The Pennsylvania State University

The Graduate School

# MODELING PROFILES OF MOTHERS' AND FATHERS' MENTAL HEALTH AND STRESS PHYSIOLOGY AND PHYSIOLOGICAL COREGULATION WITH PRESCHOOL-AGED CHILDREN

A Dissertation in

Psychology

by

Amanda M. Skoranski



Submitted in Partial Fulfillment
of the Requirements
for the Degree of

Doctor of Philosophy

December 2019

The dissertation of Amanda M. Skoranski was reviewed and approved* by the following:

Erika S. Lunkenheimer
Associate Professor of Psychology
Dissertation Adviser
Chair of Committee

Jenae M. Neiderhiser
Distinguished Professor of Psychology and Human Development and Family Studies

Ginger A. Moore
Associate Professor of Psychology

Robert W. Roeser
Professor of Human Development and Family Studies

J. Douglas Coatsworth
Professor of Human Development and Family Studies at Colorado State University
Special Member

Melvin Mark
Professor of Psychology
Department Head

*Signatures are on file in the Graduate School

## Abstract

Anxiety and depressive symptoms are the most commonly experienced mental health problems in the US. Symptoms of anxiety and depression have a biological basis in dysregulated physiological responding, particularly of the cardiac vagus, measured as respiratory sinus arrythmia (RSA), which is implicated in emotion regulation capabilities. Research has shown that anxiety and depressive symptoms can be transmitted from parent to child through dynamics of the parent-child relationship. Differences in parent-child coregulation, or the active coordination of physiological, emotional, and/or behavioral states between parent and child over time, may be one process through which emotional dysregulation and symptoms are transmitted. This dissertation project encompasses two studies that build on established research by examining interrelationships between parent mental health symptoms and physiological precursors, parent-child coregulation of physiology, and child behavioral and emotional adjustment. Study I examined person-centered profiles of mental health risk in parents using self-reported depression and anxiety symptoms as well as resting RSA and RSA stress response. These profiles are then used to predict children's emotional and behavioral adjustment. We found that membership in a higher-risk mental health and physiological regulation profile for parents predicted greater behavioral and emotional problems for children. Study II examined whether parents' profile membership impacted patterns of coregulation of RSA between parents and children. Results are discussed with regard to 1) The relationship between anxiety and depressive symptoms and underlying physiological regulation in parents; 2) The relationship between poor RSA regulation and expressed mental health symptoms in parents, processes of coregulation with young children, and transmission of risk for emotional and behavioral problems; and 3) Differences between mothers' and fathers' experience of anxiety and

depressive symptoms as well as differences in the relationship between mental health and physiological risk and interactions with young children.

## Table of Contents

## List of Tables

## List of Figures

## Acknowledgements

This material is based upon work supported by the National Institute for Child Health and Human Development under Award No. K01HD068170. Any opinions, findings, and conclusions or recommendations expressed in this publication are those of the author and do not necessarily reflect the views of the National Institute for Child Health and Human Development.

## GENERAL INTRODUCTION

Anxiety and depressive disorders are the most commonly experienced mental health problems in the United States (American Psychological Association, 2013). An estimated 19.1% of adults were diagnosed with an anxiety disorder in 2017, and approximately 1 in 3 adults will experience any anxiety disorder during their lifetimes (National Institute of Mental Health, 2017). The National Survey on Drug Use and Health found that approximately 17.3 million adults in the US experienced a major depressive episode in 2017, characterized by depressed mood, loss of interest in activities, and other problems such as sleep disturbance, excessive guilt, and problems with concentration (SAMHSA, 2017). An even greater number of individuals experience subclinical levels of anxiety and depressive symptoms. Symptoms of depression as laid out by the DSM-V include: a) depressed mood, characterized by sadness, emptiness, or lack of hope; b) loss of interest or pleasure in activities; c) significant changes in weight or loss of appetite; d) sleeping too much or too little; e) feelings of restlessness or being slowed down; f) tiredness or lower energy than normal; g) feelings of worthlessness or excessive guilt; h) lack of concentration or ability to think clearly; i) recurrent thoughts of death or suicide. The key symptom of generalized anxiety disorder is excessive worry that is difficult to control. However, associated symptoms include: a) restlessness; b) being easily fatigued; c) difficulty concentrating; d) irritability; e) muscle tension; and f) difficulty falling or staying asleep (APA; 2013). While depressive and anxiety disorders are distinct, many of the symptoms overlap, and the two are frequently comorbid (between 60-80% overlap in some studies), meaning that

individuals with anxiety problems are more likely to also have symptoms of depression, and vice versa (Lamers et al., 2011; Mineka, Watson, & Clark, 1998).

Symptoms of anxiety and depression are thought to have a biological basis in dysregulated physiological responding; in particular, disruption of cardiac vagal activity, which indexes arousal and underlies emotion regulation and social engagement (Beauchaine, 2015; Porges, 2003). In young children, cardiac vagal activity, measured as respiratory sinus arrythmia (RSA), is associated with the ability regulate emotions, and in older children and adults, RSA is related to symptoms of anxiety and depression (Beauchaine, 2001; Graziano & Derefinko, 2010). Baseline or resting RSA is thought to reflect the propensity to react to the environment, and resting RSA is higher in healthy individuals compared with those with symptoms of anxiety and/or depression (Thayer, Friedman, & Borkovec, 1995; Rottenberg, Clift, Boden, & Salmon, 2007), or children who have trouble regulating their emotions (Fox, 1989; Huffman et al., 1998). In addition, the ability to withdraw vagal tone in stressful situations (and thus mobilize coping resources) appears to be impaired in individuals who experience symptoms of anxiety and depression (Beauchaine & Thayer, 2015). In children, poor RSA withdrawal is associated with greater internalizing symptoms (Graziano & Derefinko, 2010). In adults, individuals show RSA dysregulation in either a lack of withdrawal of RSA to stress (e.g., Rottenberg et al., 2007) or excessive RSA withdrawal (i.e., showing a greater response than is necessary; Gouin, Deschenes, & Dugas, 2014; Hu et al., 2006).

