Too Stressed to Sleep: Autonomic Nervous System Reactivity and Sleep in Adolescents

By

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Abstract

The purpose of the present study was to investigate relations between autonomic nervous system (ANS) reactivity across its parasympathetic and sympathetic branches and multiple sleep parameters in adolescence. Participants were 244 adolescents (M_{age} = 15.79 years old, SD = 9.56 months; 67.2% White/European-American, 32.8% Black/African-American). Parasympathetic activity was indexed by respiratory sinus arrhythmia withdrawal (RSA-r) and sympathetic activity was indexed by skin conductance level reactivity (SCL-r), which were examined during the day in the lab and in response to a moderate stressor (the star tracing task). Sleep was examined with actigraphs in adolescents' homes for 7 consecutive nights and the following sleep parameters were derived: minutes, efficiency, and long wake episodes (LWE). Linear and non-linear relations between RSA-r or SCL-r and sleep as well as the moderating role of child sex in all examined relations were explored. Regression analysis showed that more RSA withdrawal (lower levels during task than baseline) was associated with shorter sleep, and more SLC-r (higher levels during task than baseline) was associated with shorter sleep, lower sleep efficiency, and more LWE. Assessments of quadratic effects indicated that an average level of SCL-r was associated with longer sleep, while low and high levels of SCL-r were associated with shorter sleep. Additionally, the negative association between SCL-r and sleep efficiency and the positive association between SCL-r and LWE accelerated as SCL-r increased. Finally, moderation analysis showed that associations between RSA withdrawal and SCL-r and sleep minutes and efficiency were significant only for boys. In general, results illustrate that higher

daytime physiological reactivity (increased RSA withdrawal and SCL-r) is negatively associated with sleep duration and quality for adolescents.

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Introduction

Sleep is a crucial bioregulatory system that affects children's mental and physical health. Research has shown that short and poor-quality sleep are associated with negative cognitive and academic (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010), emotion regulation (Palmer, Oosterhoff, Bower, Kaplow, & Alfano, 2018), and physical health (Knutson, 2012; Paiva, Gaspar, & Matos, 2015) outcomes. Similarly, sleep problems have been linked with socioemotional maladjustment, such as internalizing and externalizing symptoms (Gregory & Sadeh, 2012; Kelly & El-Sheikh, 2014; Shochat, Cohen-Zion, & Tzischinsky, 2014). These associations between sleep and physical and mental health outcomes have been found using various methodologies, such as subjective (e.g., Palmer et al., 2018) and objective (e.g., El-Sheikh, Saini, Gillis, & Kelly, 2019) measures of sleep, as well as cross-sectional (e.g., Paiva et al., 2015) and longitudinal (e.g., Tétreault, Bernier, Matte-Gagné, & Carrier, 2019) designs, suggesting robust relationships.

Despite the critical role sleep plays in optimal functioning, a striking number of adolescents do not sleep enough. Research has shown that up to 25% of adolescents obtain less than six hours of sleep on school nights (Roberts, Roberts, & Duong, 2009), compared to the 8 to 10 hours recommended by the National Sleep Foundation (Hirshkowitz et al., 2015). Keyes and colleagues (2015) reported that the sharpest decline in rates of nearly-adequate sleep duration (i.e., seven hours) occurs around the age of 15, when only 24-25% of adolescents regularly obtain adequate sleep. One major explanation for insufficient sleep during adolescence is a circadian rhythm shift, paired with early school start times (Carskadon, 2011). As children move into adolescence, their sleep phase is shifted to later in the evening due to later melatonin release.

This later sleep phase, combined with early school start times, leaves a small window for adolescents to obtain restorative sleep.

Beyond this circadian shift that affects sleep among all adolescents, individual differences in stress exposure and stress reactivity may contribute to variability in adolescent sleep. That is, some adolescents are even more susceptible to sleep problems due to excessive stress exposure and maladaptive reactions to that stress that are incompatible with the calmness required for sleep (Dahl, 1996). For example, levels of peer stress increase in early adolescence, and early adolescents who experience more peer victimization report heightened sleep problems over time (Tu, Spencer, El-Sheikh, & Erath, 2017). In addition, some youth struggle to negotiate autonomy and control with their parents (Smetana & Daddis, 2002) or report increasing academic stress (Carskadon, 2011). These external stressors may incur wear and tear on the body and dysregulate biological systems, such as basic stress response systems and sleep (McEwen & Stellar, 1993). Adolescents may be particularly sensitive to stressors because of various hormonal changes and rapid synaptic pruning in areas of the brain associated with emotional and cognitive processes, such as the limbic system and prefrontal cortex (PFC; Eiland & Romeo, 2013; Spear, 2000). Therefore, it follows that stress exposure and responses may contribute to variability in adolescent sleep.

The autonomic nervous system (ANS) is a key component of the stress response system that controls visceral organs and metabolic resources. Despite the centrality of the ANS in stress responding, very few studies have examined associations between reactivity in the parasympathetic (PNS) and sympathetic (SNS) branches of the ANS and sleep in adolescence. Building on a scant body of evidence, the present study examined associations between adolescents' daytime PNS and SNS reactivity to a well-established lab stressor (i.e., star-tracer;

Mirror Tracer; Lafayette Instrument Co., Lafayette, IN; (Philbrook, Erath, Hinnant, & El-Sheikh, 2018; Stanger, Abaied, Wagner, & Sanders, 2018) and their objective sleep duration and quality, as measured by actigraphy. The star-tracing task is moderately challenging and frustrating and mirrors the kind of cognitive and emotional challenge that adolescents commonly encounter at school, thereby capturing a pattern of ANS responsivity that may resemble adolescents' stress responses outside of the lab. Actigraphy allows for non-invasive, objective sleep measurement in the home, and can estimate multiple sleep parameters with accuracy (Sadeh, 2015). The current study used actigraphy to assess both sleep duration (indexed by actual sleep minutes) and sleep quality (indexed by sleep efficiency and long-wake episodes; LWE) along a continuum in a community sample of adolescents.

Literature Review

Rationale and Conceptual Framework Linking the ANS and Sleep

The ANS has two branches: The PNS, known for rest and digest functions, and the SNS, known for inducing fight, flight or freeze behaviors. These two branches work together to control internal organs and involuntary bodily functions, such as cardiovascular and electrodermal activity, which provide biological resources to adequately respond to stressors and adapt to the environment. Adolescents with less adaptive ANS responses to normative, acute or chronic stress during the day may experience prolonged stress and disruptions in bioregulation during the night, which can be reflected in disrupted sleep (Bagley, Kelly, Buckhalt, & El-Sheikh, 2015; El-Sheikh & Buckhalt, 2005).

Concurrent dysregulation of the ANS responses and sleep can be explained with reference to the central autonomic network (CAN). As discussed by Thayer and Lane (2009), the CAN has control over the heart via the vagus nerve, which is under inhibitory control via

neurons from the PFC area. These same neurons from the PFC also apply inhibitory control on the amygdala, preventing consistent biologically- and socially-taxing sympathetic or excitatory activity that disrupts sleep. Importantly, Walker and van der Helm (2009) noted that the regulation of sleep and other physiological arousal systems are all connected by areas in the PFC. Because of shared PFC networks that govern both sleep and ANS functioning, dysregulation in one of these bioregulatory systems may be related to dysregulation in the other. Indeed, it is important to note that while the present study conceptualizes ANS reactivity as a predictor of sleep, this relationship is likely bidirectional. That is, while less optimal physiological stress responses may affect sleep, poor sleep may increase stress levels and maladaptive responses to stress (Meerlo, Sgoifo, & Suchecki, 2008).

