Autonomic physiology of social anxiety in preadolescence

by

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Abstract

The present study sought to build on prior research by examining within-system (sympathetic nervous system (SNS) x SNS) and cross-system (SNS x parasympathetic nervous system) interactions between measures of autonomic reactivity to social stress as predictors of social anxiety. The physiological responses (respiratory sinus arrhythmia, RSA; skin conductance level, SCL; and pre-ejection period, PEP) of 123 early adolescents (*Mage* = 12.03 years) were measured continuously during a lab protocol designed to simulate common peer evaluation experiences. Reactivity scores were examined as predictors of social anxiety, as were the interactions between autonomic reactivity scores. Preadolescents completed measures of global social anxiety and real-time, context-specific social anxiety during the peer-evaluative stress protocol. Analyses revealed only a trend-level association between RSAR and global social anxiety, consistent with previous findings of blunted RSAR among socially anxious youth. In addition, whereas cross-system interactions were not associated with either measure of social anxiety, the interaction between PEPR and SCLR was associated with context-specific social anxiety. A significant positive association was observed between PEPR and context-specific social anxiety at higher levels of SCLR but not lower levels of SCLR. Similarly, a significant positive association was observed between SCLR and context-specific social anxiety at higher levels of PEPR but not lower levels of PEPR. An increase in SCL and shortening of PEP may reflect SNS hyperreactivity to social-evaluative stress and contribute to concurrent social anxiety.

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List of Abbreviations

ANS	Autonomic nervous system
BAS	Behavioral activation system
BIS	Behavioral inhibition system
HPA	Hypothalamic -pituitary-adrenal (axis)
HR	Heart rate
PEP	Pre-ejection period
PEPR	Pre-ejection period reactivity
PNS	Parasympathetic nervous system
RSA	Respiratory sinus arrhythmia
RSAR	Respiratory sinus arrhythmia reactivity
SA	Social anxiety
SAM	Sympathetic-adrenal-medullary
SCL	Skin conductance level
SCLR	Skin conductance level reactivity
SNS	Sympathetic nervous system

I. INTRODUCTION

Social anxiety is one of the most common disorders and forms of psychopathology worldwide, with rates typically peaking in early to middle adolescence (Biedel & Turner, 2007). Anxiety disorders, and elevated levels of social anxiety specifically, are associated with significant impairment in academic, social, and emotional functioning across childhood and adolescence (Kingery, Erdley, Marshall, Whitaker, & Reuter, 2010). Social anxiety is particularly debilitating in social situations and is often associated with self-isolation and withdrawn or avoidant behaviors among children and adolescents (Biedel & Turner, 2007).

The relationship between social anxiety and impairment across a variety of domains can be explained, in part, by the mediating role of physiological responses to stress which shape behavioral output in challenging or stressful contexts (Beauchaine, 2001; Porges, 2007). Several theories posit that abnormal autonomic activity is characteristic of individuals with high levels of anxiety and may be related to many aspects of anxious symptomology (Beauchaine, 2001; Friedman, 2007; Gray & McNaughton, 2000; Porges, 2007). However, the precise nature of the association between autonomic activity and anxiety remains unclear and support for these theories has been mixed.

Research has not determined the extent to which anxiety is characterized by abnormal autonomic functioning (e.g., Beauchaine, 2001; Porges, 2007) or normative autonomic activity and abnormal cognitions or behaviors (e.g., Mauss et al., 2013). This picture is even less clear with regards to social anxiety, since there is a relative paucity of research on autonomic functioning in individuals with elevated levels of social anxiety or social phobia. Fortunately, this body of literature is growing, and researchers have started to examine the psychophysiology of social anxiety using a variety of measures of the autonomic nervous system (ANS).

The three measures of ANS activity that are commonly used when examining associations between physiological functioning and various forms of psychopathology are respiratory sinus arrhythmia (RSA), electrodermal activity or skin conductance level (SCL), and pre-ejection period (PEP; see below for more detail). While some have studied associations between psychopathology and physiological functioning in individual systems (e.g., Schmitz, Kramer, Tuschen-Caffier, Heinrichs, & Blechert, 2011; Schmitz, Tuschen-Caffier, Wilhelm, & Blechert, 2013), recent studies have started to use increasingly complex analytical methods such as examining cross- and within-system interactions as predictors of internalizing symptoms (e.g., El-Sheikh, Erath, Buckhalt, Granger & Mize, 2008; El-Sheikh et al., 2013; Philbrook, Erath, & El-Sheikh, 2018; Wadsworth et al., 2019). Accordingly, the goal of the present study was to build on the extant literature by examining interactions between three commonly-used measures of ANS activity as predictors of social anxiety in a sample of preadolescents.

Psychophysiology – The autonomic nervous system and its subdivisions

Polyvagal theory describes the autonomic nervous system (ANS) as a physiological system that provides rapid shifts in arousal that facilitate adaptive and flexible responses to changes in the environment (Porges, 2007). The ANS itself is comprised of two different branches: the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). The PNS and SNS play opposing yet complementary roles in the body and often work together to meet the demands of a given environment or situation. The PNS is commonly referred to as the “rest and digest” or “feed and breed” system while the SNS is most often remembered for its role in “fight-or-flight” behaviors.

Parasympathetic nervous system

The PNS serves as a "brake" (via the vagus or 10^{th} cranial nerve) that decelerates heart rate and promotes calmness, attentional focus, and social engagement under normal, non-threatening circumstances (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 PNS activity (Porges, 2007). In the context of challenging environmental demands, decreased parasympathetic input - or vagal withdrawal – facilitates increases in arousal (e.g., an increase in heart rate) which allow for sustained attention and increased engagement. However, when this response is insufficient, further activation of the SNS may occur, which facilitates mobilization or inhibition (depending on the circumstances), and, at the extreme, fight-flight-or-freeze responses (Beauchaine, 2001; Porges, 2007).

Sympathetic nervous system

Though a reduction in vagal output can, in and of itself, increase arousal and mobilize metabolic resources to meet environmental demands, the SNS plays a critical role in facilitating adaptive behavioral responses in threatening or challenging environmental contexts. The SNS acts as a metaphorical gas pedal which complements the "vagal brake" described above. Vagal withdrawal can be conceptualized as taking one's foot off the brake, while activation of the SNS can be thought of as stepping on the gas pedal. This allows for larger shifts in arousal and further mobilization of metabolic resources to meet the demands of an increasingly challenging or threatening environment. Thus, as opposed to being two independent and contradictory branches of the ANS, the PNS and SNS act as complementary systems that support flexible behavioral output.

