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Parental Problem Drinking and Children's Adjustment: Are Associations Moderated by Patterns of Sympathetic and Parasympathetic Nervous System Activity?

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THESIS

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of Science in the
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By

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ABSTRACT OF THESIS

Parental Problem Drinking and Children's Adjustment: Are Associations Moderated by Patterns of Sympathetic and Parasympathetic Nervous System Activity?

Parental problem drinking (PPD) is associated with various forms of child psychopathology, including hyperactivity, conduct disorder, delinquency, depression and anxiety. However, not all children share the same risk for developing adjustment problems in the context of PPD. In this study, we examined patterns of sympathetic and parasympathetic nervous system activity account for differential susceptibility to the adverse effects of PPD in middle childhood. We found that reciprocal SNS activation protects against child internalizing symptoms in the context of mother problem drinking. We also found consistent interactions between PNS and SNS in predicting child internalizing problems. Coinhibition is linked to more internalizing symptoms including anxiety and depression. This study provides further support for Autonomic Space Theory and demonstrates the importance of taking both PNS and SNS into account when studying physiological response to stress.

KEYWORDS: Parental Problem Drinking, Autonomic Space Theory, Sympathetic Nervous System, Parasympathetic Nervous System, Child Adjustment

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Chapter One: Introduction

Parental problem drinking (PPD, see Table 1.1 for all abbreviations) is associated with various forms of child psychopathology, including hyperactivity, conduct disorder, delinquency, depression and anxiety (West & Prinz, 1987). However, not all children share the same risk for developing adjustment problems in the context of PPD. Understanding of the individual differences that convey increased or decreased vulnerability is important. Psychophysiological stress response may be such an individual difference variable (Calkins, 1997; Calkins, & Fox, 1992; El-Sheikh, 2005), but it has rarely been considered in the context of PPD. The current study will determine whether patterns of sympathetic and parasympathetic nervous system activity account for differential susceptibility to the adverse effects of PPD in middle childhood.

The Autonomic Nervous System and Polyvagal Theory

The sympathetic (SNS) and parasympathetic (PNS) nervous systems are two branches of the autonomic nervous system, which in mammals is designed to adjust bodily activity to meet the demands of environmental challenges. Both branches have influence on many of the same organs, including heart, pupils, stomach, and lungs, but their influence is complimentary (Andreassi, 2007). The PNS dominates the body at rest, while the SNS dominates during emergencies, preparing for the "fight-or-flight" response. The PNS increases salivary secretions and digestive secretion, decreases heart rate, and constricts the pupils, while the SNS has the opposite effects (Andreassi, 2007).

Polyvagal Theory (Porges, 2007) proposes three different systems in mammals which operate in stressful conditions: social communication, mobilization and

immobilization. These systems correspond to the three different vagal systems: the PNS, the SNS, and the reptilian vagus. These systems are referred to as vagal because they operate through the vagus nerve, the 10th cranial nerve, which includes motor fibers innervating the heart and thoracic and abdominal viscera, and sensory fibers from these regions. The social communication system (PNS) enables rapid inhibition and disinhibition of physiological arousal through the myelinated component of the vagus nerve, promoting social engagement. Activity of the PNS can be indexed via respiratory sinus arrhythmia (RSA), fluctuations in heart rate that accompany breathing (Porges, 2007). The mobilization system (SNS) prepares the body for "fight or flight" situations (Porges, 2007) by increasing physiological arousal. Because it increases sweating, it can be indexed via fluctuations in the conductivity of the surface of the skin (electrodermal activity, EDA). The immobilization system (reptilian vagus) operates through the unmylineated portion of the vagus nerve and generates the "freezing" response which is frequently seen in reptiles and is of less interest in humans (Porges, 1995; 2007) .

PNS and SNS influence can be observed through baseline level at rest or changes from baseline to challenge situations, also known as reactivity. We can measure PNS function via baseline RSA (also termed vagal tone) or RSA reactivity (also termed vagal reactivity). Vagal reactivity can be differentiated based on the direction of change from baseline: vagal withdrawal is decreased RSA from baseline, reflecting reduced PNS activity and increased arousal, and vagal augmentation is increased RSA from baseline, reflecting enhanced PNS activity and decreased arousal (El-Sheikh et al., 2009; Porges, 2007). Vagal withdrawal is generally regarded as a more adaptive response than vagal

augmentation under stress because it increases metabolic outputs required to cope with stress (Porges, 2007).

SNS activity can be assessed via baseline EDA or EDA reactivity. There are two general measures of EDA: skin conductance level (SCL) and skin conductance response (SCR). SCL refers to the tonic measure of conductivity resulting from sweat produced by eccrine sweat glands. Eccrine glands are located throughout the surface of the skin, but have higher concentrations on the palms of the hands. They are innervated solely by the SNS (Andreassi, 2007). Skin conductance response (SCR) refers to sudden increases in skin conductance that occur momentarily but not in response to any event in the environment. Greater SNS activity is associated with more pronounced skin conductance responses. Changes in SCL and SCR in response to stress, referred to as skin conductance level reactivity (SCLR) or skin conductance response reactivity (SCRR), can also be observed (Fowles, 2008).

The Autonomic Nervous System and Child Psychosocial Adjustment

There has been a substantial amount of research linking patterns of autonomic activity and children's adjustment problems, although the great majority of these prior studies examined the PNS and SNS separately. In his review of the literature, Beauchaine (2001) concluded that lower baseline RSA is related to both externalizing problems and internalizing problems across different ages. Both adolescents and children in middle childhood with aggressive conduct disorder showed attenuated baseline RSA in comparison to a control group, although no such difference was found in preschoolers (Bauchaine, Gatzke-kopp, & Mead, 2007). In terms of vagal reactivity, Calkins, Graziano

and Kean (2007) found that children at risk for combined internalizing and externalizing problems at 5 years old showed greater vagal withdrawal after emotionally and behaviorally challenging tasks compared to children with low behavior problems and children only at risk for externalizing problems. Boyce et al. (2001) also found that children with internalizing symptoms between 6-7 years old showed greater vagal withdrawal. However, Calkins and Keane (2004) failed to replicate this result. In their study, greater RSA withdrawal at age 2 was related to lower emotional negativity and fewer externalizing behavior problems at age 4.5 (Calkins & Keane, 2004). Interestingly, Hinnant and El-Sheikh (2009) found that neither baseline RSA nor RSA reactivity at age 6 years significantly predicted children's internalizing and externalizing problems at 8 years of age. Instead, the interaction between baseline RSA and RSA reactivity to a social stressor significantly predicted children's internalizing problems (Baseline RSA was positively associated with internalizing problems only for children who showed RSA augmentation during an argument) and externalizing problems (Baseline RSA was positively associated with child externalizing problems for children who showed RSA withdrawal, while negatively associated with child externalizing problems for children who showed RSA augmentation) (Hinnant & El-Sheikh, 2009). The conflicting results indicate further specification of the association between PNS function and child maladjustment is needed. Associations may vary depending on child age, different measures of PNS activity, family context, and statistical method used. However, simultaneous consideration of PNS and SNS functioning may also clarify associations.