PNS and Sleep

Conceptual models and empirical evidence suggest that PNS activity may be related to sleep. In normal, non-threatening situations, the PNS applies a "brake" via the myelinated vagal nerve, which helps the body maintain calmness and homeostasis by slowing heart rate and reducing physiological arousal (Porges, 2007). According to the Polyvagal Theory, the ANS generally responds first to stressors with the more evolutionarily recent system, the ventral vagus, and if such a response is not sufficient, the more primitive and energy demanding SNS is engaged (Porges, 2007). This tonic control on the heart, via the ventral vagus, is known as vagal tone. Respiratory sinus arrhythmia (RSA), a non-invasive measure of how heart rate varies during spontaneous breathing, is a good measure of vagal tone and may reflect emotion regulation at the physiological level (Porges, 2007).

In threatening or stressful situations, the "vagal brake" can be withdrawn, which increases metabolic rates and arousal and thereby provides resources (e.g., blood flow, oxygen)

to cope with stress. This is known as vagal withdrawal or suppression, characterized by decreases in RSA from baseline in response to a stressor. Conversely, vagal augmentation, which refers to increases in RSA from baseline to challenge (i.e., RSA augmentation), is characterized by a slowing of heart rate, and may reflect disengagement that undermines coping with challenge or stress (Moore & Calkins, 2004).

Importantly, high vagal tone under normal circumstances and moderate vagal withdrawal during a challenging or threatening situation may reflect flexible and adaptive responses to the environment. Specifically, research has shown that in community samples, higher vagal tone and more vagal withdrawal to stressful events is associated with lower levels of internalizing and externalizing problems (Beauchaine, 2001; El-Sheikh, Harger, & Whitson, 2001), as well as better cognitive performance (Forman-Alberti & Hinnant, 2016; Scrimin et al., 2019). However, research has shown that excessive vagal withdrawal can reflect dysregulation and unabated sympathetic influence, and may be related to high levels of anxiety (Beauchaine, 2001).

A higher level of RSA withdrawal has been associated with better child behavioral and psychological outcomes (Graziano & Derefinko, 2013; Hastings et al., 2008). Although evidence is scant, there is some support for an association between RSA withdrawal to challenges and sleep. For example, adults who experienced acute sleep deprivation exhibited less RSA withdrawal to a go/no-go task, n-back task, and reaction time task (Zhong et al., 2005). Furthermore, depressed and non-depressed adults who exhibited lower levels of RSA withdrawal to a speech task and cold exposure, had poorer subjective sleep quality (Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014). This association also appears in children: More RSA withdrawal to a reaction time task has been linked with less subjective sleepiness, more total sleep time, and less sleep activity (El-Sheikh & Buckhalt, 2005). Additionally, infants who

exhibited less RSA withdrawal during the Still Face Paradigm had more maternal-reported sleep problems at 18 months compared to children who exhibited more RSA withdrawal (Gueron-Sela et al., 2017).

Of note, however, some inconsistent findings have been reported in studies of RSA reactivity and sleep. That is, one study with undergraduate students found that greater reductions in high-frequency HRV (another measure of PNS activity analogous to RSA) in response to worry induction was related to more sleep disturbances over the course of a semester (MacNeil et al., 2017). Another study found that less RSA withdrawal was associated with less sleep activity for 9-year-old children, in the context of maternal depression (Keller, Kouros, Erath, Dahl, & El-Sheikh, 2014). To our knowledge, no study has examined associations between RSA reactivity and sleep during adolescence, when sleep duration decreases and social and environmental demands increase.

Even less research has examined how baseline RSA and RSA reactivity can work together to predict sleep. One study found that children with both lower vagal tone and more vagal withdrawal exhibited poorer sleep quality (El-Sheikh, Erath, & Bagley, 2013). These results are consistent with the possibility that extremely low RSA levels in the context of stress reflects dysregulation (Beauchaine, 2001). That is, the combination of low baseline vagal tone and further vagal withdrawal may produce cardiac over-arousal, which may be related to high anxiety.

SNS and Sleep

The SNS is commonly known for serving the "fight, flight, or freeze" functions in the presence of a threat by providing physiological resources, such as increased heart rate and oxygen flow throughout the body (Boucsein, 1992). Skin conductance level (SCL) refers to

electrodermal activity that is governed by sweat glands connected to the ANS via cholinergic fibers in the SNS, with no PNS input (Boucsein, 1992; Fowles, 1986). SCL is a measure of the behavioral inhibition component of the SNS and reflects anxious arousal related to risk assessment and sensitivity to potential negative consequences (Beauchaine, 2001). SCL reactivity, or SCL-r, refers to increases in electrodermal activity from resting to challenging conditions, and therefore increased SNS activation.

Research has shown that higher SCL-r is associated with anxiety (Weems, Zakem, Costa, Cannon, & Watts, 2005), fearfulness (Fowles, Kochanska, & Murray, 2000), and inhibitory behaviors and responses (Matthys, van Goozen, Snoek, & van Engeland, 2004). Conversely, lower SCL-r may be indicative of under-arousal or disinhibition that underlies some externalizing symptoms and conduct problems (Gregson, Tu, & Erath, 2014; Hinnant, Erath, Tu, & El-Sheikh, 2016). In addition, some research suggests that lower baseline SCL (SLC-b) and lower SCL-r are associated with depression, representing affect blunting and loss of motivation (El-Sheikh & Arsiwalla, 2011).

Past research has found a bidirectional association between sleep and SNS activity (Meerlo et al., 2008). Specifically, when individuals have increased sympathetic arousal, they may experience increases in sleep latency and decreases in sleep quality. In turn, this poor sleep may increase stress levels, and therefore sympathetic arousal, again affecting sleep (Meerlo et al., 2008). Despite this logical association, remarkably few studies have assessed how SNS reactivity during waking hours and sleep are related for adolescents.

Some research has shown that sleep-deprived adults experience less arousal as measured by skin resistance level (the inverse of SCL; Miró, Cano-Lozano, & Buela-Casal, 2002), although other studies have illustrated the opposite. Indeed, one study found that after 24 hours

of sleep deprivation, participants had significantly higher SNS reactivity, as measured by SCL-r to a difficult perception task with false feedback, compared to participants with adequate sleep (Liu, Verhulst, Massar, & Chee, 2015). In addition, a study with college undergraduates showed that both normal and troubled sleepers with higher SCL-r to stress-elicitation tasks reported poorer sleep, as measured by self-reported sleep latency, number of night awakenings, length of night awakenings, and sleep duration (Waters, Adams, Binsk, & & Varnado, 1993). That is, more arousal, as measured by higher SCL-r, appeared to interfere with sleep quality and duration.

In addition to SCL, a few studies have assessed how cardiac pre-ejection period (PEP), another measure of SNS activity, and sleep are related in children. One study, with a sample of 11-year-olds, found that lower PEP (i.e., more SNS activity) during a modified Trier Social Stress Task was associated with poorer sleep indicated by more LWE, sleep activity, and worse sleep efficiency (Bagley & El-Sheikh, 2014). Moreover, it has been found that children that have better sleep show greater increases in PEP over time (i.e., decreased SNS activity; El-Sheikh, Hinnant, & Philbrook, 2017). Taken together, these results suggest that greater SNS reactivity to stress, as characterized by lower PEP levels, is associated with poorer sleep.

Based on an extensive literature review, only one study to our knowledge has assessed the direct relation between SCL-r and sleep in children. Results showed that school-aged children with higher SCL-r to a modified Stroop Test exhibited poorer sleep behaviors such as sleepwalking and nightmares (Fisher & Rinehart, 1990). Thus, the small number of studies assessing SCL and sleep suggest that stronger SCL reactivity to challenges may be incompatible with sleep.

Exploratory Sex-Related Moderation Effects

Girls and boys tend to report different emotional responses to stress. In general, empirical evidence shows that adolescent girls report seeking more social engagement and instrumental support in response to stress compared to boys (Donaldson, Prinstein, Danovsky, & Spirito, 2000; Ryan & Shim, 2012). Adolescent girls also tend to ruminate on stressors more, have stronger emotional expressions of stress, and have more emotional intensity compared to boys (for review see El-Sheikh & Buckhalt, 2005; Rose & Rudolph, 2006). These emotional and psychological responses to stress may be outward expressions of physiological responses to stress (Porges, 2007).