Two measures of SNS activity are commonly used by researchers who study ANS functioning. The first measure is electrodermal activity or skin-conductance level (SCL), which reflects shifts in the level of electrical conductance across the surface of the skin due to changes in sweating (typically measured on the palm of the hand). SCL is a well-validated index of the behavioral inhibition system (BIS; Gray, 1987), a brain network that inhibits or interrupts behavior in the face of possible threat or punishment and plays a major role in the neuropsychology of anxiety (Beauchaine, 2001; Gray & McNaughton, 2000). High SCL-reactivity (SCLR) to stressors may reflect anxious arousal, whereas low SCLR may reflect fearlessness or impulsivity in challenging or threatening circumstances (Beauchaine, 2001). However, moderate-to-high SCLR may also reflect engagement and contextually appropriate inhibitory control efforts (Sheppes, Catran, & Meiran, 2009), which may explain why the association between anxiety levels and SCLR tends to vary considerably across studies.

The second common measure of SNS activity is pre-ejection period (PEP). PEP is a measure of the contractile force of the left ventricle and β-adrenergic input to the heart (Beauchaine, 2001). More specifically, it is a measure of the amount of time between the start of ventricular depolarization – or QRS complex – and the moment of aortic valve opening. Shorter PEP reflects higher levels of β-adrenergic influences on the heart while longer PEP reflects lower levels of β-adrenergic activity and less SNS activation. PEP is unchanged by vagal blockade (Newlin & Levenson, 1979), which suggests that RSA and PEP are measures of distinct aspects of cardiac functioning (i.e., changes in heart rate vs. changes in contractile force, respectively).

When measured in response to a carefully selected reward or incentive, PEP may serve as a peripheral marker of mesolimbic dopamine activity and an index of the behavioral activation system (BAS; Gray, 1987; Beauchaine, 2001). The BAS is sensitive to approach/reward situations, which suggests that the SNS facilitates responses to appetitive/rewarding situations as well as punishments, threats, and negative challenges. However, when PEP is measured in the context of stressful or challenging situations, it is no longer considered a “pure” measure of mesolimbic dopaminergic activity (Beauchaine, 2001) and may instead serve as a more general measure of β-adrenergic influences on the heart and an index of effortful task engagement (Elliot, Dweck, & Yeager, 2017; Kelsey, 2012). A number of studies have examined PEP-reactivity (PEPR) in response to stressors or challenges (e.g., Alkon, Boyce, Neilands, & Eskenazi, 2017; Coulombe, Rudd, & Yates, 2019; McLaughlin et al., 2014; Stifter, Dollar, & Cipriano, 2011; Suurland et al., 2018), while others have measured PEPR in response to a reward or incentive (e.g., Beauchaine, Gatzke-Kopp, & Mead, 2007; Beauchaine et al., 2013; Hinnant et al., 2016). Unfortunately, studies that examine PEP as a predictor of psychosocial functioning are relatively scarce compared to studies that use measures such as RSA or SCL. In addition, PEP has not been examined as a predictor of social anxiety, highlighting the importance of the present study.

Existing theoretical and empirical literature

Several theories posit that high levels of anxiety may be associated with abnormal parasympathetic activity and autonomic inflexibility, especially in childhood and adolescence and in response to stressors (Beauchaine, 2001; Porges, 2007). Although these theories typically refer to more general forms of anxiety, the extant literature on social anxiety seems to partially support this notion, with some studies finding evidence for blunted physiological reactivity and recovery among socially anxious individuals, as well as hyperarousal that may be limited to baseline measures. In children and adolescents, elevated social anxiety has been associated with blunted cardiac and parasympathetic responding to social stress (Nikolić, de Vente, Colonessi, &

Bögels, 2016; Schmitz et al., 2011; 2013), as well as elevated baseline levels of cardiac and electrodermal activity (e.g., Asbrand, Blechert, Nitschke, Tuschen-Caffier, & Schmitz; 2016; Kramer, Asbrand, & Tuschen-Caffier, 2012; Nikolić et al., 2016; Schmitz et al., 2011; 2013).

In a particularly relevant study, Schmitz et al. (2011) found that children with Social Phobia exhibited higher baseline levels of SNS activation (i.e., electrodermal activity), higher heart rates, and lower levels of PNS activity (i.e., low basal RSA levels). They also found evidence for blunted parasympathetic reactivity to a social stress task and a slower heart rate recovery following the task relative to healthy control children. These results were later corroborated by the same research group using a sample of children with subclinical levels of anxiety, such that children who were high in social anxiety demonstrated higher baseline sympathetic activity, blunted cardiac and parasympathetic reactivity to a speech task, and a slower heart rate (HR) recovery after the speech task (Schmitz et al., 2013).

In contrast to these studies, a number of researchers have found that socially-anxious and non-anxious individuals exhibit similar patterns of autonomic activity in young adult (college-aged) and adult samples (e.g., Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014; Mauss, Wilhelm, & Gross, 2003). In addition, many have observed a biased perception of bodily symptoms in individuals with social anxiety (Anderson & Hope, 2009; Schmitz, Blechert, Kramer et al., 2012; Mauss et al., 2003; Klumbies et al., 2014), which could indicate that people with high levels of social anxiety are simply more aware of physiological changes in response to social stress and do not necessarily exhibit different patterns of autonomic activity/reactivity. Modest and inconsistent associations between social anxiety and psychophysiology also seem to parallel the broader literature on emotion response coherence, in which self-reports of emotion are either not associated or only modestly associated with individual physiological measures

(Evers et al., 2014; Hollenstein & Lanteigne, 2014). Thus, it is unclear whether physiological differences among socially anxious individuals only exist in childhood and adolescence, do not exist at all, or if contradictory findings are the result of methodological differences or limitations across studies.

