 2001).

Some have argued that social interactions and social stressors may activate both "flight-fight-or-freeze" (FFFS) and BAS neural pathways among socially anxious individuals, as these individuals view social situations as highly threatening yet potentially rewarding (Corr, 2002; Kimbrel, 2008). Corr (2002) posits that FFFS and BAS activity may have antagonistic and facilitating effects on behavior under certain conditions, and that resulting approach-avoidance conflicts may influence BIS activity which is postulated to underlie the emotion of anxiety. Thus, given that the BAS may partially inhibit FFFS responses to threatening stimuli, socially anxious individuals who are high on both FFFS and BIS sensitivity and *low* on BAS sensitivity may experience the most severe levels of anxiety in social situations (Corr, 2002; Kimbrel, 2008).

Empirical evidence to corroborate this notion, though sparse, has been found in studies using self-report measures of BAS/BIS sensitivity (Kimbrel, Nelson-Gray, & Mitchell, 2012; Morgan, van Honk, Hermans, Scholten, Stein, & Kahn, 2009). Specifically, both studies found that higher self-reported BIS and lower BAS were either directly or indirectly (via cognitive biases) related to increased social anxiety, however these results have yet to be replicated using corresponding physiological measures.

The tendency of many clinicians and researchers alike is often to equate social anxiety with both *hyperarousal* and *hyperreacivity* in social situations, where hyperarousal refers to consistently elevated levels of arousal (both at rest and during stressors) and hyperreactivity refers to significantly stronger physiological responses to stress (i.e., a greater or "excessive" increase in arousal from baseline to social stress tasks). Additionally, several psychophysiological theories suggest that anxiety disorders may be accompanied by excessive autonomic arousal - as evidenced by high sympathetic activation - resulting from a tendency to perceive normative situations as threatening (Beauchaine, 2001; Thayer & Lane, 2000). Though there is some evidence to suggest that social anxiety may indeed be characterized by *hyperarousal* in childhood and adolescence, few researchers, if any, have found evidence for *hyperreactivity* in the context of social stress. For example, Erath et al. (2012) examined peer victimization as a potential moderator of the association between preadolescents' social anxiety and physiological arousal in the context of peer stress. Preadolescents (ages 10-12; $n = 63$) participated in a conversation-based peer evaluation task, and the authors found that social anxiety was more strongly associated with physiological hyperarousal (i.e., higher HR, lower RSA) among preadolescents who experienced higher levels of peer victimization. In a similar study, Kramer et al. (2012) compared 8-12 year old children diagnosed with Social Phobia ($n =$

41), to matched healthy controls (*n* = 40). Children were exposed to the Trier Social Stress Test for Children (TSST-C), and no differences in reactivity were observed for HR, salivary alpha-amylase (sAA), or cortisol responses between socially anxious and non-anxious children. However, children with Social Phobia did demonstrate elevated HR levels throughout the baseline, stress, and recovery periods, relative to healthy control children, providing evidence for chronic hyperarousal in socially anxious youth. There is even evidence that social anxiety in children as young as the age of 5 may be characterized by autonomic hyperarousal in the context of social stress, as Nikolić et al. (2016) found that higher EDA and reduced heart rate variability (HRV) were associated with greater social anxiety in a sample of 4.5 year old children (*n* = 110).

Several psychophysiological theories posit that various anxiety disorders may also be characterized by abnormal parasympathetic activity and autonomic inflexibility (Beauchaine, 2001; Friedman, 2007; Porges, 2007). In fact, several studies have found that children and adolescents with social anxiety may actually show blunted cardiac or autonomic reactivity in response to social-stress tasks, in conjunction with baseline hyperarousal. In a study conducted by Schmitz et al. (2011), children (ages 8-12) diagnosed with Social Phobia (*n* = 30) were compared to healthy control children (*n* = 26), while exposed to the Trier Social Stress Test for Children (TSST-C). At baseline, children with Social Phobia demonstrated higher levels of sympathetic activation (i.e., electrodermal activity), higher heart rates, and lower levels of parasympathetic activity (as indexed by low basal RSA levels). Effect sizes were medium to large indicating a relatively strong relationship between social anxiety and physiological measures. Additionally, the authors found evidence for blunted parasympathetic reactivity to the TSST-C in SP children, and a slower heart rate recovery after the task relative to healthy control children.

Intriguingly, in a subsequent study, Schmitz et al. (2013) found similar results in a population of children with subclinical levels of social anxiety. Children (ages 10-12) were split into high (n = 20) and low social anxiety (n = 20) categories based on scores from the revised Social Anxiety Scale for Children (SASC-R), and physiological measures were obtained before, during, and after a standardized speech task. Moderate effects were observed between social anxiety and physiological measures, such that children who were high in social anxiety demonstrated higher baseline sympathetic activity, blunted cardiac and parasympathetic reactivity to the speech task, and a slower HR recovery after the speech task. Thus it appears that autonomic functioning in socially anxious youth may be characterized by baseline hyperarousal (i.e., low RSA, high SNS activity, high HR), blunted cardiac and parasympathetic reactivity to social stress, and slower autonomic recovery following social stress.

Taken together, these findings appear to provide evidence in support of a model of autonomic inflexibility in socially anxious youth. Additionally, the aforementioned studies provide some evidence to support the notion of hyperarousal in individuals with social anxiety, though this may be limited to baseline measures of physiology. Overall, the available research generally suggests baseline hyperarousal and limited physiological reactivity and recovery among highly anxious individuals. However, these findings are by no means conclusive and have yet to be applied to a variety of physiological parameters or replicated across different child and adolescent samples. Furthermore, several researchers have found contradictory evidence in young adult (college-aged) and adult samples (e.g., Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014; Mauss, Wilhelm, & Gross, 2003; 2004), which suggests that physiological differences may only exist in childhood and adolescence, or that contradictory findings may be the result of methodological differences or limitations across studies. Therefore, the question of whether or

not meaningful physiological differences exist between preadolescents with higher and lower levels of social anxiety remains unanswered, highlighting the need for the present study. It is our aim to build upon the psychophysiological literature in a variety of ways and to shed light on this controversial and unsolved area of research.

The Present Study

In the present study, physiological responses were measured during a lab protocol in which participants were asked to lead a conversation while under evaluation by fictitious peer judges, and to consider a response to the peer judges after receiving feedback that they were not chosen as one of the best performers during the conversation activity. Interactive situations that include mild negative social feedback (e.g., instances of peer rebuff) are particularly common during adolescence and may better reflect actual social challenges faced by socially anxious youth than speech or non-social tasks. Likewise, conversation-based tasks and instances of peer rebuff may represent anxiety-provoking stressors that are more salient to individuals with social anxiety, whereas speech and performance anxiety are more common among a variety of individuals. Thus, the social interaction protocol utilized in the current study may help to eliminate overlap between socially anxious preadolescents, and preadolescents who have high levels of speech or performance anxiety. The ecological validity of the task used in the present study is further evident when one considers that participants are being assessed during a developmental period when peer evaluation and social feedback are particularly salient.

In addition to the ecological validity of our peer-evaluative lab protocol, the present study included the assessment of multiple physiological parameters. To date, many psychophysiological studies have utilized only a small number of physiological measures, thereby limiting the extent to which researchers can capture small, yet meaningful differences

across physiological systems. By including measures of both sympathetic (SCL and PEP) and parasympathetic (RSA) functioning, as well as more general autonomic measures (HR), the current study sought to provide a more detailed understanding of the distinct dimensions of ANS activity associated with social anxiety in early adolescence. Moreover, to our knowledge, no other study has examined physiological measurements of both BIS (SCL) and BAS (PEP) activity in relation to social anxiety. BAS activity among anxious individuals has been largely overlooked, despite the possibility that assessments of BAS activity may be critical to developing a more complete and nuanced understanding of the psychophysiology of social anxiety.

In the present study, participants' physiological responses were measured across a full lab protocol and in the context of two unique "stages" of social stress (within the same lab protocol), each of which consisted of a pre-task, stress, and recovery period. After a baseline period, preadolescents were asked to hold a conversation with a gender-matched research assistant, while under the assumption that they were being evaluated by three fictitious peer judges (referred to as the "peer evaluation period"). Following the peer evaluation period (i.e., the first social stressor) and a brief waiting period, preadolescents were informed that the peer judges chose two other fictitious participants as the best performers in the conversation task. Participants were then told that they may be able to reconnect with the peer judges and were given 3 minutes to consider their potential responses to the peer judges. This 3-minute period is referred to as the "peer rebuff" period, which was followed by a recovery period and served as a second type of social stress within the lab protocol. Thus, the first "stage" of social stress included the baseline period, peer evaluation period, and subsequent waiting period; whereas the second stage of social stress included the waiting period, peer rebuff period, and a final recovery period (see Appendix D for further description of each stage; a more detailed account of the

entire lab protocol is also provided in the *Method* section). Examining whether social anxiety predicted change in physiological activity across the full lab protocol and during two separate stages of pre-task, stress, and recovery allowed us to test for corroborating results and consider differences across two related social stressors.

In contrast to this approach, previous studies have typically aggregated measures of physiological functioning across time periods (i.e., tasks) during which meaningful change may occur. This technique may overlook subtle within-period changes that help distinguish adolescents with higher and lower levels of social anxiety. By conducting growth modeling analyses with multiple physiological measurements within each period (i.e., three 1-minute measurements within each period), the present study was able to examine associations between social anxiety and changes in ANS responses across pre-task periods, social challenges, and recovery periods.

Furthermore, although relatively few studies exist, the majority of psychophysiological studies on social anxiety in adolescence have examined associations between global self-report measures of social anxiety and different aspects of physiological functioning. The current study, however, utilized context-specific – or "real-time" – measures of anxiety, in conjunction with global self-report measures. Both measures of social anxiety were used to complement one another, as context-specific measures may be more precise and may provide a more accurate depiction of anxiety levels experienced during the lab protocol, whereas global self-report measures may be more reliable and generalizable. Correlations between global and context-specific measures of social anxiety are surprisingly low, indicating that each measure may be tapping into unique dimensions of anxiety. Thus, by using two complementary measures of social anxiety, the present study attempted to clarify whether models of hyperarousal or

autonomic inflexibility are more applicable to preadolescents who report anxiety in real-time social stress situations, or to preadolescents who report more social anxiety in general.

By addressing these limitations and building on prior studies, our goal was to advance the current understanding of autonomic functioning in socially anxious youth. We hypothesized that preadolescents with higher levels of social anxiety would exhibit autonomic hyperarousal and inflexibility as measured through HR, RSA, SCL, and PEP, and that all findings would be replicated across context-specific and global measures of social anxiety. First, we hypothesized that social anxiety would be associated with higher initial levels and less quadratic growth in HR across the full lab protocol, and for both stages of social stress. That is, at lower levels of social anxiety, we anticipated that HR would be lower during the pre-task periods, increase during stress periods, and decrease during recovery periods. In contrast, at higher levels of social anxiety, we expected higher pre-task HR and minimal change across the stress and recovery periods, such that anxious preadolescents would demonstrate a relatively high and stable HR pattern across the lab protocol.

Second, we hypothesized that social anxiety would be associated with lower initial levels and less quadratic growth in RSA across the full lab protocol and during both stages of social stress. That is, at lower levels of social anxiety, we predicted that RSA would change non-linearly, such that RSA would be higher during pre-task periods, decrease during the stress periods, and increase during the recovery periods. In contrast, at higher levels of social anxiety, we anticipated lower pre-task RSA levels and minimal change across the stress and recovery periods, such that socially anxious youth would exhibit a relatively low and stable pattern of RSA across the lab protocol.

Third, we hypothesized that social anxiety would be associated with higher initial levels but not growth in SCL across the full lab protocol and during the first stage of social stress. That is, we hypothesized that SCL would be lower during the baseline period at lower levels of social anxiety relative to higher levels of social anxiety, whereas growth in SCL would be similar at lower and higher levels of social anxiety, such that preadolescents would exhibit increases in SCL during the stress period and decreases during the recovery period. Analyses for the second stage of social stress were considered exploratory, as limited empirical evidence precluded the formulation of hypotheses regarding "passive" social stressors (i.e., peer rebuff experiences) and SCL in particular.

Conceptually, two different patterns of physiological activity during the second stage of social stress seemed equally plausible. First, some have argued that in the context of mild-to-moderate stress, SCL may reflect engagement and inhibitory control efforts rather than anxiety (Sheppes et al., 2009). Thus, during the second stage of social stress, growth in SCL could be similar at lower and higher levels of social anxiety if preadolescents are similarly engaged with the stressor, such that SCL increases during the stress period and decreases during the recovery period. In contrast, several theories of social anxiety posit that SCL is indeed a marker of anxiety (e.g., Beauchaine, 2001) and that instances of peer rejection may be particularly stressful for individuals with high levels of social anxiety (Beidel & Turner, 2007). Consequently, social anxiety may be associated with more quadratic growth in SCL during the second stage of social stress. That is, at higher levels of social anxiety, SCL would be expected to increase during the stress period and decrease during the recovery period; whereas at lower levels of social anxiety, we would expect minimal change across the stress and recovery periods during the second stage of social stress.

Finally, we hypothesized that social anxiety would be associated with shorter initial levels and less quadratic growth in PEP across the full lab protocol and for both stages of social stress. That is, at lower levels of social anxiety, we expected PEP to change non-linearly, such that PEP would be shorter during pre-task periods, further shorten (i.e., decrease) during the stress periods, and lengthen (i.e., increase) during the recovery periods. In contrast, at higher levels of social anxiety, we anticipated longer pre-task PEP levels and minimal change across the stress and recovery periods, such that socially anxious preadolescents would exhibit a relatively stable pattern of longer PEP across the lab protocol (i.e., consistently low levels of BAS-related sympathetic activity). To aid in the interpretation of hypotheses and results involving PEP we feel that it is important to note that "shorter" PEP refers to *higher* levels of BAS-related sympathetic activity (i.e., a shorter period of time between the start of ventricular depolarization – or QRS complex – and the moment of aortic valve opening), whereas "longer" PEP refers to *lower* levels of BAS-related sympathetic activity.

II. METHOD

Participants

One hundred-twenty-three (123) fifth and sixth graders (*M*age = 12.03 years, *SD* = .64) and one parent per preadolescent (82% biological mothers, 67% married) participated in the study. The sample of preadolescents included 50% males and 58.5% European Americans, 35% African Americans, and 6.5% of other ethnicities. The modal annual family income was between $35,001 and $50,000; 21% reported an income of less than $20,000, and 24% reported an income of more than $75,000.

Procedures

Participants were recruited in two cohorts separated by one year through flyers sent home with fifth and sixth grade students at five elementary schools in the southeastern United States. Parents who responded to the flyers were provided with information about the study, including details of the lab protocol, and were scheduled for a research visit via telephone. Preadolescents and their parents visited the lab for assessment during the summer. The lab visit lasted approximately two hours, and parents and preadolescents were compensated monetarily for their participation. Following an introduction and consent procedures, parents completed questionnaires and preadolescents participated in lab activities while their physiological responses were monitored and recorded. After completing lab activities, preadolescents were debriefed and given a brief snack break before completing questionnaires. All study procedures were approved by the University Institutional Review Board.

The lab protocol included *baseline*, *peer evaluation*, *waiting*, *peer rebuff*, and *recovery* components. Following a 5-minute acclimation period used to help preadolescents adjust to the physiological equipment, participants' physiological responses were measured during a 3-minute baseline period. During this period, preadolescents were instructed to sit quietly and look at pictures of nature scenery on a nearby computer screen until a research assistant returned to the room. After the acclimation and baseline periods, participants responded to several interview questions (e.g., "how difficult do you expect the conversation activity to be?") and were instructed to try their best to lead a 3-minute conversation with an adult research assistant (RA; same sex) as if they were meeting an unfamiliar, same-age peer for the first time. To lead the conversation, preadolescents were told that they could talk about themselves, ask questions about the RA, or talk about anything they wished. They were told that the conversation would be

viewed via one-way Skype (an internet-based video-chat program) by three same-age, same-sex peer judges, who were actually fictitious. Preadolescent participants were informed that the peer judges would decide how well they performed in the conversation activity compared to two other participants the peer judges had allegedly watched via Skype. The *peer evaluation* period refers to the 3-minute conversation activity.

Following the peer evaluation period, preadolescents responded to several interview questions about the conversation activity and were told that they would soon receive a response from the peer judges via Skype indicating who they chose as the best performers. The 3 minutes after post-conversation interview questions is referred to as the *waiting period*. After the waiting period participants received a text message via Skype, ostensibly from the peer judges, indicating that the peer judges chose the other two participants as the best performers in the conversation activity. Participants were then told that they may have a chance to change the peer judges' opinions by speaking directly to them through Skype. The *peer rebuff* period refers to the 3 minutes following the feedback from the peer judges, during which participants waited and considered their potential response to the peer judges. Following the peer rebuff period and several interview questions, the task was ended by informing participants that the Skype connection to the peer judges had been lost and preadolescents were asked to sit quietly for 3 minutes, similar to the baseline period. The *recovery* period refers to the 3 minute period after the lab protocol had been ended. Following the recovery period, physiological equipment was removed and participants were carefully debriefed using a process debriefing procedure, informed by Underwood (2005) and Hubbard (2005). Specifically, participants were led to their own conclusion that the peer judges were not real, and the rationale for deception and purpose of the study were discussed with participants.