Research has supported that mental health symptoms can be transmitted from parents to children through behavioral and physiological mechanisms during parent-child interactions (Tronick, 1989). Differences in parent-child coregulation patterns (i.e., the coordination of parents' and children's behavioral, emotional, and/or physiological states over time; Olson &

Lunkenheimer, 2009) are observed when parents experience mental health symptoms (Tronick & Beeghly, 2011). For instance, parent-child dyads in which parents' experience symptoms of depression or anxiety also display less attunement (i.e., time-dependent concordance) in their behavioral and emotional states (Beebe & Lachman, 1998; Feldman et al., 2009). This is important because the interactive process of coregulation can facilitate the development of effective emotion regulation skills for young children (Calkins, 2011; Feldman, 2012). Research has suggested that parent-child attunement of RSA is also observed between healthy parent-child dyads, promoting the development of emotion regulation for children (Davis, West, Bilms, Morelen, & Suveg, 2018). However, physiological attunement can be disrupted in parent-child dyads who are at higher risk for mental health problems (Lunkenheimer et al., 2015; Lunkenheimer et al., 2018). As such, coregulation of cardiac vagal tone may be one way in which symptoms of anxiety and depression are transmitted from parent to child in early childhood.

## Physiological Basis for Mental Health Problems

**Respiratory sinus arrhythmia.** Anxiety and depressive symptoms are thought to arise from maladaptive emotion regulation processes that have a biological basis in physiological stress reactivity. In particular, measures of respiratory sinus arrhythmia (RSA) hold promise as reliable biomarkers for psychopathology because levels of RSA are thought to reflect a biological propensity to regulate emotions (Beauchaine, 2015). RSA is a measure of cardiac vagal activity, which reflects engagement of the parasympathetic nervous system (PNS). The PNS is one branch of the autonomic nervous system which functions to maintain physiological homeostasis and enables growth and restoration. The PNS works in concert with the sympathetic branch of the autonomic nervous system (SNS), which is responsible for the "fight-flight"

response. The PNS and SNS innervate many of the same visceral structures and have historically been thought to serve antagonistic functions (e.g., the PNS slows heart rate while the SNS speeds up heart rate). We know now that autonomic regulation is more complicated, with patterns of reciprocal activation, coactivation, and coinhibition of the two branches determining behavior (Berntson, Cacioppo, & Quigley, 1991). Nevertheless, in well-regulated individuals, it would be expected that the PNS is the dominant influence during times of rest while the SNS becomes dominant during times of stress or challenge that require mobilization and preparation for action. In contexts of perceived safety, the PNS acts as a "brake" on the heart via the vagus nerve, slowing heart rate and presumably inhibiting the fight-flight response (Porges, 2007). Conversely, when an individual perceives a threat, or interprets a situation as stressful, the vagal brake is withdrawn, facilitating mobilization of resources for the defensive response of the SNS (Porges, 2007).

RSA has gained popularity in psychophysiological research due to the fact that it is one of the few measures that can isolate activity of the PNS from overall autonomic reactivity (Berntson, Cacioppo, & Quigley, 1993). While both the PNS and the SNS contribute to variability in heart rate, the PNS acts to quickly regulate heart rate with changing contexts, while the SNS acts more slowly. Thus, cardiac vagal tone can be captured by measuring high-frequency heart rate variability, specifically within the frequency band associated with respiration, which serves as a basis for quantification of RSA. Ability to isolate vagal activity is important given the functional significance of cardiac vagal tone. Theories regarding its importance (e.g., Polyvagal Theory, Porges, 2007; Neuro-visceral Integration Model, Thayer & Lane, 2000) have placed cardiac vagal tone at the heart of processes of emotional expression, social engagement, and self-regulation. According to these theories, vagal pathways are part of a

greater neuro-visceral network which includes connections to muscles of the face and head, enabling the spontaneous expressions of emotion that are universally recognized in humans (e.g., smiles, scowls, frowns; Porges, 1991; 2007). Connectivity between vagal pathways and the prefrontal cortex have also been proposed, suggesting that RSA may serve as a periphery measure of self-regulatory executive processes (Thayer & Lane, 2000; 2009). The rapid inhibition and disinhibition of the vagal brake also suggest that withdrawal of RSA may be one of the fastest and most flexible physiological markers of stress, reflecting quick adaptation to different social environments and contexts (Porges, 2007). Coordination between RSA and the hypothalamic-pituitary axis have also been observed, such that increased RSA may be linked with decreases in the release of the stress hormone cortisol (Porges, 2003).

**Mental health symptoms and RSA.** The role of the vagus in social communication, self-soothing, and inhibition of the fight-flight response make it a candidate biomarker for psychopathology, as symptoms of mental health problems reflect deficiencies in these areas (Porges, 2003). Prior research supports the hypothesis that greater levels of RSA at rest and reliable withdrawal of RSA during times of stress are related to emotional regulation and social competence, whereas lower RSA and unreliable modulation of RSA are associated with poor emotion regulation, and with psychopathology in extreme cases (Beauchaine, 2001; Porges, 2007). Features of poor vagal control, including impaired social awareness, dysregulation, and emotional reactivity, are present in profiles of many mental health problems (Porges, 2007). For example, the inability to inhibit sympathetic and adrenocortical arousal during times of relative safety (i.e., when context does not call for such responses) is both a symptom of poor vagal control and a key feature of anxiety disorders (Porges, 2007). Depressive disorders also include

symptoms that would be consistent with compromised vagal control, such as unresponsive social behavior and fewer facial expressions and instances of shared gaze (Rottenberg, 2007).