In an attempt to address some of these inconsistencies, a growing number of researchers have utilized multiple measures of ANS activity when examining associations between social anxiety and physiological functioning (e.g., Kaeppler & Erath, 2017; Schmitz et al., 2011; 2013). The psychophysiology of social anxiety may be too complex to be explained by activity or dysfunction within a single physiological system, and several researchers have argued for the use of a multi-system or interactive analytical approach. For example, Bauer et al. (2002) assert that "substantial advances can be made by investigating patterns of physiological responses among multiple, concurrent systems rather than individual response systems." In line with this thinking, multi-system analyses would constitute a more detailed and comprehensive examination of the link between social anxiety and physiological functioning than analyses of activity within an individual system alone.

Several studies have utilized this analytic approach and examined cross-system and within-system interactions as predictors of psychosocial adjustment (e.g., Clark, Skowron, Giuliano, & Fisher, 2016; El-Sheikh, Erath, Buckhalt, Granger, and Mize, 2008; El-Sheikh et al., 2009; El-Sheikh, Keiley, Erath, & Dyer, 2013; Philbrook et al., 2018; Salomon, Matthews, & Allen, 2000; Stifter et al., 2013). Examples of cross-system models include interactions between parasympathetic and sympathetic branches of the ANS (e.g., RSA x SCL; see El-Sheikh et al., 2009; 2013; Philbrook et al., 2018), while examples of within-system models include interactions between hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medullary

(SAM) activity (Wadsworth et al., 2019) as well as HPA activity and SCL (e.g., El-Sheikh et al., 2008). Surprisingly, while there is ample evidence that multiple types of arousal inherently relate and contribute to anxious symptomatology (McNaughton & Gray, 2000), cross- and within-system analyses are uncommon and relatively new compared to single-system analyses. In addition, studies that examine these types of interactions as predictors of social anxiety are essentially non-existent. Accordingly, the goal of the present study was to examine interactions between three measures of ANS reactivity (RSAR, SCLR, and PEPR) as predictors of social anxiety among a sample of preadolescents.

Cross-system coordination: Reciprocal vs. non-reciprocal autonomic responses

Even though each component of the ANS can operate somewhat independently and affects physiological responses in different ways, there is growing evidence that reciprocal cross-system responses to stressors or challenges are associated with better psychosocial functioning than non-reciprocal or conflicting responses (e.g., El-Sheikh et al., 2009; Erath & El-Sheikh, 2015; Philbrook et al., 2018; Salomon, Matthews, & Allen, 2000; Stifter et al., 2013). Reciprocal responses occur when multiple, often opposing systems (e.g., PNS and SNS) operate in a synergistic fashion to produce coordinated shifts in arousal (e.g., both systems increase arousal), while non-reciprocal responses occur when these systems produce conflicting or opposing output. Non-reciprocal or conflicting responses may indicate dysfunction or poor physiological regulation, especially in the context of anxiety-provoking situations (Beauchaine et al., 2007; El-Sheikh et al., 2009; Philbrook et al., 2018).

To date, much of the literature on reciprocal vs. non-reciprocal autonomic responses has focused on interactions between the PNS and SNS, as measured by RSA and SCL. This is perhaps unsurprising since the PNS and SNS exert relatively opposing influences on arousal and

RSA and SCL are the most commonly-used measures of each of these systems (Beauchaine, 2001; Porges, 2007). In these studies, increases in RSA and decreases in SCL (or vice versa) reflect reciprocal responses while concurrent increases or decreases in both RSA and SCL reflect non-reciprocal or conflicting responses. As expected, studies have shown that reciprocal response patterns are more adaptive than non-reciprocal responses and are associated with better psychosocial functioning in most cases (e.g., El-Sheikh et al., 2009; Erath & El-Sheikh, 2015; Philbrook et al., 2018)

However, several studies found that the pattern of effects varied as a function of sex and risk factors such as marital conflict (e.g., El-Sheikh et al., 2013; Philbrook et al., 2018). For example, El-Sheikh et al. (2013) measured autonomic activity in response to a star-tracing task and found evidence for a 4-way interaction between sex, marital conflict, RSA, and SCL. Their analyses revealed that girls with either low basal RSA coupled with low basal SCL, or the combination of increasing RSA and decreasing SCL in response to a stress task (i.e., reciprocal PNS activation), reported higher levels of anxiety and depression in the context of elevated marital conflict. In contrast, Philbrook et al. (2018) found that non-reciprocal responses to the same star tracing task (i.e., co-activation of PNS and SNS) were predictive of increases in internalizing and externalizing symptoms in the context of high levels of marital conflict. These somewhat inconsistent findings suggest that it is important for researchers to control for variables that may influence results, or, if they have the power to do so, consider potential moderating variables and test complex interactions.

Within-system coordination: Reciprocal vs. non-reciprocal sympathetic responses

In contrast to the findings outlined above, the few studies that have examined within-system interactions as predictors of anxiety or internalizing symptoms have typically found that reciprocal SNS responses are maladaptive rather than adaptive (El-Sheikh et al., 2008; Wadsworth et al., 2019; see Bauer et al., 2002 for a theoretical overview). For example, El-Sheikh et al. (2008) found a positive association between basal cortisol levels and internalizing as well as externalizing problems among children with higher SCL, but not among children with low SCL. Similarly, Wadsworth et al. (2019) found that the combination of high SAM and HPA reactivity scores was related to higher levels of internalizing problems while the combination of low SAM and HPA recovery scores predicted elevated externalizing problems. Thus, it appears that reciprocal SNS responses (potentially reflecting excessive SNS activity/reactivity) and non-reciprocal PNS x SNS responses are related to elevated internalizing symptoms.

Unfortunately, very few (if any) studies have examined interactions between PEP and SCL or RSA and PEP. This makes it difficult to draw inferences directly from the literature about the ways in which reciprocal or non-reciprocal patterns of cardiac (i.e., RSAR x PEPR) and SNS (i.e., SCLR x PEPR) responses relate to psychosocial functioning and social anxiety in particular. However, based on the findings from the literature outlined above, a reasonable hypothesis is that reciprocal SNS responses (i.e., high SNS reactivity across measures) and non-reciprocal PNS x SNS responses will be related to high levels of social anxiety. In the case of SNS hyper-reactivity (i.e., high PEPR and high SCLR), excessive arousal in the context of a social-evaluative stress task may increase preadolescents' social anxiety by directing attention away from the task at hand and toward threat cues or internal cues/symptoms such as a rapid heart rate. In partial contrast, non-reciprocal PNS x SNS responses could contribute to high levels of social anxiety by providing ambiguous physiological signals and limited support for responses to social stress.