Measures

Social Anxiety. Social anxiety (SA) was assessed in two ways (see Appendix B). As a measure of *global social anxiety*, preadolescents completed the Social Anxiety Scale for Adolescents (SAS-A; La Greca & Lopez, 1998), an 18-item self-report measure (e.g., "I feel that others make fun of me"; "I feel shy even with peers I know very well"), with items rated on a 5-point scale (1 = *not at all*, 5 = *all the time*). Internal consistency of the SAS-A was good (α = .92). At the *context-specific* level, SA was assessed with a composite of two items from the peer evaluation task. Participants were asked to rate their anxiety on a 5-point scale (1 = *not at all*, 5 = *very much*) before and after the conversation activity (e.g., "how nervous or anxious are you about the conversation activity?" and "how nervous or anxious were you during the activity?"). The two items were moderately correlated (r = .58, p < .001) and were averaged to create a lab-based, *context-specific social anxiety* measure.

Physiological assessment. HR, RSA, SCL, and PEP were measured continuously in 1-minute intervals during acclimation (5 minutes; not used in analyses), resting baseline (3 minutes), speaking baseline (reading aloud with an RA; 3 minutes; not used in analyses), peer evaluation (3 minutes), waiting (3 minutes), peer rebuff (3 minutes), and recovery (3 minutes) periods (see Appendix C for descriptive statistics of each measure). Because the assent process included a description of the peer stress protocol, baseline measurements of physiological parameters may be conceptualized more accurately as *pre-task levels*, which were influenced by some level of anticipatory stress. Peer stress levels of physiological parameters were not collected for three participants because they chose not to participate in the peer stress procedures or their uncomfortable appearance led us to forego the peer stress period.

HR and RSA. HR and RSA data acquisition followed standard guidelines (Bernston et al., 1997) using a Bioamp data acquisition system (MindWare Technologies, Inc., Gahanna, OH). Electrocardiography data were collected through disposable silver/silver-chloride (Ag-AgCl) electrodes (1½" foam sensor, 7% chloride gel) placed on participants' right clavicle and left and right rib by a same-sex RA. HR scores were quantified as heart beats per minute with MindWare HRV analysis software. RSA scores were quantified using the spectral analysis method (Berntson et al., 1997) with MindWare HRV analysis software and expressed in units of $\ln(ms^2)$. Very few artifacts were detected and corrected manually using standard procedures (Berntson et al., 1997).

SCL. Data acquisition followed standard guidelines using a MindWare data acquisition system and MindWare EDA analysis software (MindWare Technologies, Gahanna, OH). Skin conductance (units = microsiemens or μS) was measured with two disposable Ag-AgCl electrodes (1½" x 1" foam, 0% chloride gel) placed on the palm of the non-dominant hand. Participants were seated throughout the physiological assessment, and a taped loop in electrode lead cables was used to further limit movement artifacts. SCL data were not included for 12 participants due to measurement artifacts.

PEP. Cardiac pre-ejection period (PEP) was derived from cardiac data using a modified lead-II configuration (Berntson et al., 1997) and thoracic impedance data using a four-spot impedance configuration (Berntson & Cacioppo, 2004). These data were collected using Ag-AgCl electrodes (1 ½" foam, 7% chloride gel; MindWare Technologies, Inc., Gahanna, OH). To measure cardiac data, electrodes were placed on the right clavicle and left and right ribs. Thoracic impedance was measured using electrodes placed at the apex and base of the thorax and dual electrodes were placed on the back, approximately 1 ½ inches above and below the thorax

electrodes. Data were quantified using MindWare IMP analysis software and are measured in milliseconds (ms).

Demographic variables. Gender, ethnicity, and grade level were represented by dichotomous variables (male = 0, female = 1; non-African American = 0, African American = 1; 5^{th} grade = 0, 6^{th} grade = 1, respectively), and parents reported annual household income on a 6-point scale (1 = *Less than $10,000* to 6 = *More than $75,000*).

Plan of Analysis

The aim of the current study was to identify differences in physiological functioning among preadolescents with varying degrees of social anxiety. We used growth modeling in MPlus (Muthen & Muthen, 2012) to examine whether social anxiety predicted the intercept (i.e., pre-task level) and linear or quadratic growth in four physiological parameters (HR, SCL, RSA, and PEP) across pre-task, social stress, and recovery periods. These physiological parameters were measured continuously and scored in 1-minute intervals across a 3-minute baseline period, 3-minute social stress periods (i.e., peer evaluation or peer rebuff period), and 3-minute recovery periods (i.e., waiting period and true recovery period) following each social stress period. The first set of growth models predicted the intercept and linear and quadratic terms for each physiological parameter across the entire lab protocol, which included the baseline, peer evaluation, waiting, peer rebuff, and recovery periods. The second set of growth models predicted the intercept and linear and quadratic terms for each physiological parameter during the first stage of social stress, which included the baseline, peer evaluation, and first recovery (i.e., waiting) period. The final set of growth models predicted the intercept and linear and quadratic terms for each physiological parameter during the second stage of social stress, which included the waiting (pre-task), peer rebuff, and final recovery period. Note that the waiting

period served both as a recovery period during the first stage of social stress as well as a pre-task period during the second stage (see Appendix D for a description of the two stages of social stress). All three sets of growth analyses were conducted separately for global and context-specific measures of social anxiety. In addition, age, sex, ethnicity, and income were included as control variables if they were significantly correlated with the physiological outcome variable. Specifically, we controlled for the effects of age, sex, ethnicity, or income on the intercept of a particular growth model if they were correlated with the average level of the physiological outcome variable during baseline (for models using the full lab protocol and during stage one) or during the waiting period (for stage two). Likewise, demographic variables were included as covariates for the linear and quadratic slopes if they were correlated with reactivity to or recovery from either social stressor (i.e., reactivity to or recovery from either the peer evaluation or peer rebuff period) for the physiological outcome variable.

III. RESULTS

Descriptive statistics and correlations are shown in Tables 1-4. A series of unconditional and conditional growth models were fit using Mplus version 7.0 (Muthén & Muthén 1998-2012) to examine intercepts and growth in each physiological variable (i.e., HR, SCL, RSA, and PEP) across the full lab protocol, and during two unique stages of social stress. Mplus allows for the inclusion of respondents with missing data by utilizing full information maximum likelihood (FIML) estimation, which sorts observations into missing data patterns and estimates growth parameters using all available data. It was hypothesized that the average growth trajectory for each physiological variable would be quadratic in each set of analyses, such that participants would exhibit increases in physiological arousal during social stress and subsequent decreases in arousal during recovery periods. Accordingly, each set of growth model analyses compared the

fit of unconditional linear and quadratic models (models with no predictors other than time) using a $\Delta\chi^2$-test, and the variance of growth parameters was examined to identify those that could be predicted by the substantive predictor variables (i.e., measures of social anxiety and relevant control variables).

Growth parameters (i.e., the intercept, linear slope, and quadratic slope) were examined individually to determine if measures of social anxiety were significant predictors of each parameter after controlling for relevant demographic variables. This is slightly different from the typical approach of conducting a $\Delta\chi^2$-test to determine the effect of a predictor variable on all three growth parameters at once, which allows a researcher to comment on the extent to which a variable predicts *overall* growth in the outcome. However, by examining the effect of social anxiety on each individual growth parameter (with all growth parameters included in the model), we were able to identify models in which measures of social anxiety were significant predictors of some of the growth parameters (e.g., just the quadratic slope) but not all. This approach may be particularly useful for the purposes of the present study, as individuals with high and low levels of social anxiety may differ in their pre-task levels of arousal but show similar linear or quadratic growth in their physiological responses over time, or vice versa.

In the final fitted growth models, residual variances for the repeated measures of the physiological outcome variable were allowed to covary across one time point to improve model fit. Growth models in which social anxiety was a significant predictor of at least one growth parameter were plotted to illustrate the fitted trajectories of prototypical preadolescents at high (+1SD) and low (-1SD) levels of either context-specific or global social anxiety. Fit statistics for these models can be found in Table 5, and corresponding unstandardized coefficients are presented in Tables 6 and 7.

Heart Rate

No significant correlations were observed between global or context-specific social anxiety and heart rate variables (see Table 1). However, females demonstrated higher overall heart rates across the lab protocol, and greater heart rate reactivity to the peer rebuff period. Similarly, income was positively associated with heart rate reactivity to the peer evaluation period (i.e., the conversation task) at the bivariate level, and older preadolescents demonstrated greater heart rate reactivity to and recovery from the peer evaluation period. An unconditional linear growth model for the *full lab protocol* fit the data relatively poorly (χ^2/df = 7.12, TLI = .84, RMSEA = .22), with adequate fit indicated by RMSEA less than .10 and a χ^2/df ratio of less than 5 (Keiley, Keller, & El-Sheikh, 2009). The addition of a quadratic term provided a significant improvement in fit ($\Delta\chi^2$ = 50.25, Δdf = 4, Critical χ^2 = 9.49), thus, growth in heart rate across the full lab protocol was modeled as quadratic (χ^2/df = 6.91, TLI = .84, RMSEA = .22). Parameter estimates of mean growth levels and the variances for each growth factor can be found in Table 8. On average, preadolescents exhibited increases in heart rate in response to the social stressors and decreases in heart rate following social stress. Significant variance was observed in the growth parameters which allowed us to examine context-specific and global social anxiety as predictors of each parameter.

Across the full lab protocol, context-specific anxiety was associated with the linear (β = .33, *B* = .16, *S.E.* = .07, *p* = .027) and quadratic (β = -.41, *B* = -.01, *S.E.* = .01, *p* = .029) slopes for heart rate, but not the intercept (β = -.06, *B* = -.71, *S.E.* = 1.05, *p* = .497), after controlling for sex, age, and income (see Table 6). Context-specific anxiety explained 7.5% of the variance in the linear slope for heart rate, and 13.8% in the quadratic slope beyond control variables. The final fitted growth model, which allowed residual variances for repeated measures of heart rate to

covary across one time period, provided an excellent fit to the data (see Table 5). As is shown in Figure 1, preadolescents with higher context-specific anxiety exhibited increases in heart rate in response to the social stressors (M_{linear} = .36, *S.E.* = .20, p = .075) and heart rate recovery following social stress ($M_{quadratic}$ = -.03, *S.E.* = .01, p = .022). In contrast, preadolescents who reported lower context-specific anxiety demonstrated a very stable heart rate pattern across the lab protocol (M_{linear} = -.00, *S.E.* = .19, p = .984; $M_{quadratic}$ = -.01, *S.E.* = .01, p = .572). Global social anxiety, on the other hand, was not significantly associated with the intercept (β = .02, B = .28, *S.E.* = 1.51, p = .854), linear slope (β = -.03, B = -.02, *S.E.* = .12, p = .857), or quadratic slope for heart rate (β = -.02, B = .00, *S.E.* = .01, p = .911) after controlling for sex, age, and income.

At the stage-specific level of analysis, an unconditional quadratic model provided a poor fit to the data for the *first stage* of social stress (χ^2/df = 14.12, TLI = .80, RMSEA = .33), but the addition of a quadratic term led to an improvement in fit over an unconditional linear model ($\Delta\chi^2$ = 69.35, Δdf = 4, Critical χ^2 = 9.49). On average, preadolescents exhibited increases in heart in response to the peer evaluation period, and heart rate recovery following the stressor. However, significant variance was only observed in the intercept for heart rate during this stage (see Table 8). Thus, the linear and quadratic slopes could not be predicted by the substantive predictor variables. No significant findings emerged for either context-specific or global social anxiety during this stage after controlling for relevant demographic variables. To preserve space, these null results are not presented.

During the *second stage* of social stress, an unconditional quadratic model provided a borderline acceptable fit (i.e., χ^2/df < 5 but RMSEA > .10) to the data (χ^2/df =3.54, TLI = .96, RMSEA = .15), and the addition of a quadratic term led to a significant improvement in fit over

the unconditional linear model ($\Delta\chi^2 = 27.42$, $\Delta df = 4$, Critical $\chi^2 = 9.49$). Significant mean-level growth in heart rate was not observed during the second stage of social stress; however, significant variance existed in all growth parameters (see Table 8). Controlling for the effects of sex, context-specific anxiety was associated with the quadratic slope for heart rate at the non-significant trend level (β = -.22, B = -.04, *S.E.* = .02, p = .065), explaining an additional 4.5% of the variance in the quadratic slope (see Table 6). However, context-specific anxiety was not significantly related to the intercept (β = -.07, B = -.74, *S.E.* = .92, p = .423) or the linear slope for heart rate (β = .18, B = .25, *S.E.* = .16, p = .121; see Table 5 for final model fit statistics). Despite the trend-level effect of context-specific anxiety on the quadratic slope for heart rate, we chose to present and interpret these results because predicting variance in a quadratic term is often quite difficult and trend-level effects may still be meaningful given our relatively small sample size. Nevertheless, this finding should be interpreted cautiously.

Follow-up analyses revealed that preadolescents with higher levels of context-specific anxiety exhibited increases in heart rate during the peer rebuff period (see Figure 2; M_{linear} = .91, *S.E.* = .50, p = .068) and a decrease in heart rate following social stress ($M_{quadratic}$ = -.11, *S.E.* = .05, p = .04). In contrast, preadolescents with lower levels of context-specific anxiety showed no growth in heart rate across the second stage of social stress (M_{linear} = -.00, *S.E.* = .19, p = .984; $M_{quadratic}$ = -.01, *S.E.* = .01, p = .572). These results are quite consistent with the overall pattern of heart rate responses identified across the full lab protocol, which suggest that preadolescents with higher context-specific anxiety may exhibit stronger heart rate responses to, and greater recovery from, social stress than those who are lower in context-specific anxiety. Counter to these findings, global social anxiety was not associated with the intercept (β = -.07, B = -.91, *S.E.* = .1.31, p = .488), linear slope (β = .13, B = .24, *S.E.* = .24, p = .318), or quadratic slope for heart

rate (β = -.16, *B* = -.04, *S.E.* = .03, *p* = .194) during the second stage of social stress when controlling for sex. Thus, context-specific anxiety is uniquely related to heart rate responses in the present study.

Skin Conductance

As shown in Table 2, global and context-specific measures of social anxiety were not correlated with skin conductance variables. However, older children had higher overall skin conductance levels, while African American preadolescents exhibited reduced SCL reactivity to the peer evaluation period and lower skin conductance levels across the lab protocol. The latter finding is consistent with prior research demonstrating higher skin conductance levels among European Americans relative to African Americans (El-Sheikh, Keiley, & Hinnant, 2010). An unconditional quadratic growth model for the *full lab protocol* fit the data poorly (χ^2/df = 11.9, TLI = .74, RMSEA = .30); however, the addition of a quadratic term provided a significant improvement in fit over an unconditional linear model ($\Delta\chi^2$ = 327.93, Δdf = 4, Critical χ^2 = 9.49). At the mean level, preadolescents exhibited increases in SCL in response to the social stressors and a decrease in SCL following social stress. Significant variance was observed in the growth parameters which allowed us to examine context-specific and global social anxiety as predictors of each parameter (see Table 8).

Global social anxiety was significantly associated with the quadratic slope for SCL across the full lab protocol (β = .26, *B* = .01, *S.E.* = .01, *p* = .034), but not the intercept (β = -.05, *B* = -.24, *S.E.* = .45, *p* = .589) or the linear slope (β = -.11, *B* = -.04, *S.E.* = .05, *p* = .335) after controlling for the effects of sex, age, and ethnicity (see Table 7). Global social anxiety explained 6.3% of the variance in the quadratic slope for SCL beyond control variables (see Table 5 for final model fit statistics). As seen in Figure 3, preadolescents with higher levels of

global social anxiety exhibited slightly reduced SCL reactivity to the social stressors (M_{linear} = .64, *S.E.* = .08, $p < .001$) and blunted SCL recovery following social stress ($M_{quadratic}$ = -.04, *S.E.* = .01, $p < .001$) compared to children with lower global social anxiety (M_{linear} = .71, *S.E.* = .09, $p < .001$; $M_{quadratic}$ = -.05, *S.E.* = .01, $p < .001$). In contrast to these results, context-specific anxiety was not significantly associated with the intercept (β = .05, *B* = .17, *S.E.* = .34, p = .610), linear slope (β = .05, *B* = .02, *S.E.* = .03, p = .648), or quadratic slope for heart rate (β = -.08, *B* = -.01, *S.E.* = .00, p = .510) after controlling for sex, age, and ethnicity.

At the stage-specific level of analysis, an unconditional quadratic model provided a poor fit to the data for the *first stage* of social stress (χ^2/df = 24.8, TLI = .64, RMSEA = .44), however the addition of a quadratic term led to an improvement in fit over an unconditional linear model ($\Delta\chi^2$ = 136.33, Δdf = 4, Critical χ^2 = 9.49). On average, preadolescents exhibited increases in SCL during the peer evaluation period that decelerated or tapered off following the stressor. Significant variance was also observed in each of the growth parameters during this stage (see Table 8). Controlling for the effects of age and ethnicity, global social anxiety was associated with the linear (β = -.26, *B* = -.18, *S.E.* = .10, p = .07) and quadratic slopes for SCL (β = .36, *B* = .02, *S.E.* = .01, p = .067) at the non-significant trend level (see Table 7), explaining an additional 6.8% of the variance in the linear slope for SCL and 13.1% of the variance in the quadratic slope (see Table 5 for final model fit statistics). However, global social anxiety was not a significant predictor of the intercept for SCL during the first stage of social stress (β = -.01, *B* = -.02, *S.E.* = .47, p = .971). Although these effects are indeed non-significant (at p <.05), we chose to present and interpret these results for the reasons described in previous sections. However, these findings should also be interpreted cautiously.