Gender differences in psychological responses to stress may affect or reflect gender differences in bioregulatory activity. One study found that older adolescent girls ruminated more than boys, which negatively affected girls' sleep through depressive symptoms (Chow, Homa, & Amersdorfer, 2017). Additionally, another study showed that girls had significantly greater developmental increases (pre- and post-puberty) in cortisol responses to the Trier Social Stress Test and star tracing task compared to boys, whose cortisol responses stayed relatively stable across development (Stroud, Papandonatos, D'Angelo, Brush, & Lloyd-Richardson, 2017). Because of gender differences in emotional responses to stress, and the connection between emotion and sleep, gender differences in the association between adolescents' ANS reactivity and sleep may exist.

Exploratory Non-Linear Effects

As noted above, past results generally indicate that more RSA withdrawal and less SCL-r are associated with better sleep for both adults (Bylsma et al., 2014; Waters et al., 1993) and children (Fisher & Rinehart, 1990; Gueron-Sela et al., 2017). However, there have been some mixed results, particularly with RSA withdrawal as some studies have found that less RSA

withdrawal is associated with better sleep (Keller et al., 2014; MacNeil et al., 2017). Additionally, lower SCL-r may reflect a less active behavioral inhibition system characterized by fearlessness and insensitivity to negative consequences (Fowles et al., 2000), which may contribute to conduct problems and antisocial behavior (Frick & Morris, 2004). These results suggest potential risks of extremely low and high RSA withdrawal and SCL-r, such that moderate physiological reactivity may be most conducive to sleep.

Current Study

The present study examined associations between adolescents' RSA and SCL reactivity to the star tracing task and multiple sleep parameters measured with actigraphy in a community sample. Most of the existing studies on ANS activity and sleep have included younger children or adults. However, adolescents may face heightened risk for sleep problems because of normative biological changes (e.g., hormonal, neural, circadian shift) as well as the increased social and environmental demands. Developmental changes in stress and sleep during adolescence call for research to examine associations between daytime physiological regulation and sleep problems (short duration, less efficiency, and more LWE), thus building on the scant body of work with younger children.

In line with research suggesting that a higher level of vagal withdrawal to moderate stress tends to be associated with better sleep in children (e.g. El-Sheikh, Hinnant, & Erath, 2015), we hypothesized that greater RSA withdrawal would be associated with longer sleep duration (measured in minutes) and better sleep quality (measured as higher sleep efficiency and fewer LWE) in adolescents. Additionally, in line with research suggesting that higher SCL-r is associated with poorer sleep in children (e.g. Fisher & Rinehart, 1990), we hypothesize that greater SCL-r would be associated with fewer sleep minutes, less sleep efficiency, and more

LWE in adolescents. Tests of gender as a moderator of these associations as well as tests for non-linear associations between physiological reactivity and all sleep parameters were considered exploratory and no hypotheses were proposed. Consistent with the suggestion that multiple sleep parameters should be used to accurately depict sleep (Sadeh, 2015), we used actigraphy to measure sleep duration (actual sleep minutes) and sleep quality (sleep efficiency, and LWE).

Method

Participants

The current study included participants from the fourth wave of the Family Stress and Youth Development: Bioregulatory Effects project, a larger longitudinal study, which was approved by the university's institutional review board. The analytic sample (n = 244) included adolescents who had data for either the predictor (RSA and SCL) or the outcome (actigraphic sleep). The analytic sample was comprised of 52.9% girls and 47.1% boys, with a mean age of 15.79 years old (SD = 9.56 months). Representative of the small town and semi-rural southeastern community from which they were sampled, 67.2% of the adolescents were White/European-American (EA), and 32.8% are Black/African-American (AA).

Procedures

Youth sleep was examined with actigraphs during the regular school year, excluding holidays. Actigraph watches were delivered to the adolescents' home and they were instructed to place the watch on their non-dominant wrist before going to sleep for 7 consecutive nights. Adolescents also completed a sleep diary and sleep log in order to corroborate actigraphy data. Youth visited the laboratory to examine their ANS reactivity during the star tracer task (Mirror Tracer; Lafayette Instrument Co., Lafayette, IN). There was an average of 4.02 (SD = 12.33) days between the last night of watch wear and the adolescents' lab visit.

To examine RSA, disposable electrodes were placed on the adolescents' torso to measure electrocardiogram (ECG) activity and respiration. To assess SCL, two electrodes were placed on the palm of the non-dominant hand. After a three-minute adaptation period during which adolescents were asked to sit quietly to adjust to the lab, baseline RSA and SCL were assessed for three minutes while the adolescent once again sat quietly in the lab. Then, they performed a star-tracing task for three minutes, which involved tracing the shape of a star while looking at its reflection in a mirror (Mirror Tracer; Lafayette Instrument Co., Lafayette, IN). The task is moderately stressful and has been used to evoke ANS reactivity in youth including RSA-r (Liew, Johnson, Smith, & Thoemmes, 2011; Philbrook et al., 2018) and SCL-r (Allen & Matthews, 1997; Erath, El-Sheikh, Hinnant, & Cummings, 2011).

Measures

RSA.

Youths' RSA was collected and analyzed with equipment and software from MindWare Technologies Ltd (Gahanna, OH). Electrocardiogram data were collected via the MW1000A acquisition system (MindWare Technologies) in accordance with standard guidelines (Berntson et al., 1997). Data were sampled at 1000 Hz. RSA scores were quantified using spectral analysis (Berntson et al., 1997) with MindWare HRV analysis software (version 3.0.21) as the natural log of the variance in heart period within the respiratory frequency range (.15-.40 Hz) and expressed in units of $\ln(ms^2)$. The data were inspected for artifacts and missing R peaks on the basis of improbable IBIs. Missing or misplaced R peaks were inserted or altered manually. Data were scored in one-minute intervals and averaged across the three-minute baseline and the three-minute star-tracing task. RSA-r was calculated as a difference score (RSA star-tracing minus

RSA baseline), such that higher RSA-r scores reflect greater RSA augmentation, whereas lower RSA-r scores reflect greater RSA withdrawal.

SCL.

To assess SCL, two silver/silver-chloride (Ag-AgCl) electrodes (1"× 1" foam, 0 % chloride gel) were placed on the non-dominate palm. The electrodes were not allowed to touch and were taped down to ensure a consistent signal. SCL data were sampled at 1000 Hz via the MW1000A acquisition system (MindWare Technologies). SCL data was quantified with MindWare EDA analysis software (Electrodermal Activity Version 3.0.21), where data were analyzed in 1-min intervals (units = microsiemens or μS). SCL was averaged across the 3 min baseline assessment, as well as the 3-minute star-tracing task. SCL-r was computed as a difference score (SCL star-tracing minus baseline SCL), such that positive scores represent an increase in SCL, and therefore increased SNS activity, and negative scores represent decreases in SCL, and therefore decreases in SNS reactivity.

Sleep.

Sleep was assessed using actigraphy, which is a reliable method for estimating sleep duration and quality when used for consecutive nights. Sleep was examined using the validated Motionlogger Octagonal Basic actigraphs (Ambulatory Monitoring Inc., Ardsley, NY.), which measured motion during sleep in one-minute epochs using the zero-crossing mode. Sadeh's scoring algorithm was used to derive the various sleep parameters (Sadeh, Sharkey, & Carskadon, 1994). Additionally, we used sleep diaries to corroborate objective sleep data (Acebo et al., 1999).