SNS activity is also linked to child adjustment. Children with disruptive behavior disorders between 8 to 12 years old showed lower baseline SCL compared to the matched

control group (van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000). The same association between baseline SCL and child psychopathology applied to children in the same age range with oppositional defiant disorder, attention-deficit/hyperactivity disorder and comorbid disorders (Snoek, van Goozen, Matthys, Buitelaar, & van Engeland, 2004). In terms of SCL reactivity, it is positively related to child anxiety and depression (Weems, Zakem, Costa, Cannon, & Watts, 2005). Overall, lower baseline SCL is associated with externalizing problems and greater SCL reactivity is related to internalizing problems.

Interactions between the SNS and PNS

Initially, researchers viewed the opposite effects of the PNS and SNS (down-regulation vs. up-regulation of arousal, respectively) as indicative of their pattern of interaction. Whenever the PNS is active, the SNS must deactivate. Whenever the SNS is active, the PNS will deactivate. However, theory about the interaction between the SNS and PNS has advanced from this single continuum model to a two-dimensional autonomic space model (Berntson, Cacioppo & Quigley, 1991). This model is depicted in Figure 1.1. The first dimension (vertical axis in Figure1.1) represents PNS influence, which falls along a continuum from low to high. The second dimension (horizontal axis in Figure1.1) represents SNS influence, which also falls along a continuum from low to high. This leads to three general patterns of autonomic influence: coupled reciprocal modes, coupled nonreciprocal modes and uncoupled modes. Coupled reciprocal modes include (1) reciprocal PNS activation, with increasing PNS response and decreasing SNS response and (2) reciprocal SNS activation, with increasing SNS response and decreasing

PNS response (Berntson et al., 1991). These two patterns represent the expected patterns of influence according to the earlier one-dimensional model. The PNS and SNS affect the visceral organs in the same direction, maximizing the range of target organ responses (Berntson et al., 1991). Coupled nonreciprocal modes are those in which the PNS and SNS change in the same direction, thereby working in opposition to each other. There are two types of coupled nonreciprocal patterns: (1) coactivation occurs when both the SNS and PNS are simultaneously activated, meaning that the SNS is pushing increased arousal while the PNS is pushing for decreased arousal, while (2) coinhibition occurs when both the SNS and PNS are simultaneously deactivated, meaning that the PNS is pushing for increased arousal while the SNS is pushing for decreased arousal. Uncoupled modes represent responses in one branch of an ANS system with the absence of response in other branch (Berntson et al., 1991).

Empirical research has documented the existence of these three patterns of autonomic influence. Reciprocal SNS activation was the most prevalent mode, with an average of 75% of children and adolescents exhibiting this pattern across three different stress-eliciting tasks: reaction time, mirror-tracing and social competence interview. Prevalence of other patterns varied from task to task (Salomon, Matthews, & Allen, 2000). Response patterns were also highly consistent across three different tasks, with more than 90% of the participants showing the same pattern in two out of the three tasks. Children and adolescents exhibiting coinhibition reported significantly higher levels of family conflict than coactivators (Salomon et al., 2000). However, another study documented different prevalence rates of the different autonomic patterns. Coinhibition and reciprocal SNS activation were the most common autonomic modes in children

between 3-6 years old across three different studies, while coinhibition and reciprocal PNS activation were most common in response to stress in children between 7-8 years old (Alkon et al., 2003). These results indicate that the prevalence of autonomic mode may differ based child age.

Autonomic Activity in the Context of Family Risk

A number of studies have examined individual differences in autonomic activity as vulnerability or protective factors in the context of family risk. Less efficient PNS function and over-reacting SNS function exacerbate the association between family risk and child maladjustment. For example, El-Sheikh and Whitson (2006) found greater vagal withdrawal while overhearing an argument is a protective factor against internalizing problems otherwise associated with marital conflict among elementary school-age children and young adolescents. Higher SCL reactivity during a stress-eliciting task exacerbates the association between parental depressive symptoms and child internalizing symptoms, externalizing symptoms, and social problems as reported by parents (Cummings, El-Sheikh, Kouros & Keller, 2007). Higher SCL reactivity also serves as a risk factor for child internalizing and externalizing symptoms in the context of marital conflict (El-Sheikh, 2005b; El-Sheikh, Keller, & Erath, 2007).

However, marital conflict predicts increased externalizing problems for boys with lower SCL reactivity (El-Sheikh, Keller, & Erath, 2007). Erath, El-Sheikh and Cummings (2009) also discovered that lower SCL reactivity is a risk factor for children to develop externalizing problems in the context of harsh parenting. Thus, some findings suggest lower SCL reactivity as a risk factor, while others suggest higher SCL reactivity as a risk

factor. These mixed findings may be due to a number of factors, including the lack of simultaneous investigation of the SNS and PNS activity. There has been very little research on interactions between the SNS and PNS in the context of family stress. One exception is provided by El-Sheikh and colleagues (2009), who focused on child externalizing problems in the context of marital conflict. Across three independent samples, they found marital conflict predicted more externalizing problems, including delinquency and aggression, when children in middle childhood showed coinhibition and coactivation of the SNS and PNS. At the same time, reciprocal SNS activation served as a protective factor for child aggression and attention deficit/hyperactive behaviors in the context of aggressive marital conflict.

Expanding Research to the Context of PPD

West and Prinz (1987), in their summary of research published between 1975 and 1985, demonstrated that parental alcoholism is related to a wide variety of child psychopathology symptoms, including hyperactivity and conduct disorder, substance abuse, delinquency, anxiety and depression. Since then, research has continued to observe such associations, and has begun examining potential mediators. For example, PPD predicted greater marital conflict one year later, which was related to less parental warmth and more parental psychological control, which predicted child externalizing and internalizing problems two years later (Keller, Cummings, Davis, & Mitchell, 2008). Higher level of family cohesion and adaptability and child-parent attachment security are two protective factors against child maladjustment in the context of PPD (El-Sheikh, & Buckhalt, 2003). Marital conflict, parent-child conflict and maternal depression mediate

the association between father problem drinking and child internalizing and externalizing problems (El-Sheikh, & Flanagan, 2001). However, little is known about whether child psychophysiological response to stress works as a vulnerability or protective factor in the context of PPD. The only exceptions are studies by El-Sheikh (2001; 2005a) demonstrating the effect of child vagal tone and vagal withdrawal. A higher level of vagal withdrawal while listening to an audiotaped argument was a protective factor against children's externalizing, internalizing and social problems otherwise associated with PPD (El-Sheikh, 2001). A longitudinal follow-up demonstrated that lower vagal tone combined with higher PPD at age 9 predicted higher internalizing problems at age 11 (El-Sheikh, 2005a). However as suggested by the autonomic space model, PNS and SNS do not function independently when coping with stress. It is important to take both into consideration when we explore how child psychophysiological response to stress moderates the association between PPD and child maladjustment. No research to date has examined this question.