Low resting RSA may reflect emotional inflexibility; when levels are already low in every-day contexts, the rapid withdrawal of RSA in stressful situations is not available as a regulatory strategy (Beauchaine, 2001). Low resting RSA may also mean that inhibitory control of the fight-flight response is chronically compromised in individuals with mental health disorders (Porges 2007). From a dynamic systems perspective, healthy systems are characterized by adaptive variability, while pathological systems are characterized by predictability and rigid cycles (Friedman, 2006). This perspective would also place low baseline RSA in the profile for psychopathology, as low RSA would represent lower high-frequency heart rate variability and may also reflect inflexibility of the physiological stress response (Friedman, 2006). Likewise, the vagal response to stress may be altered in individuals with symptoms of psychopathology. Excessive withdrawal of RSA to stress has been linked with emotional lability (i.e., greater intensity and reactivity of emotional responses) and thus may reflect risk for psychopathology (Beauchaine, 2015). On the other end of the spectrum, a blunted RSA response or augmented RSA in the face of stress may also mark psychopathology, reflecting emotional inflexibility and inappropriate stress response (Porges, 2003; 2007). Thus, a moderate level of RSA suppression characterizes healthy individuals, with dysfunction reflected in the extremes of each end, either too much or too little suppression of RSA in response to stress (Beauchaine, 2015).

Early in development, research has found associations between levels of RSA and emotion regulation (ER), or ability to monitor, evaluate, and modify emotional responses in accordance with one's goals (Beauchaine, 2001; Thompson, 1994). Relationships between RSA and emotion regulation are observed as early as infancy. Infants with greater baseline RSA show

more appropriate emotional response to both positive and negative events (Fox, 1989). Infants with higher baseline RSA may also be more easily soothed by parents when experiencing negative emotion (Huffman et al., 1998). By contrast, lower RSA withdrawal to stress in infancy predicts greater behavioral and social problems by the preschool period (Brooker et al, 2013; Dale et al., 2011; Graziano, Keane, & Calkins, 2007). In preschool-aged children, higher baseline RSA predicted declines in parent-rated negativity across childhood (Blandon, Calkins, Keane, & O'Brien, 2010). Higher baseline RSA followed by greater RSA withdrawal during an anger-inducing task was associated with better regulation of anger and reduced aggression among 4-year-old children (Miller et al., 2013). Toddlers who showed less RSA withdrawal during cognitive and emotional challenges also displayed greater unregulated negative affect and less employment of regulatory behaviors during stressful situations (Calkins & Dedmon, 2000). Dysregulation of RSA has been linked with behavior problems and aggression in infants, toddlers, and preschool-aged children (Calkins & Dedmon, 2000; Calkins, Graziano, & Keane, 2007; Calkins & Keane, 2004; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996).

Accounts differ as to whether dysfunctions in vagal control represent a general biomarker for all symptoms of psychopathology (i.e., poor ER), or whether there are differences in patterns of RSA across discrete diagnoses, for instance, between anxiety and depressive disorders (Beauchaine, 2001; Beauchaine, 2015; Beauchaine & Thayer, 2015). According to the Tripartite Model (Clark & Watson, 1991), anxiety and depression share a general distress factor which accounts for the overlap in symptoms and frequent comorbid diagnoses. However, each disorder also has unique characteristics that differentiate the two. While depression is characterized by low positive affect (i.e., anhedonia), anxiety is characterized by heightened autonomic arousal. The model is supported by factor analytic studies examining the relatedness between specific

subscales of various questionnaire measures (Anderson & Hope, 2008; Dunbar, Ford, Hunt, & Der, 2000; Watson et al., 1995). Subscales measuring symptoms of physiological hyperarousal (e.g., tension, shortness of breath, dizziness, dry mouth, "butterflies" in the stomach, and feelings of panic) appear distinct from the general distress factor and are more characteristic of anxiety than depression. Such symptoms of autonomic arousal involve activity of both the PNS and SNS, and it is unclear whether the type of arousal associated with anxiety stems from overactivity of the SNS, underactivity of the PNS, or an interaction between the two. It is possible that since RSA reflects emotional dysfunction, RSA patterns may underlie the general distress factor, and differentiation of the two disorders stems from differences in SNS and adrenocortical function (Beauchaine & Thayer, 2015). This idea is supported by research that suggests low RSA paired with poor RSA withdrawal predict both internalizing and externalizing symptoms throughout childhood and adolescence (Graziano & Derefinko, 2013). However, one study that attempted to test the validity of the tripartite model found that lower resting RSA in youths (aged 10-13) correlated with parent-reported anxiety symptoms, but not depressive symptoms, providing support for the specific quality of autonomic arousal in anxiety during the transition to puberty (Greaves-Lord et al., 2007).

The extant research suggests that there is a link between RSA and symptoms of anxiety and depression, however, the relationship between expressed symptoms and underlying physiological functioning is likely more complicated than what has previously been represented. It is possible that inconsistent findings, such that higher RSA is linked to depressive and anxiety symptoms in some studies but not others, or that the RSA stress response may show differentiation between depression and anxiety in some cases, but not others, stem from different clustering of symptoms within patient and community samples (Rottenberg et al., 2002).

Heterogeneity may be observed even within diagnostic categories, as well as within individuals who express subclinical levels of anxiety and/or depression (Beauchaine, 2015). To capture that level of differentiation, new statistical techniques need be employed. Thus, it is the goal of the first study of this proposal to examine how symptoms of anxiety and depression and physiological reactivity organize within individuals in a community sample of parents of young children using latent profile analysis (LPA). LPA allows for the differentiation of subgroups of individuals who differ based on their responses on a variety of indicators. In this case, indicators will include resting RSA, RSA stress reactivity, and symptoms of anxiety and depression.