In the analytic sample, many adolescents (36.0%) had seven nights of valid actigraphy data, 28.5% of adolescents had six nights, 19.0% had five nights, 8.2% had four nights, 3.7% had

three nights, 1.2% had two nights, .4% had one night, and 3.00% of youth had no usable actigraphy data. Reasons for missing data were primarily forgetting to wear the watch, excluding nights of medication use for chronic illnesses (usually for allergies or respiratory infections), and a few cases of either lack of consistency between actigraphy-based and diary-based sleep onset data (used to validate actigraphy) or actigraph malfunction. Consistent with past suggestions (Meltzer, Montgomery-Downs, Insana, & Walsh, 2012), at least five nights of actigraphy data are needed to ensure adequate estimation of sleep. Therefore, participants' sleep data was not included if they had less than five nights of actigraphy data, and full information maximum likelihood estimation (FIML) was used to handle missing data.

Three sleep variables were derived. Sleep duration was indexed by sleep minutes, which is the number of minutes from actigraphy-based sleep onset to wake time excluding awakenings. Two well-recognized sleep quality parameters were derived: sleep efficiency, or the percentage of epochs (minutes) scored as sleep between sleep onset and waking, and LWE, which refers to the number of scored wake episodes that were longer than five minutes each.

Additional Measures.

To account for possible confounds, we controlled for various demographic factors: age, race, sex, income-to-needs ratio, and BMI. Sex was dichotomized such that 0 = female and 1 = male. Race was also dichotomized such that 0 = EA/White (White) and 1 = AA/Black (AA/Black). Furthermore, age was measured in months. BMI was assessed with a Tanita scale for weight and stadiometer for height, then z-scored. Finally, income-to-needs was derived using mother-reported family income, family size, and the federal poverty guidelines at the time of collection (U.S. Department of Commerce). An income-to-needs ratio of <1 was considered below the poverty line (14.4% of participants), 1-2 was considered at or near the poverty line

(29.6%), 2-3 was considered lower middle class (21.6%), 3-4 was considered middle class (25%), and ≥4 was considered upper middle class (9.3% of participants). Finally, in accordance with the law of initial values, which stipulates that baseline physiological functioning places limits on reactivity capacity, we also controlled for baseline levels of RSA and SCL (Hinnant, Philbrook, Erath, & El-Sheikh, 2018; Wilder, 1967).

Plan of Analysis

Preliminary tests for distributions and correlations were conducted in SPSS. Path models were fit to test the hypotheses in AMOS, which uses FIML estimation. Analyses were performed separately for RSA-r and SCL-r, for each sleep parameter, and for exploratory interactions (i.e., physiological reactivity x sex and non-linear effects of physiological reactivity). Variables were entered in a series of nested path models, rather than simultaneously, so that the main effects of physiological reactivity (the primary aims of the study) are interpretable (i.e., not obscured by the inclusion of interaction terms). Demographic and baseline physiological (RSA or SCL) variables were entered in the first model. A physiological reactivity variable (RSA-r or SCL-r) was added in the second model. In the third model, either the quadratic physiological reactivity term (physiological reactivity squared) or the interaction between physiological reactivity and sex was added (each in a separate model). A fourth model that tested sex as a moderator of the nonlinear effects of physiological reactivity was included but yielded no significant results, and therefore no additional information is presented. Predictor and control variables that were significantly correlated in preliminary analyses were covaried with each other in all models. Coefficients are presented at the model of entry in the tables below.

All continuous predictors and controls were mean-centered for moderation analyses to limit multicollinearity and facilitate plotting. Significant interactions between physiological

reactivity and sex were plotted at +/- 1 *SD* of the predictor (ANS reactivity) using the Preacher interaction utility (Preacher, Curran, & Bauer, 2006) to show the associations between physiological reactivity and sleep variables for girls and boys. The Preacher interaction utility uses a test of simple slopes within regions of significance to determine if slopes are significantly different from zero. Quadratic effects of physiological reactivity were plotted using Microsoft Excel.

Results

Preliminary Results

Adolescents exhibited significant decreases in RSA and increases in SCL from baseline to the star tracing task (RSA $t(221) = 3.81$, $p < .001$; SCL $t(141) = -9.29$, $p < .001$). In the present sample, 53.2% of adolescents exhibited RSA withdrawal, while 46.8% exhibited RSA augmentation. Additionally, 88.7% of youth showed increases in SCL and 11.3% showed decreases in SCL. Boys had significantly fewer sleep minutes ($t(199) = 3.62$, $p < .0001$; M_{Girls} = 419.84 minutes, M_{Boys} = 392.36 minutes), poorer sleep efficiency ($t(199) = 2.77$, $p < .01$; M_{Girls} = 92.13, M_{Boys} = 89.55), and more LWE ($t(199) = -2.30$, $p < .05$; M_{Girls} = 2.06, M_{Boys} = 2.62). However, there were no significant differences between boys and girls for RSA baseline, SCL baseline, RSA-r, or SCL-r. Black/AA youth had higher baseline RSA compared to White youth ($t(220) = -2.08$, $p < .05$; M_{Black} = 7.13, M_{White} = 6.79). Additionally, White youth had higher baseline SCL ($t(149) = 4.99$, $p < .001$; M_{Black} = 5.89, M_{White} = 10.22) and more sleep minutes ($t(199) = 3.03$, $p < .01$; M_{Black} = 388.61 minutes, M_{White} = 414.25 minutes) compared to Black youth.

All means, standard deviations and correlations of main study variables can be found in Table 1. Several demographic variables were correlated with physiological or sleep variables. In

addition to the sex and race differences reported above, continuous demographic variables were correlated with some physiological and sleep variables. Higher income-to-needs ratio was correlated with higher baseline SCL, more sleep minutes, more sleep efficiency, and less LWE. Adolescent zBMI was correlated with higher baseline RSA and decreases in RSA during the star tracer. In addition, several correlations among physiological and sleep variables emerged. SCL-r was positively correlated with LWE and negatively correlated with sleep efficiency. SCL-r^2 was significantly negatively correlated to sleep minutes and sleep efficiency as well as significantly positively correlated with LWE. Sleep minutes was moderately correlated with higher sleep efficiency and lower LWE, and sleep efficiency and LWE were strongly negatively correlated.

RSA-r Predicting Sleep

Direct effects.

All RSA-r models met at least two out of three model fit criteria ($\chi 2$ close to 0 with a non-significant *p*-value, comparative fit index (CFI) ≥ .95, and root mean square error of approximation (RMSEA) ≤ .08; (Hu & Bentler, 1998). Controlling for demographic variables and baseline RSA, RSA-r to the star tracing task was not significantly related to sleep efficiency or LWE. However, RSA-r was significantly related to sleep minutes (B = 10.25, SE = 4.48, p < .05, R^2 = .16; see Table 2 for all model results) such that more RSA withdrawal was associated with fewer sleep minutes.

RSA-r^2.

There were no significant non-linear associations between RSA-r^2 and any objective sleep parameters.

RSA-r x sex.

Assessment of sex as a moderator revealed that there was a significant interaction between RSA-r and sex predicting sleep minutes (B = 24.73, SE = 8.51, $p < .001$, R^2 = .23). The interaction plot and test of simple slopes indicated that there was a significant positive association between RSA-r and sleep minutes for boys, which illustrated that boys who experienced more RSA withdrawal to the star tracing task had fewer sleep minutes (see Figure 1a.) than those who exhibited less RSA withdrawal. There was not a significant association between RSA-r and sleep minutes for girls, who had relatively long sleep regardless of RSA-r. Predicted means illustrated that at higher levels of RSA-r (i.e., RSA augmentation), boys and girls had relatively high and similar number of sleep minutes (M_{Girls} = 423.95 minutes; M_{Boys} = 418.68 minutes). At lower levels of RSA-r (i.e., RSA withdrawal), predicted means show that girls slept 48.27 minutes longer than boys (M_{Girls} = 427.94 minutes; M_{Boys} = 379.67 minutes).