Follow-up analyses revealed that the skin conductance levels of preadolescents with low

global social anxiety increased during the peer evaluation period and began to taper off during the waiting period (see Figure 4; M_{linear} = 1.20, *S.E.* = .22, $p < .001$; $M_{quadratic}$ = -.08, *S.E.* = .02, $p < .001$). In contrast, preadolescents with higher levels of global social anxiety exhibited a continuous increase in SCL across the first stage of social stress, with limited reductions in the slope of SCL during the waiting period (M_{linear} = .80, *S.E.* = .20, $p < .001$; $M_{quadratic}$ = -.04, *S.E.* = .02, $p = .068$). Thus, preadolescents who are lower in global social anxiety may start to recover from social stress during the waiting period, while those with higher levels of global social anxiety may continue to increase in their sympathetic arousal. Context-specific anxiety, on the other hand, was not a significant predictor of the intercept ($\beta = .06$, $B = .22$, *S.E.* = .35, $p = .523$), linear slope ($\beta = -.02$, $B = -.01$, *S.E.* = .08, $p = .879$), or quadratic slope for SCL ($\beta = .03$, $B = .00$, *S.E.* = .01, $p = .835$) during the first stage of social stress, after controlling for age and ethnicity.

During the *second stage* of social stress, an unconditional quadratic model fit the data relatively poorly (χ^2/df =8.37, TLI = .90, RMSEA = .25), though the addition of a quadratic term provided a significant improvement in fit over an unconditional linear model ($\Delta\chi^2 = 55.75$, $\Delta df = 4$, Critical $\chi^2 = 9.49$). Significant mean-level growth in SCL was observed during the second stage of social stress, such that preadolescents exhibited increases in SCL in response to the peer rebuff period that decelerated during the recovery period. Significant variance was observed in the intercept and quadratic slope for SCL during the second stage of social stress, but not the linear slope (see Table 8). Thus, only the intercept and quadratic slope could be predicted by the substantive predictor variables, and the variance of the linear slope was constrained to zero in subsequent models. Controlling for the effects of age and ethnicity, global social anxiety was significantly associated with the quadratic slope for SCL ($\beta = .26$, $B = .01$, *S.E.* = .02, $p = .018$) but not the intercept ($\beta = -.05$, $B = -.27$, *S.E.* = .49, $p = .588$; see Table 7), explaining an

additional 6.5% of the variance in the quadratic slope beyond control variables and main effects (see Table 5 for final model fit statistics).

Consistent with the SCL growth models described above, preadolescents with higher levels of global social anxiety showed continuous increases in SCL across the second stage of social stress and blunted SCL recovery following the peer rebuff period (see Figure 5; M_{linear} = .17, *S.E.* = .05, $p < .001$; $M_{quadratic}$ = -.01, *S.E.* = .01, $p = .17$), whereas those with lower global social anxiety exhibited increases in SCL during the peer rebuff period and much larger decreases in SCL (i.e., greater recovery) following the stressor (M_{linear} = .17, *S.E.* = .05, $p < .001$; $M_{quadratic}$ = -.03, *S.E.* = .01, $p = .001$). Also consistent with prior models, context-specific anxiety was not a significant predictor of the intercept ($\beta = .04$, $B = .16$, *S.E.* = .36, $p = .666$) or quadratic slope for SCL ($\beta = .01$, $B = .00$, *S.E.* = .01, $p = .933$) during the second stage of social stress, after controlling for age and ethnicity.

Thus, in the present study, SCL responses were uniquely related to the global measure of social anxiety just as heart rate responses were exclusively related to the context-specific measure of anxiety. Across growth models assessing preadolescents' skin conductance responses, global social anxiety was related to slightly reduced SCL reactivity to stress and, perhaps more importantly, blunted SCL recovery following social stress. Each model showed that preadolescents with higher levels of global social anxiety exhibited continuous increases in SCL, even after the cessation of social stressors, with limited recovery in SCL during waiting and recovery periods.

Respiratory Sinus Arrhythmia

No significant correlations were observed between global or context-specific social anxiety and respiratory sinus arrhythmia (RSA) variables (see Table 3). However, older

preadolescents had lower RSA across most of the lab protocol, particularly during the baseline and rebuff periods. No significant findings emerged for growth models examining RSA responses across the *full lab protocol*, or during the *second stage* of social stress. To preserve space, only the growth models examining RSA responses during the *first stage* of social stress are reported and interpreted, and null results are not presented.

An unconditional quadratic model provided a borderline acceptable fit to the data (i.e., $\chi^2/df < 5$ but RMSEA > .10) for the *first stage* of social stress ($\chi^2/df = 4.64$, TLI = .86, RMSEA = .17), and the addition of a quadratic term led to a significant improvement in fit over an unconditional linear model ($\Delta\chi^2 = 39.73$, $\Delta df = 4$, Critical $\chi^2 = 9.49$). Preadolescents showed no mean-level change in RSA during this stage of social stress, however significant variance was observed in each of the growth parameters (see Table 8). Context-specific anxiety was associated with the linear (β = -.21, *B* = -.06, *S.E.* = .04, *p* = .073) and quadratic slopes for RSA (β = .21, *B* = .01, *S.E.* = .01, *p* = .060) at the non-significant trend level (see Table 6), explaining an additional 4.3% of the variance in the linear slope and 4.5% of variance in the quadratic slope (see Table 5 for final model fit statistics), but was not associated with the intercept for RSA (β = .10, *B* = .09, *S.E.* = .09, *p* = .342).

Follow-up analyses revealed that preadolescents with higher levels of context-specific anxiety exhibited reductions in RSA (i.e., vagal withdrawal) in response to the peer evaluation period and subsequent increases in RSA during the waiting period (see Figure 6; M_{linear} = -.07, *S.E.* = .05, *p* = .235; $M_{quadratic}$ = .01, *S.E.* = .01, *p* = .254). In contrast, those with lower levels of context-specific anxiety showed increases in RSA (i.e., vagal augmentation) in response to the peer evaluation period and decreases in RSA during the waiting period (M_{linear} = .07, *S.E.* = .06, *p* = .180; $M_{quadratic}$ = -.01, *S.E.* = .01, *p* = .133). However, these findings should be interpreted

cautiously given that the observed effects are indeed non-significant trends. Furthermore, though analyses indicate that growth in RSA during this stage differs at lower and higher levels of context-specific anxiety, the conditional linear and quadratic slopes are non-significant for preadolescents with high and low context-specific anxiety. Although it is not uncommon to find significant interactions with non-significant conditional slopes, this does make the interpretation of results slightly more difficult. Global social anxiety, on the other hand, was not a significant predictor of the intercept (β = .07, *B* = .08, *S.E.* = .13, *p* = .529), linear slope (β = .10, *B* = .04, *S.E.* = .04, *p* = .351), or quadratic slope for RSA (β = -.18, *B* = -.01, *S.E.* = .01, *p* = .212).

Pre-Ejection Period

No significant correlations were observed between global social anxiety and pre-ejection period (PEP) variables (see Table 4). However, higher levels of context-specific anxiety were correlated with greater reductions in PEP in response to the peer rebuff period (i.e., PEP shortening). In addition, ethnicity was positively related to average baseline PEP, sex was associated with greater reductions in PEP in response to the peer evaluation period, and income was related to greater reductions in PEP following the peer evaluation period at the bivariate level. No significant findings emerged for growth models examining PEP responses across the full lab protocol, or during either stage of social stress. To preserve space, these null results are not presented.

IV. DISCUSSION

The present study used growth modeling to examine whether changes in the physiological responses of preadolescents across a full lab protocol, and during two unique stages of social stress, were predicted by measures of context-specific and global social anxiety. Preadolescents' physiological responses were assessed in the context of a salient peer-evaluative

social stress protocol using four well-validated measures of ANS activity. Specifically, RSA was used as a measure of PNS responses, SCL as a measure of BIS activity, PEP as a measure of BAS activity, and HR as a measure of overall ANS activity.

Though inconsistencies exist, the literature on the psychophysiology of social anxiety suggests that high levels of social anxiety among children and adolescents may be characterized by autonomic inflexibility and blunted physiological reactivity to social stress, as well as elevated baseline levels of ANS arousal. Thus, we hypothesized that high levels of social anxiety would be associated with 1) higher HR, higher SCL, lower RSA, and longer PEP during pre-task periods, 2) blunted or reduced growth in physiological responses across the full lab protocol and during both stages of social stress for measures of HR, RSA, and PEP, and 3) no differences in growth in SCL across the full lab protocol and during the first stage of social stress (no hypotheses were offered for the second stage of social stress). In contrast to our hypotheses, neither context-specific nor global social anxiety was significantly associated with growth in PEP or initial levels for any of the physiological outcome variables. Context-specific anxiety was uniquely related to HR responses across the full lab protocol and during the second stage of social stress, as well as RSA responses during the first stage of social stress. Similarly, global social anxiety was uniquely related to SCL responses across the lab protocol and during both stages of social stress. Though results were inconsistent with our hypotheses and fit statistics for the final fitted growth models ranged from slightly less than adequate to good (see Table 5), each finding is deserving of additional attention.

In the present study, higher levels of context-specific anxiety, but not global social anxiety, were associated with more dynamic heart rate responses across the full lab protocol and during the second stage of social stress. Intriguingly, this is inconsistent with what was

hypothesized, as preadolescents with higher context-specific anxiety exhibited stronger heart rate responses to social stress and greater heart rate recovery compared to those with lower context-specific anxiety. Furthermore, this is in contrast to findings reported by Schmitz et al. (2013), who found blunted cardiac reactivity among socially anxious preadolescents using a global measure of social anxiety. However, though our results may provide evidence for more dynamic heart rate responses among preadolescents who report high levels of context-specific anxiety, they may also be consistent with a model of hyper-reactivity to social stress. It is worth noting that the social stressors used in the present study may elicit only mild-to-moderate stress responses. Thus, preadolescents with higher context-specific anxiety may be exhibiting a stronger HR response than is warranted given the demands of the lab protocol.

Preadolescents with higher context-specific anxiety also displayed greater increases in heart rate in response to the peer rebuff period specifically, which may support the notion that instances of peer rejection tend to be particularly stressful for individuals with high levels of social anxiety (Beidel & Turner, 2007). These individuals may view the peer rebuff period as being particularly challenging or aversive, and corresponding increases in ANS activity (i.e., HR) may facilitate appropriate social responses under mildly stressful circumstances. In contrast, preadolescents with lower levels of context-specific anxiety may not view the peer rebuff period, or the lab protocol as a whole, as particularly challenging or stressful, which may help to explain the limited increases in overall arousal observed during social stress.

Also of interest is that preadolescents with higher context-specific anxiety exhibited greater reductions in heart rate following social stress, which may indicate greater feelings of relief upon the cessation of social stressors compared to individuals with lower context-specific anxiety. These findings are contrary to existing evidence for slower heart rate recovery after

social stress among preadolescents with high social anxiety (e.g., Schmitz et al., 2011; 2013), though prior results were found using global or retrospective reports of social anxiety rather than context-specific measures. However, the present study failed to replicate these findings with a similar measure of global social anxiety, and found the opposite pattern of results using a context-specific measure of anxiety. Thus, it is possible that different patterns of heart rate responses may be specific to the type of stressor or measure of social anxiety that is used in a given study. On the other hand, observed effects were modest in size in the present study, and the heart rate responses of preadolescents with higher and lower levels of context-specific anxiety only differed by several beats per minute. Accordingly, an important direction for future research will be to distinguish the physiological responses that are associated with high context-specific anxiety from those that have been associated with global social anxiety.

A growing number of studies have also found evidence for increased sympathetic arousal among children with high levels of social anxiety, using measures of electrodermal responding (e.g., Asbrand, Blechert, Nitschke, Tuschen-Caffier, & Schmitz; 2016; Nikolić, de Vente, Colonessi, & Bögels, 2016; Schmitz et al., 2011) or other measures of SNS activity. In contrast to these findings and our hypotheses, the present study found no relationship between PEP responses or initial levels of SCL and either measure of social anxiety. In addition, we found evidence for slightly reduced skin conductance responses to social stress and reduced SCL recovery among preadolescents with higher global social anxiety compared to those with lower social anxiety. Though relevant research is sparse, there is some evidence that socially anxious individuals may have difficulties down-regulating their autonomic arousal following social stress (e.g., Schmitz et al., 2011; 2013). However, to our knowledge, this is the first study to observe reduced recovery among socially anxious preadolescents using a measure of skin conductance or

electrodermal responding, or to utilize a measure of PEP. Unfortunately, autonomic recovery and PEP responses have been largely overlooked and understudied in the extant literature on social anxiety. Consequently, research examining autonomic recovery in conjunction with baseline arousal and reactivity to stress may be particularly informative, as different aspects of physiological responses (e.g., reactivity or recovery) may be uniquely related to anxiety disorders and to social anxiety in particular. In addition, utilizing PEP as a measure of SNS activity that is complementary to SCL or electrodermal responses is an important direction for future research.

It is important to note that although SCL and PEP are both measures of the SNS, SCL is thought to reflect BIS activity whereas PEP may be a marker of BAS activity. Some have suggested that social anxiety may be characterized by high BIS and low BAS activation (Corr, 2002; Kimbrel, 2008), and this has generally been supported in studies using self-reports of BIS/BAS sensitivity (e.g., Kimbrel et al., 2012; Morgan et al., 2009). However, results obtained using corresponding physiological measures are in opposition to these findings, and are consistent with the notion that self-report and physiological measures of BIS and BAS activity are largely independent from one another (Brenner, Beauchaine, Sylvers, 2005). Thus, self-reports of behaviors that are associated with BIS or BAS sensitivity may be more strongly related to social anxiety than corresponding physiological responses, though this claim needs to be tested directly among socially anxious individuals before definitive conclusions can be drawn.

RSA results were partially consistent with heart rate analyses, but did not support the notion that anxiety disorders, and social anxiety in particular, may be characterized by abnormal parasympathetic activity. Though several studies have found evidence for lower basal RSA or blunted parasympathetic reactivity to social stress (e.g., Schmitz et al., 2011; 2013), global social

anxiety was not related to initial levels or growth in RSA in the present study, which is in line with research suggesting that parasympathetic activation may not be as strongly related to social anxiety as previously thought (e.g., Asbrand et al., 2016; Gerlach, Wilhelm, & Roth, 2003). In addition, in the present study higher context-specific anxiety was related to greater vagal withdrawal in response to the peer evaluation period, which was inconsistent with our hypotheses. However, this result provides additional support for the argument that preadolescents who were high in context-specific anxiety might have demonstrated stronger ANS responses than were warranted given the mild-to-moderately stress inducing nature of the current lab protocol. In contrast, those who reported lower context-specific anxiety actually exhibited increases in RSA (i.e., vagal augmentation) in response to the peer evaluation period, which is thought to support attentional focus and spontaneous social engagement behaviors (Porges, 2007). Although additional studies have observed greater parasympathetic reactivity among socially anxious individuals (e.g., Stein, Asmundson, & Chartier, 1994), the general consensus seems to be that social anxiety may be related to lower basal RSA and blunted parasympathetic reactivity, particularly in the context of social stress. Thus, additional research is needed to clarify the relationship between various measures of social anxiety and parasympathetic responses.

In addition to its contributions, several limitations of the present study should be noted. First, although growth modeling may be particularly useful in examining the dynamic physiological responses of individuals across several stressors or an entire lab protocol, latent growth modeling is typically best suited for larger samples (Preacher, Wichman, Briggs, & MacCallum, 2008). Consequently, some of the trend-level effects observed in the present study may be a product of limited power to detect small to moderate effects using complex growth

analyses. In support of this argument, effects sizes observed across analyses ranged from small to moderate which is typical in studies with intensive methods (e.g., physiological data, observational data) and consistent with empirical evidence suggesting that self-reports of emotion are often only modestly associated with physiological measures (Evers et al., 2014; Hollenstein & Lanteigne, 2014). Nevertheless, all trend-level results should be interpreted cautiously and future research should work to replicate and extend these findings using larger samples.

Second, in the present study the baseline measurements used in growth models for the full lab protocol and during the first stage of social stress were likely influenced by some level of anticipatory stress. Accordingly, an important direction for future research will be to utilize baseline measures that are devoid of anticipatory stress. However, this may prove to be difficult as even the presence of an unfamiliar person in a more ecologically valid setting (e.g., inside the home) may influence physiological measures to some degree.

It should also be noted that all analyses were cross-sectional and thus conclusions regarding causality or the direction of association(s) among variables cannot be made. Longitudinal analyses examining the influence of physiological responses on the development of social anxiety over time may be particularly informative, as it is currently unclear if observed physiological differences are an antecedent or a consequence of anxiety disorders, or if there is a bidirectional relationship between anxiety disorders and physiological responses over time. In addition, although the laboratory-based peer-evaluative stress protocol was designed to be ecologically valid (e.g., face-to-face interpersonal interaction, developmentally salient stressor), adaptive responses to peer-evaluative stress in the laboratory may not generalize to a natural peer environment. Research examining potential sex or ethnic group differences in the relationship

between social anxiety and physiological responses will also be an important, as there is some evidence that females may view social situations as particularly important during this developmental period relative to males (Rose & Rudolph, 2006), and may differ from males in certain aspects of their physiology at various points in the lifespan (e.g., Greaves-Lord et al., 2010; Koenig & Thayer, 2016).