**Intergenerational Transmission of Anxiety and Depression**

The presence of depressive and/or anxiety symptoms in parents remains a key risk factor for the development of depressive and anxiety disorders in children (Hammen, 2009; Joormann, Eugène, & Gotlib, 2008; Turner, Beidel, & Epstein, 1991). Children of parents who display symptoms of anxiety and/or depression show greater internalizing (e.g. withdrawal, shyness) and externalizing (e.g., overactivity, aggression) symptoms in early childhood (Goodman et al., 2011). By middle childhood and adolescence, children of depressed parents also experience greater rumination and negative attributions, both considered symptoms of major depressive disorder (Gotlib, Joorman, & Foland-Ross, 2014). Prior research suggest that genetics certainly play a role in the transmission of depressive and anxiety disorders from parents to children. For instance, risk of developing a depressive disorder increases threefold when an individual has a first-degree relative with an already diagnosed disorder (Tsuang & Faraone, 1990). However, environmental factors predominantly explain the link between subclinical levels of depression and anxiety in parents and children, which are more common than formal diagnoses (for review, see Goodman & Gotlib, 1999). As such, understanding the potentially modifiable mechanisms

that contribute to the transmission of mental health symptoms from parent to child is critical for developing effective preventive interventions. Evidence supports the idea that mental health symptoms can be transmitted from parents to children through mechanisms of the parent-child relationship, as described below (Tronick, 1989).

**Parenting.** The presence of depressive and anxiety symptoms in parents impact the parenting behaviors and emotions that are expressed during parent-child interactions. Symptoms of depression such as excessive sadness, anhedonia, and withdrawal from the social environment have a direct impact on the content of parents' interactions with children (Goodman & Gotlib, 1999; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Parent-child interactions are characterized by greater sadness, conflict, and hostility when parents experience elevated symptoms of depression (Lovejoy et al., 2000). Withdrawal from daily activities may lead parents who experience symptoms to be less able to structure the child's environment to promote positive developmental outcomes, for instance, offering less support for children's growing autonomy during the preschool years (Cicchetti & Schneider-Rosen, 1986). Rumination, which refers to the unproductive rehearsal of thoughts related to negative mood or experiences (Joorman & Gotlib, 2010), is commonly experienced by individuals with depressive symptoms and may distract parents from attending to children's needs (Goodman & Gotlib, 1999; Psychogiou & Parry, 2014). This lack of attention towards children may cause parents with depressive symptoms to over- or underestimate their children's abilities (Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). As such, parents with depressive symptoms were found to place unrealistic expectations on their young children, thus overwhelming their immature systems for managing difficult emotions (Zahn-Waxler et al., 1990).

Parent-child interactions are also impacted by parents' symptoms of anxiety, which can cause parents to be less able to meet children's needs. Similar to parents with depressive symptoms, research has shown that parents with anxiety may become withdrawn during parent-child interactions, offering less assistance, acknowledgement, or praise to their children while completing difficult tasks (Woodruff-Borden, Morrow, Bourland, & Cambron, 2002). Mothers with anxiety symptoms may also possess a parenting style characterized by intrusive behaviors (i.e., attempting to help a child complete a task when they are capable of doing it alone; Feldman, Granat, Pariente, Kanety, Kuint, & Gilboa-Schechtman, 2009). Mothers with anxiety symptoms may display a hypervigilant attention towards their children, which can lead to interference with children's attempts at self-regulation, intervening before the child is able to handle a challenge for themselves (Beebe & Lachman, 1998). Mothers reporting higher anxiety and shyness may be over overprotective and less supportive of children's autonomy with their preschool-aged children (Root, Hastings, & Rubin, 2016). Parents with anxiety symptoms may respond to threats in their children's environment more strongly, modeling this anxious processing style to their children (Field & Lester, 2010). One recent study showed that parents' anxiety predicted greater attention to threat for children a few years later, putting them more at risk for developing an anxiety disorder (Aktar, Van Brockstaele, Pérez-Edgar, Wiers & Bögels, 2019). Parents with higher levels of anxiety symptoms may also have difficulty regulating their own emotions, which in turn could perpetuate the development of internalizing symptoms in children (Han, Lei, Qian, Li, & Wang, 2016; Kerns, Pincus, McLaughlin, & Comer, 2017).

**Child emotion regulation.** Poorer parenting practices may perpetuate the development of anxiety and depressive symptoms in children by impacting ER ability (Thompson, 1994). Depressive and anxiety disorders are among the most commonly diagnosed in children, but rates

remain low until adolescence (<1-2%), with the exception of separation anxiety disorder (APA, 2013; CDC, 2019). However, young children can show early signs of anxiety and depression which may become worse as they age, for example, difficulties regulating emotion appropriately (i.e., not under- or over-regulating) may serve as a precursor to psychopathology in children (Fox & Calkins, 2003). Children who experience both a greater level of negative emotion and have difficulty regulating this emotion are more likely to develop depression. Studies have shown this effect as early as the preschool years (Reinfjell et al., 2016), and ER continues to moderate relationships between negative emotion and depression into late childhood and adolescence (Verstraeten, Vasey, Raes, & Bijttebier, 2009). While most children experience sadness during an unpleasant situation, a child at risk for developing depression may have difficulty resolving this sadness, instead experiencing excessive and persistent negative emotion (Cole, Luby, & Sullivan, 2008). In children as young as 5, experiencing a loss gives way to hopelessness and pessimism when children have been exposed to parents' depressive symptoms (Murray, Woolgar, Cooper, & Hipwell, 2001). Older children at-risk for developing depressive symptoms show negative attentional biases (i.e., remembering negative content more clearly, having difficulty inhibiting the processing of negative stimuli; Gotlib et al., 2014). Similarly, children with anxiety symptoms experience a greater intensity of negative emotion and have difficulty utilizing effective ER strategies (Carthy, Horesh, Apter, & Gross, 2010). Older children who have been diagnosed with an anxiety disorder may have difficulty managing worry, sadness, and anger (Suveg & Zeman, 2004).

Although parent's anxiety and depressive symptoms can impact the development of ER and symptoms in children throughout the lifespan, their influence may be particularly important during the period of early childhood (i.e., between the ages of 0-5). Parents' support for

children's ER is particularly important during the first few years of life when children have limited capacity for self-regulation (Calkins, 2011). In addition, exposure to parental symptoms at early ages increases the likelihood that children will develop maladaptive strategies for interacting with the affected parent, which may extend to their interactions with others and become internalized as poorer self-regulation (Tronick, 1989). These ideas are supported by a meta-analysis of the effects of maternal depressive symptoms on behavior from infancy to early adulthood, which revealed that maternal symptoms had a greater detrimental impact on children's internalizing, externalizing, and general psychopathology symptoms, as well as negative affect and behavior, when children were first exposed to symptoms at younger ages (Goodman et al., 2011).