The Present Study

There is now evidence that analyses of cross- and within-system ANS interactions may provide a more nuanced and detailed examination of the psychophysiology of internalizing symptoms (El-Sheikh et al., 2008; 2013; Philbrook et al., 2018, Wadsworth et al., 2019). Although a considerable portion of the extant literature has focused on interactions between RSA and SCL (or the PNS and SNS), interactions between other measures of ANS reactivity (e.g., PEPR x SCLR) may enhance understanding of the psychophysiology of internalizing symptoms. Accordingly, the goal of the present study was to test main and interactive effects of RSA reactivity (RSAR), SCL reactivity (SCLR), and PEP reactivity (PEPR), on measures of social anxiety.

By utilizing several well-validated measures of autonomic functioning that have been thoroughly described in the literature (i.e., RSA, PEP, and SCL), the present study addresses pressing questions about the psychophysiology of social anxiety. In addition, measurement of ANS responses during a peer-evaluative social stress protocol in the present study may better reflect actual social challenges faced by socially anxious youth than other tasks that are commonly used in the literature, such as speech or non-social tasks. Indeed, there is considerable evidence that interactive situations that include evaluation and mild negative social feedback (e.g., instances of peer rebuff) are especially common and salient during adolescence (Somerville. 2013; Westenberg et al., 2007). Furthermore, previous studies of associations between ANS activity and anxiety have also typically utilized young-adult (e.g., college-aged) or adult samples, thereby limiting the extent to which researchers are able to address the development of internalizing disorders among children and adolescents. We built on the existing literature by examining autonomic activity and social anxiety during a developmental period in which social anxiety is particularly common and influential for children (i.e., during the transition from late childhood to early adolescence).

The present study also advanced the existing literature by utilizing two different measures of social anxiety: a commonly-used “global” measure and a real-time or “context-specific” measure (see Kaeppler & Erath, 2017 and Su, Pettit, & Erath, 2016 for other descriptions/applications of context-specific measures of social anxiety). Typically, social anxiety is assessed using global measures that are administered under normal, relaxed conditions. However, social information-processing theory posits that social information is context-specific (Pettit and Mize, 2007), which means that the level of social anxiety that is captured by context-specific measures may differ from that which is captured by commonly-used global measures. For example, in the context of ongoing social stress, it is possible that preadolescents’ social anxiety levels may be exacerbated relative to the levels of anxiety captured by global measures. Thus, context-specific and global measures of social anxiety may be used to complement each other and to examine whether significant findings replicate across measures of social anxiety.

In the present study, we hypothesized that there would be a positive association between PEPR and social anxiety at higher levels of SCLR but not lower levels SCLR. In addition, we hypothesized that there would be a positive association between RSAR and social anxiety at lower levels of SCLR or PEPR and a negative association at higher levels of SCLR or PEPR. Put another way, we hypothesized that SNS hyper-reactivity (i.e., high SCLR and high PEPR) and non-reciprocal PNS x SNS responses (i.e., high RSAR and low SLCR/PEPR, or vice versa) would be related to higher levels of social anxiety.

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. Participants were also asked to rate how nervous/anxious they were about the conversation activity immediately prior to the peer evaluation period (i.e., the 3-minute conversation activity).

Following the peer evaluation period, participants were asked to rate how anxious/nervous they were during the conversation activity. They also 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, preadolescents were asked to sit quietly for 3 minutes, similar to the baseline period (this is referred to as the recovery period). Following the recovery period, physiological equipment was removed, and participants were carefully debriefed using a process debriefing procedure, informed by Underwood (2005).

Measures

Social Anxiety. Social anxiety (SA) was assessed in two ways. As a measure of global social anxiety, preadolescents completed the Social Anxiety Scale for Adolescents (SAS-A; La Greca and 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 = 0.92). At the context-specific level, social anxiety 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*) immediately 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 ($r = 0.58$, $p < 0.001$) and averaged to create a lab-based, context-specific measure of social anxiety (Kaeppler & Erath, 2017; Su, Pettit, & Erath, 2016).

Physiological assessment. RSA, SCL, and PEP were measured continuously in 1-minute intervals during acclimation (5 minutes), resting baseline (3 minutes), speaking baseline (reading aloud with an RA; 3 minutes), peer evaluation (3 minutes), waiting (3 minutes), peer rebuff (3 minutes), and recovery (3 minutes) periods. 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.

Respiratory Sinus Arrhythmia (RSA). 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. RSA scores were quantified using the spectral analysis method (Berntson et al., 1997) with MindWare HRV analysis software as the natural log of the variance in heart period within age-adjusted respiratory frequency bands (e.g., .23-.50 for 11-12 year-olds; Shader et al., 2018), expressed in units of $\ln(ms^2)$. Very few artifacts were detected and corrected manually using standard procedures (Berntson et al., 1997). RSA reactivity (RSAR) refers to the residualized change score from the pre-task period (i.e., resting baseline period) to the peer-evaluative stress period. The residualized change score is the residual of the regression of RSA during the social stress task on pre-task RSA (Burt & Obradovic, 2013). In the present study, we multiplied RSAR values by negative 1, such that higher RSAR scores indicate greater reductions in RSA (i.e., greater vagal withdrawal) while lower RSAR scores indicate blunted PNS reactivity or vagal augmentation from the pre-task period to the peer-evaluative stress period.

Skin Conductance Levels (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. To account for initial (i.e., pre-task) values when examining reactivity, SCLR was computed as the residualized change score from the pre-task period to the peer-evaluative stress period, such that higher SCLR scores indicate greater increases in SCL in response to peer-evaluative stress.

Pre-ejection Period (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 measured in milliseconds (ms). To account for initial (i.e., pre-task) values when examining reactivity, PEPR was computed as the residualized change score from the pre-task period to the peer-evaluative stress period. In the present study, we multiplied PEPR values by negative 1, such that higher PEPR scores indicate a greater shortening of PEP while lower PEPR scores indicate a lengthening of PEP (i.e., a decrease in β-adrenergic influences on the heart) in response to peer-evaluative stress.