The current study also utilized data from a community sample and findings do not necessarily generalize to preadolescents with clinical levels of social anxiety. However, the mean total score on the SAS-A (i.e., global social anxiety) in the current sample was slightly higher than the mean scores reported for community samples in previous studies (Epkins, 2002; Flanagan, Erath, & Bierman, 2008; Inderbitzen-Nolan & Walters, 2000; Morris & Masia, 1998), and 29.5% of the sample scored above the approximate clinical threshold of 50 on the SAS-A, which is used to identify children and adolescents likely to manifest clinically significant social anxiety (La Greca, 1999). Thus, the present study represents a wide range of social anxiety levels.

Finally, additional research is needed to determine if similar results would be observed in studies that use non-social tasks, or in the context of social stress tasks that are more-anxiety provoking and elicit larger physiological responses. An important direction for future research is to compare the physiological responses of individuals with higher and lower social anxiety across a variety of different stressors (e.g., orthostatic tests, cognitive tasks, mild social stress tasks, and highly anxiety-provoking social stress tasks) within the same study. Though inconsistent findings in the broader literature on social anxiety may indeed be due to limitations or differences in the methods and analyses used across studies, they may also point to the need for a model of response specificity among socially anxious individuals. Perhaps observed

differences in physiological responses are dependent upon the type of task that is used in a given study, similar to what was found in a study comparing ANS reactivity among patients with current or remitted depression and anxiety to healthy controls (Hu, Lamers, de Geus, & Penninx, 2016). For example, as a form of anxiety that is specifically social, it is possible that differences in the physiological responses of highly socially anxious and less anxious individuals may be more consistently observed in the context of social stress than in response to cognitive or non-social tasks.

Furthermore, though social anxiety is a relatively context-specific anxiety disorder (i.e., limited to social contexts), considerable variability exists in the social situations that individuals with social anxiety identify as stressful or anxiety-provoking. Accordingly, individuals who report experiencing greater social anxiety in situations that are more closely related to the stressor or task that is used in a study may show patterns of physiological responding that are different from those who score high on retrospective or global measures of social anxiety. Indeed, in the present study preadolescents who reported higher levels of context-specific anxiety differed in their physiological responses from those who reported high levels of global social anxiety. In fact, those with high context-specific anxiety displayed physiological responses that were more consistent with models of hyper-reactivity to stress than models of autonomic inflexibility. Measures of context-specific anxiety may better assess more normative experiences of anxiety, whereas measures of global social anxiety may better assess more problematic anxiety. Indeed, we recently found that lower teacher-reported social competence is correlated with global social anxiety but not context-specific anxiety in the same sample (Kaeppler & Erath, 2016). Similarly, in the present study context-specific anxiety was associated with HR reactivity and recovery, a commonly-reported sensation in social evaluation situations. In

contrast, global social anxiety, which is less normative because it occurs across time and familiar and unfamiliar social situations, was not associated with high HR reactivity, consistent with the lack of evidence for autonomic hyper-reactivity among highly socially anxious children and adolescents.

There is also evidence for subgroups of socially anxious individuals (e.g., Erath et al., 2012; Gazelle, 2008; Kaeppler & Erath, 2016), which means that the physiological responses of individuals who score high on the same measure of social anxiety may differ according to another factor such as levels of peer victimization or coping strategies. For example, Erath et al. (2012) found that social anxiety was associated with heightened cardiac activity (i.e., low RSA and high HR) for preadolescents who reported high levels of peer victimization but not for those who reported lower levels of peer victimization. Therefore, a more nuanced psychophysiological model that considers additional individual attributes, social context, and differences in the way that social anxiety is assessed may be critical to understanding the etiology and development of social phobia and non-clinical social anxiety.

To our knowledge, this is the first study to use a growth modeling approach to examine the dynamic physiological responses of socially anxious preadolescents in the context of social stress. Growth modeling analyses may allow researchers to examine the overall pattern of physiological responses across a full lab protocol or in response to specific stressors, and may complement MANOVA's or regression analyses that have typically been used in previous studies. If results of the present study are replicated and extended, theoretical and clinical implications are possible. Research along these lines may inform theoretical models of context-dependent differences in the physiological responses of socially anxious individuals, or individuals with anxiety disorders more generally. In addition, findings from the present study

may elucidate patterns of physiological responses to stress that are helpful in identifying children and adolescents with social anxiety. Given the link between ANS responses and social behavior that has been described by a number of authors, a logical next step may be to study the impact of abnormal physiological responses on social behavior among anxious individuals in greater detail. By applying information from nuanced psychophysiological studies of social anxiety to studies of corresponding social behavior, future research may be able to identify specific aspects of physiological stress responses that undermine important social behaviors and negatively influence the lives of individuals with social anxiety.

References

Alvares, G. A., Quintana, D. S., Kemp, A. H., Van Zwieten, A., Balleine, B. W., Hickie, I. B., & Guastella, A. J. (2013). Reduced heart rate variability in Social Anxiety Disorder: Associations with gender and symptom severity. *Plos ONE*, 8(7), 1-9.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: American Psychiatric Publishing.

Anderson, E. R., & Hope, D. A. (2009). The relationship among social phobia, objective and perceived physiological reactivity, and anxiety sensitivity in an adolescent population. *Journal of Anxiety Disorders*, 23(1), 18-26.

Anderson, E. R., Veed, G. J., Inderbitzen-Nolan, H. M., & Hansen, D. J. (2010). An evaluation of the applicability of the tripartite constructs to social anxiety in adolescents. *Journal of Clinical Child and Adolescent Psychology*, 39(2), 195-207.

Asbrand, J., Blechert, J., Nitschke, K., Tuschen-Caffier, B., & Schmitz, J. (2016). Aroused at home: Basic autonomic regulation during orthostatic and physical activation is altered in children with social anxiety disorder. *Journal of Abnormal Child Psychology*. Advance online publication. doi:10.1007/s10802-016-0147-7

Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*, 13(2), 183-214.

Beidel, D. C. (1991). Social phobia and overanxious disorder in school-age children. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30(4), 545-552.

Beidel, D. C., Turner, S. M., & Dancu, C. V. (1985). Physiological, cognitive and behavioral aspects of social anxiety. *Behaviour Research and Therapy*, 23(2), 109-117.

Beidel, D. C., & Turner, S. M. (2007). *Shy children, phobic adults: Nature and treatment of social phobia.* Washington, D.C.: American Psychological Association.

Berntson, G. G., Bigger, J. T., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, 34, 623–648.

Berntson, G. G., & Cacioppo, J. T. (2004). Heart rate variability: Stress and psychiatric conditions. In M. Malik & A. J. Camm (Eds.), *Dynamic electrocardiography* (pp.57-64). New York, NY: Blackwell.

Bertson, G. G., Quigley, K. S., & Lozano, D. (2007). Cardiovascular psychophysiology. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bernston (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 182-210). New York, NY: Cambridge University Press.

Bradley, M. M., & Lang, P. J. (2007). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary, & G. G. Bernston (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 581-607). New York, NY: Cambridge University Press.

Brenner, S. L., Beauchaine, T. P., & Sylvers, P. D. (2005). A comparison of psychophysiological and self-report measures of BAS and BIS activation. *Psychophysiology*, 42(1), 108-115.

Chavira, D. A., Stein, M. B., Bailey, K., & Stein, M. T. (2004). Child anxiety in primary care: Prevalent but untreated. *Depression and Anxiety*, 20(4), 155-164.

Copeland, W. E., Wolke, D., Lereya, S. T., Shanahan, L., Worthman, C., & Costello, E. J. (2014). Childhood bullying involvement predicts low-grade systemic inflammation into adulthood. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 111(21), 7570-7575.

Corr, P. J. (2002). J. A. Gray's reinforcement sensitivity theory: Tests of the joint subsystems hypothesis of anxiety and impulsivity. *Personality and Individual Differences,* 33(4), 511-532.

Costanzo, P., Miller-Johnson, S., Wencel, H., & March, J., (1995). *Anxiety disorders in children and adolescents.* (1st ed., pp. 82-108). New York, NY, US: Guilford Press.

Cruz, J. E., Emery, R. E., & Turkheimer, E. (2012). Peer network drinking predicts increased alcohol use from adolescence to early adulthood after controlling for genetic and shared environmental selection. *Developmental Psychology,* 48(5), 1390-1402.

Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry,* 47(2), 85-95.

Eckman, P. S., & Shean, G. D. (1997). Habituation of cognitive and physiological arousal and social anxiety. *Behaviour Research and Therapy,* 35(12), 1113-1121.

Edelmann, R. J., & Baker, S. R. (2002). Self-reported and actual physiological responses in social phobia. *British Journal of Clinical Psychology,* 41(1).

El-Sheikh, M., Keiley, M., & Hinnant, J. B. (2010). Developmental trajectories of skin conductance level in middle childhood: Sex, race, and externalizing behavior problems as predictors of growth. *Biological Psychology,* 83(2), 116-124.

Erath, S. A., & Tu, K. M. (2014). Peer stress in preadolescence: Linking physiological and coping responses with social competence. *Journal of Research on Adolescence,* 24, 757-771.

Erath, S. A., Flanagan, K. S., & Bierman, K. L. (2007). Social anxiety and peer relations in early adolescence: Behavioral and cognitive factors. J*ournal of Abnormal Child Psychology,* 35, 405–416.

Erath, S. A., Tu, K. M., & El-Sheikh, M. (2012). Socially anxious and peer-victimized preadolescents: "Doubly primed" for distress? *Journal of Abnormal Child Psychology,* 40, 837-848.

Evers, C., Hopp, H., Gross, J. J., Fischer, A. H., Manstead, A. R., & Mauss, I. B. (2014). Emotion response coherence: A dual-process perspective. *Biological Psychology,* 9, 843-849.

Friedman, B. H. (2007). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology,* 74(2), 185-199.

Gazelle, H. (2008). Behavioral profiles of anxious solitary children and heterogeneity in peer relations. *Developmental Psychology,* 44, 1604-1624.

Gerlach, A. L., Mourlane, D., & Rist, F. (2004). Public and private heart rate feedback in social phobia: A manipulation of anxiety visibility. *Cognitive Behaviour Therapy*, 33, 36-45.

Gerlach, A. L., Wilhelm, F. H., Gruber, K., & Roth, W. T. (2001). Blushing and physiological arousability in social phobia. *Journal of Abnormal Psychology*, 110(2), 247-258.

Gerlach, A. L., Wilhelm, F. H., & Roth, W. T. (2003). Embarrassment and social phobia: The role of parasympathetic activation. *Journal of Anxiety Disorders,* 17(2), 197-210.

Gray, J. A. (1987). Perspectives on anxiety and impulsivity: A commentary. *Journal of Research in Personality,* 21, 493-509.

Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system* (2nd ed.). New York: Oxford University Press.

Greaves-Lord, K., Tulen, J., Dietrich, A., Sondeijker, F., van Roon, A., Oldehinkel, A., & Huizink, A. (2010). Reduced autonomic flexibility as a predictor for future anxiety in girls from the general population: The TRAILS study. *Psychiatry Research,* 179(2), 187–193.

Greco, L. A., & Morris, T. L. (2005). Factors influencing the link between social anxiety and peer acceptance: Contributions of social skills and close friendships during middle childhood. *Journal of Behavior Therapy,* 37, 197–205.

Grossman, P., Wilhelm, F. H., Kawachi, I., & Sparrow, D. (2001). Gender differences in psychophysiological responses to speech stress among older social phobics: Congruence and incongruence between self-evaluative and cardiovascular reactions. *Psychosomatic Medicine,* 63(5), 765-777.

Hofmann, S. G., Newman, M. G., Ehlers, A., & Roth, W. T. (1995). Psychophysiological differences between subtypes of social phobics. *Journal of Abnormal Psychology,* 104, 224-231.

Hollenstein, T., & Lanteigne, D. (2014). Models and methods of emotional concordance. *Biological Psychology*, 981-985.

Hu, M. X., Lamers, F., de Geus, E., & Pennix, B. (2016). Differential autonomic nervous system reactivity in depression and anxiety during stress depending on type of stressor. *Psychosomatic Medicine.* Advance online publication. doi: 10.1097/PSY.0000000000000313

Hubbard, J. A. (2005). Eliciting and measuring children's anger in the context of their peer interactions: Ethical considerations and practical guidelines. *Ethics and Behavior*, 15, 247–258.

Kashdan, T. B., & Herbert, J. D. (2001). Social anxiety disorder in childhood and adolescence: Current status and future directions. *Clinical Child and Family Psychology Review,* 4(1), 37-61.

Keiley, M. K., Keller, P. S., & El-Sheikh, M. (2009). Effects of physical and verbal aggression, depression, and anxiety on drinking behavior of married partners: A prospective and retrospective longitudinal examination. *Aggressive Behavior,* 35(4), 296-312.

Kimbrel, N. A. (2008). A model of the development and maintenance of generalized social phobia. *Clinical Psychology Review,* 28(4), 592-612.

Kimbrel, N. A., Nelson-Gray, R. O., & Mitchell, J. T. (2012). BIS, BAS, and bias: The role of personality and cognitive bias in social anxiety. *Personality & Individual Differences,* 52(3), 395-400.

Kingery, J. N., Erdley, C. A., Marshall, K. C., Whitaker, K. G., & Reuter, T. R. (2010). Peer experiences of anxious and socially withdrawn youth: An integrative review of the developmental and clinical literature. *Clinical Child and Family Psychology Review,* 13(1), 91-128.

Klumbies, E., Braeuer, D., Hoyer, J., & Kirschbaum, C. (2014). The reaction to social stress in Social Phobia: Discordance between physiological and subjective parameters. *Plos ONE,* 9(8), 1-11.

Koenig, J., & Thayer, J. F. (2016). Sex differences in healthy human heart rate variability: A meta-analysis. *Neuroscience and Biobehavioral Reviews,* 64, 288-310.

Kramer, M., Seefeldt, W. L., Heinrichs, N., Tuschen-Caffier, B., Schmitz, J., Wolf, O. T., & Blechert, J. (2012). Subjective, autonomic, and endocrine reactivity during social stress in children with Social Phobia. *Journal of Abnormal Child Psychology,* 40(1), 95-104.

Kushner, M. G., Sher, K. J., & Beitman, B. D. (1990). The relation between alcohol problems and the anxiety disorders. *American Journal of Psychiatry,* 147, 685-695.

La Greca, A. M., & Harrison, H. M. (2005). Adolescent peer relations, friendships, and romantic relationships: Do they predict social anxiety and depression? *Journal of Clinical Child and Adolescent Psychology,* 34(1), 49-61.

La Greca, A. M., & Lopez, N. (1998). Social anxiety among adolescents: Linkages with peer relations and friendships. *Journal of Abnormal Child Psychology,* 26, 83–94.

La Greca, A. M., & Stone, W. L. (1993). Social Anxiety Scale for Children-Revised: Factor structure and concurrent validity. *Journal of Clinical Child Psychology,* 22, 17–27.

Matthews, K. A., Manuck, S. B., & Saab, P. G. (1986). Cardiovascular responses of adolescents during a naturally occurring stressor and their behavioral and psychophysiological predictors. *Psychophysiology,* 23(2), 198-209.

Mauss, I. B., Wilhelm, F. H., & Gross, J. J. (2003). Autonomic recovery and habituation in social anxiety. *Psychophysiology,* 40(4), 648-653.

Mauss, I. B., Wilhelm, F. H., & Gross, J. J. (2004). Is there less to social anxiety than meets the eye? Emotion experience, expression, and bodily responding. *Cognition & Emotion,* 18(5), 631-62.

Miers, A. C., Blöte, A. W., Sumter, S. R., Kallen, V. L., & Westenberg, P. M. (2011). Subjective and objective arousal correspondence and the role of self-monitoring processes in high and low socially anxious youth. *Journal of Experimental Psychopathology,* 2, 531–550.

Morgan, B. E., van Honk, J., Hermans, E. J., Scholten, M. M., Stein, D. J., & Kahn, R. S. (2009). Gray's BIS/BAS dimensions in non-comorbid, non-medicated social anxiety disorder. *World Journal of Biological Psychiatry*, 10(4), 925-928.

Muthén, L. K., & Muthén, B. O. (1998-2012). *Mplus User's Guide* (7th Ed.). Los Angeles, CA: Muthén & Muthén.

Nikolić, M., de Vente, W., Colonnesi, C., & Bögels, S. M. (2016). Autonomic arousal in children of parents with and without social anxiety disorder: a high-risk study. *Journal of Child Psychology and Psychiatry.* Advance online publication. doi: 10.1111/jcpp.12563

Parker, J. G., Rubin, K. H., Price, J., & DeRosier, M. E. (1995). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti & D. Cohen (Eds.), *Developmental Psychopathology*: Vol. 2. Risk, disorder, and adaptation (pp. 96–161). New York: Wiley.