**Family dynamics.** The majority of research in the field has focused on how symptoms of anxiety and depression affect parenting in mothers. However, family systems perspectives suggest that parenting does not exist in a bubble; and in two-parent households, it is important to consider how mothers and fathers symptoms interact and influence the development of ER in children (Cox & Paley, 1997). Research on fathers suggests that fathers' depressive and anxiety symptoms have a similar negative effect on parenting practices and child emotional outcomes as do mothers' symptoms (Bögels & Phares, 2008; Sweeney & MacBeth, 2016; Wilson & Durbin, 2010). The practices of one parent are likely to influence the relationship between the child and the co-parent (Cox & Paley, 1997). Parents may adopt the parenting practices and strategies of their partners, and similar levels of negative parenting (e.g., intrusion) have been observed between mothers and fathers with the same child (Barnett, Deng, Mills-Koonce, Willoughby, & Cox, 2008). Symptoms of anxiety and depression that are experienced by fathers may affect the quality of the relationship between mothers and children, and vice versa (Field, Houssain, &

Malphurs, 1999; Goodman, 2008). For example, depressed mood in mothers had a negative impact on the father's parenting and vice versa, and the influence of each parent interacted to predict emotional symptoms in children, with the highest level of symptoms observed for children with negative experiences with both mothers and fathers (Malmberg & Flouri, 2011). In cases where both parents are depressed, paternal depression exacerbates the negative effects of maternal depression on child behavior problems, however, interactions with healthy fathers may buffer the negative impact of mothers' depressive symptoms on child outcomes (Mezulis, Hyde, & Clark, 2004). Thus, it is important to understand the influence of both mothers' and fathers' symptoms of anxiety and depression on children's ER and development of psychopathology symptoms.

**Dyadic parent-child coregulation.** Dysfunctional ER strategies likely emerge in the context of the family system where multiple factors interact, including parental psychopathology, temperamental vulnerabilities in the child, family stress, and support from other caregivers (Goodman & Gotlib, 1999). Despite these systemic influences, relatively little research on parents' mental health symptoms and child ER has been dedicated to understanding the dyadic, interpersonal context, which involves dynamic contributions from both parent and child (Lovejoy et al., 2000). This is an oversight, given that the form and organization of behavioral patterns observed during parent-child interactions may serve a significant role in either transmitting mental health symptoms to children, or protecting children from them.

Early in life, parental support is provided in part through the process of parent-child *coregulation*, which refers to the active organization and coordination of parents' and children's emotional, behavioral, and physiological states in real time (Olson & Lunkenheimer, 2009). Over time, parents and children develop a series of reliable patterns for interacting with one another,

and in the case of elevated depressive and anxiety symptoms in parents, maladaptive patterns of coregulation may emerge (Tronick & Beeghly, 2011). Symptoms of depression and anxiety have been associated with greater cognitive inflexibility (Barrett & Fleming, 2011), which may increase the likelihood that parents and children get "stuck" in maladaptive systems of coregulation. For example, greater negativity on the part of parents may prompt coercive cycles, wherein behavioral dysregulation in children is met by punitive over-control and subsequent disengagement from parents, creating a positive feedback loop promoting future negative responses (Patterson, 2002).

One pattern of coregulation the parent-child dyadic system that is particularly important for promoting self-regulation in young children is the temporal coordination of elements of the interaction, referred to as synchrony or *attunement* (Feldman, 2007). From very early in development, parents match infants' affective states through shared gaze and emotional expressions during face-to-face interactions. This matching requires that the parent perceive, understand, and respond sensitively to the child's emotional states (Feldman, 2012; Tronick & Beeghly, 2011). Attunement in parent-child behavior is characterized by parental behaviors that are well-coordinated with infants' social cues so that the interaction is not overstimulating, but still provides the child with useful input for forming and understanding social relationships (Feldman, 2012). In typical parent-child dyads, the process of coregulation involves mismatches as well as attunement, for example, a parent may not return her infant's smile, or a parent's effort to assist a child may be perceived as overwhelming and intrusive. However, healthy parent-child relationships are characterized by greater attunement on average than higher risk dyads, and healthy dyads quickly return to a state of attunement following periods of mismatch (Tronick, 1989). The accumulation of experiences of attunement with a parent over time allows the child to

develop trust in the support of others, which leads to the development of confidence in managing physiological and emotional states, and in turn supports future efforts at emotion regulation. Conversely, children who experience less sensitive parenting, and therefore fewer opportunities for attunement, may be at risk for developing maladaptive strategies of self-regulation that persist into adolescence and adulthood, making them vulnerable to developing mental health issues later in life (Tronick & Beeghly, 2011).

Patterns of parent-child coregulation differ when parents display symptoms of anxiety and depression. This is observed at the behavioral level as parents' difficulty achieving and maintaining attunement with their children. High-risk parenting has been characterized by excessive maternal engagement to the point of intrusion when mothers experience anxiety symptoms, and with minimal engagement when depressive symptoms are present (Feldman et al., 2009). Parental sensitive engagement is necessary to conform to the needs of the child and facilitate attunement (Feldman et al., 2009). However, sensitivity is lowered when mothers display symptoms of anxiety and/or depression, which can lead to inappropriate maternal behavior that is not coordinated with the child (Beebe & Lachman, 1998). Parents who display symptoms of depression tend to be withdrawn and inconsistent in their interactions with their children, limiting opportunities for emotional attunement (Tronick, 1989; Tronick & Beeghly, 2011). Parents with higher depressive symptoms may also struggle to coordinate their behavior with their children's behavior, resulting in poorer scaffolding that may in turn contribute to emotional dysregulation in children (Hoffman, Crnic, & Baker, 2006). Parents who display symptoms of anxiety may either be self-absorbed, which also results in difficulty establishing attunement with a relational partner, or they may be hypervigilant to the point of intrusion, again disrupting efforts at attunement (Beebe et al., 2011). For example, mothers with anxiety may

engage in excessive "chase" behavior, attempting to regain their infant's attention when they turn away from the parent to engage in self-soothing behavior, thus disrupting efforts at self-regulation (Beebe & Lachmann, 1998). Parents with depression and anxiety symptoms may also have a more difficult time reestablishing attunement following a behavioral or emotional mismatch (Tronick, 1989).