Demographic variables. Sex, ethnicity, and grade level were represented by dichotomous variables (male = 0, female = 1; non-African American = 0, African American = 1; 5th grade = 0, 6th 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*).

III. RESULTS

Descriptive statistics and correlations are shown in Table 2. On average, preadolescents reported moderate levels of global social anxiety (*M* = 42.48 out of a possible score of 90) and context-specific social anxiety (*M* = 2.77 out of a possible score of 5). In addition, paired sample t-tests showed that, on average, preadolescents exhibited significant reactivity from the baseline period to the social-evaluative stress task for each of our measures of ANS reactivity. More specifically, preadolescents exhibited a decrease in RSA, $t(119) = 7.21$, $p < .001$, a shortening of PEP (i.e., decrease in PEP), $t(112) = 3.87$, $p < .001$, and an increase in SCL, $t(107) = -15.55$, $p < .001$, from baseline to the stress task. Two outliers were identified for PEPR (>3SD), and analyses were conducted including outliers and with the outliers re-coded to the next highest value. No significant differences emerged between analyses, so results are not reported in the following sections.

In line with previous studies on anxiety and internalizing symptoms, we found a significant positive correlation between sex and global social anxiety, such that girls reported higher levels of global social anxiety than boys. In addition, there were positive correlations between sex and RSAR as well as PEPR, which suggests that girls exhibited higher levels of RSAR and PEPR than boys. Similarly, we found a positive correlation between income and RSAR, indicating that preadolescents in families with higher household incomes exhibited higher levels of RSAR. Ethnicity and SCLR were negatively correlated, such that African American participants exhibited lower SCLR than non-African American participants.

Regression models were fit using AMOS (which allows for Full Information Maximum Likelihood estimation with missing data; Arbuckle, 2009) to test independent associations linking measures of reactivity for each physiological variable with two measures of social anxiety. Interactive effects were also explored to examine cross-system (i.e., RSAR x PEPR or RSAR x SCLR) and within-system coordination (i.e., PEPR x SCLR) for all autonomic measures of reactivity. Continuous control and predictor variables were mean-centered, and the following demographic variables were included in all analyses: sex, ethnicity (non-African American vs. African American), grade, and household income.

Each regression analysis included 1) all demographic variables, 2) reactivity scores for a given combination of autonomic measures (i.e., RSAR and SCLR, RSAR and PEPR, or SCLR and PEPR), 3) the interaction between these reactivity scores, and 4) a measure of context-specific or global social anxiety as the outcome variable. For significant interactions, simple intercepts and slopes were computed according to standard procedures for interaction effects (Dearing & Hamilton, 2006). Slopes represent associations between a given measure of autonomic reactivity (e.g., PEPR) and social anxiety at higher (+1SD) and lower (-1SD) levels of reactivity in another measure of autonomic functioning (e.g., SCLR). To fully describe interactions, we also tested associations between the autonomic reactivity variable initially designated as the moderator and social anxiety at higher and lower levels of the autonomic reactivity variable initially designated as the predictor (Roisman et al., 2012).

Independent associations

After controlling for demographic variables, PEPR and SCLR were unrelated to preadolescents' global social anxiety. However, a trend-level association emerged between RSAR and global social anxiety, β = -.161, B = -.168, SE = .099, p = .094, such that lower RSAR (i.e., lower vagal withdrawal) was associated with higher global social anxiety (and vice versa). In contrast, all three measures of autonomic reactivity (RSAR, PEPR, and SCLR) were unrelated to context-specific social anxiety. To conserve space, statistics for null results are not presented in text (see Table 3 for regression results).

Interactive associations predicting global social anxiety

None of the interactions between measures of autonomic reactivity were associated with global social anxiety. Null results are not presented in text, though model statistics are presented in Table 3.

Interactive associations predicting context-specific anxiety

Only the interaction between PEPR and SCLR emerged as a significant predictor of context-specific anxiety, β = .252, B = .016, SE = .005, p = .004, explaining 5.7% of the variance above and beyond control variables and main effects. Simple slopes analyses revealed a significant positive association between PEPR and context-specific social anxiety at higher levels of SCLR, β = .261, B = .047, SE = .014, p = .001, and no association between PEPR and context-specific social anxiety at lower levels of SCLR, β = -.093, B = -.016, SE = .015, p = .258 (see Figure 1). To further elucidate the nature of the interaction between PEPR and SCLR, we conducted simple slopes analyses for the association between SCLR and context-specific social anxiety at high (+1SD) and low (-1SD) levels of PEPR. These analyses also revealed a positive association between SCLR and context-specific social anxiety at high levels of PEPR, β = .336, B = .139, SE = .044, p = .001, and no association at lower levels of PEPR, β = -.132, B = -.051, SE = .052, p = .329.

IV. DISCUSSION

The goal of the present study was to examine independent and interactive associations between measures of autonomic reactivity and social anxiety in preadolescence. Novel elements of the study include assessments of physiological reactivity to peer-evaluative stress, analyses of interactions within (i.e., PEPR x SCLR) and across (i.e., RSAR x PEPR and RSAR x SCLR) physiological systems (i.e., SNS and PNS), and measures of context-specific and global social anxiety. Results provided some support for hypotheses but also suggested that the psychophysiological of social anxiety requires further study.

Consistent with previous findings of blunted vagal withdrawal among socially anxious youth (e.g., Schmitz et al., 2011; 2013), we found a trend-level negative association between RSAR and global social anxiety when controlling for relevant demographic variables (see Table 3), which suggests that lower levels of vagal withdrawal are marginally associated with higher levels of global social anxiety (or vice versa). In contrast, we found a trend-level positive correlation between RSAR and context-specific social anxiety (see Table 2), though bivariate associations should be interpreted cautiously. Thus, associations between RSAR and social anxiety may differ as a function of the type of measure (e.g., context-specific vs. global).