Patterson, M. L., & Ritts, V. (1997). Social and communicative anxiety: A review and meta-analysis. In B. R. Burleson (Ed.), *Communication yearbook 20* (pp. 262–303). Thousand Oaks, CA: Sage.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, 74(2), 116-143.

Preacher, K. J., Wichman, A. L., Briggs, N. E., & MacCallum, R. C. (2008). *Latent Growth Curve Modeling.* Thousand Oaks, CA: Sage

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, 35(8), 741-756.

Rubin, K. H., & Burgess, K. B. (2001). Social withdrawal and anxiety. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 407–434). New York: Oxford University Press.

Schmitz, J., Blechert, J., Kramer, M., Asbrand, J., & Tuschen-Caffier, B. (2012). Biased perception and interpretation of bodily anxiety symptoms in childhood social anxiety. *Journal of Clinical Child and Adolescent Psychology*, 41(1), 92-102.

Schmitz, J., Kramer, M., Tuschen-Caffier, B., Heinrichs, N., & Blechert, J. (2011). Restricted autonomic flexibility in children with social phobia. *Journal of Child Psychology and Psychiatry,* 52(11), 1203-121.

Schmitz, J., Tuschen-Caffier, B., Wilhelm, F., & Blechert, J. (2013). Taking a closer look: Autonomic dysregulation in socially anxious children. *European Child & Adolescent Psychiatry,* 22(10), 631-640.

Schneier, F. R., Johnson, J., Horning, C. D., Liebowitz, M. R., & Weissman, M. M. (1992). Social phobia: Comorbidity and morbidity in an epidemiology sample. *Archives of General Psychiatry*, 49, 282–288.

Sheppes, G., Catran, E., & Meiran, N. (2009). Reappraisal (but not distraction) is going to make you sweat: Physiological evidence for self-control effort. *International Journal of Psychophysiology*, 71, 91–96.

Siess, J., Blechert, J., & Schmitz, J. (2014). Psychophysiological arousal and biased perception of bodily anxiety symptoms in socially anxious children and adolescents: A systematic review. *European Child & Adolescent Psychiatry*, 23(3), 127-142.

Sigurdson, J., Wallander, J., & Sund, A. (2014). Is involvement in school bullying associated with general health and psychosocial adjustment outcomes in adulthood? *Child Abuse & Neglect*, 7(1), 173-178.

Somerville, L. H. (2013). The teenage brain: Sensitivity to social evaluation. *Current Directions in Psychological Science,* 22(2), 121-127.

Stein, M. B., Asmundson, G. G., & Chartier, M. (1994). Autonomic responsivity in generalized social phobia. *Journal of Affective Disorders,* 31(3), 211-221.

Stein, M. B., Tancer, M. E., Gelernter, C. S., Vittone, B. J., & Uhde, T. W. (1990). Major depression in patients with social phobia. *American Journal of Psychiatry,* 147, 637-639.

Stroud, L., Foster, E., Handwerger, K., Papandonatos, G. D., Granger, D., Kivlighan, K. T., & Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stress. *Development and Psychopathology,* 21, 47–68.

Stuart, J., & Jose, P. E. (2014). Is bullying bad for your health? The consequences of bullying perpetration and victimization in childhood on health behaviors in adulthood. *Journal of Aggression, Conflict and Peace Research,* 6(3), 185-195.

Sumter, S. R., Bokhorst, C. L., Miers, A. C., Van Pelt, J., & Westenberg, P. M. (2010). Age and puberty differences in stress responses during a public speaking task: Do adolescents grow more sensitive to social evaluation? *Psychoneuroendocrinology,* 35, 1510–1516.

Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders,* 61(3), 201-216.

Thibodeau, M. A., Gómez-Pérez, L., & Asmundson, G. G. (2012). Objective and perceived arousal during performance of tasks with elements of social threat: The influence of anxiety sensitivity. *Journal of Behavior Therapy and Experimental Psychiatry,* 43(3), 967-974.

Underwood, M. K. (2005). Observing anger and aggression among preadolescent girls and boys: Ethical dilemmas and practical solutions. *Ethics and Behavior,* 15, 235–245.

van den Bos, E., de Rooij, M., Miers, A. C., Bokhorst, C. L., & Westenberg, P. M. (2014). Adolescents' increasing stress response to social evaluation: Pubertal effects on cortisol

and alpha-amylase during public speaking. *Child Development,* 85, 220-236.

Vasey, M. W., & Daleiden, E. L. (1996). Information-processing pathways to cognitive interference in childhood. In I. G. Sarason, G. Pierce, & B. Sarason (Eds.), *Cognitive interference: Theory, methods, and findings* (pp. 117–138). Hillsdale, NJ: Erlbaum.

Vasey, M. W., El-Hag, N., & Daleiden, E. L. (1996). Anxiety and the processing of emotionally-threatening stimuli: Distinctive patterns of selective attention among high- and low-test-anxious children. *Child Development,* 67, 1173-1185.

Vernberg, E. M., Abwender, D. A., Ewell, K. K., & Beery, S. H. (1992). Social anxiety and peer relationships in early adolescence: A prospective analysis. *Journal of Clinical Child Psychology,* 21, 189–196.

Westenberg, P. M., Drewes, M. J., Goesdhart, A. W., Siebelink, B. M., & Treggers, P. D. A. (2004). A developmental analysis of self-reported fears in late childhood through mid-adolescence: Social evaluative fears on the rise? *Journal of Child Psychology and Psychiatry,* 45, 481-495.

Westenberg, P. M., Gullone, E., Bokhorst, C. L., Heyne, D.A, & King, N. J. (2007). Social evaluation fear in childhood and adolescence: Normative developmental course and continuity of individual differences. *British Journal of Developmental Psychology*, 25, 471–483.

Wittchen, H., Stein, M. B., & Kessler, R. C. (1999). Social fears and social phobia in a community sample of adolescents and young adults: Prevalence, risk factors, and co-morbidity. *Psychological Medicine,* 29(2), 309-323.

APPENDIX A

Literature Review

Description and prevalence of social anxiety

Anxiety disorders are among the most common psychiatric disorders experienced by children and adolescents. Social anxiety in particular is the most commonly diagnosed anxiety disorder in this age group, with lifetime prevalence rates of approximately 5-12% (Chavira, Stein, Bailey, & Stein, 2004; Kashdan & Herbert, 2001). Although descriptions of social anxiety disorder can be traced back to the writings of Hippocrates, formal diagnostic criteria for Social Anxiety Disorder – also known as Social Phobia – were not included in the *Diagnostic and Statistical Manual of Mental Disorders* until the publication of its third edition in the 1980's (Beidel & Turner, 2007). The *DSM-V* characterizes Social Phobia as "a marked and persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others" (American Psychiatric Association, 2013).

The age of onset for Social Phobia can be as early as 8 years of age (Chavira et al., 2004; Wittchen, Stein, & Kessler, 1999), illustrating the importance of studying social anxiety in child and adolescent samples. Though a variety of researchers have studied social anxiety in adult and clinical populations, relatively few studies have examined social anxiety in subclinical child or adolescent samples. The tendency to focus on clinical populations has drawbacks, as even in normative samples elevated levels of social anxiety in early adolescence have been associated with significant impairment in academic, social, and emotional functioning (Kingery et al., 2010). Furthermore, research conducted exclusively within clinical populations is limited in the information it can afford about the development of social anxiety from subclinical to clinical levels. Similarly, a focus on studying social anxiety exclusively in adulthood may also be

misguided, as rates of social anxiety peak in early to middle adolescence (Beidel & Turner, 2007), coinciding with increasing concerns of peer acceptance and social evaluation (Westenberg, Drewes, Goesdhart, Siebelink, & Treggers, 2004).

The intense discomfort experienced by those with social anxiety in various social contexts can have a strong negative impact on day-to-day functioning and may place anxious individuals at an increased risk for other socio-emotional problems that are associated with negative physical and mental health outcomes. For example, social anxiety has consistently been linked to an increased risk of peer victimization (Kingery et al., 2010), which is associated with a myriad of negative health outcomes. As concerns of peer evaluation increase in adolescence, negative peer experiences may become increasingly harmful, particularly for those with social anxiety (Erath, Tu, & El-Sheikh, 2012). Children who experience victimization during adolescence are at an increased risk of alcohol abuse (Cruz, Emery, & Turkheimer, 2012), depression (Stuart & Jose, 2014), low quality relationships with a spouse/partner (Sigurdson, Wallander, & Sund, 2014), and low-grade systemic inflammation (Copeland et al., 2014) in adulthood. Thus, social anxiety and subsequent negative social experiences in childhood and adolescence may set into motion physiological and psychological processes that influence health and well-being across the lifespan. Furthermore, adolescence may represent a developmental period during which individuals with social anxiety are particularly at risk for the development of social, emotional, and physical health problems that may persist well into adulthood.

Psychophysiological theories of anxiety/social anxiety

By utilizing increasingly sophisticated technologies as a means of observing and measuring psychophysiological responses, researchers have started to build a more detailed understanding of the various mechanisms and underlying pathways that influence physiological

functioning. Psychophysiological theories of social anxiety and anxiety more generally focus on the various components of the autonomic nervous system (ANS), and suggest that ANS functioning may influence various aspects of human behavior. Polyvagal Theory, for example, describes the potential link between autonomic functioning and social behavior (Porges, 2007). Polyvagal Theory posits that the ANS promotes adaptive behavioral responses to changes in environmental demands. Flexible ANS functioning allows an individual to quickly modify their behavior in response to stress, and return to or maintain homeostasis under non-threatening conditions (Porges, 2007).

The ANS is comprised of two distinct systems, the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). The PNS promotes growth and the maintenance of homeostasis, and is often referred to as the "rest and digest" or "feed and breed" system. Complementary to the PNS, the SNS facilitates mobilization of metabolic resources under stressful or threatening conditions, and is commonly referred to as the "fight or flight system" (Porges, 2007). The PNS exerts a tonic influence on the heart which helps to slow the heart rate and facilitate calmness, attentional focus, and social engagement under normal circumstances. Activation of the SNS, however, is more metabolically "costly" and is thus typically reserved for threatening situations or situations in which the response of the PNS is insufficient to meet environmental demands (Porges, 2007; Thayer & Lane, 2000). Moreover, sympathetic influences on cardiac output are relatively slow compared to PNS influences on the heart, which allow for a much more rapid response to environmental demands.

The parasympathetic nervous system is itself comprised of two related yet unique neural systems: the myelinated vagus and unmyelinated vagus. The myelinated vagus originates from source nuclei located in the nucleus ambiguus and is related to social communication, self-

soothing, and the inhibition of arousal (Porges, 2007); whereas the unmyelinated vagus is responsible for vegetative functions such as digestion, as well as orientation responses and immobilization under threatening conditions (Beauchaine, 2001). The unmyelinated vagus is phylogenetically more primitive than the SNS and myelinated vagus, and is found in most vertebrates. Porges (2007) proposed a hierarchy of ANS responses, such that phylogenetically newer subsystems of the ANS are activated first in response to stress, followed by activation of more primitive subsystems if the initial response is insufficient. The myelinated vagus is the primary PNS component examined in the present study and its influence on the heart can be monitored via respiratory sinus arrhythmia (RSA), a commonly used measure of PNS activity that reflects high frequency heart rate variations during the respiratory cycle (Porges, 2007).

Polyvagal Theory also describes an evolutionary integration of the neural networks that regulate vagal (PNS) activity with neural networks that regulate the muscles of the face and head, commonly referred to as the Social Engagement System or "face-heart connection" (Porges, 2007; Thayer & Lane, 2000). Thus, a link between PNS functioning and social communication and behavior has been theorized, such that the myelinated vagus facilitates social engagement behaviors and calm behavioral states by slowing the heart rate, inhibiting SNS activity, and attenuating HPA-axis responses. This calming PNS influence has been referred to as "the vagal brake," which allows for spontaneous engagement or disengagement through the rapid inhibition or disinhibition of PNS influence to the heart, depending upon current environmental demands. Additionally, the myelinated vagus contributes to social engagement by influencing the muscles of the face and head that are responsible for listening, facial expressions, and regulating eye gaze (Porges, 2007). Consequently, because the Social Engagement System

and myelinated vagus are highly integrated, atypical development of the Social Engagement System may be reflected in social, behavioral, and autonomic functioning (Porges, 2007).

Several psychophysiological theories have proposed that anxiety disorders may indeed be characterized by abnormal autonomic functioning. Building on Polyvagal theory, Thayer and Lane (2000) describe a model of emotion regulation and dysregulation that views the PNS as but one component of autonomic functioning, attentional regulation, and affective processes. Thayer and Lane (2000) propose that flexible and adaptive physiological and behavioral responses involve the selection of an appropriate response given current environmental demands, as well as the inhibition of other, less appropriate responses. Therefore, psychological disorders may either reflect an inability to "choose" an appropriate physiological or behavioral response, *or* an inability to inhibit inappropriate or irrelevant responses; both of which may be manifested as inflexibility at the autonomic level (Thayer & Lane, 2000). At the cognitive level, the inability to inhibit inappropriate responses or disregard irrelevant information may manifest as hypervigilance towards internal or external threat cues. Thus, psychological disorders such as social anxiety may be partially characterized by the allocation of attentional resources towards irrelevant internal or external cues, which may be influenced by abnormal autonomic functioning.

Blunted PNS reactivity and low baseline vagal tone may reflect a lack of flexibility both at the behavioral and autonomic level, as baseline vagal tone and vagal reactivity are associated with attentional focus and emotional regulation (Porges, 2007; Beauchaine, 2001, Thayer & Lane, 2000). The PNS, however, is but one component of the ANS and alone cannot fully account for changes in heart rate, a commonly used index of overall autonomic functioning. Additionally, a model of autonomic functioning that ignores SNS influences cannot adequately

differentiate between various psychological disorders that are characterized by similar PNS functioning, such as anxiety disorders and depression. For example, it has been posited that anxiety disorders, depression, and aggression are all characterized by low baseline vagal tone; however, each represents a unique disorder or psychological state. Thus, only by examining both PNS and SNS functioning can researchers begin to build a richer and more nuanced understanding of what may differentiate individuals with various disorders at the physiological level (Beauchaine, 2001).

To address this limitation, Beauchaine (2001) proposed an integrated model of autonomic functioning that combines information from Polyvagal Theory and Gray's motivational theory. Gray's motivational theory posits that behavior is governed by three distinct, yet interrelated brain systems: 1) the fight, flight, or freeze system (FFFS), 2) the reward system, or Behavioral Activation System (BAS), and 3) the punishment system, or Behavioral Inhibition System (BIS). The FFFS is regarded as the "defensive avoidance" subsystem of the brain and is associated with emotions such as fear, panic, and rage (Kimbrel, 2008). Furthermore, hyperreactivity in the FFFS has been hypothesized to be related to various anxiety disorders such as panic disorder and specific phobia (Gray & McNaughton, 2000). In contrast, heightened BAS activity may be associated with appetitive/approach behaviors, including negative approach behaviors such as impulsive responding, and positive approach behaviors such as social participation. Heightened BIS activity, however, may be associated with passive or fearful behaviors such as depression, introversion, and anxiety (Beauchaine, 2001). Though SNS arousal is associated with both BIS and BAS activity, Beauchaine (2001) suggests that electrodermal activity (i.e., SCL) may be uniquely related to the BIS, while preejection period (PEP) may be uniquely associated with BAS activity. However, researchers have reported increases in electrodermal activity during

appetitive activation (Bradley & Lang, 2007), illustrating the theoretical and speculative nature of such arguments.

As a whole, psychophysiological theories posit that anxiety disorders may indeed be characterized by both excessive BIS activation and reduced vagal tone, though empirical evidence to corroborate this notion has been sparse and somewhat inconsistent. Similar patterns of autonomic functioning have also been theorized among individuals with depression, which may elucidate some of the mechanisms responsible for high comorbidity rates between the two disorders (Beauchaine, 2001). Furthermore, the role of BAS functioning in both anxiety disorders and depression remains unclear, and studies examining both BIS and BAS activity may be critical towards building an understanding of what differentiates individuals with either disorder.

To date, very few studies have examined the role of BAS functioning in the development of anxiety disorders; however, some have theorized that anxiety disorders may be characterized by high levels of FFFS and BIS sensitivity and low levels of BAS sensitivity (Corr, 2002; Kimbrel, 2008). Corr (2002) suggested that FFFS and BAS activity may have antagonistic and facilitating effects on behavior under certain conditions. Thus, given that the BAS may partially inhibit FFFS responses to threatening stimuli, socially anxious individuals who are high on both FFFS and BIS sensitivity and *low* on BAS sensitivity are thought to experience the most severe levels of anxiety in social situations (Corr, 2002; Kimbrel, 2008). Though very few in number, several studies have produced corroborating evidence to suggest that social anxiety may indeed be characterized by concurrent BIS hyperarousal and BAS hypoarousal (Kimbrel et al., 2012; Morgan et al., 2009). However, these results were found using self-report measures of BAS/BIS sensitivity and have yet to be replicated using corresponding physiological measures. Thus, in an

attempt to build upon the current literature and to develop a more comprehensive understanding of ANS functioning among individuals with varying levels of social anxiety, the current study will examine physiological measures of BIS activity (SCL), BAS activity (PEP), and parasympathetic activity (RSA).