The Current Study

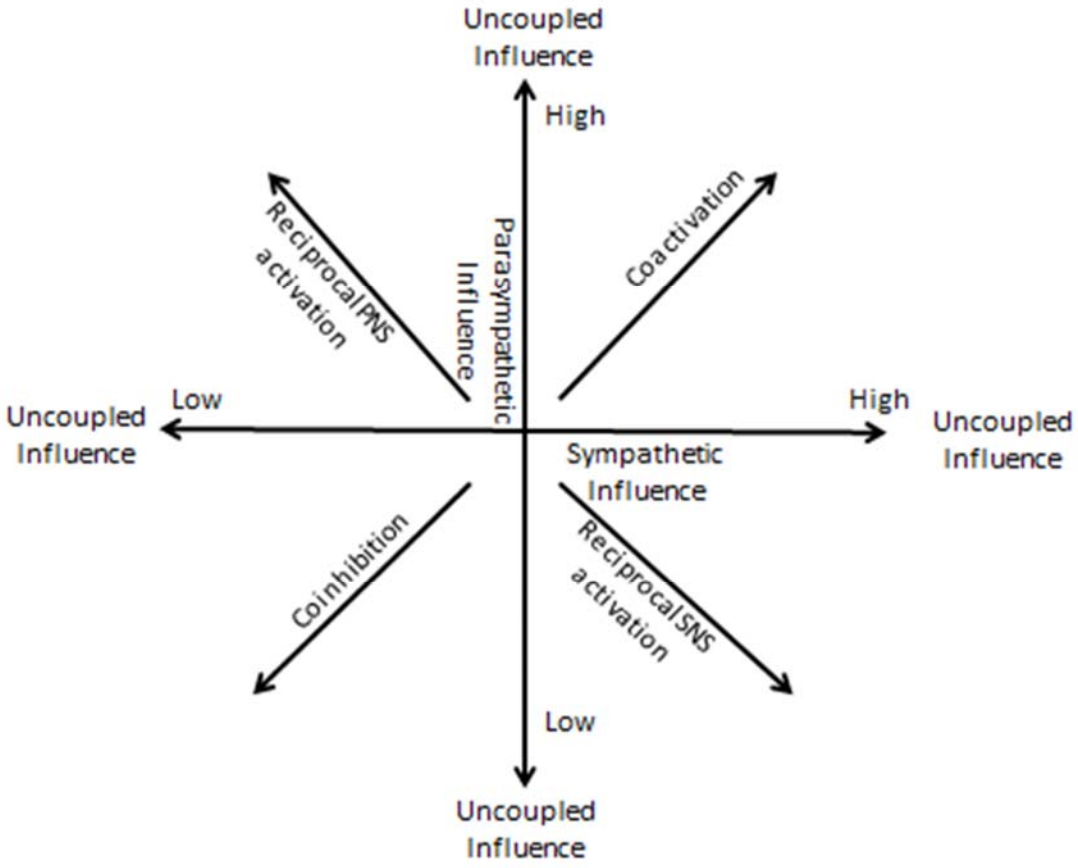
The current study examined whether ANS activity moderates the association between PPD and child internalizing and externalizing symptoms, using the perspective of the autonomic space model. As an initial goal, we examined the frequency of different autonomic response patterns in the sample, and determined whether response patterns were associated with child age. We used the approach to classifying children employed by Salomon et al. (2000) so that a comparison can be made to prior research. Next, we hypothesized that reciprocal modes of activation (higher PNS activity combined with

lower SNS activity or lower PNS activity combined with higher SNS activity) served as a protective factor for children's maladjustment in the context of PPD. The two different reciprocal modes may serve different functions: PNS activation may enhance social engagement and calm during stress; SNS activation enhances preparation for potentially dangerous or threatening situations. On the other hand, the nonreciprocal modes, including coinhibition and coactivation of PNS and SNS were hypothesized to serve as risk factors for child maladjustment in the context of PPD. These patterns of ANS activity represented an inefficient physiological response that may prevent adequate responding to stress. We had no specific hypotheses with regard to the role of uncoupled PNS and SNS activity, as this has rarely been examined in prior research. For our assessment of autonomic response patterns, we examined reactivity in both systems, and treat these values as continuous, in order to be consistent with and compare results to El-Sheikh and colleagues (2009).

Table 1.1 Key terminologies and abbreviation

Abbreviation	Full term	Definition
ANS	autonomic nervous system	
PNS	parasympathetic nervous system	One branch of ANS, dominates the body at rest
SNS	sympathetic nervous system	One branch of ANS, dominates the body in emergency
RSA	respiratory sinus arrhythmia	Fluctuation in heart rate due to breathing; index of PNS
RSAR	respiratory sinus arrhythmia reactivity	Residualized RSA change score in stressful task from baseline value
EDA	electrodermal activity	Fluctuation in the conductivity of the surface of the skin; index of SNS
SCL	skin conductance level	Mean skin conductance value across task
SCLR	skin conductance level reactivity	Residualized SCL change score in stressful task from baseline SCL
SCR	skin conductance response	Difference between peak skin conductance value and trough skin conductance value to nonspecific stimulus
SCRR	skin conductance response reactivity	Residualized SCR change score in stressful task from baseline SCR
PPD	parental problem drinking	
	Reciprocal PNS activation profile	Increasing PNS function (vagal augmentation) and decreasing SNS function (decreasing EDA)
	Reciprocal SNS activation profile	Decreasing PNS function (vagal withdrawal) and increasing SNS function (increasing EDA)
	Coinhibition ANS profile	Decreasing PNS function (vagal withdrawal) and decreasing SNS function (decreasing EDA)
	Coactivation ANS profile	Increasing PNS function (vagal augmentation) and increasing SNS function (increasing EDA)

Figure 1.1
Autonomic space theory



Chapter Two: Methods

Participants

Participants are 296 families from two larger research projects investigating parents' drinking, child stress response and child sleep. Both studies use an alcohol consumption screening questionnaire to obtain samples with sufficient variability in alcohol consumption. Participants were classified as light drinkers, moderate drinkers and heavy drinkers based on the frequency of drinking and the amount of drinking on each occasion. In total, 108 were classified as light drinkers, 83 were moderate drinkers and 105 were heavy drinkers. Participants include parents who have cohabitated for at least two years and their child between 6-12 years of age (M=8.58, SD=1.95; 50.9% are female) with no developmental delay or sleep disorder. 74.6% of the participants are white, 12.5% are African American, 0.68% are Asian, 0.34% is Hispanic/Latino and the rest are mixed or other race. 89.8% of the children live with their biological mother. Mothers on average have 16 years of education and the median family annual income is between $40,000 - $54,999. Only one child from each family participated if more than one child was qualified.

Procedures

Both larger studies were approved by the Internal Review Board of the University of Kentucky. Procedures used for this study are identical across both projects. Informed consent was obtained from parents and informed assent was obtained from children. Families came to campus to participate. Parents completed a series of questionnaires on a computer separately in different rooms. The child was attached to physiological recording equipment, including a Pizzo belt below the chest (for measurement of respiratory

changes), electrodes on the right collarbone, the bottom left rib and the bottom right rib (for assessment of RSA), and two electrodes on the palm of child's non-dominant hand (for assessment of EDA). Children were left alone to sit on a couch for 3 minutes to get a baseline measure of physiological activity. The child then completed a mirror-tracing task, which is a well-established laboratory task for eliciting autonomic reactivity (Cummings et al., 2007; El-Sheikh et al., 2009; Hinnant & El-Sheikh, 2009; Salomon et al., 2000). In the mirror-tracing task, the child needed to trace the image of a star on a sheet of paper by only looking at the image through a mirror. This mirror-tracing task lasted for 3 minutes. Children also completed a series of questionnaires via interview.

Measures

RSA Data Acquisition and Scoring

Standard guidelines (Berntson et al., 1991) were used to measure RSA. A custom bioamplifier from Mindware Technologies (BioNex Model 3711-08; Gahanna, OH) was used and the signal was digitized with the Mindware acquisition system BioLab 2.5 at a sampling rate of 1,000 readings per second. The bioamplifier was set for ECG filtering with half power cutoff frequencies of .5 and 45 Hz and the signal was amplified with a gain of 500. The ECG signal was then processed using an analysis system from Mindware, HRV 3.0.10. 10. R-waves were identified by an automated algorithm and misidentified R-waves were corrected manually.