Results also showed that there was a significant interaction between RSA-r and sex predicting sleep efficiency (B = 2.28, SE = 1.10, $p < .05$, R^2 = .20). Test of simple slopes revealed that there was a significant positive association between RSA-r and sleep efficiency for boys, such that boys who experienced more RSA withdrawal had poorer sleep efficiency (see Figure 1b). Again, there was no significant association for girls, who tended to have relatively high levels of sleep efficiency independent of RSA-r (see Figure 2). Predicted means indicated that at higher levels of RSA-r, girls and boys had similar sleep efficiency (M_{Girls} = 92.02; M_{Boys} = 91.16). However, at lower levels of RSA-r, girls had relatively higher sleep efficiency compared to boys (M_{Girls} = 93.10; M_{Boys} = 88.28).

Finally, there was a marginal interaction between RSA-r and sex predicting LWE (B = -.52, SE = .30, $p < .10$, R^2 = .12). However, test of simple slopes did not reveal significant associations between RSA-r and LWE for either boys or girls (see Figure 1c).

SCL-r Predicting Sleep

Direct effects.

All models met previously mentioned fit criteria. Controlling for demographic variables and baseline SCL, SCL-r to the star tracing task was marginally associated with fewer sleep minutes (B = -3.28, SE = 1.99, $p < .10$, R^2 = .17; see Table 3 for all model results), significantly associated with lower sleep efficiency (B = -.93, SE = .23, $p < .001$, R^2 = .18), and significantly associated with more LWE (B = .22, SE = .06, $p < .001$, R^2 = .14).

SCL-r^2.

There was a significant non-linear association between SCL-r^2 and sleep minutes (B = -1.98, SE = .35, $p < .001$). Specifically, at average levels of SCL-r, adolescents had the most sleep minutes ($M_{minutes}$ = 500.32; see Figure 2a), whereas sleep minutes were lower at high ($M_{minutes}$ = 473.21) and low levels of SCL-r ($M_{minutes}$ = 480.96). There was also a significant non-linear association between SCL-r^2 and sleep efficiency (B = -.13, SE = .05, $p < .01$), such that the strength of the negative association between SCL-r and sleep efficiency accelerated as SCL-r increased (see Figure 2b). Predicted means showed the poorest sleep efficiency at very high levels of SCL-r ($M_{efficiency}$ = 76.93) and the highest sleep efficiency at lower SCL-r ($M_{efficiency}$ = 81.47), with similar levels of efficiency at average SCL-r ($M_{efficiency}$ = 80.69). Finally, there was a significant non-linear association between SCL-r^2 and LWE (B = .03, SE = .01, $p < .05$), such that the positive association between SCL-r and LWE accelerated as SCL-r increased (see Figure 2c). Predicted means indicated that at higher levels of SCL-r, youth had the highest amount of LWE (M_{LWE} = 6.69). At lower levels of SCL-r, adolescents experienced the least amount of LWE (M_{LWE} = 5.55) with a similar amount of LWE at average SCL-r (M_{LWE} = 5.81).

SCL-r x sex.

Tests of sex as a moderator indicated that there was a significant interaction between SCL-r and sex predicting sleep minutes (B = -9.09, SE = 3.97, $p < .05$, $R^2 = .21$). Tests of simple slopes revealed that there was a significant negative association between SCL-r and sleep minutes for boys, such that boys who exhibited greater SCL-r had fewer sleep minutes (see Figure 3a). Similar to RSA-r models, there was not a significant association between SCL-r and sleep minutes for girls, such that girls had relatively high sleep minutes regardless of SCL-r. Predicted means showed that at lower levels of SCL-r, girls and boys had similar sleep minutes (M_{Girls} = 420.60 minutes; M_{Boys} = 415.54 minutes). However, at higher levels of SCL-r, girls slept 46.92 minutes longer than boys (M_{Girls} = 426.06 minutes; M_{Boys} = 379.14 minutes).

Additionally, there was a marginal interaction between SCL-r and sex predicting sleep efficiency (B = -.83, SE = .50, $p < .10$, $R^2 = .16$). Tests of simple slopes showed that there was a significant negative association between SLC-r and sleep efficiency, such that boys who exhibited greater SCL-r had less sleep efficiency (see Figure 3b). There was not a significant association between SCL-r and sleep efficiency for girls such that girls had similar levels of sleep efficiency regardless of SCL-r. Predicted means illustrated that at lower levels of SCL-r, girls and boys had similar levels of sleep efficiency (M_{Girls} = 93.63; M_{Boys} = 92.61). In contrast, predicted means indicated that at higher levels of SCL-r, girls had more sleep efficiency than boys (M_{Girls} = 91.43; M_{Boys} = 86.57).

There was not a significant interaction between SCL-r and sex predicting LWE.

Discussion

The present study examined relations between RSA-r and SCL-r to a moderate stressor (star tracing task) and several nighttime sleep parameters (sleep minutes, sleep efficiency, and LWE) in adolescents. In addition, exploratory analyses examined the possibility of adolescent

sex as a moderator of relations as well as non-linear associations between ANS reactivity and sleep. Based on previous literature with children and adults (Bylsma et al., 2014; Fisher & Rinehart, 1990; Gueron-Sela et al., 2017; Waters et al., 1993), we hypothesized that more RSA withdrawal and less SCL-r would be associated with better sleep across all parameters. In line with our hypotheses, linear effects indicated that more SCL-r was associated with less sleep efficiency, less sleep minutes, and more LWE. In addition, non-linear associations were found and mainly showed that associations between SCL-r and poorer sleep (efficiency, LWE) accelerated (i.e., strengthened) at higher levels of SCL-r, further confirming the connection between stronger SNS reactivity to challenge and poorer sleep. However, contrary to hypotheses a higher level of RSA withdrawal was associated in a linear fashion with fewer sleep minutes. Additionally, the linear associations linking RSA withdrawal and SCL-r with sleep minutes and efficiency were significant only for boys. Overall, results indicate that stronger physiological reactions to stressors (i.e., more vagal withdrawal and SCL-r) during the day are associated with poorer sleep quality and quantity for adolescents.

A small number of studies have examined the relationship between ANS responses while awake and sleep in young children and adults. This is the first study, to our knowledge, that examined this association in adolescents. Adolescence is a unique developmental period marked by shifts in sleep, emotion-regulatory capacity, and stress. Adolescents tend to get significantly less sleep compared to children and adults, due to a delayed melatonin release as well as social factors such as early school start times (Carskadon, 2011). Adolescents also experience rapid neuronal changes that widen the gap between intensity of emotional experiences and degree of cognitive control (Spear, 2000). Finally, adolescents have significant shifts in relationships, such that adolescents seek more autonomy from parents and more approval from peers, which can be

sources of significant stress (LaFontana & Cillessen, 2010; Smetana & Daddis, 2002). These developmental changes make adolescence a distinct period that warrants separate study, including in the area of stress responding and sleep.

A direct linear effect was detected between RSA withdrawal with sleep minutes. Contrary to our hypothesis, more RSA withdrawal was associated with shorter sleep. Although this is not consistent with some studies (El-Sheikh & Buckhalt, 2005; El-Sheikh et al., 2015), there has been some support for relations between higher levels of RSA withdrawal and sleep problems in children. For example, Keller and colleagues (2014) found that in the context of maternal depression, less RSA withdrawal was associated with less sleep activity for 9-year-old children. Additionally, one study with college students found that greater reductions in high-frequency HRV in response to worry induction was associated with more subjective sleep disturbances over the course of a semester (MacNeil et al., 2017). The PNS serves rest and digest functions, providing tonic control over the body's arousal systems. Because high vagal tone reduces arousal and inhibits or counteracts SNS activity, extreme vagal withdrawal increases arousal and allows relatively greater SNS influence, potentially leading to feelings of panic and anxiety that interfere with sleep (Bagley et al., 2015; Beauchaine, 2001; Dahl, 1996).

Consistent with our hypothesis, there was a direct linear association between SCL-r and sleep. More SCL-r was associated with shorter sleep, less sleep efficiency, and more LWE. These results build on the adult literature which has illustrated that adults who exhibited higher SCL-r to stress-elicitation tasks reported shorter and poorer quality sleep (Waters et al., 1993). To our knowledge, only one study assessed the direct association between SCL-r and sleep in children (Fisher & Rinehart, 1990), and findings from that investigation are consistent with

present study's results. Specifically, school-aged children with higher SCL-r to a modified Stroop Test had more sleep problems such as sleepwalking and nightmares.