Cognitive Models of Anxiety/Social Anxiety

Cognitive models of anxiety and social phobia more specifically, implicitly minimize potential physiological differences among individuals with high and low levels of anxiety, instead emphasizing the role that distorted cognitions play in the development and maintenance of anxiety disorders. For example, Rapee and Heimberg (1997) argue that distorted or biased processing of social information and internal cues are largely responsible for the development and maintenance of social anxiety in social-evaluative situations. However, though underlying physiological mechanisms are often not the focus of such models, psychophysiological theories such as those described above may be highly related to cognitive models and thus may contribute to a more integrative and comprehensive understanding of anxiety disorders. As previously discussed, Thayer and Lane (2000) suggest that inflexibility at the physiological level may manifest as inflexibility at the cognitive and behavioral levels. Additionally, inflexible physiological responses may also reflect the allocation of attentional resources towards irrelevant internal or external cues (Thayer & Lane, 2000), as is often described in cognitive models of social anxiety and anxiety disorders more generally. Therefore, it may be beneficial to think of cognitive and psychophysiological models of anxiety as being complementary rather than competing theoretical models. Indeed, future theoretical papers may wish to continue to integrate information from psychophysiological theories, such as Polyvagal theory and Gray's motivational theory, with information from existing cognitive models of anxiety.

Emotion Concordance/Discordance

Integration of cognitive and psychophysiological theories of anxiety may prove to be complex, however, as empirical evidence for a model of "response coherence" across various systems has been largely inconsistent (Evers et al., 2014). Response coherence models posit that emotions are the result of coordinated actions across physiological, behavioral, and experiential systems. However, in contrast to these models, empirical evidence suggests that self-reports of emotion are often either not associated, or only modestly associated with physiological measures (Evers et al., 2014; Hollenstein & Lanteigne, 2014), findings which are paralleled in the literature on social anxiety and psychophysiology. For example, in child and adolescent samples, associations between self-reported levels of social anxiety and various measures of physiological functioning have been relatively inconsistent (Siess, Blechert, & Schmitz, 2014). Additionally, in a meta-analysis conducted by Patterson and Ritts (1997), large effects were observed between social anxiety and cognitive and behavioral measures, while small to modest effects were observed for physiological measures. Such results have continued to puzzle researchers and have led to the development of two competing schools of thought: 1) that social anxiety is largely influenced by inflexible or abnormal physiological functioning, or 2) that social anxiety is characterized by relatively normative physiological functioning and a biased perception of bodily symptoms.

To address these inconsistencies, Evers et al. (2014) proposed a dual-process perspective, whereby response coherence may be prevalent *within* but not across two distinct systems. The dual-process perspective describes an automatic response system (i.e., rapid, unconscious physiological and cognitive responses) and a reflective response system (i.e., conscious behavioral and cognitive responses), which may operate relatively independently of one another

(Evers et al., 2014). Thus, according to this perspective, modest and inconsistent associations between social anxiety and psychophysiology may either reflect limitations in methods or analyses across studies, or discordance across dimensions of emotion, such that abnormal automatic responses are not consistently reflected at a more conscious level.

Results in Adults

Among adults, social anxiety has often been associated with chronic hyperarousal as evidenced by chronically elevated heart rates and blood pressure at rest and during stress tasks (Beidel, Turner, & Dancu, 1985; Davidson, Marshall, Tomarken, & Henriques, 2000; Eckman & Shean, 1997; Gerlach, Mourlane, & Rist, 2004; Gerlach, Wilhelm, & Roth, 2003; Gerlach, Wilhelm, Gruber, & Roth, 2001; Kramer, Seefeldt, Heinrichs, Tuschen-Caffier, Schmitz, Wolf, & Blechert, 2012; Matthews, Manuch, & Saab, 1986), though others have failed to produce similar results (Beidel, 1991; Edelmann & Baker, 2002; Hofmann, Newmann, Ehlers, & Roth, 1995; Miers, Blote, Sumter et al., 2011). However, inconsistent findings may be partially due to limitations and methodological differences across studies, as studies vary in their sample sizes and in the nature of the anxiety provoking tasks used to assess physiological responses. Thus, it is unclear whether or not the current literature supports a model of hyperarousal in socially anxious adults.

In contrast to these findings, the relationship between social anxiety and physiological reactivity/recovery has been relatively more consistent. Across a variety of studies, there has typically been no association between self-reported social anxiety and differences in physiological reactivity or recovery among socially anxious and non-anxious adults. This is somewhat perplexing given that the literature on social anxiety in children and adolescents has revealed a variety of associations between several physiological measures and self-reported

social anxiety; though these results have also been inconsistent. To our knowledge, only two studies have provided evidence for differences in physiological reactivity among socially anxious adults. Alvares et al. (2013) found reduced heart rate variability while at rest among a sample of socially anxious female adults (M_{age} = 25.66 years) relative to healthy controls, whereas Grossman, Wilhelm, Kawachi, and Sparrow (2001) found evidence for increased autonomic reactivity among older female adults with social anxiety. Other studies, however, have failed to produce corroborating evidence and have found no differences in reactivity or recovery across a variety of physiological parameters among individuals with varying degrees of social anxiety (Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014; Thibodeau, Gomez-Perez, & Asmundson, 2012; Mauss, Wilhelm, & Gross, 2003; Mauss, Wilhelm, & Gross, 2004).

Though these findings appear to be relatively consistent they are by no means conclusive, as the aforementioned studies vary tremendously in their sample sizes, age ranges, physiological measures, participant levels of social anxiety (e.g., clinical vs. healthy controls, or high vs. low social anxiety), and methods used to obtain physiological measurements (e.g., while at rest, while giving an impromptu speech, during a typing task). Additionally, several of the studies utilized limited autonomic measures such as heart rate (HR) or heart rate variability (HRV), which have been criticized as comparatively broad measures of autonomic functioning that do not capture potential underlying differences in PNS and SNS activity. Thus, replication of these findings using similar methodologies is needed before reliable conclusions can be drawn regarding the psychophysiology of socially anxious adults.

Results in Children and Adolescents

Similar to the literature on physiological functioning in socially anxious adults, several studies have reported hyperarousal among children and adolescents with high levels of social

anxiety as evidenced by chronically elevated HR. For example, in a study conducted by Erath, Tu, and El-Sheikh (2012), researchers examined peer victimization as a potential moderator of the association between preadolescents' social anxiety and physiological arousal in the context of peer stress. Preadolescents' (ages 10-12; n = 63) participated in a conversation-based peer evaluation task, and the authors found that social anxiety was more strongly associated with physiological hyperarousal (i.e., higher HR, lower RSA) among preadolescents who experienced higher levels of peer victimization. In a similar study, Kramer et al. (2011) compared 8-12 year old children diagnosed with Social Phobia (n = 41), to matched healthy controls (n = 40). Children were exposed to the Trier Social Stress Test for Children (TSST-C), and no differences in reactivity were observed for HR, salivary alpha-amylase (sAA), or cortisol responses between socially anxious and non-anxious children. However, children with Social Phobia demonstrated elevated HR levels throughout the baseline, stress, and recovery periods, relative to healthy control children, providing evidence for consistent hyperarousal in socially anxious youth (for additional evidence, see Anderson, Veed, Inderbitzen-Nolan, & Hansen, 2010).

However, these results are equivocal as recent studies by Anderson and Hope (2009), and Schmitz et al. (2012) failed to produce similar findings in adolescent samples. Furthermore, though evidence for chronic hyperarousal among socially anxious youth is inconsistent, two studies have also demonstrated *baseline* hyperarousal among preadolescent samples as evidenced by elevated HR and sympathetic arousal (Schmitz, Kramer, Tuchen-Caffier, Heinrichs, & Blechert, 2011; Schmitz, Tuschen-Caffier, Wilhelm, & Blechert, 2013). Unfortunately, to our knowledge, these represent the only studies that measure the dynamic physiological responses of socially anxious youth from baseline, through social challenges and recovery periods. As a

result, it is important to view these findings with caution prior to their replication in independent child and adolescent samples.

In contrast to the literature on physiology among socially anxious adults, the majority of psychophysiological studies in childhood and adolescence appear to provide evidence for a relationship between social anxiety and autonomic reactivity or recovery across a variety of physiological parameters. Though there is a dearth of studies examining physiological responses in socially anxious youth, existing studies have provided some evidence to suggest that high levels of social anxiety in childhood and adolescence may be characterized by low basal RSA and blunted PNS reactivity (Erath et al., 2012; Schmitz et al., 2011; Schmitz et al., 2013; Greaves-Lord et al., 2010), chronically elevated SNS activity (Schmitz et al., 2013), and a slower HR recovery after exposure to a social-stress task (Schmitz et al., 2011; 2013).

Two studies conducted by Schmitz et al. (2011 and 2013) provide perhaps the most compelling evidence of concurrent hyperarousal and autonomic inflexibility among socially anxious youth, and thus warrant additional attention. In the first study conducted by Schmitz et al. (2011), children (ages 8-12) diagnosed with Social Phobia (n = 30) were compared to healthy control children (n = 26), while exposed to the Trier Social Stress Test for Children (TSST-C). At baseline, children with Social Phobia exhibited higher levels of sympathetic activation, $t(50) = 2.34$, $p = .024$, $d = .64$, higher heart rates, $t(54) = 2.06$, $p = .044$, $d = .55$, and lower levels of parasympathetic activity (as indexed by low basal RSA levels), $t(54) = 2.23$, $p = .030$, $d = .67$. Additionally, the authors found evidence for blunted parasympathetic reactivity in SP children during the TSST-C, $F(2, 104) = 3.68$, $p = .028$, $f = .26$, and a slower heart rate recovery after the task, $F(1,54) = 4.24$, $p = .044$, $f = .24$, relative to healthy control children.

Intriguingly, in a subsequent study, Schmitz et al. (2013) found similar results in a population of children with subclinical levels of social anxiety. Children (ages 10-12) were split into high (HSA; $n = 20$) and low social anxiety (LSA; $n = 20$) groups based on scores from the revised Social Anxiety Scale for Children (SASC-R), and physiological measures were obtained before, during, and after a standardized speech task. The authors found that HSA children exhibited higher baseline sympathetic activity, $F(1,38) = 5.67$, $p = .022$, $\eta^2 = .130$, blunted cardiac reactivity, $t(38) = 2.56$, $p = .014$, $d = .81$, and parasympathetic reactivity during the speech task, $t(38) = 2.00$, $p = .052$, $d = .64$, and a slower HR recovery after the speech task, $t(38) = 2.28$, $p = .028$, $d = .73$. Thus, in accordance with the two aforementioned studies, it appears that flexible and adaptive autonomic functioning may be characterized by high basal parasympathetic activity and low basal sympathetic activity, as well as moderate vagal withdrawal and potential increases in sympathetic activity in response to specific environmental demands.

Nevertheless, contradictory evidence does exist, as several researchers have failed to find differences in physiological reactivity or recovery among youth with varying levels of social anxiety (Anderson & Hope, 2009; Anderson, Veed, Inderbitzen-Nolan, & Hansen, 2010; Kramer et al., 2012). However, though contradictory evidence is present in the literature on social anxiety in childhood and adolescence, a common methodological limitation in these studies must be addressed. Among the three studies that reported no differences in physiological reactivity among socially anxious youth, each utilized limited physiological measures by assessing only HR/HRV and/or blood pressure. Both HR/HRV and measures of blood pressure have produced largely inconsistent results across a variety of samples with anxiety disorders (see Seiss et al., 2014), which is perhaps not surprising as each is a non-specific measure of autonomic

functioning. HR/HRV does not capture potential meaningful differences in the autonomic components that contribute to overall cardiac functioning (i.e., the PNS and SNS). Similarly, measures of blood pressure may be influenced by both SNS and PNS functioning (see Berntson, Quigley, & Lozano, 2007), and SNS activity may be more accurately assessed through electrodermal (SCL) and/or PEP measurements.

The results summarized above should be interpreted with caution as, similar to the literature on social anxiety in adulthood, studies varied in sample size, physiological measures, participant levels of social anxiety (e.g., clinical vs. healthy controls, or high vs. low social anxiety), and methods used to obtain physiological measurements (e.g., during orthostatic test, while giving a speech). Therefore, the role of physiological functioning in the development and maintenance of social anxiety in childhood and adolescence remains unclear, highlighting the need for the present study. The aim of the current study is to build upon prior research in a variety of novel ways in an attempt to address several of the limitations of previous studies and to shed light on what appears to be an important, yet controversial topic.

APPENDIX B

Lab-based/context-specific Social Anxiety

Pre-activity Questions

	Not at all	A little	Somewhat	Pretty much	Very much
1. How nervous or anxious are you about the conversation activity?	1	2	3	4	5

Post-activity Questions (continued)

	Not at all	A little	Somewhat	Pretty much	Very much
1. How anxious or nervous were you about the conversation activity?	1	2	3	4	5

Social Anxiety Scale for Adolescents

Read each of the following sentences carefully and circle the number that best describes how you feel.

	Not at all	Hardly ever	Some-times	Most of the time	All the time
1. I worry about doing something new in front of others.	0	1	2	3	4
2. I worry about being teased.	0	1	2	3	4
3. I feel shy around people I don't know.	0	1	2	3	4
4. I only talk to people I know really well.	0	1	2	3	4
5. I feel that peers talk about me behind my back.	0	1	2	3	4
6. I worry about what others think of me.	0	1	2	3	4
7. I'm afraid that others will not like me.	0	1	2	3	4
8. I get nervous when I talk to peers I don't know very well.	0	1	2	3	4
9. I worry about what others say about me.	0	1	2	3	4
10. I get nervous when I meet new people.	0	1	2	3	4
11. I worry that others don't like me.	0	1	2	3	4
12. I am quiet when I'm with a group of people.	0	1	2	3	4
13. I feel that others make fun of me.	0	1	2	3	4
14. If I get into an argument, I worry that the other person will not like me.	0	1	2	3	4
15. I'm afraid to invite others to do things with me because they might say no.	0	1	2	3	4
16. I feel nervous when I'm around certain people.	0	1	2	3	4
17. I feel shy even with peers I know very well.	0	1	2	3	4
18. It's hard for me to ask others to do things with me.	0	1	2	3	4

APPENDIX C

Descriptive Tables for Physiological Measures

Variable	Mean	SD	Skew	Min	Max	N
HR Baseline A – Minute 1	82.05	12.66	.718	54.23	125.48	120
HR Baseline A – Minute 2	83.40	12.57	.566	52.72	126.19	120
HR Baseline A – Minute 3	83.50	12.37	.647	57.88	124.62	120
HR Conversation – Minute 1	90.05	14.70	.962	59.53	139.72	117
HR Conversation – Minute 2	87.47	14.04	1.00	59.31	137.26	117
HR Conversation – Minute 3	87.78	13.63	1.10	60.58	139.73	115
HR Waiting Period – Minute 1	82.01	11.17	.240	52.65	116.31	110
HR Waiting Period – Minute 2	82.09	10.95	.348	51.06	118.19	111
HR Waiting Period – Minute 3	82.03	11.18	.292	49.25	121.02	111
HR Rebuff Period – Minute 1	81.40	12.23	.509	53.03	115.95	110
HR Rebuff Period – Minute 2	82.70	12.65	.756	52.75	128.05	111
HR Rebuff Period – Minute 3	83.13	12.34	.721	56.66	129.44	110
HR Recovery – Minute 1	81.24	11.27	.569	55.20	116.99	110
HR Recovery – Minute 2	81.16	11.81	.535	51.56	120.15	110
HR Recovery – Minute 3	81.32	11.85	.403	51.36	116.74	109
HR Baseline A Average	82.99	12.40	.655	54.94	125.43	120
HR Baseline B Average	85.81	11.77	.469	57.04	120.64	119
HR Conversation Average	88.47	13.91	1.023	59.97	138.61	117
HR Waiting Period Average	82.04	10.88	.300	51.00	118.51	111
HR Rebuff Average	82.42	12.15	.649	54.15	123.61	111
HR Recovery Average	81.29	11.44	.497	52.71	117.96	110

Variable	Mean	SD	Skew	Min	Max	N
RSA Baseline A – Minute 1	6.98	1.13	-.004	3.73	9.67	123
RSA Baseline A – Minute 2	6.80	1.22	-.745	2.02	9.20	123
RSA Baseline A – Minute 3	6.85	1.16	-.481	2.57	9.30	123
RSA Conversation – Minute 1	6.81	1.25	-.226	2.99	10.17	120
RSA Conversation – Minute 2	6.92	1.14	-.432	1.95	9.97	120
RSA Conversation – Minute 3	6.93	1.10	-.199	2.82	10.26	119
RSA Waiting Period – Minute 1	7.04	1.08	-.072	4.31	9.39	114
RSA Waiting Period – Minute 2	6.96	1.06	.160	4.78	9.61	114
RSA Waiting Period – Minute 3	6.82	1.14	-.163	3.07	9.37	114
RSA Rebuff Period – Minute 1	7.07	1.04	.171	4.49	9.60	114
RSA Rebuff Period – Minute 2	6.92	1.22	-.214	3.64	9.63	114
RSA Rebuff Period – Minute 3	6.90	1.04	.225	4.13	9.89	114
RSA Recovery – Minute 1	7.16	1.01	-.037	4.30	9.79	118
RSA Recovery – Minute 2	7.10	1.07	-.016	4.72	9.57	118
RSA Recovery – Minute 3	7.04	1.02	-.210	4.58	9.07	117
RSA Baseline A Average	6.88	1.05	-.348	3.28	9.34	123
RSA Baseline B Average	7.05	1.03	-.004	3.33	9.86	123
RSA Conversation Average	6.88	1.10	-.233	2.59	10.00	120
RSA Waiting Period Average	6.94	.99	.110	4.24	9.26	114
RSA Rebuff Average	6.96	.99	.224	4.56	9.69	114
RSA Recovery Average	7.10	.91	.017	4.95	9.39	118