Baseline RSA was the average RSA level across the 3 minute baseline period. RSA was also averaged across the 3 minute mirror-tracing task. RSA reactivity was computed as a residualized change score between baseline RSA and RSA during the task

(residuals were saved from a regression in which task RSA was predicted by baseline RSA).

EDA Data Acquisition and Scoring

Standard guidelines were also used to measure EDA. A custom bioamplifier from Mindware Technologies (BioNex Model 3711-08; Gahanna, OH) was used and the signal was digitized with the Mindware acquisition system BioLab 2.5 at a sampling rate of 1,000 readings per second. The bioamplifier was set for EDA filtering with half power cutoff frequencies of 1 and 45 Hz and the signal was amplified with a gain of 10. The EDA signal was then processed using an analysis system from Mindware, EDA 3.0.9. Both SCR and SCL were obtained from the EDA recording. SCR was acquired as the difference between peak skin conductance value and the trough skin conductance value (for each momentary response) and SCL was acquired as the mean skin conductance value across the task. Baseline SCL (or SCR) was the average SCL (or SCR) across the 3 minute baseline period. SCL (or SCR) was also averaged across the 3 minute mirror-tracing task. SCLR (skin conductance level reactivity) or SCRR (skin conductance response reactivity) was computed as a residualized change score between baseline and the value during the task (residuals were saved from a regression in which task value was predicted by baseline value).

Parental Problem Drinking

Parental problem drinking was measured with the Alcohol Use Disorders Identification Test (AUDIT), a 10-item questionnaire (Saunders, Aasland, Babor, & Grant, 1993). Each parent reported on self and partner drinking behavior. Questions ask

about the frequency of alcohol consumption and the hazardous results due to alcohol consumption (for example, “How often do you have a drink containing alcohol?”; “How often during the last year have you failed to do what was normally expected of you because of drinking?”). All 10 items were summed up after recoding. The Cronbach’s α varies from .80 to .87 among self-report and partner report. Mother problem drinking and father problem drinking were both averaged across self-report and partner report because they were highly correlated (r=.80, df=233, p<.001; r=.76, df=237, p<.001, respectively).

Child Adjustment

Both parents completed the Child Behavior Checklist (CBCL) (Achenbach, 1991) subscales for internalizing and externalizing behaviors. Each parent reported how much the listed 58 behaviors described the child in the past 6 months on a 3 point Likert Scale from 0 “not true” to 2 “very true or often true”. The externalizing subscale includes measures of child aggression (example items are “bragging, boasting”, “cruelty, bullying or meanness to others”) and delinquency (example items are “lying or cheating”, “steals at home”). Cronbach’s α=. 87 for mother report and α=.89 for father report for externalizing subscale. The internalizing subscale includes measurement of social withdrawal, for example “would rather be alone than with others”, “refuses to talk”; somatic complaints for example “feels dizzy”, “overtired” and anxiety/depression, for example “cries a lot”, “feels he/she has to be perfect”. Cronbach’s α=.84 for mother report and α=.85 for father report for internalizing subscale. We used T scores because of the number of predictors in the models and leaving out gender and age could reduce the complexity of the models. 19.7% of children showed internalizing symptoms in the clinical range and 8.1% in the subclinical range based on mother report (19.0% and 8.1%

respectively on father report). 10.5% of the children had externalizing symptoms in the clinical range and 5.7% in the subclinical range based on mother report (11.2% and 4.4% respectively on father report).

Children completed the Child Depression Inventory-Revised (CDI), a 15-item questionnaire (Kovacs, 1978). All items were summed up after reverse coding. Cronbach's α=.80. The child reported "Yes", "Sometimes", or "No" to each item based on if that sentence sounded like to him/her. An example item is "I often worry about something bad happening to me".

Children also completed the Revised Children's Manifest Anxiety Scale (RCMAS), a 37-item questionnaire (Reynolds, & Richmond, 1978). The child reported "Yes" or "No" to each statement they heard. We used three subscales: child physiological symptoms of anxiety (for example, "Often I have trouble getting my breath", "Often I feel sick in my stomach"), worry/oversensitivity (for example, "I get nervous when things do not go the right way for me") and social concerns (for example, "I feel that others do not like the way I do things"). The three subscales were summed together as one indicator of anxiety in the present study with a Cronbach's α=.91.

Analyses

We used SAS 9.3 to analyze the data. Missing data were replaced by means at the item level if less than 2 items were missing in the same scale. After the mean replacement, we calculated missing rate at the scale level. The missing rate was 7.5% in AUDIT for self-report and 9.8% for partner report reported by the female. For male report, the missing rate was 14.9% for both self-report and partner report. The missing rate was 1.7% for CDI and 2.7% for RCMAS. 5.4% of data were missing in both

internalizing and externalizing subscales of CBCL in mother report and 9.5% in father report.

We had a substantial amount of missing data for the physiological measures, especially for EDA data, due to malfunction of the equipment. The missing rate is 34.24% for EDA and 17.29% for RSA. Due to the higher percentage of missing EDA data, we examined if the children with EDA measures and without differed on any other variables in this study. No significant differences were observed.

Classification of Child Autonomic Patterns

Classification of child autonomic pattern was based on Salomon et al (2000) procedures. We subtracted the RSA and EDA average response during the mirror tracing task from the corresponding baseline measure to index the physiological response due to stress. Only children who changed from baseline to challenge by at least .25 standard deviations were considered responders, while children within .25 standard deviation were non-responders. Responders were classified as reciprocal SNS responders if they had positive EDA response with negative RSA response (indicating higher arousal). Children were classified as reciprocal PNS responders if they had negative EDA response and positive RSA response. Children were classified as coactivators if they had positive EDA response and positive RSA response. Children were classified as coinhibitors if they had negative EDA response and negative RSA response. Rates of children in each category were examined and mean differences in child age between groups were tested using a one-way ANOVA. Two sets of classifications were examined, one in which SCL was the measure of EDA, and one in which SCR was the measure of EDA.

PPD x Autonomic Pattern Interactions.

Hypotheses were also tested with hierarchical multiple regression by treating RSAR and SCLR or SCRR as continuous variables, as in El-Sheikh et al. (2009). The initial step included demographic information like child gender, age and family income (except for predicting externalizing problems and internalizing problems because T scores were used). The second step added PPD. Separate models were fit for mother PD and for father PD. The third step added EDA reactivity and RSA reactivity. Separate models were fit for SCLR and SCRR as the index of EDA reactivity. The fourth step added two-way interactions: PPD x SCLR (or SCRR), PPD x RSAR, SCLR (or SCRR) x RSAR. The fifth step added the three-way interaction: PPD x SCLR (or SCRR) x RSAR. All variables were mean centered before computing cross-products. Significant interactions were probed using an online utility (www.quantpsy.org; Preacher, Curran, & Bauer, 2006), and were plotted at +/- 1 SD of the interacting variables. These procedures were followed for the prediction of child internalizing symptoms and externalizing symptoms.