Attunement is also observed at the biological level across many physiological systems, including vagal control, heart rate, and hormonal systems through the release of cortisol (Davis, West, Bilms, Morelen, & Suveg, 2018). Attunement at the behavioral level is supported by physiological systems that have evolved to serve affiliative functions, e.g., coordination of heart rate and daily sleep-wake cycles (Feldman, 2007). Cycles of mutual reinforcement are established between the physiological and behavioral levels. As parents and children match each other's emotional expressions and behaviors, attunement also occurs in their complementary biological systems, including autonomic arousal, hormonal release, and brain activation (Feldman, 2012). Attunement at the physiological level may in turn promote behavioral and emotional synchrony, producing the feeling of security that is experienced within relationships that are characterized by organized attachment (Feldman, 2007). Feldman and colleagues (2011) found that during dyadic interaction, mothers adapted their heart rate to that of their infant's, and infants in turn coordinated their heart rate with mothers within lags of less than 1 s, establishing biological attunement in the acceleration and deceleration of heart rate. Coupling between mothers' and infants' heart rate was stronger during periods of matching emotional expressions (i.e., mutual positivity; Feldman, Magori-Cohen, Galili, Singer, & Louzoun, 2011).

Importantly for the development of emotion regulation, attunement of cardiac vagal activity, measured using RSA, has been observed in healthy parent-child dyads, particularly

during the developmental period of early childhood (Davis et al., 2018). For instance, Lunkenheimer and colleagues (2015) modeled moment-to-moment shifts in RSA for parents and preschool-aged children during a challenging interaction task. Results revealed that parent RSA predicted child RSA across time, even after accounting for fluctuations in each individual's RSA. Parent-child coregulation of RSA may support the development of adaptive vagal control in young children, thus promoting their ability to manage physiological and emotional arousal and engage in healthy social interactions. Research has shown that better RSA regulation in children is associated with improved attunement with parents at the behavioral level. In infants, well-regulated RSA response to threat was related to greater mother-child synchronous play (Moore et al., 2009; Moore & Calkins, 2004). Conversely, infants with lower RSA show fewer instances of behavioral and emotional matching during face-to-face interaction with parents (Feldman & Eidelman, 2007). Children also show higher baseline RSA and better regulated RSA stress responses when working in collaboration with a parent than when working alone (Calkins, Graziano, Berdan, Keane, & Degnan, 2008). Improved RSA regulation in parents promotes efforts at both behavioral and physiological attunement as well. For example, mothers who showed an appropriate RSA response to stress were also more sensitive and responsive to infants' social cues (Moore et al., 2009). Thus, RSA regulation and attunement between parents and children seem to be related and facilitate one another.

As previously discussed, depressive and anxiety symptoms in parents may lead to disruption of attunement between parents and children at the behavioral level (Beebe & Lachman, 1998; Tronick & Beeghly, 2011). Due to the interdependence of behavioral and physiological attunement, disruption of behavioral attunement may also extinguish attunement in physiological systems (Feldman, 2012). Prior research lends support to this idea by

demonstrating that maternal sensitive engagement (which is reduced when symptoms of anxiety or depression are elevated) is associated with stronger attunement in mother-child RSA, while maternal behavioral disengagement is associated with weakening coordination, and disruption of attunement at very high levels (Skoranski, Lunkenheimer, & Lucas-Thompson, 2018). A parent who has difficulty regulating their autonomic arousal may become a "moving target" for the child's attempts to coordinate vagal activity, resulting in maladaptive patterns of self-regulation. Woody and colleagues (2016) examined RSA concordance in mothers and adolescent children during a talking task among patients with MDD and control participants. They found that parent-child dyads where mothers had MDD were characterized by negative synchrony in RSA, such that increases in RSA for mothers were met with decreases in RSA for children, and vice versa (Woody, Feurer, Sosoo, Hastings, & Gibb, 2016). However, RSA concordance is not always dampened in contexts of risk, in some cases, dysfunction in dyadic relationships may result in greater attunement of physiological states, which may also convey risk through excessive coupling and transmission of poor regulatory strategies (Gray, Lipschutz, & Scheeringa, 2017; Smith, Woodhouse, Clark, & Skowron, 2016). For example, Suveg and colleagues (2016) found increased attunement in heart rate variability for mothers and children in dyads who exhibited greater risk (as measured using a composite score that including maternal psychopathology). However, higher behavioral attunement and child self-regulation were associated with lower physiological attunement in this group, suggesting that coordination of physiological systems might actually be conferring additional risk in the already high-risk group (Suveg, Shaffer, & Davis, 2016).

Research to date suggests that symptoms of depression and anxiety in parents have an impact on the degree of attunement obtained by parent-child dyads, however, the nature of that

relationship remains unclear. For some dyads, physiological attunement may be disrupted, which would impact children's self-regulation by reducing opportunities to engage in coregulation of physiology with parents (Woody et al., 2016). For others, attunement may either be strengthened or unchanged by symptoms, but may actually convey additional risk when children are coregulating with a parent affected by symptoms of anxiety and depression (Suveg et al., 2016). No studies have yet examined whether parents' self-regulation of RSA is related to the degree of attunement with children. However, it may be expected that parents who have trouble regulating their RSA would be poor interactive partners for children, resulting in either reduced attunement (Moore et al., 2009), or again conveying poorly regulated physiological responsivity to children through means of coregulation. The second study of this proposal will approach these questions by examining how mental health profiles in parents (including RSA reactivity and symptoms of anxiety and depression) relate to and predict attunement in RSA between parents and young children. Relationships between attunement and children's emotion regulation and internalizing symptoms will also be explored to determine whether increased attunement in the context of mental health risk is more detrimental to children's outcomes.