In addition to these findings, interactive analyses revealed a positive association between PEPR and context-specific social anxiety at higher levels of SCLR, but no association between PEPR and context-specific social anxiety at lower levels of SCLR. Similarly, follow-up analyses indicated that high SCLR is associated with higher context-specific social anxiety at higher levels of PEPR but not at lower levels of PEPR. Thus, the highest levels of context-specific social anxiety were apparent when both PEPR and SCLR were elevated (see Figure 1), providing support for the hypothesis that SNS hyper-reactivity is related to higher levels of social anxiety. A concurrent increase in SCL and shortening of PEP reflects SNS-mediated arousal that may direct attention toward internal (e.g., sweating, heart rate) or external (e.g., negative facial expression) cues of threat. Although the present study did not include measures of preadolescents' attention (e.g., through eye-tracking), increased attention to threat cues and internal cues/somatic symptoms has been linked with elevated social anxiety and increased SNS activity across several studies (e.g., Barry, Vervliet, & Hermans, 2015; Buckner, Maner, & Schmidt, 2010; Heeren, Peschard, & Philippot, 2012; Klumpp & Amir, 2009; Staugaard, 2010).

Whereas higher reactivity across SNS measures was associated with concurrent anxiety in the context of social evaluation, SNS-mediated increases in arousal are not necessarily maladaptive. Indeed, studies have found that SCLR to peer-evaluative stress may reflect engagement and underlie socially competent behavior among socially anxious preadolescents (e.g., Kaeppler & Erath, 2017) and reflect inhibitory control efforts during emotional challenge tasks (Sheppes et al., 2009). Rather, our findings suggest that the combination of heightened reactivity across multiple dimensions of SNS functioning (i.e., SNS hyper-reactivity) carries psychological risk, at least insofar as this response is related to context-specific social anxiety. These results are consistent with several other studies in which SNS hyper-reactivity (e.g., the coupling of high HPA-axis and SAM reactivity) was related to higher levels of anxiety or internalizing symptoms (El-Sheikh et al., 2008; Wadsworth et al., 2019). However, the present study is the first to examine the interaction between PEPR and SCLR as a predictor of social anxiety (or any measure of anxiety or internalizing symptoms for that matter), and it will be important to test for replication in other studies.

Our analyses also showed that direct associations between social anxiety and individual measures of ANS activity are tenuous and inconsistent. This is consistent with studies that find limited evidence for direct effects between measures of social anxiety and individual physiological variables (e.g., Mauss et al., 2003). The present study did find marginal evidence for an association between blunted RSA withdrawal and higher levels of global social anxiety, which is consistent with two key studies by Schmitz et al. (2011; 2013), but no significant associations between individual physiological variables and either measure of social anxiety emerged.

Associations between measures of physiological functioning and social anxiety may differ across studies for several reasons. First, the type of stress task used to elicit physiological responses may impact the results of a given study. For example, Schmitz et al. (2011; 2013) used the Trier Social Stress Task to study autonomic reactivity among preadolescents, while we used a social-evaluative stress task that may elicit different physiological or emotional responses. Second, the decision to examine measures of baseline activity, reactivity to stress, or recovery from stress may account for different results across studies. In the studies by Schmitz et al. (2011; 2013), many significant findings emerged in analyses of baseline levels of ANS activity and physiological recovery. In contrast, we focused exclusively on measures of ANS reactivity and interactions between these measures. Future research should provide a more comprehensive analysis of the relationship between social anxiety and baseline measures of ANS functioning as well as physiological recovery.

Results of the present study also point to the significance of the social anxiety measurement approach. As noted above, results partially confirmed the hypothesis that SNS hyper-reactivity would be related to higher levels of social anxiety, but only through analyses that included a context-specific rather than global measure of social anxiety. Important differences may exist between commonly-used global measures of social anxiety and real-time measures of social anxiety collected during exposure to social-evaluative challenge or stress. The correlation between the context-specific and global measures of social anxiety was non-significant in the present study ($r = .13$, see Table 2); however, another study with a similar social-evaluative stress protocol reported a modest positive correlation between context-specific and global measures of social anxiety ($r = .31$; Su et al., 2016). The benefit of using a global measure of social anxiety is that measures are well-validated and refer to a wide range of times and circumstances, enhancing generalizability, compared to real-time, context-specific measures. On the other hand, global measures may not accurately reflect the level of social anxiety that individuals experience in the context of specific, salient social challenges. Accordingly, an important direction for future research will be to elucidate the physiological correlates and developmental significance of context-specific measures of social anxiety as compared to global measures.

The finding that SNS hyper-reactivity is related to context-specific but not global social anxiety also provides some evidence bearing on questions about response coherence across physiological and subjective measures of emotion (see Evers et al., 2014). Many studies that have examined associations between social anxiety and psychophysiological variables have assessed physiological responses in a certain time and context, but assessed subjective anxiety under relaxed conditions with global measures that refer to a variety of times and situations (e.g., Asbrand et al., 2016; Klumbies et al., 2014; Kramer et al., 2012; Mauss et al., 2003; Nikolić et al., 2016; Schmitz et al., 2011; 2013). Evers et al. (2014) posited that mismatched assessments of physiological and subjective emotional responses is one reason for inconsistent results in studies of response coherence. Indeed, we found a significant interactive association linking physiological and subjective measures of emotional responses when responses were assessed concurrently in the same social-evaluative context, but not when social anxiety was measured globally. However, several hypothesized associations between physiological responses and context-specific social anxiety did not emerge, and there are a variety of other perspectives on the nature of response coherence and reasons for inconsistencies across studies (see Hollenstein & Lanteigne, 2014 for an overview). Thus, our results should be interpreted as one piece of evidence that contributes to the broader literature.

In contrast to hypotheses and results of prior studies (e.g., El-Sheikh et al., 2009; 2013; Philbrook et al., 2018), interactions between measures of PNS and SNS reactivity were not associated with global or context-specific social anxiety in the present study. Inconsistencies across studies could be due to differences in the type of task that was used to assess reactivity (e.g., star-tracing vs. social-evaluative stress task) or the outcome measures that were used (i.e., context-specific and global social anxiety vs. generalized anxiety and internalizing symptoms). In addition, PNS x SNS interactions may be related to internalizing symptoms, or social anxiety specifically, under certain environmental conditions or primarily among girls or boys. For example, Philbrook et al. (2018) found that non-reciprocal PNS x SNS reactivity to a star-tracing task (i.e., increases in RSA and increases in SCL) predicted elevated internalizing symptoms only among teens who reported exposure to relatively high levels of marital conflict. Similarly, El-Sheikh et al. (2013) found that PNS x SNS interactions predicted internalizing symptoms only among girls who experienced high levels of marital conflict. Accordingly, an important direction for future research will be to examine whether PNS x SNS interactions predict social anxiety in particular stress or demographic contexts. Such analyses require large samples due to the statistical power needed for reliable detection of subgroup differences or complex interactions.