Several non-linear associations between SCL-r and sleep qualified some aforementioned linear relations. Analyses revealed that the negative association between SCL-r and sleep efficiency accelerated as SCL-r increased. Sleep efficiency differed by more than a half standard deviation at high (+ 1 *SD*) SCL-r compared to low (-1 *SD*) SCL-r. A similar pattern of effects was found for LWE, such that the positive association between SCL-r and LWE accelerated as SCL-r increased. Differences in LWE at high versus low SCL-r were almost a full standard deviation, suggesting robust differences. This pattern of results suggests that the more extreme SNS responding during the day is damaging to sleep.

A non-linear association was also found between sleep minutes and SCL-r. However, unlike associations with efficiency and LWE, results indicated that adolescents who exhibited average SCL-r had the longest sleep, while those who exhibited high and low SCL-r had a similar, shorter sleep duration. Predicted means showed that differences in sleep minutes at high SCL-r versus average SCL-r was half a standard deviation, and there was almost half a standard deviation difference in sleep minutes at low SCL-r and average SCL-r.

Past research shows that SNS activation may be beneficial in the context of stress. For example, higher SCL-r was found to protect against externalizing behavior for children in the context of harsh and permissive parenting, as higher SCL-r reflects sensitivity to negative consequences in threatening circumstances (Erath, El-Sheikh, & Cummings, 2009; Hinnant, Erath, Shimizu, & El-Sheikh, 2019). Low ANS reactivity may indicate insufficient signal or physiological resources for effective coping with immediate environmental demands, potentially

prolonging the negative effects of stress, consistent with the non-linear association between SCL-r and sleep minutes.

However, when stress response systems are deployed too often or too intensively, adolescents may experience allostatic load. Adolescents face multiple stressors both internally through shifting biology and externally though school, peer, and family experiences. Adolescents who exhibit strong responses to a moderate stressor, such as the star tracer task, may exhibit similar reactions to frequent stressors that are common in adolescence. Thus, youth with higher RSA withdrawal and SCL-r may experience allostatic load, such that bioregulatory systems that support sleep and other regulatory behaviors become worn down. Despite the apparent benefits in the context of shorter-term stress (Cui et al., 2015), RSA withdrawal in response to a chronic stressor may be damaging. For example, in the context of a chronic stressor (i.e., harsh parenting), children who exhibited more RSA withdrawal to a laboratory stressor showed decreases in vagal tone across time (Hinnant, Erath, & El-Sheikh, 2015), suggesting a decreased ability of the PNS to provide necessary tonic control over arousal systems.

Allostatic load may prolong both psychological and physiological stress, therefore leading to a spillover effect into the night, affecting sleep. Based on the CAN framework put forth by Thayer and Lane (2009), the sleep system and ANS share neuronal networks in the PFC, suggesting that hyper-arousal in the ANS would impact sleep. Indeed, high arousal is incompatible with sleep (Bagley et al., 2015; Dahl, 1996). The present findings further support these propositions and empirical findings by illustrating that high vagal withdrawal and high SNS reactivity interfere with sleep duration and quality for adolescents.

The current study found some support for adolescent sex as a moderator of the linear associations linking RSA withdrawal and SCL-r with sleep. Results showed that, for boys only,

more RSA withdrawal was associated with fewer sleep minutes and less sleep efficiency. Additionally, for boys only, more SCL-r was associated with shorter and less efficient sleep. At higher levels of both RSA withdrawal and SCL-r, boys and girls differed on both sleep minutes and efficiency by almost a full standard deviation, suggesting robust differences. At lower levels of RSA withdrawal and SCL-r, boys and girls had similar sleep minutes and sleep efficiency.

While these interactions were exploratory, there are several possible explanations for why the association between ANS responding and sleep appears stronger for boys. Past research has shown that girls tend to report more stress during adolescence (Hankin, Mermelstein, & Roesch, 2007) and have stronger outward expressions of emotions (El-Sheikh & Buckhalt, 2005; Rose & Rudolph, 2006). Additionally, girls are more likely to seek outside support during times of distress (Donaldson et al., 2000; Ryan & Shim, 2012). Increased reporting, expression, and support-seeking behaviors during stress suggest that girls may be more consciously aware of their stress, which may facilitate more conscious coping with stress. Greater conscious coping with stress may override physiological reactions to stress, such that girls are less affected by stronger ANS responses. Importantly, research has shown that engagement with a stressor can moderate the effects of physiological reactivity. For example, Connor-Smith and Compas (2004) found that higher heart rate reactivity was associated with poorer physical health among adolescents who reported disengaged coping but not engaged coping. Similarly, another study illustrated that there was a significant association between lower RSA withdrawal and lower social competence for youth exhibiting disengaged responses to peer stress, but not engaged responses (Erath & Tu, 2014). Thus, engaged coping responses may dampen the effects of physiological responses on behavioral or biological outcomes.

In contrast to girls, boys exhibit less support seeking behaviors, reporting of stress, and weaker emotional expressions (Donaldson et al., 2000; Hankin et al., 2007; Rose & Rudolph, 2006), opening up the possibility for boys to be more affected by physiological stress responses and for those responses to be prolonged, creating an ANS profile that is likely to affect sleep (Thayer & Lane, 2009). In line with this, future work should attempt to explain sex differences in the associations between physiology and sleep. For example, research might examine if conscious coping strategies do indeed account for sex differences in the association between ANS responding and sleep. Future research might also examine if ANS recovery after a stressor mediates sex differences in associations between physiological reactivity and sleep.

Another possible explanation for sex as a moderator involves differences in sleep duration and quality. Because girls exhibited longer and higher-quality sleep, it is possible that boys stand to benefit more from less severe ANS reactions to stress. That is, compared to boys, girls appear closer to the "ceiling" of good sleep, whereas boys have more room for improvement on the basis of less severe ANS responses to daytime stress. However, it is important to note that research is decidedly mixed on sex differences in the effects of psychophysiology. Thus, future research should aim to replicate the results of the present study to better understand the reliability of the sex differences.

The current results should be considered in the context of its strengths and limitations. The analyses were cross sectional, and therefore no causal conclusions can be drawn about the relationship between ANS responsivity and sleep or the direction of the relationship. In addition, sample characteristics limit generalizability. For example, results may be different in a majority ethnic minority sample because of increased stress from to discrimination (Yip, 2018), or in a sample with younger or older adolescents because of age differences in sleep (Keyes et al.,

2015). While the present study did assess a well-validated moderate stressor, the star tracing task, it is possible that a different stressor, perhaps a peer evaluation task such as the Trier Social Stress Test, would be more salient for adolescents given their strong concerns about social evaluation.

Despite these limitations, there are several notable strengths of the present study. First, sleep was measured objectively with actigraphy, allowing for the assessment of multiple sleep parameters and accurate estimation of sleep (Sadeh, 2015). Second, the present study was based on a large sample size, especially given the assessment of physiological reactivity, and utilized validated measurements for both RSA and SCL. Finally, this is the first study to our knowledge that assessed quadratic effects between physiological reactivity and sleep.

Considering that few studies that have assessed the relationship between ANS reactivity and sleep in general, there are many possible avenues for future research. Including the previously mentioned directions for future research, future studies should assess the relationship longitudinally to further explicate direction of findings as well as to begin exploring causal mechanisms. Importantly, while this study does conceptualize ANS reactivity as the predictor and sleep as the outcome, sleep problems may predict psychophysiology. Indeed, one study found that higher RSA withdrawal in conjunction with higher SCL-r predicted better sleep across time for children, suggesting a benefit of higher physiological reactivity for sleep (Erath & El-Sheikh, 2015), in contrast to results of the present study. Therefore, the present results could be interpreted in the opposite direction: poorer sleep may lead to higher PNS and SNS reactivity.