## STUDY I: LATENT CLASS ANALYSIS OF MENTAL HEALTH RISK IN MOTHERS AND FATHERS AND RELATIONS TO EMOTIONAL SYMPTOMS IN CHILDREN

Mental health problems have become increasingly common over the past several decades (Twenge et al., 2010). Anxiety and depressive disorders are the most commonly experienced mental health conditions by adults in the United States (American Psychological Association, 2013). An even greater number of individuals experience subclinical levels of anxiety and depressive symptoms (APA, 2013), such as depressed mood, loss of interest in activities, sleep and eating disturbances, and problems with concentration, to name a few. Among this population, approximately 68% of women are mothers and 57% are fathers (Mental Health Foundation, 2019). Both mothers and fathers are at risk for developing perinatal depression and anxiety in the months after child birth (Gaynes et al., 2005; Paulson & Bazemore, 2010). This could be due to parenthood presenting additional stressors to individuals' lives (Crnic & Low, 2002; Deater-Deckard, 1998). Parent mental health is of particular interest because anxiety and depressive symptoms may negatively impact children's outcomes through poor parenting practices, low self-regulation ability, dysfunctional parent-child interaction patterns, and modeling anxious and depressive styles (Tronick, 1989; Tronick & Beeghly, 2011).

Symptoms of anxiety and depression are thought to have a biological basis in dysregulated physiological activity, in particular, dysregulated functioning of the parasympathetic nervous system on the heart, which is measured using respiratory sinus arrythmia (RSA; Berntson, Cacioppo, & Quigley, 1993). Beginning early in development, RSA appears to provide support for emotion regulation (ER) capabilities (i.e., the ability to monitor, evaluate, and modify emotional responses in accordance with one's goals; Beauchaine, 2001; Thompson, 1994). Dysregulated RSA may reflect low ER skills, which in turn can develop into

mental health problems by adulthood (Beauchaine, 2015; Beauchaine & Thayer, 2015). Studies seem to suggest that dysregulation in RSA is associated with both anxiety and depressive symptoms, however, it remains unclear whether different patterns of RSA dysregulation give rise to anxiety versus depressive symptoms respectively, and whether dysregulation is always observed along with heightened symptoms. The associations between behavioral/emotional symptoms and underlying physiology may be more complicated than simple one-to-one relations, and therefore may require more sophisticated methods to disentangle. Accordingly, the present study examined profiles of mental health risk using a person-centered approach and including indices of symptoms of anxiety and depression and activation of RSA. We also tested whether parents' belonging to a higher-risk profile versus a low-risk profile was related to emotional and behavioral problems for their 4-year-old children.

**Respiratory Sinus Arrythmia**

Anxiety and depressive symptoms are thought to arise from maladaptive emotion regulation (ER) processes that have a biological basis in physiological stress reactivity. Measures of respiratory sinus arrhythmia (RSA) hold promise as reliable biomarkers for psychopathology symptoms (Beauchaine, 2015; Beauchaine & Thayer, 2015). RSA is a measure of cardiac vagal activity, which reflects engagement of the parasympathetic nervous system on the heart (Berntson et al., 1993). When an individual is at rest, parasympathetic engagement is typically higher, indicated by higher levels of RSA (Porges, 1991). At higher levels, RSA acts as a "brake," slowing heart rate during times of rest (Porges, 1991). When an individual experiences stress or challenge, the "brake" is withdrawn (i.e., RSA decreases), allowing the fight-flight response to become active and heart rate to speed up in order to respond to environmental demands (Porges, 2007). Thus, in well-regulated individuals, it would be expected that RSA

would be higher during times of rest and lowered or withdrawn during instances of stress or challenge.

Theories regarding the importance of RSA (e.g., Polyvagal Theory, Porges, 2007; Neuro-visceral Integration Model, Thayer & Lane, 2000) have placed it at the heart of processes of emotional expression, social engagement, and self-regulation. According to these theories, RSA is part of a greater neuro-visceral network enabling the spontaneous expressions of emotion that are universally recognized in humans (e.g., smiles, scowls, frowns; Porges, 1991; 2007). Connections with the prefrontal cortex have also been proposed, suggesting that RSA may serve as a periphery measure of self-regulatory executive processes (Thayer & Lane, 2000; 2009). The withdrawal of RSA may be one of the fastest and most flexible physiological markers of stress, reflecting quick adaptation to different social environments and contexts (Porges, 2007). Coordination between RSA and the hypothalamic-pituitary axis have also been observed, such that increased RSA may be linked with decreases in the release of the stress hormone cortisol (Porges, 2003).

**Mental Health and RSA**

The role of RSA in social communication, self-regulation, and inhibition of the fight-flight response make it a candidate biomarker for psychopathology, as symptoms of mental health problems reflect deficiencies in these areas (Porges, 2003). For example, the inability to inhibit sympathetic and adrenocortical arousal during times of relative safety (i.e., when context does not call for such responses) is both a symptom of low resting RSA and a key feature of anxiety disorders (Porges, 2007). Depressive disorders also include symptoms that would be consistent with low resting RSA and less RSA withdrawal to stress, such as unresponsive social behavior and fewer facial expressions and instances of shared gaze (Rottenberg, 2007).