Chapter Three: Results

Assumptions check

We found minor violations of the assumption of normality of residuals in some of the models. The residuals were not homoscedastic against parental problem drinking because the majority of participants exhibited lower levels of problem drinking. However, regression analysis is robust to moderate violations of assumptions (Cohen, Cohen, West & Aiken, 2003). In addition, no non-linear transformation would significantly reduce violations and would further complicate the interpretation of the results. Thus, no non-linear transformations were used in the analysis.

Classification of autonomic nervous patterns and age differences

Using SCR as an indicator of SNS, 37 children were classified as coinhibitors, 7 coactivators, 48 reciprocal SNS responders, 6 reciprocal PNS responders and 86 non-responders. Child age did not differ between different autonomic nervous patterns, $F(4, 177)=1.08$, $p=.37$. We also examined if different ANS profiles were related to different level of PPD and child maladjustment. We found different ANS profiles were significantly related to different level of mother problem drinking, $F(4, 175)=3.13$, $p=.02$. Post-hoc follow up test using Bonferroni corrections found that reciprocal PNS activation group showed significantly more mother problem drinking than any other group, that is, in comparison to nonresponders, 95% CI=[.12, 7.79]; reciprocal SNS activators, 95% CI=[.41, 8.31]; coinhibitors, 95% CI=[.71, 8.70] and coacativators, 95% CI=[.15, 10.26]. No other pairwise comparisons were significant. ANS profile also significantly predicted child internalizing symptoms reported by father, $F(4, 165)=3.87$, $p=.005$. Pairwise post-hoc comparisons revealed that coinhibitors showed significantly more internalizing

symptoms than non-responders, 95% CI=1.16, 12.76]. No other pairwise comparisons turned out to be significant. ANS profiles were not related to other independent or dependent variables in this study.

Using SCL as an indicator of SNS, 6 children were classified as coinhibitors, 7 coactivators, 65 reciprocal SNS responders, 2 reciprocal PNS responders, and 104 non-responders. Child age did not differ between different autonomic nervous patterns, $F(4, 177)=0.53$, $p=.712$. ANS profiles based on SCL did not predict any independent or dependent variables in the study.

Predicting child internalizing and externalizing symptoms

We found a significant interaction between mother problem drinking, RSAR and SCRR in predicting child internalizing symptoms reported by mother, $\beta=.36$, $p<.001$. See Table 3.1. Further probing of this three-way interaction showed that on average, children experience higher internalizing symptoms in the context of mother PD, unless children exhibit reciprocal SNS reactivity, meaning RSA withdrawal and increasing SCR. See Figure 3.1. This suggested that reciprocal SNS activation served as a protective factor against child internalizing symptoms in the context of mother PD.

No other significant three way interactions between EDA, RSAR and PPD were found. See Tables 3.2, 3.3 and 3.4. However, some two-way interactions between PNS and SNS were found. Child RSAR marginally interacted with SCLR in predicting child internalizing symptoms reported by mother, $\beta=.15$, $p=.088$ for the model including mother PD and $\beta=.19$, $p=.061$ for the model including father PD. See Table 3.1. Further probing the interactions showed similar patterns across these two models. Lower SCLR activity combined with lower RSAR was associated with significantly higher child

internalizing problems reported by mother. This demonstrated that ANS coinhibition was a risk factor for child internalizing problems. See Figure 3.2 for the interaction controlling for mother PD.

Predicting child self-report of depression

We found patterns of interaction between child RSA and EDA, including SCRR and SCLR, in predicting child depression reported by the child, controlling for mother PD and father PD, β=.16, *p*=.051; β=.20, *p*=.018; β=.17, *p*=.055; β=.22, *p*=.032 respectively. See Table 3.5. Across the four different models, lower EDA reactivity combined with RSA withdrawal predicted significantly higher child depression. These results replicated the findings above that ANS coinhibition served as a risk factor for child depression. See Figure 3.3 as an example.

Predicting child self-report of anxiety

Similar interactions between RSA and EDA, including SCR and SCL were also seen in predicting child anxiety in models controlling for mother PD and father PD, β=.92, *p*=.002; β=.56, *p*=.007; β=.80, *p*=.022; β=.49, *p*=.037 respectively. See Table 3.6. Further probing of these interactions showed similar patterns across models. At lower level of EDA reactivity, as RSAR increased, child physiological symptoms of anxiety decreased, while at higher levels of EDA reactivity, as RSAR increased, child physiological symptoms of anxiety increased. This meant that coinibition and coactivation patterns of ANS were associated with higher child anxiety. See Figure 3.4 as an example.

Table 3.1 Predicting child internalizing symptoms reported by mother

Variable and step	Model1		Model2		Model3		Model4	
Step 1	*B*	β	*B*	β	*B*	β	*B*	β
Family income	-0.42	-.13	-0.39	-.12	-0.31	-.10	-0.28	-.09
Father PD					0.22	.11	0.25	.12
Mother PD	0.46^	.16	0.39	.14				
ΔR^2	.028^		.028^		.017		.017	
Step 2								
RSAR	-0.60	-.05	-1.15	-.10	-0.10	-.08	-0.57	-.05
SCLR			-0.29	.08			-0.25	-.07
SCRR	-0.26	-.06			-0.18	-.04		
ΔR^2	.007		.010		.007		.009	
Step 3								
Mother PD*RSAR	0.74	.19	0.24	.06				
Mother PD*SCLR			-0.19	-.11				
Mother PD*SCRR	-0.14	-.16						
Father PD*RSAR					0.02	.01	0.17	.06
Father PD*SCLR							0.04	.05
Father PD*SCRR					0.06	.05		
RSAR*SCLR			0.52^	.14			0.67^	.19
RSAR*SCRR	0.68	.12			0.61	.11		
ΔR^2	.012		.023		.009		.015	
Step 4								
Mother PD*RSAR*SCLR			0.07	.04				
Mother PD*RSAR*SCRR	0.35*	.36						
Father PD*RSAR*SCLR							0.21	.18
Father PD*RSAR*SCRR					0.12	.07		
ΔR^2	.034*		.001		.004		.016	

Note: * $p<.05$, ^ $p<.1$. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity;

Table 3.2 Predicting child externalizing symptoms reported by mother

Variable and step	Model1		Model2		Model3		Model4	
Step 1	*B*	β	*B*	β	*B*	β	*B*	β
Family income	-0.82	-.24	-0.80*	-.23	-0.73**	-.21	-0.71**	-0.21
Father PD					0.03	.02	0.13	.06
Mother PD	0.01	.00	0.05	.02				
ΔR^2	.055*		.055*		.053*		.053*	
Step 2								
RSAR	0.06	.01	0.03	.00	0.13	.01	0.57	.05
SCLR			-0.18	-.05			-0.07	-.02
SCRR	0.13	.03			0.17	.04		
ΔR^2	.000		.001		.000		.001	
Step 3								
Mother PD*RSAR	-0.34	-.08	-0.37	-.09				
Mother PD*SCLR			-0.09	-.05				
Mother PD*SCRR	-0.15	-.17						
Father PD*RSAR					0.12	.04	0.25	.09
Father PD*SCLR							0.19*	.20
Father PD*SCRR					0.11	.09		
RSAR*SCLR			-0.08	-.02			0.03	.01
RSAR*SCRR	-0.20	-.03			-0.10	-.02		
ΔR^2	.009		.008		.008		.040^	
Step 4								
Mother PD*RSAR*SCLR			0.08	.04				
Mother PD*RSAR*SCRR	0.15	.15						
Father PD*RSAR*SCLR							0.12	.10
Father PD*RSAR*SCRR					-0.00	-.00		
ΔR^2	.006		.001		.000		.005	