Indeed, several studies have found that sleep does impact reactivity to stress (Walker, 2009). For example, one study showed that adults who experienced acute sleep deprivation exhibited more SNS arousal (Liu et al., 2015). Additionally, children who experience better sleep

show decreases in SNS arousal (measured by PEP) across time (El-Sheikh et al., 2017). However, research has also shown that poor sleep can predict less physiological arousal. One study showed that adults who experienced acute sleep deprivation exhibited less RSA withdrawal to several stressful tasks (Zhong et al., 2005), suggesting that poor sleep may impact the ability to mobilize biological resources to address a challenge. Concerning SCL, research has illustrated that adults subjected to sleep deprivation experience less SNS arousal (Miró et al., 2002). Considering these results, future research with adolescents should explicate the direction of effects with longitudinal designs.

Additionally, future models should assess sympathovagal balance, exploring how the PNS and SNS together can affect sleep in adolescents. Of note, past work has emphasized the importance of coordination between systems. Erath and El-Sheikh (2015) found that when the PNS and SNS drive arousal in the same direction in the context of a stressor (i.e., higher RSA withdrawal and SCL-r), school-aged children have better quality and longer sleep across time. It is also important to continue to investigate non-linear associations between physiological reactivity and sleep, considering the mixed results in the literature (e.g. Bylsma et al., 2014; Fisher & Rinehart, 1990; MacNeil et al., 2017; Miró et al., 2002). Finally, given that research has shown that both PNS and SNS reactivity are related to adolescent mental health (Beauchaine, 2001; Weems et al., 2005), and that mental health is related to sleep (Gregory & Sadeh, 2016), future work might consider how mental health mediates to the relationship between ANS reactivity and sleep.

Overall, the current findings illustrate that more physiological reactivity during the day negatively impacts objective nighttime sleep for adolescents. This relationship appears to be non-linear for SCL-r, with highest levels SCL-r associated with less sleep efficiency and more LWE.

Interestingly, average levels of SCL-r were associated with the highest levels of sleep minutes suggesting that mild to moderate reactivity may support sleep. Linear associations were moderated by sex, such that both more RSA withdrawal and SCL-r were associated with fewer sleep minutes and less sleep efficiency for boys only. This is the first study, to our knowledge, to explore sex differences and non-linear associations between ANS reactivity and sleep during adolescence, and future research is essential to confirm or challenge the results.

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Table 1.
Correlations, Means, and Standard Deviations of Main Study Variables.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. RSA-b	-													
2. RSA-r	-.29**	-												
3. RSA-r^2	.13	-.63**	-											
4. SCL-b	-.10	-.17*	.05	-										
5. SCL-r	.05	-.09	.07	-.27**	-									
6. SCL-r^2	.02	-.13	.05	-.08	.66**	-								
7. Sleep Minutes	.04	.12	-.10	.17	-.10	-.22*	-							
8. Sleep Efficiency	.05	.04	.03	.05	-.22*	-.24**	.59**	-						
9. Long-Wake Episodes	-.05	.03	-.05	-.03	.21*	.23*	-.41**	-.92**	-					
10. Sex	-.08	.04	.02	-.05	-.07	-.04	-.25**	-.19**	.16*	-				
11. Age in Years	-.06	-.04	.06	.09	-.02	-.02	-.15*	.004	-.04	.21**	-			
12. SES	-.02	-.01	.01	.25**	.01	-.01	.17*	.16*	-.15*	-.004	-.05	-		
13. zBMI	.26**	-.20**	.04	.13	-.07	-.04	-.03	-.04	.03	-.03	-.05	-.09	-	
14. Race	.14*	-.06	.10	-.38**	.06	-.02	-.21**	-.10	.05	-.05	-.03	-.28**	.14*	-
M	6.91	-.22	.80	9.13	1.85	8.69	407.23	90.97	2.31	-	15.27	2.39	.88	-
SD	1.15	.87	1.66	4.98	2.30	15.29	54.55	6.68	1.74	-	.85	1.31	.98	-

Note: RSA = respiratory sinus arrhythmia; RSA-b = baseline respiratory sinus arrhythmia; RSA-r = respiratory sinus arrhythmia reactivity; SCL- skin conductance level; SCL-b = baseline skin conductance level; SCL-r = skin conductance level reactivity; 407.23 minutes equates to 6.79 hours; sex: 0 = Female, 1 = Male; zBMI = z-scored body mass index; race: 0 = White, 1 = Black.
* $p < .05$.
** $p < .01$.

Table 2.
Test for Associations between RSA and Sleep

		Sleep Minutes				Sleep Efficiency				Long Wake Episodes			
	N	b_1	*SE*	β	R^2	b_1	*SE*	β	R^2	b_1	*SE*	β	R^2
Model 1: Demographics	244				.15				.08				.06
RSA-b		3.59	3.41	.08		.44	.43	.08		-.10	.11	-.07	
Sex		-26.18***	7.35	-.24		-2.77**	.93	-.21		.61	.24	.18	
Age		-.42	.38	-.07		.07	.05	.10		-.02	.01	-.11	
Income:Needs		5.27~	2.88	.13		.74*	.36	.15		-.18	.10	-.14	
zBMI		-.99	3.96	-.02		-.33	.50	-.05		.06	.13	.03	
Race		-25.41**	8.05	-.22		-1.10	1.02	-.08		.11	.27	.03	
Model 2: Reactivity	244				.17				.08				.06
RSA-r		10.25*	4.48	.16		.58	.57	.08		.03	.15	.01	
Model 3: Interactions*	244												
RSA-r x Sex		24.73**	8.51	.27	.23	2.28*	1.10	.20	.16	-.52	.30	-.18	.12
RSA-r^2		1.07	2.89	.03	.18	.56	.37	.14	.10	-.08	.10	-.08	.07

Note: Path coefficients are presented at the model of entry. In model 3, exploratory interactions (either RSA x Sex and RSA-r^2) were tested in separate models. RSA = respiratory sinus arrhythmia; RSA-b = baseline respiratory sinus arrhythmia; RSA-r = respiratory sinus arrhythmia reactivity; SCL- skin conductance level; SCL-b = baseline skin conductance level; SCL-r = skin conductance level reactivity; sex: 0 = Female, 1 = Male zBMI = z-scored body mass index; race: 0 = White, 1 = Black.

~ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .00$.

Table 3.
Test for Associations between SCL and Sleep

		Sleep Minutes				Sleep Efficiency				Long Wake Episodes			
	N	b_1	*SE*	β	R^2	b_1	*SE*	β	R^2	b_1	*SE*	β	R^2
Step 1: Demographics	244				.16				.08				.06
SCL-b		1.66~	.99	.15		.06	.13	.05		-.003	.03	-.01	
Sex		-25.49***	7.33	-.23		-2.75**	.93	-.21		.62*	.25	.18	
Age		-.48	.38	-.08		.06	.05	.09		-.02	.01	-.11	
Income:Needs		4.39	2.91	.11		.69~	.37	.14		-.18~	.10	-.13	
zBMI		-1.30	3.85	-.02		-.26	.49	-.04		.04	.13	.02	
Race		-16.29~	9.06	-.14		-.69	1.14	-.05		.07	.30	.02	
Step 2: Reactivity	244				.17				.18				.14
SCL-r		-3.28~	1.99	-.14		-.93***	.23	-.32		.22***	.06	.28	
Step 3: Interactions*	244												
SCL-r x Sex		-9.09*	3.97	-.23	.21	-.83~	.50	-.17	.16	.13	.13	.10	.12
SCL-r^2		-1.98***	.35	-.54	.32	-.13**	.05	-.29	.19	.03*	.01	.23	.15

Note: Path coefficients are presented at the model of entry. In model 3, exploratory interactions (either RSA x Sex or RSA-r^2) were tested in separate models. RSA = respiratory sinus arrhythmia; RSA-b = baseline respiratory sinus arrhythmia; RSA-r = respiratory sinus arrhythmia reactivity; SCL- skin conductance level; SCL-b = baseline skin conductance level; SCL-r = skin conductance level reactivity; sex: 0 = Female, 1 = Male zBMI = z-scored body mass index; race: 0 = White, 1 = Black.