Variable	Mean	SD	Skew	Min	Max	N
SCL Baseline A – Minute 1	6.55	4.12	.960	1.05	18.90	110
SCL Baseline A – Minute 2	6.18	4.12	.942	1.07	17.16	109
SCL Baseline A – Minute 3	6.13	4.25	.783	.97	16.62	110
SCL Conversation – Minute 1	11.22	5.26	.703	1.79	31.33	117
SCL Conversation – Minute 2	10.38	4.85	.813	2.16	29.46	118
SCL Conversation – Minute 3	10.05	4.90	.885	2.28	29.10	118
SCL Waiting Period – Minute 1	8.90	4.59	.836	1.44	23.05	113
SCL Waiting Period – Minute 2	8.67	4.64	.661	1.29	21.26	113
SCL Waiting Period – Minute 3	8.77	4.79	.698	1.18	23.54	112
SCL Rebuff Period – Minute 1	9.86	4.98	.635	1.35	23.89	111
SCL Rebuff Period – Minute 2	9.50	4.78	.649	2.03	23.59	111
SCL Rebuff Period – Minute 3	9.52	4.85	.685	1.36	26.73	112
SCL Recovery – Minute 1	9.25	4.52	.634	2.16	21.47	116
SCL Recovery – Minute 2	8.91	4.45	.699	1.93	22.35	115
SCL Recovery – Minute 3	8.93	4.58	.756	1.83	23.62	114
SCL Baseline A Average	6.23	4.07	.848	1.05	16.86	111
SCL Baseline B Average	9.06	4.57	.808	1.42	26.22	115
SCL Conversation Average	10.50	4.98	.773	2.18	29.97	119
SCL Waiting Period Average	8.76	4.65	.726	1.37	22.62	113
SCL Rebuff Average	9.58	4.85	.646	1.36	24.74	112
SCL Recovery Average	8.98	4.48	.697	1.97	22.48	116

Variable	Mean	SD	Skew	Min	Max	N
PEP Baseline A – Minute 1	118.29	16.29	.075	74.30	161.20	116
PEP Baseline A – Minute 2	119.56	16.79	.315	74.80	163.95	116
PEP Baseline A – Minute 3	118.79	16.32	.183	72.60	162.85	116
PEP Conversation – Minute 1	114.88	15.77	-.045	77.60	150.55	113
PEP Conversation – Minute 2	116.77	17.24	.008	75.95	164.50	113
PEP Conversation – Minute 3	116.34	16.68	-.193	75.55	157.90	112
PEP Waiting Period – Minute 1	116.47	15.60	.152	71.95	161.75	108
PEP Waiting Period – Minute 2	117.86	16.17	.102	69.75	161.20	107
PEP Waiting Period – Minute 3	117.75	15.96	.312	73.60	166.70	108
PEP Rebuff Period – Minute 1	117.28	16.39	.056	73.05	163.95	107
PEP Rebuff Period – Minute 2	117.34	16.80	.263	73.60	171.10	107
PEP Rebuff Period – Minute 3	116.19	15.64	.200	70.85	162.30	107
PEP Recovery – Minute 1	117.02	17.02	-.153	63.30	154.60	110
PEP Recovery – Minute 2	117.31	16.05	-.071	64.80	161.75	110
PEP Recovery – Minute 3	118.51	15.73	.303	71.95	170.00	110
PEP Baseline A Average	118.88	15.77	.168	74.25	160.37	116
PEP Baseline B Average	119.21	16.16	.189	77.85	165.23	116
PEP Conversation Average	115.97	16.29	-.102	77.93	157.65	113
PEP Waiting Period Average	117.37	15.48	.179	71.77	163.22	108
PEP Rebuff Average	116.94	15.64	.183	72.50	165.78	107
PEP Recovery Average	117.67	15.91	.063	67.37	161.78	111

APPENDIX D

Periods used in the two "stages" of social stress

STAGE	Periods included				
Full Lab Protocol	**Baseline**	**Peer Evaluation**	**Waiting**	**Peer Rebuff**	**Recovery**
Stage 1	**Baseline**	**Peer Evaluation**	**Waiting**	**_**	**_**
Stage 2	**_**	**_**	**Waiting**	**Peer Rebuff**	**Recovery**

Table 1

Descriptive Statistics and Correlations Between Heart Rate and Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Global SA	-														
2. CS SA	.13	-													
3. Sex	.19*	.12	-												
4. Ethnicity	.07	.17+	.06	-											
5. Grade	-.17+	.07	-.07	-.46**	-										
6. Income	-.17+	-.02	.02	-.52**	.25**	-									
7. HR Baseline	.02	-.06	.16+	-.04	.04	.03	-								
8. HR Evaluation	-.01	.02	.20*	-.10	.16	.12	.85**	-							
9. HR Waiting	-.08	-.04	.24*	-.06	-.01	.08	.95**	.83**	-						
10. HR Rebuff	.06	.01	.34**	-.02	-.01	.11	.93**	.86**	.94**	-					
11. HR Recovery	.04	-.01	.28**	-.07	.00	.10	.94**	.81**	.96**	.93**	-				
12. HR react 1	-.09	.14	.15	-.17+	.23*	.23*	.00	.53**	.11	.15	.07	-			
13. HR rec 1	-.03	-.08	.03	.12	-.21*	-.11	.46**	.00	.56**	.41**	.50**	-.67**	-		
14. HR react 2	.10	.11	.23*	.00	.14	.07	.09	.22*	.00	.33**	.08	.30**	-.36**	-	
15. HR rec 2	-.05	-.17+	-.04	.01	-.11	.04	.20*	.06	.23*	.00	.36**	-.20*	.35**	-.66**	-
Mean (SD)	42.8 (14.6)	2.77 (1.12)	50%	35%	.61 (.49)	.00 (1.55)	82.9 (12.4)	88.5 (13.9)	82.0 (10.9)	82.4 (12.2)	81.3 (11.4)	.00 (7.37)	.00 (6.09)	.00 (3.86)	.00 (4.12)
Skew	.35	.40	.02	.64	-.46	-.45	.66	1.02	.30	.65	.50	.99	-.24	.68	-.31

Note: SA = social anxiety; Sex, ethnicity, and age were measured using dichotomous variables (i.e., Male = 0, Female = 1; Non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1, respectively); HR = heart rate; react 1 = reactivity from baseline to peer evaluation period; rec 1 = recovery from peer evaluation period to waiting period; react 2 = reactivity from waiting period to rebuff period; rec 2 = recovery from the rebuff period to the true recovery period; Rows 7 through 11 represent average scores during each period; $^{+}p < .10$, $*p < .05$, $**p < .01$

Table 2

Descriptive Statistics and Correlations Between Skin Conductance and Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Global SA	-														
2. CS SA	.13	-													
3. Sex	.19*	.12	-												
4. Ethnicity	.07	.17+	.06	-											
5. Grade	-.17+	.07	-.07	-.46**	-										
6. Income	-.17+	-.02	.02	-.52**	.25**	-									
7. SCL Baseline	-.09	.03	-.09	-.27**	.22*	.00	-								
8. SCL Evaluation	-.14	.00	-.15	-.45**	.23*	.18+	.77**	-							
9. SCL Waiting	-.11	.00	-.11	-.42**	.21*	.16+	.88**	.92**	-						
10. SCL Rebuff	-.10	.03	-.12	-.42**	.24*	.16	.84**	.94**	.96**	-					
11. SCL Recovery	-.02	-.01	-.08	-.44**	.22*	.17+	.80**	.89**	.96**	.96**	-				
12. SCL react 1	-.07	-.01	-.17+	-.31**	.03	.18+	.00	.64**	.35**	.43**	.44**	-			
13. SCL rec 1	.10	-.05	.08	-.07	.04	-.01	.44**	.00	.40**	.26**	.36**	-.56**	-		
14. SCL react 2	-.01	.05	-.01	-.10	.17+	.10	-.06	.21*	.00	.28**	.12	.37**	-.46**	-	
15. SCL rec 2	.16+	-.08	.11	-.10	-.10	.03	.05	-.02	.14	.00	.28**	.00	.39**	-.50**	-
Mean (SD)	42.8 (14.6)	2.77 (1.12)	50%	35%	.61 (.49)	.00 (1.55)	6.23 (4.07)	10.50 (4.98)	8.76 (4.65)	9.58 (4.85)	8.98 (4.48)	.00 (3.09)	.00 (1.86)	.00 (1.33)	.00 (1.28)
Skew	.35	.40	.02	.64	-.46	-.45	.85	.77	.73	.65	.70	.70	-.01	1.72	-.19

Note: SA = social anxiety; Sex, ethnicity, and age were measured using dichotomous variables (i.e., Male = 0, Female = 1; Non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1, respectively); SCL = skin conductance level (units = µS); react 1 = reactivity from baseline to peer evaluation period; rec 1 = recovery from peer evaluation period to waiting period; react 2 = reactivity from waiting period to rebuff period; rec 2 = recovery from the rebuff period to the true recovery period; Rows 7 through 11 represent average scores during each period; $^{+}p < .10$, $*p < .05$, $**p < .01$

Table 3

Descriptive Statistics and Correlations Between Respiratory Sinus Arrhythmia and Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Global SA	-														
2. CS SA	.13	-													
3. Sex	.19*	.12	-												
4. Ethnicity	.07	.17+	.06	-											
5. Grade	-.17+	.07	-.07	-.46**	-										
6. Income	-.17+	-.02	.02	-.52**	.25**	-									
7. RSA Baseline	.08	.05	.09	.06	-.20*	.07	-								
8. RSA Evaluation	.14	-.07	.00	.02	-.17+	-.03	.70**	-							
9. RSA Waiting	.04	.08	-.02	.12	-.14	.00	.83**	.67**	-						
10. RSA Rebuff	.07	.05	-.02	.11	-.22*	-.01	.81**	.77**	.86**	-					
11. RSA Recovery	.02	.03	-.01	.11	-.16+	-.04	.80**	.69**	.85**	.83**	-				
12. RSA react 1	.10	-.14	-.10	-.03	-.05	-.12	.00	.72**	.10	.26**	.14	-			
13. RSA rec 1	-.09	.17+	.02	.11	-.06	.04	.51**	.00	.74**	.46**	.52**	-.49**	-		
14. RSA react 2	.06	-.04	.00	.00	-.18+	-.03	.19*	.38**	.00	.52**	.19*	.33**	-.34**	-	
15. RSA rec 2	.00	.05	.03	-.05	.05	-.02	.22*	.11	.26**	.00	.56**	-.06	.24**	-.42**	-
Mean (SD)	42.8 (14.6)	2.77 (1.12)	50%	35%	.61 (.49)	.00 (1.55)	6.88 (1.05)	6.88 (1.10)	6.94 (.99)	6.96 (.99)	7.10 (.91)	.00 (.79)	.00 (.74)	.00 (.51)	.00 (.51)
Skew	.35	.40	.02	.64	-.46	-.45	-.35	-.23	.11	.22	.02	-.20	.16	-.85	.28

Note: SA = social anxiety; Sex, ethnicity, and age were measured using dichotomous variables (i.e., Male = 0, Female = 1; Non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1, respectively); RSA = respiratory sinus arrhythmia (units = ln[ms^2]); react 1 = reactivity from baseline to peer evaluation period; rec 1 = recovery from peer evaluation period to waiting period; react 2 = reactivity from waiting period to rebuff period; rec 2 = recovery from the rebuff period to the true recovery period; Rows 7 through 11 represent average scores during each period; $^+p < .10$, $*p < .05$, $**p < .01$

Table 4

Descriptive Statistics and Correlations Between Pre-Ejection Period and Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Global SA	-														
2. CS SA	.13	-													
3. Sex	.19*	.12	-												
4. Ethnicity	.07	.17+	.06	-											
5. Grade	-.17+	.07	-.07	-.46**	-										
6. Income	-.17+	-.02	.02	-.52**	.25**	-									
7. PEP Baseline	.12	.10	-.13	.19*	.04	-.12	-								
8. PEP Evaluation	.06	.06	-.18+	.08	.13	-.03	.91**	-							
9. PEP Waiting	.04	.13	-.12	.12	.11	-.14	.93**	.90**	-						
10. PEP Rebuff	.04	.05	-.13	.14	.07	-.16	.90**	.87**	.96**	-					
11. PEP Recovery	.10	.11	-.11	.12	.06	-.12	.93**	.91**	.98**	.95**	-				
12. PEP react 1	-.05	-.09	-.20*	-.13	.14	.10	.00	.41**	.15	.14	.12	-			
13. PEP rec 1	.01	.16+	.17+	.13	-.07	-.20*	.27**	.00	.43**	.41**	.45**	-.57**	-		
14. PEP react 2	-.01	-.23*	-.08	.08	-.07	-.12	.02	.01	.00	.28**	.03	.00	-.03	-	
15. PEP rec 2	.04	.17+	-.07	-.02	.06	.00	.23*	.26**	.20*	.00	.31**	.13	-.07	-.72**	-
Mean (SD)	42.8 (14.6)	2.77 (1.12)	50%	35%	.61 (.49)	.00 (1.55)	118.8 (15.8)	116.0 (16.3)	117.4 (15.5)	116.9 (15.6)	117.7 (15.9)	.00 (6.73)	.00 (6.69)	.00 (4.41)	.00 (4.93)
Skew	.35	.40	.02	.64	-.46	-.45	.17	-.10	.18	.18	.06	-1.64	1.80	-.72	-.20

Note: SA = social anxiety; Sex, ethnicity, and age were measured using dichotomous variables (i.e., Male = 0, Female = 1; Non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1, respectively); PEP = pre-ejection period (measured in ms); react 1 = reactivity from baseline to peer evaluation period; rec 1 = recovery from peer evaluation period to waiting period; react 2 = reactivity from waiting period to rebuff period; rec 2 = recovery from the rebuff period to the true recovery period; Rows 7 through 11 represent average scores during each period; $^{+}p < .10$, $*p < .05$, $**p < .01$

Table 5

Final Fitted Models Demonstrating Inter-Individual Differences in Growth in Heart Rate, Skin Conductance Level, and Respiratory Sinus Arrhythmia

Model	χ^2	*df*	χ^2/df	TLI	RMSEA
I. Heart Rate: Full Protocol predicted by CS Social Anxiety	317.30	149	2.13	.95	.09
II. Heart Rate: Stage 2 predicted by CS Social Anxiety	114.81	42	2.73	.96	.12
III. SCL: Full Protocol predicted by Global Social Anxiety	733.32	138	5.31	.86	.19
IV. SCL: Stage 1 predicted by Global Social Anxiety	325.59	50	6.51	.86	.20
V. SCL: Stage 2 predicted by Global Social Anxiety	236.18	58	4.07	.93	.16
VI. RSA: Stage 1 predicted by CS Social Anxiety	165.95	48	3.46	.86	.14

Note: Only models in which a measure of anxiety was signficantly associated with at least one growth parameter are reported

Table 6

Estimated Non-standardized Growth Parameters from the Final Fitted Models for the Growth Analysis of Heart Rate and Respiratory Sinus Arrhythmia Predicted by Context-Specific Anxiety

	Heart Rate – Full Protocol	Heart Rate – Stage 2	RSA – Stage 1
Intercept (S.E.)	82.402 (1.165)***	77.698 (1.671)***	7.145 (.147)***
Increments to true initial status:			
CS Anxiety	-.714 (1.051)	-.740 (.924)	.086 (.090)
Sex	-	5.509 (2.048)**	-
Age	-	-	-.394 (.176)*
Income	-	-	-
Model R^2%	.4%	6.2%	4.4%
Linear Slope (S.E.)	.179 (.178)	.635 (.463)	.004 (.039)
Increments to rate of true linear change:			
Context-Specific Anxiety	.164 (.074)*	.246 (.159)	-.062 (.035)$^{+}$
Sex	.210 (.168)	.874 (.360)*	-
Age	-.132 (.178)	-	-
Income	.117 (.053)*	-	-
Model R^2%	25.8%	13.3%	6%
Quadratic Slope (S.E.)	-.020 (.012)	-.070 (.049)	-.001 (.005)
Increments to rate of true quadratic change			
Context-Specific Anxiety	-.011 (.005)*	-.037 (.020)$^{+}$	.008 (.004)$^{+}$
Sex	-.006 (.012)	-.094 (.046)*	-
Age	.007 (.013)	-	-
Income	-.006 (.004)	-	-
Model R^2%	25.3%	11.7%	5.5%

Note: S.E. = Standard Error; Sex and age were measured using dichotomous variables (i.e., Male = 0, Female = 1; 5th grade = 0, 6th grade = 1); $^{+}p < .10$, $*p < .05$, $**p < .01$, $***p < .001$

Table 7

Estimated Non-standardized Growth Parameters from the Final Fitted Models for the Growth Analysis of Skin Conductance Level Predicted by Global Social Anxiety