Note: * $p<.05$, ^ $p<.1$. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity;

Table 3.3 Predicting child internalizing symptoms reported by father

Variable and step	Model1		Model2		Model3		Model4	
Step 1	*B*	β	*B*	β	*B*	β	*B*	β
Family income	-0.08	-.02	-0.09	-.03	-0.08	-.02	-0.10	-.03
Father PD					0.02	.01	-0.01	-.00
Mother PD	0.03	.01	0.04	.01				
ΔR^2	.001		.001		.000		.000	
Step 2								
RSAR	-2.30*	-.17	-2.22^	-.17	-2.11^	-.16	-1.98^	-.15
SCLR			-0.20	-.05			-0.32	-.08
SCRR	-0.33	-.07			-0.37	-.07		
ΔR^2	.040*		.038*		.039*		.036^	
Step 3								
Mother PD*RSAR	0.18	.04	0.22	.05	0.10	.03	0.09	.02
Mother PD*SCLR			0.05	.02			-0.06	-.05
Mother PD*SCRR	0.07	.07			0.01	.01		
Father PD*RSAR								
Father PD*SCLR								
Father PD*SCRR								
RSAR*SCLR			-0.27	-.07			-0.08	-.02
RSAR*SCRR	-0.28	-.04			-0.19	-.03		
ΔR^2	.004		.007		.003		.008	
Step 4								
Mother PD*RSAR*SCLR			-0.02	-.01				
Mother PD*RSAR*SCRR	-0.08	-.08						
Father PD*RSAR*SCLR							0.10	.07
Father PD*RSAR*SCRR					0.09	.05		
ΔR^2	.001		.000		.002		.003	

Note: * $p<.05$, ^ $p<.1$. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity;

Table 3.4 Predicting child externalizing symptoms reported by father

Variable and step	Model1		Model2		Model3		Model4	
Step 1	*B*	β	*B*	β	*B*	β	*B*	β
Family income	-0.77*	-.22	-0.71*	-.20	-0.77**	-.22	-0.76**	-.21
Father PD					-0.04	-.01	-0.19	-.08
Mother PD	-0.15	-.05	-0.07	-.02				
ΔR^2	.041*		.041*		.047*		.047*	
Step 2								
RSAR	-0.89	-.07	-0.42	-.03	-0.43	-.03	-0.42	-.03
SCLR			-0.60^	-.16			-0.65*	-.18
SCRR	-0.67^	-.14			-0.74^	-.15		
ΔR^2	.023		.033^		.022		.034^	
Step 3								
Mother PD*RSAR	-0.58	-.14	-0.06	-.01				
Mother PD*SCLR			0.13	.07				
Mother PD*SCRR	-0.00	-.00						
Father PD*RSAR					0.06	.02	0.06	.02
Father PD*SCLR							0.01	.01
Father PD*SCRR					0.03	.02		
RSAR*SCLR			0.00	.00			0.02	.01
RSAR*SCR	0.03	.00			0.04	.01		
ΔR^2	.011		.004		.001		.001	
Step 4								
Mother PD*RSAR*SCLR			0.01	.01				
Mother PD*RSAR*SCRR	-0.21	-.21						
Father PD*RSAR*SCLR							-0.03	-.02
Father PD*RSAR*SCRR					-0.06	-.03		
ΔR^2	.009		.000		.000		.000	

Note: * p<.05, ^ p<.1. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity.

Table 3.5 Predicting child depression reported by child

Variable and step	Model1		Model2		Model3		Model4	
Step 1	*B*	β	*B*	β	*B*	β	*B*	β
Male	-0.63	-.06	-0.59	-.06	-0.55	-.05	-0.63	-.06
Child age	-0.19	-.07	-0.14	-.05	-0.21	-.07	-0.16	-.06
Family income	-0.13	-.07	-0.09	-.05	-0.13	-.07	-0.08	-.04
ΔR^2	.013		.013		.013		.013	
Step 2								
Father PD					0.03	.03	0.03	.02
Mother PD	0.10	.06	0.12	.08				
ΔR^2	.000		.000		.000		.000	
Step 3								
RSAR	-0.50	-.07	-0.30	-.04	-0.31	-.05	-0.21	-.03
SCLR			-0.24	-.12			-0.22	-.11
SCRR	-0.39^	-.15			-0.40^	-.15		
ΔR^2	.010		.009		.011		.009	
Step 4								
Mother PD*RSAR	-0.43	-.19	-0.25	-.11				
Mother PD*SCLR			-0.05	-.06				
Mother PD*SCRR	-0.01	-.02						
Father PD*RSAR					-0.11	-.07	-0.08	-.05
Father PD*SCLR							-0.02	-.04
Father PD*SCRR					0.00	.01		
RSA*SCLR			0.41*	.20			0.44*	.22
RSA*SCRR	0.54^	.16			0.56^	.17		
ΔR^2	.044^		.047*		.034		.041^	
Step 5								
Mother PD*RSAR*SCLR			0.05	.05				
Mother PD*RSAR*SCRR	-0.05	-.09						
Father PD*RSAR*SCLR							0.04	.06
Father PD*RSAR*SCRR					-0.01	-.01		
ΔR^2	.002		.002		.000		.002	

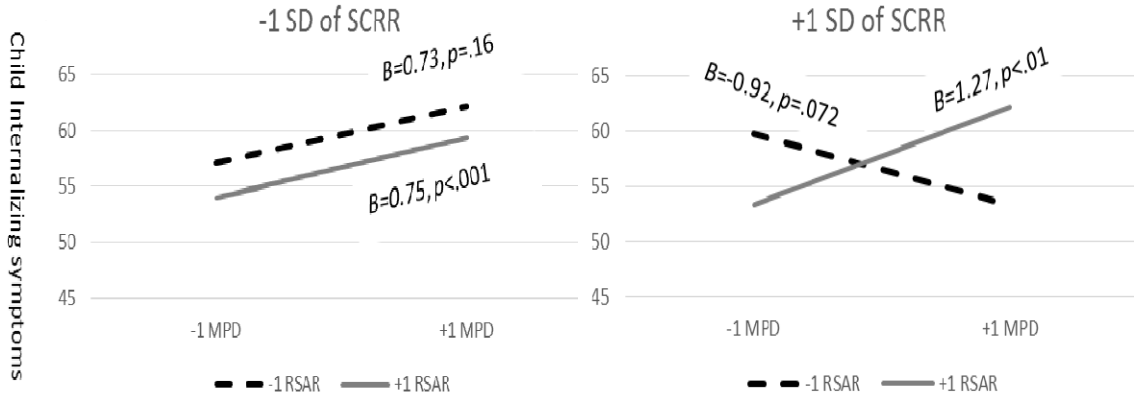
Note: * p<.05, ^ p<.1. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity.