Despite its advancements, the present study has several limitations that should be addressed. First, baseline measurements used in regression analyses were likely influenced by some level of anticipatory stress, limiting variability in physiological reactivity that could be linked with social anxiety. Indeed, in a prior study with the first cohort of the present sample, we found that global social anxiety was associated with higher cardiovascular arousal (higher heart rate, lower RSA) beginning during the baseline period and continuing throughout the stress periods, though only at high levels of peer victimization experiences (Erath, Tu, & El-Sheikh, 2012). To address this limitation, future studies should attempt to utilize baseline measures that are devoid of anticipatory stress.

It should also be noted that all analyses were cross-sectional and 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 social anxiety, or if the association is bidirectional.

In addition, the social-evaluative stress task used in the present study may elicit only mild to moderate stress responses as well as moderate levels of context-specific anxiety, on average. Additional research is needed to determine if similar results would be observed in the context of social stress tasks that are more-anxiety provoking and elicit larger physiological responses. To reconcile differences across studies, it would also be informative to compare results across a variety of stress tasks (e.g., cognitive tasks, mild social stress tasks, and highly anxiety-provoking social stress tasks) within the same study. Differences in physiological responses across levels of social anxiety may be dependent upon the type of task that is used, similar to what was found in a study comparing ANS reactivity among patients with current or remitted depression and anxiety to healthy controls (Hu et al., 2016).

Finally, the present study used a community sample, and it is possible that findings would not generalize to preadolescents with clinical levels of social anxiety. However, the mean total score on the SAS-A (i.e., 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 (Olivares et al., 2002). Thus, the present study represents a wide range of levels of social anxiety and the sample contains a substantial proportion of highly anxious youth when compared to samples in related studies.

To our knowledge, this study is the first examination of interactions between measures of autonomic reactivity to social stress as predictors of social anxiety. If our findings are replicated, results could be informative for psychophysiological models of social anxiety, as researchers in this area have only recently begun to utilize a multi-system, interactive analytic approach. The methods of the present study are also applicable to the study of other forms of psychopathology. Future research would likely benefit from a shift towards an interactive, multi-system examination of psychophysiological responses to stress. Such research will contribute to an increasingly sophisticated understanding of the role of physiological responding in the development and maintenance of various forms of internalizing psychopathology, and social anxiety in particular.

Findings from the present study may also have important clinical implications. For example, SNS hyper-reactivity to social stressors (i.e., high levels of reactivity across multiple SNS measures) could play a role in the development of anxiety disorders by contributing to an increase in preadolescents' context-specific social anxiety and influencing social functioning or attention to threat cues. Indeed, an important follow-up to the present study will be to examine the impact of context-specific social anxiety on preadolescents' attention (e.g., through eye-tracking), social behavior (e.g., through measures of social competence), and risk of developing an anxiety disorder. In addition, our results could prove useful for biofeedback protocols aimed at teaching preadolescents to modulate SNS-mediated arousal in real time, which, in turn, may regulate anxious symptoms. However, further research will be required before firm conclusions can be drawn about the relationship between social anxiety and ANS activity.

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Table 1 – *Defining Reciprocal and Non-Reciprocal Responses*

	Reciprocal vs. Non-Reciprocal	Effects on Overall Arousal	General Effects on the Body	Other Terms
PNS x SNS Interactions				
Decrease in RSA x Decrease in SCL	Non-reciprocal	Little-or-no change	Increase in HR, decrease in sweating	Autonomic coinhibition
Decrease in RSA x Increase in SCL	Reciprocal	Increase	Increase in HR, increase in sweating	Reciprocal sympathetic activation
Increase in RSA x Decrease in SCL	Reciprocal	Decrease	Decrease in HR, decrease in sweating	Reciprocal parasympathetic activation
Increase in RSA x Increase in SCL	Non-reciprocal	Little-or-no change	Decrease in HR, increase in sweating	Autonomic coactivation
SNS x SNS Interactions				
Lengthening of PEP x Decrease in SCL	Reciprocal	Decrease	Weaker contractions of the heart, decrease in sweating	Reciprocal SNS coinhibition
Lengthening of PEP x Increase in SCL	Non-reciprocal	Little-or-no change	Weaker contractions of the heart, increase in sweating	-
Shortening of PEP x Decrease in SCL	Non-reciprocal	Little-or-no change	Stronger contractions of the heart, decrease in sweating	-
Shortening of PEP x Increase in SCL	Reciprocal	Increase	Stronger contractions of the heart, increase in sweating	Reciprocal SNS coactivation
Cardiac Interactions				
Decrease in RSA x Lengthening of PEP	Non-reciprocal	Little-or-no change	Increase in HR, weaker contractions of the heart	-
Decrease in RSA x Shortening of PEP	Reciprocal	Increase	Increase in HR, stronger contractions of the heart	Reciprocal cardiac upregulation
Increase in RSA x Lengthening of PEP	Reciprocal	Decrease	Decrease in HR, weaker contractions of the heart	Reciprocal cardiac downregulation
Increase in RSA x Shortening of PEP	Non-reciprocal	Little-or-no change	Decrease in HR, strong contractions of the heart	-

Note: SCL reflects electrical conduction across the surface of the skin rather than sweating per se. Similarly, PEP may be interpreted as the strength of left ventricular contraction or speed of a given contraction (as opposed to stronger vs. weaker contractions per se). RSA = respiratory sinus arrhythmia (units = ln[ms2]), SCL = skin conductance level (units = μS), PEP = pre-ejection period (unit = ms), HR = heart rate.