	SCL – Full Protocol	SCL – Stage 1	SCL – Stage 2
Intercept (S.E.)	7.939 (.774)***	5.962 (.806)***	9.853 (.851)***
Increments to true initial status:			
Global Social Anxiety	-.243 (.450)	-.017 (.468)	-.266 (.492)
Ethnicity	-2.853 (.835)**	-2.139 (.872)*	-3.957 (.923)***
Age	.701 (.827)	.952 (.862)	.410 (.907)
Model R^2%	14.6%	10.3%	19%
Linear Slope (S.E.)	.671 (.077)***	1.03 (.178)***	.171 (.046)***
Increments to rate of true linear change:			
Global Social Anxiety	-.043 (.045)	-.182 (.101)$^{+}$	-
Ethnicity	-.335 (.084)***	-.147 (.189)	-
Age	-.042 (.082)	-.101 (.183)	-
Model R^2%	23.8%	8.7%	-
Quadratic Slope (S.E.)	-.043 (.006)***	-.060 (.019)**	-.019 (.008)*
Increments to rate of true quadratic change			
Global Social Anxiety	.007 (.003)*	.019 (.011)$^{+}$	.007 (.003)*
Ethnicity	.019 (.006)**	-.018 (.020)	-.004 (.006)
Age	.000 (.006)	.001 (.019)	-.003 (.005)
Model R^2%	25.8%	16.5%	8%

Note: S.E. = Standard Error; Ethnicity and age were measured using dichotomous variables (i.e., Non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1); $^{+}p < .10$, $*p < .05$, $**p < .01$, $***p < .001$

Table 8

Parameter Estimates of Mean Growth Levels and Variance of Growth Factors for Final Unconditional Growth Models

	Intercept (S.E.)	Slope (S.E.)	Quadratic (S.E.)
HR – Full protocol			
Mean	82.691*** (1.130)	.237* (.099)	-.023*** (.007)
Variance	152.349*** (19.775)	.553*** (.131)	.002** (.001)
HR – Stage 1			
Mean	82.037*** (1.130)	1.287*** (.163)	-.165*** (.020)
Variance	154.161*** (19.990)	.489 (.461)	.001 (.006)
HR – Stage 2			
Mean	82.237*** (1.070)	.045 (.184)	-.018 (.023)
Variance	129.522*** (17.665)	1.941*** (.538)	.033*** (.009)
SCL – Full protocol			
Mean	7.249*** (.383)	.542*** (.041)	-.036*** (.003)
Variance	17.177*** (2.269)	.132*** (.023)	.001*** (.000)
SCL – Stage 1			
Mean	5.532*** (.383)	1.194*** (.094)	-.102*** (.010)
Variance	15.671*** (2.284)	.420** (.125)	.003* (.001)
SCL – Stage 2			
Mean	8.769*** (.426)	.261*** (.045)	-.034*** (.006)
Variance	20.845*** (2.783)	.006 (.007)	.001*** (.000)
RSA – Full protocol			
Mean	6.857*** (.091)	.013** (.004)	-
Variance	.916*** (.131)	.000 (.000)	-
RSA – Stage 1			
Mean	6.902*** (.102)	.006 (.039)	-.001 (.005)
Variance	1.014*** (.163)	.108*** (.025)	.002*** (.000)
RSA – Stage 2			
Mean	6.953*** (.093)	.015* (.007)	-
Variance	.892*** (.134)	.001 (.001)	-
PEP – Full protocol			
Mean	117.832*** (1.464)	-.022 (.043)	-
Variance	240.700*** (32.578)	.109*** (.030)	-
PEP – Stage 1			
Mean	118.745*** (1.476)	-.948** (.342)	.113* (.045)
Variance	235.197*** (33.402)	8.093*** (1.988)	.151*** (.033)
PEP – Stage 2			
Mean	117.210*** (1.469)	.016 (.053)	-
Variance	236.147*** (32.371)	.002 (.048)	-

Note: S.E. = Standard Error; HR = heart rate; SCL = skin conductance level (units = μS); RSA = respiratory sinus arrhythmia (units = ln[ms^2]); PEP = pre-ejection period (measured in ms); Mean and variance were not reported for quadratic slope if the addition of a quadratic term did not provide a significant improvement in fit over an unconditional linear model; *$p < .05$, **$p < .01$, ***$p < .001$

Figures

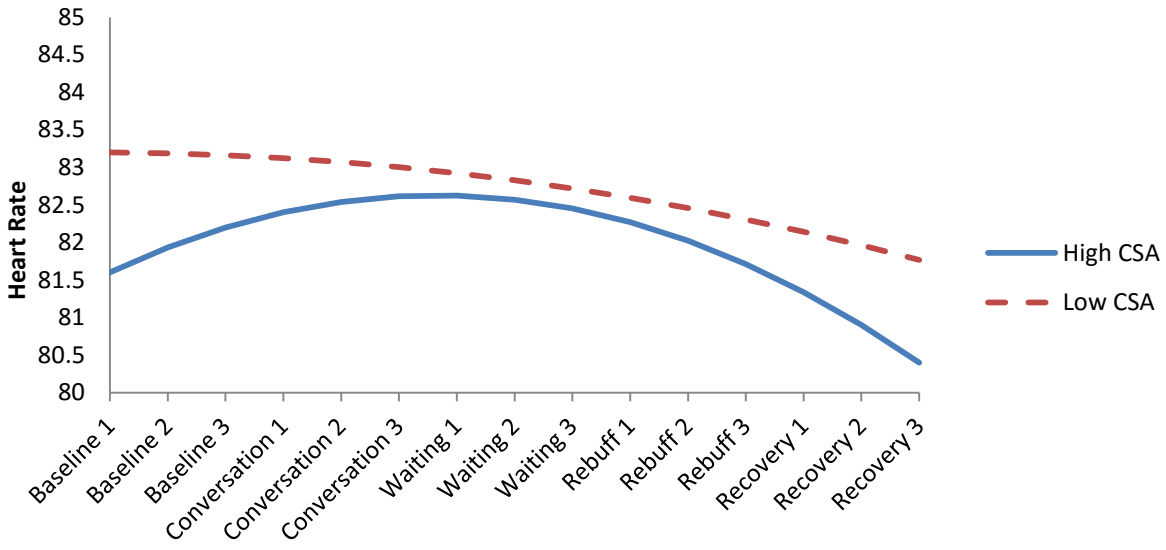

Figure 1. Average growth in heart rate across the full lab protocol for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of context-specific anxiety.

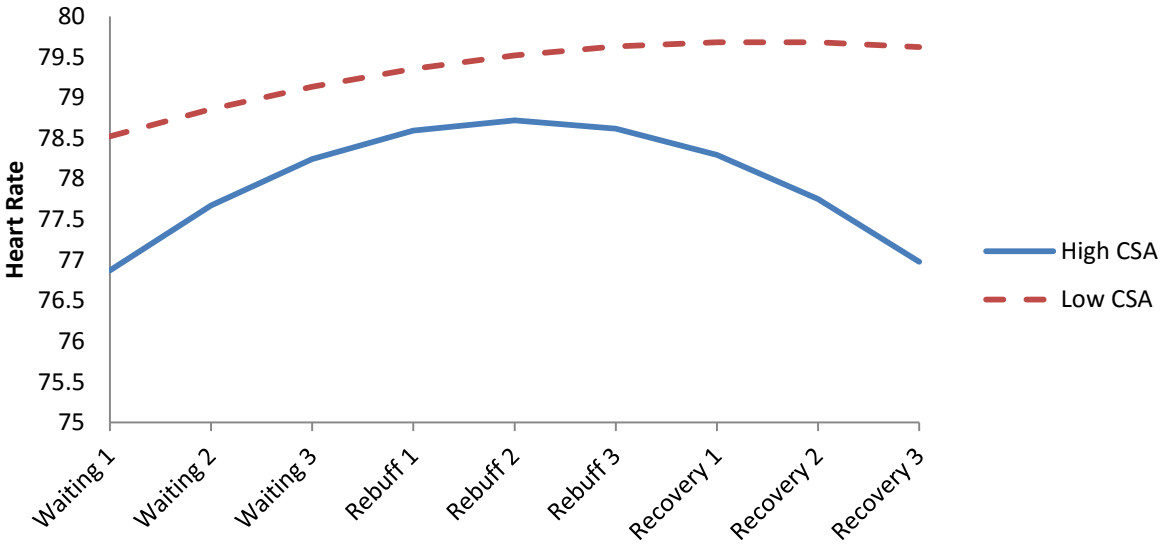

Figure 2. Average growth in heart rate across the second stage of social stress for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of context-specific anxiety.

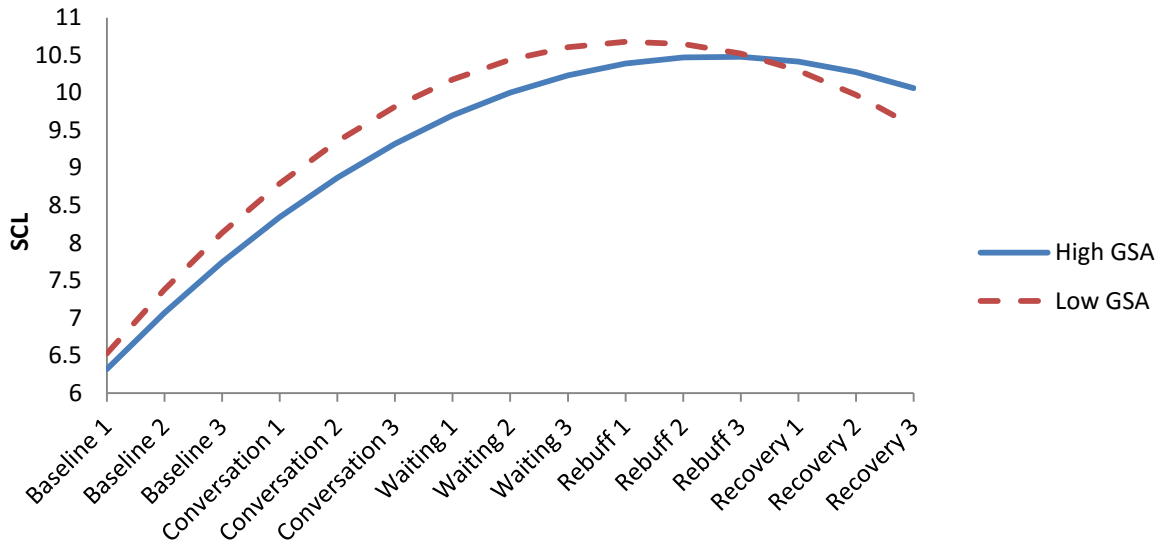

Figure 3. Average growth in skin conductance level (SCL) across the full lab protocol for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of global social anxiety.

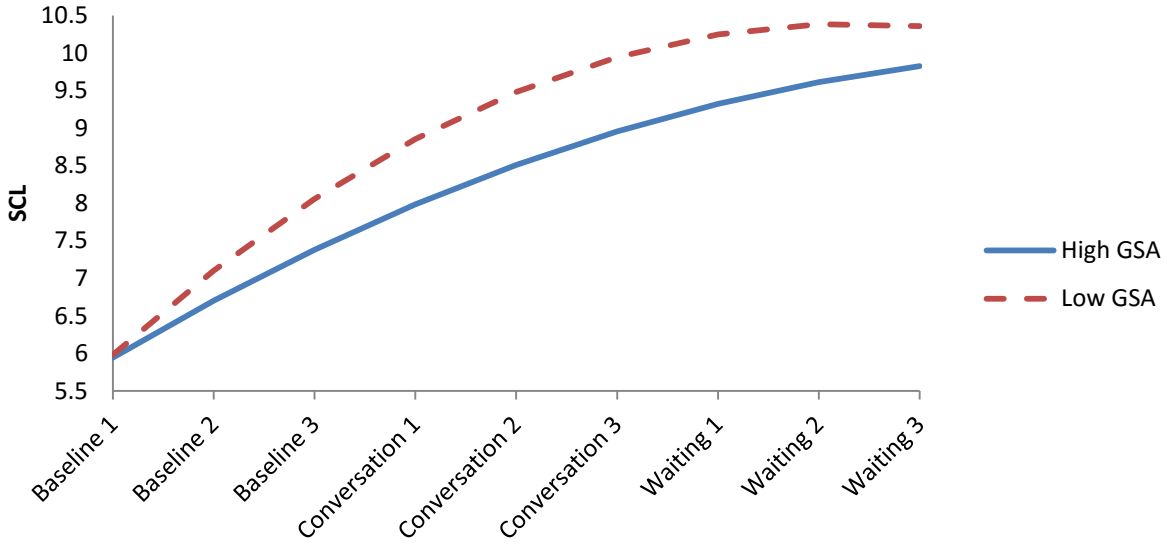

Figure 4. Average growth in skin conductance level (SCL) across the first stage of social stress for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of global social anxiety.

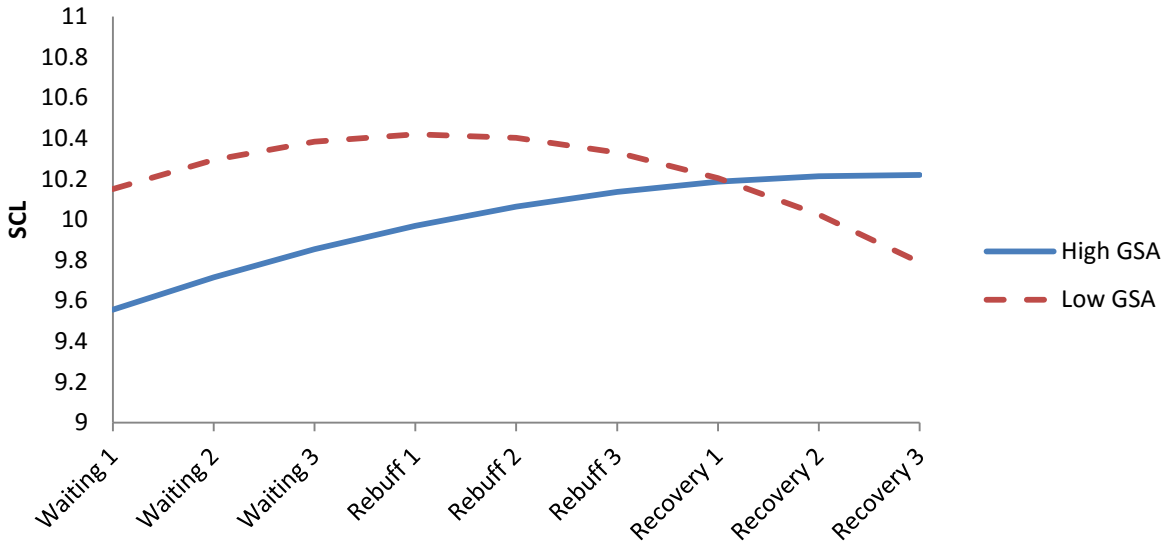

Figure 5. Average growth in skin conductance level (SCL) across the second stage of social stress for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of global social anxiety.

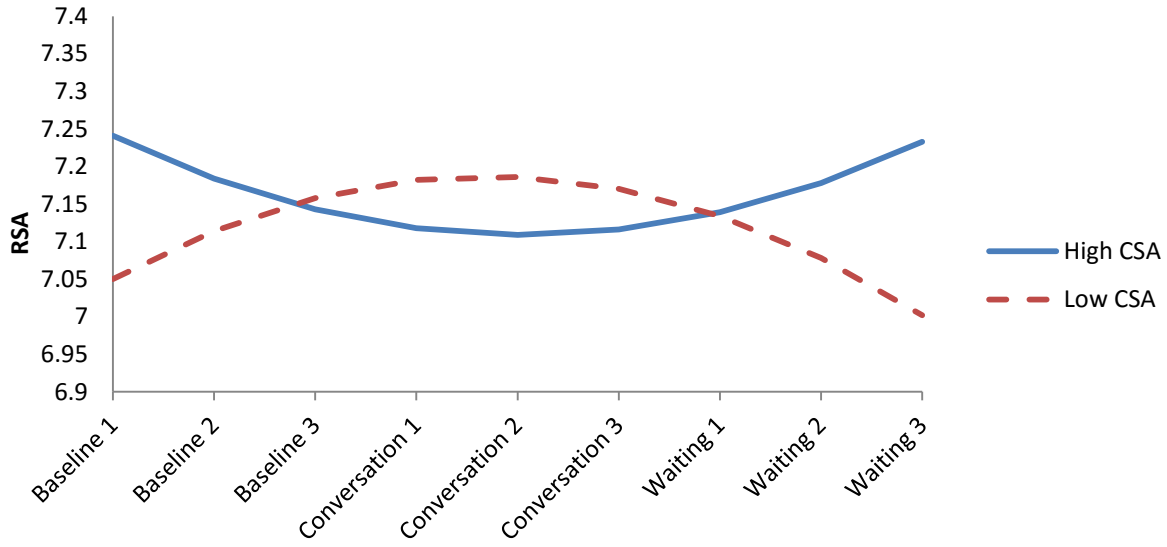

Figure 6. Average growth in respiratory sinus arrhythmia (RSA) across the first stage of social stress for prototypical preadolescents at high (+1*SD*) and low (-1*SD*) levels of context-specific anxiety.