Table 3.6 Predicting child anxiety reported by child

Variable and step	Model1		Model2		Model3		Model4	
Step 1	B	β	B	β	B	β	B	β
Male	-0.84	-.06	-0.64	-.04	-0.57	-.04	-0.57	-.04
Child age	-0.62^	-.15^	-0.62^	-.15^	-0.61^	-.15^	-0.62^	-.15^
Family income	-0.25	-.09	-0.29	-.08	-0.28	-.10	-0.23	-.09
ΔR^2	.04^		.04^		.04^		.04^	
Step 2								
Father PD					0.05	.03	0.08	.05
Mother PD	0.09	.04	0.07	.03				
ΔR^2	.00		.00		.00		.00	
Step 3								
RSAR	-0.33	-.03	0.25	.03	-0.19	-.02	0.00	.00
SCLR			-0.17	-.06			-0.17	-.06
SCRR	-0.06	-.02			-0.09	-.03		
ΔR^2	.00		.00		.00		.00	
Step 4								
Mother PD*RSAR	-0.40	-.13	-0.17	-.06				
Mother PD*SCLR			0.04	.03				
Mother PD*SCRR	0.00	.00						
Father PD*RSAR					-0.33^	-.15^	-0.27	-.12
Father PD*SCLR							0.03	.05
Father PD*SCRR					0.00	.01		
RSAR*SCLR			0.56*	.20*			0.49^	.17^
RSAR*SCRR	0.92*	.20*			0.80*	.17*		
ΔR^2	.04^		.04		.06*		.05*	
Step 5								
Mother			0.13	.09				
Mother	-0.11	-.14						
Father							.03	.04
Father					-0.04	-.03		
ΔR^2	.01		.01		.00		.01	

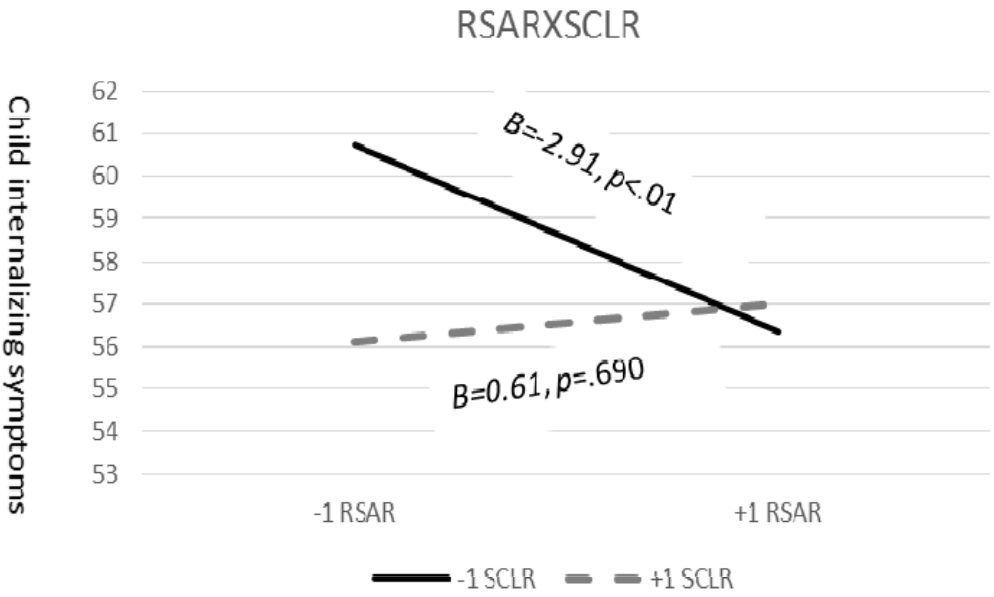
Note: * $p<.05$, ^ $p<.1$. PD= problem drinking; RSAR= respiratory sinus arrhythmia reactivity; SCLR= skin conductance level reactivity; SCRR= skin conductance response reactivity.

Figure 3.1
Three way interaction between SCRR, RSAR and Mother PD in predicting child internalizing problems



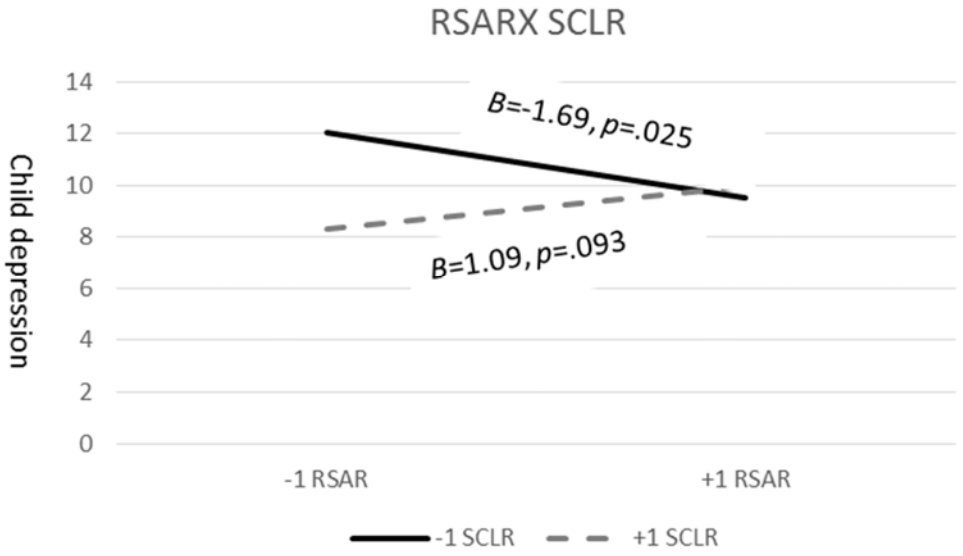
Note: SCRR=skin conductance response reactivity; RSAR= respiratory sinus arrhythmia reactivity.

Figure 3.2
Interaction between RSAR and SCLR in predicting child internalizing symptoms controlling for mother PD



Note: SCLR=skin conductance level reactivity; RSAR= respiratory sinus arrhythmia reactivity

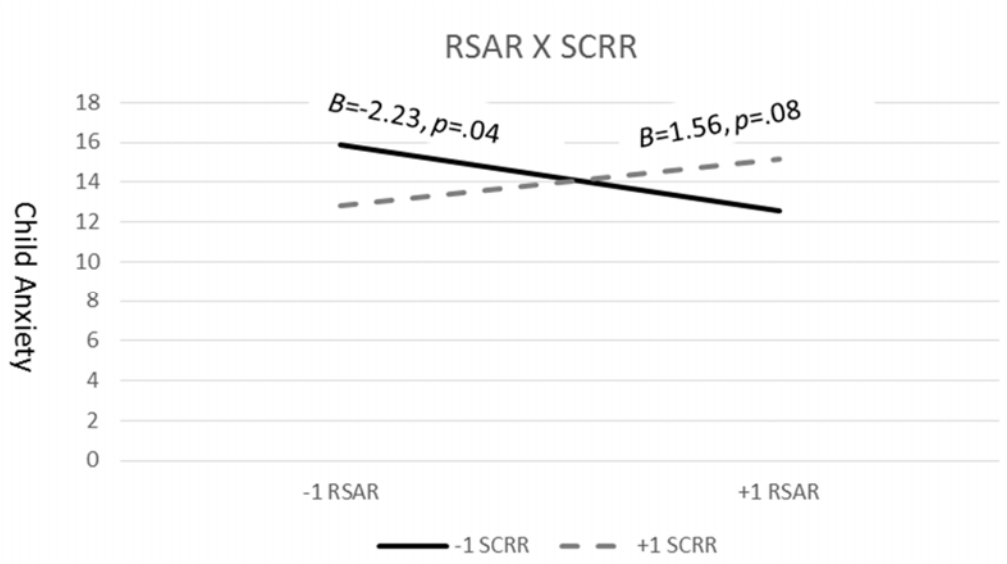
Figure 3.3
Interaction between RSAR and SCLR in predicting child depression controlling for mother PD



Note: SCLR=skin conductance level reactivity; RSAR= respiratory sinus arrhythmia reactivity

Figure 3.4

Interaction between RSAR and SCRR in predicting child anxiety controlling for mother PD



Note: SCRR=skin conductance response reactivity; RSAR= respiratory sinus arrhythmia reactivity.