~ $p < .10$.

* $p < .05$.

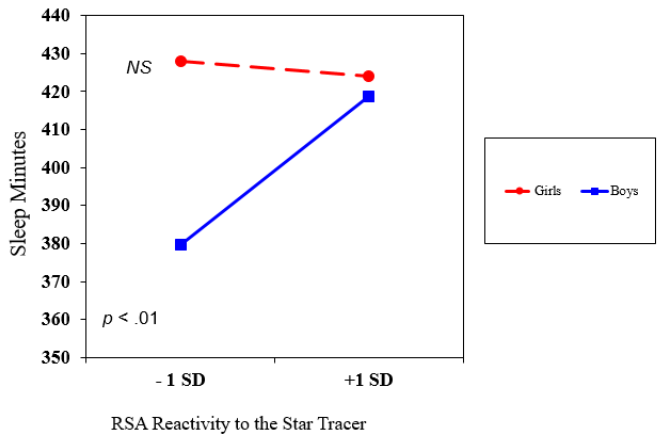
** $p < .01$.

*** $p < .00$.

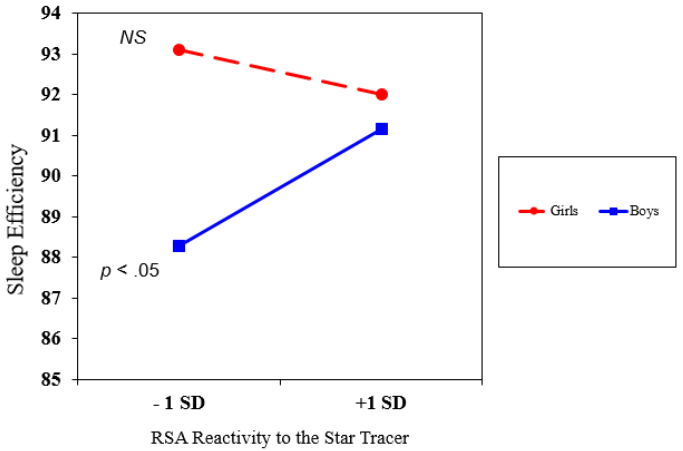
Figure 1.

Adolescent Sex as Moderator of the Association between RSA-r and Sleep

1a.



1b.



1c.

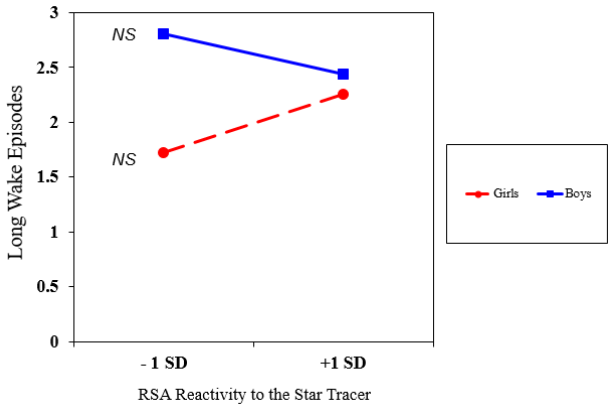
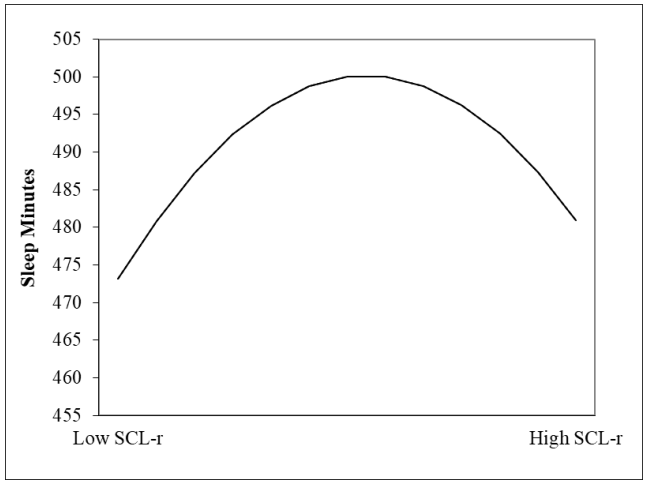


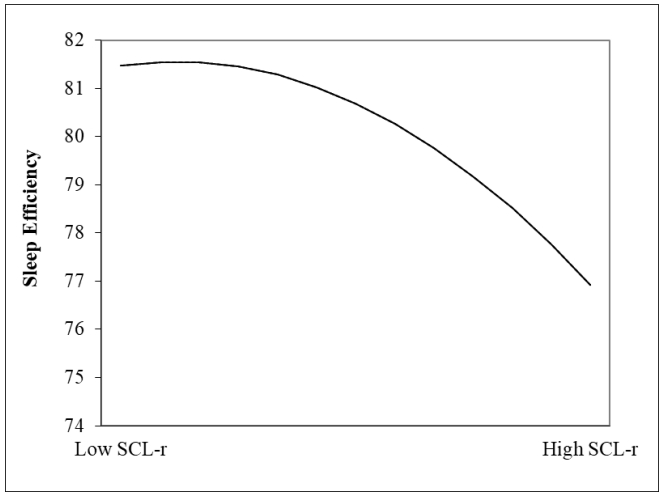
Figure 2.

Non-Linear Association between SCL-r and Sleep

2a.



2b.



2c.

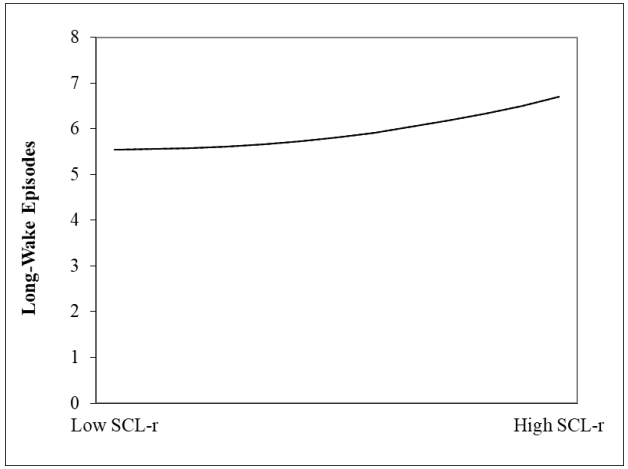
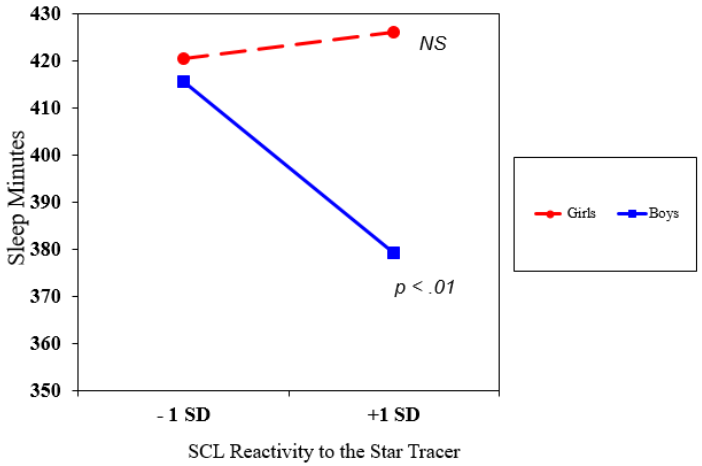


Figure 3.

Adolescent Sex as a Moderator of the Association between SCL-r and Sleep

3a.



3b.