Table 2

Descriptive Statistics and Correlations Between Study Variables

	1	2	3	4	5	6	7	8	9
1. Sex	-								
2. Ethnicity	.06	-							
3. Grade	-.07	-.46**	-						
4. Income	.02	-.52**	.25**	-					
5. GbSA	.19*	.07	-.17+	-.17+	-				
6. CSA	.12	.17+	.07	-.02	.13	-			
7. RSAR	.22*	-.11	.11	.24*	-.15	.15+	-		
8. PEPR	.20*	.13	-.14	-.09	.05	.09	.14	-	
9. SCLR	-.17	-.31**	.03	.18+	-.07	-.01	-.03	-.19+	-
Mean (SD)	50% (.50)	35% (.48)	.61 (.49)	4.13 (1.55)	42.48 (14.5)	2.77 (1.12)	.00 (.77)	.00 (6.73)	.00 (3.09)
Skew	.02	.64	-.46	-.45	.36	.40	.47	1.64	.70

Note: 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); GbSA = global social anxiety; CSA = context-specific anxiety; RSAR = respiratory sinus arrhythmia reactivity (units = ln[ms2]); PEPR = pre-ejection period reactivity (unit = ms); SCLR = skin conductance level reactivity (units = μS); $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$

Table 3

Independent and Interactive Effects Between Study Variables

	Social Anxiety		Context-Specific Anxiety	
	B (SE)	*β*	*B (SE)*	*β*
Model 1: Controls				
Sex	.31 (.14)*	.19*	.26 (.20)	0.11
Ethnicity	-.19 (.15)	-.11	.66 (.21)**	.28**
Grade	-.28 (.14)+	-.17+	.45 (.20)*	.19*
Income	-.10 (.05)*	-.18*	.05 (.06)	0.07
R^2	11%		13%	
Model 2: Main effects				
RSAR	-.17 (.09)+	-0.16+	.20 (.13)	0.13
PEPR	-.00 (.01)	-0.01	.01 (.02)	0.06
SCLR	-.01 (.02)	-0.03	.03 (.03)	0.09
Model 3: Interaction effects				
RSAR x PEPR	-.01 (.01)	-.11	.00 (.02)	0.02
ΔR^2/ Total R^2	.2% / 13.3%		0% / 13.4%	
RSAR x SCLR	.01 (.04)	0.03	-.04 (.06)	-.07
ΔR^2/ Total R^2	0% / 13.4%		.2% / 16.3%	
PEPR x SCLR	.00 (.00)	0.08	.02 (.01)**	.25**
ΔR^2/ Total R^2	1.2% / 12.6%		5.7% / 22.1%	

Note: Male = 0, Female = 1; non-African American = 0, African American = 1; 5th grade = 0, 6th grade = 1, respectively); RSAR = respiratory sinus arrhythmia reactivity (units = ln[ms2]); PEPR = pre-ejection period reactivity (unit = ms); SCLR = skin conductance level reactivity (units = μS); Coefficients are presented at step of entry; Main effects were entered one autonomic system at a time (e.g., RSAR, PEPR, and then SCLR) and each interaction was tested in a separate model; +p < .10, *p < .05, **p < .01.

Figures

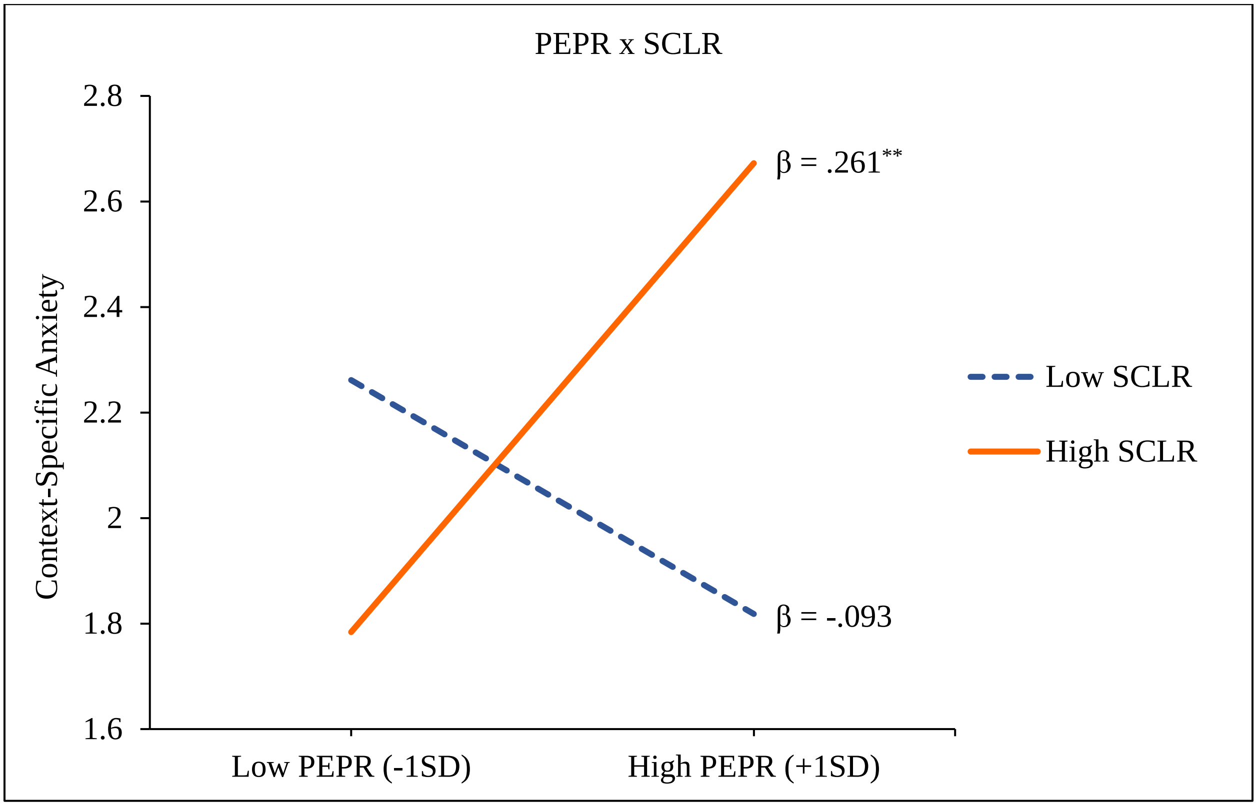

Figure 1. The interaction between PEP reactivity and SCL reactivity predicting context-specific anxiety; +p < .10, *p < .05, **p < .01.