Chapter Four: Discussion

Autonomic profiles and child age

We did not find that rates of child autonomic profiles differ across age. This is consistent with previous research documenting the stability of RSA reactivity and SCL reactivity in middle childhood (El-Sheikh, 2007; Hinnant et al., 2011) and preschoolers (Calkins & Keane, 2004). However, this is different from one of the few studies that tested the autonomic space model, which found that coinhibition and reciprocal PNS activation became more prevalent while reciprocal SNS activation became less prevalent as age increases (Alkon et al., 2003). More research is needed before we can conclude whether ANS profiles vary at different ages.

Most children in our studies are classified as non-responders. Although previous research has documented the effectiveness in eliciting stress using mirror tracing task (Cummings et al, 2007; El-Sheikh et al., 2009; Salomon et al., 2000), this laboratory task may not generate enough stress response in our participants. Except for non-responders, reciprocal SNS is the most prevalent autonomic profile. This is consistent with previous research that reciprocal SNS activation is the most prevalent ANS profile in all responders across three different stressful situations (Salomon et al., 2000).

Interactions between PPD and autonomic profiles

We found that mother problem drinking significantly interacted with skin conductance response reactivity and respiratory sinus arrhythmia reactivity in predicting child internalizing problems. Further, in general, children showed increasing internalizing symptoms under the context of mother problem drinking. However, children who showed reciprocal SNS activation profile (vagal withdrawal and increasing skin conductance

response) were the exception, they showed decreasing internalizing symptoms in the context of maternal problem drinking. So the reciprocal SNS activation profile serves as a protective factor for child internalizing symptoms under the influence of mother problem drinking. This is somewhat consistent with previous finding that vagal withdrawal serves as a protective factor against child maladjustment in the context of PPD (El-Sheikh, 2001; 2005a). According to polyvagal theory, vagal withdrawal promotes sympathetic metabolic output to prepare the human body to cope with a stressful environment (Beauchaine, 2001; Porges, 1995; 2007). This current study using the framework of autonomic space theory found that vagal withdrawal in combination with SNS activation, which results in the most metabolic output, protects children from internalizing symptoms in the context of PPD. Our study also extends the protective effect of reciprocal SNS activation profile to children's internalizing problems and to the context of PPD (El-Sheikh et al., 2009).

Although we found the interaction between autonomic profiles and PPD in predicting child internalizing symptoms, the majority of three-way interactions were not significant. Thus, caution should be taken before concluding that reciprocal SNS activation serves a protective role. More research needs to be done before we can draw the conclusion on the relation between autonomic profiles and child maladjustment in the context of PPD.

Autonomic profiles, child maladjustment and family risk factor

Although we did not find the consistent expected interactions between PPD and autonomic profiles, we did find consistent links between autonomic profiles and child

internalizing problems across mother report and child self-report. Child ANS coinhibition was consistently associated with increasing child internalizing symptoms in comparison to reciprocal PNS activation profile. This replicates the risk effect of ANS coinhibition profile, which has been related to higher perceived family conflict both from parent's and child's report and more child externalizing problems (Boyce at al., 2001; Salomon et al., 2000).

This study also found that reciprocal PNS activation profile is related to more mother problem drinking in comparison to all other ANS profiles. This is somewhat similar to previous research that documented the less optimal adjustment including sleep problems, lower emotion regulation capability and internalizing symptoms, with vagal augmentation (El-Sheikh & Buckhalt, 2005; El-Sheikh & Whitson, 2006; Gentzler, Santucci, Kovacs, & Fox, 2009). The association between reciprocal PNS activation and mother problem drinking suggests that mother problem drinking may promote the development of this autonomic response pattern and that this pattern may partially account for why mother problem drinking is associated with child adjustment problems. Future research should therefore explore the possibility that autonomic patterns may mediate associations between parental problem drinking and child internalizing and externalizing symptoms.

EDA as an indicator of SNS

This study further establishes electrodermal activity as an indicator SNS function from a perspective of autonomic space theory. It is more common to use preejection period (PEP), a cardiac measure of SNS function (Alkon, et al., 2003; Berntson et al., 1991; Boyce et al., 2001; Salomon et al., 2000). One reason that PEP has been more

popular than EDA is that when combined with RSA, the measures of SNS and PNS functions target the same organ (the heart; Berntson et al, 1991). However, El-Sheikh and colleagues (2009) used SCL as an indicator of SNS function and found that RSA and SCL significantly interacted with marital conflict in predicting child externalizing behaviors across three independent samples. El-Sheikh and colleagues (2009) therefore called for more research using EDA. The current study answers this call, and adds empirical support for the use of electordermal activity in autonomic space research.

Limitations and future research

Although this current study contributes to the literature for the reasons mentioned above, there are several limitations. First, our study is a cross-sectional design. This design limits our ability to draw any causal conclusion in our findings. We specified the model as parental problem drinking interacting with ANS profiles in predicting child maladjustment. However, it is also probable that children with adjustment problems influence parents' drinking problems by creating stress with which it is difficult for them to cope (El-Sheikh et al., 2009).

Furthermore, we only focused on the reactivity measure of SNS and PNS in this study. Berntson et al. (1991) also mentions the importance of tonic (e.g., baseline or resting) measures of autonomic nervous systems. Moreover, RSA baseline and SCL baseline do significantly interact with marital conflict in predicting child externalizing problems (El-Sheikh et al., 2009). In addition, previous research has documented the interactions between RSA baseline and RSA reactivity in predicting child maladjustment (Hinnant & El-Sheikh, 2009). Future research should explore the relations between

baseline measures of both SNS and PNS and environmental risk factors on child mental health status.

Additionally, we used residualized change scores as a measure of ANS reactivity. This procedure has been established as a standard measure of reactivity that takes into account the pre-task value (Beauchaine, 2001;Cummings et al., 2007; El-Sheikh & whitson, 2006; Erath, et al., 2009; Hinnant, et al., 2011;). However, Berntson et al. (1991) proposed the importance of tracing the change in ANS activity during the task instead of just focusing on the initial value and response at the end. In response to his suggestion, it will be beneficial to measure the status of autonomic functions at different time points throughout the laboratory task and explore if family risky factors interact with such changes overtime in predicting child adjustment (Keller & El-Sheikh, 2011).

Conclusion

In spite of the limitations of the current study, this study advances research by providing the first exploration of the interaction between PNS, SNS and parental drinking problems in predicting child maladjustment. We found some evidence that reciprocal SNS activation protects against child internalizing symptoms in the context of mother problem drinking. We also found consistent interactions between PNS and SNS in predicting child internalizing problems. Coinhibition is linked to more internalizing symptoms including anxiety and depression. This provides more empirical support for the Autonomic Space Theory, which emphasizes the importance of taking account of both PNS and SNS when exploring the physiological mechanisms underlying psychological disorder. This study also further establishes the procedure of measuring EDA as an indicator of SNS function.

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