**Anxiety and Depressive Symptoms.** Dysregulated emotion can give rise to mental health problems such as anxiety and depression in adolescence and adulthood (Beauchaine, 2001; Beauchaine, 2015). As RSA is considered a biomarker for ER, studies have focused on understanding how RSA regulation differs in individuals with anxiety and/or depression in both clinical and community samples. Accounts differ as to whether dysfunctions in RSA (and ER by association) represent a general biomarker for all symptoms of psychopathology or whether there are differences in patterns of RSA across discrete categories (e.g., between anxiety and depressive symptoms; Beauchaine, 2001; Beauchaine, 2015; Beauchaine & Thayer, 2015). According to the Tripartite Model (Clark & Watson, 1991), anxiety and depression share a general distress factor which accounts for the overlap in symptoms and frequent comorbid diagnoses. However, each disorder also has unique characteristics that differentiate the two. While depression is characterized by low positive affect (i.e., anhedonia), anxiety is characterized by heightened autonomic arousal. The model is supported by factor analytic studies examining the relatedness between specific subscales of various questionnaire measures (Anderson & Hope, 2008; Dunbar, Ford, Hunt, & Der, 2000; Watson et al., 1995).

One possibility from this model is that RSA reflects the general distress factor, which is observed in both depressive and anxious individuals (Beauchaine & Thayer, 2015). Supporting this idea, research has shown that levels of resting or average RSA tend to be lower in individuals who display greater symptoms of either depression or anxiety (Beauchaine & Thayer, 2015). Thayer and colleagues (1995) found that patients with generalized anxiety disorder (GAD) showed reduced average RSA compared to controls, an effect that held across task conditions of resting, worry, and relaxation. Watkins and colleagues found that stepwise increases in trait anxiety predicted similar stepwise reductions in resting RSA (Watkins,

Grossman, Krishnan, & Sherwood, 1998). Another study found that that patients with GAD who also showed lower resting RSA had heightened sensitivity to unpredictable threat, suggesting that individuals with low resting RSA may have more difficulty regulating anxiety symptoms. Similar results have been found in studies of individuals with depressive symptoms. One study reported that average RSA levels were generally lower in individuals with major depressive disorder (MDD) versus controls, which was true for the resting phase of the study, a non-emotional challenge task, and a psychosocial stressor (Rottenberg, Clift, Boden, & Salmon, 2007). Moser and colleagues (1998) also showed a trend towards lower average RSA for MDD patients, although this did not reach significance.

Differentiation between individuals with anxiety versus depressive symptoms may be more evident when examining patterns of RSA change during stressful situations rather than resting or average RSA levels. In one study, rumination, which entails unproductive repetitive thinking about an individual's problems and is more characteristic of depression, was associated with either low RSA withdrawal or non-existent RSA change to stress. Conversely, excessive RSA withdrawal (i.e., withdrawal that was too extreme for the situation at hand, indicating heightened arousal) was associated with worry, which is more closely related to feelings of threat and is associated with anxiety (Kircanski Waugh, Camacho, & Gotlib, 2016). Gouin and colleagues (2014) found that excessive RSA withdrawal among college students during a "free worry" task was associated with greater distress and predicted greater perceived stress during finals week (Gouin, Deschenes, & Dugas, 2014). In contrast, individuals with MDD showed an augmented RSA response (i.e., increases in RSA from resting to stressor) to a challenging psychosocial task and no RSA change during a challenging physical task (Rottenberg et al., 2007). Thus, while resting or average RSA seem to be similarly affected by symptoms of anxiety

or depression, patterns of RSA change to stress may be different, but reflect dysregulation nonetheless. For individuals with anxiety symptoms, excessive RSA withdrawal is more apparent, whereas RSA augmentation and/or no change in RSA are more characteristic of individuals with depression.

Findings do not always support this general model, however. Hu and colleagues (2006) observed excessive RSA reductions to a stressful interview among patients with both anxiety and depression. Rottenberg and colleagues (2002) found that individuals with MDD who showed *higher* resting RSA reported greater sadness and were less likely to recover from a depressive episode 6 months later. (Rottenberg, Wilhelm, Gross, & Gotlib, 2002). Another study found that significant associations between RSA and anxiety symptoms were rendered non-significant after accounting for anti-depressive medication use (Licht, de Geus, van Dyck, & Pennix, 2009); similar findings were observed in a sample of individuals with both anxiety and depressive symptoms (Hu, Lamers, de Geus, & Pennix, 2016).

**Person-centered approaches.** Differentiation may be difficult to observe in patient samples because of frequent comorbidity between anxiety and depressive disorders (Lamers et al., 2011; Mineka, Watson, & Clark, 1998). It is possible that inconsistent findings, such that lower RSA is linked to depressive and anxiety symptoms in some studies but not others, or that the RSA stress response may show differentiation between depression and anxiety in some cases, but not others, stem from different clustering of symptoms within patient and community samples (Rottenberg et al., 2002). Heterogeneity may be observed even within diagnostic categories, as well as within individuals who express subclinical levels of anxiety and/or depression (Beauchaine, 2015). In community samples, where individuals may report subclinical

levels of symptoms of anxiety, depression, or both, it may be easier to differentiate typical RSA responses from those that signify mental health risk.

The extant research discussed above suggests that there is a link between RSA and symptoms of anxiety and depression, however, the relationship between expressed symptoms and underlying physiological functioning is likely more complicated than what has previously been represented. To capture that level of differentiation, new statistical techniques need be employed. Thus, the first goal of this study is to examine how symptoms of anxiety and depression and physiological reactivity organize within individuals in a community sample of parents of young children using a person-centered approach. Person-centered approaches have been utilized to examine multiple predictive factors as a cluster rather than individual variables, for example, analyzing the joint effect of different forms of parent emotion socialization on children's problem behaviors (Hernandez, Smith, Day, Neal, & Dunsmore 2018; Miller et al., 2015). This method condenses a large amount of information into one variable, which increases overall power for the resulting model (Larsen & Hoff, 2006). One recent study utilized a person-centered approach to determine profiles of emotion regulation which included physiological factors as well as observed emotion expression (Turpyn, Chapllin, Cook, & Martelli, 2015). However, no studies have utilized such an approach to examine how physiological regulation (i.e., RSA activity) and mental health symptoms organize in parents of young children.

**Mental Health and Parenting**

When parents experience symptoms of depression and/or anxiety, children are at a greater risk for developing a mental health problem themselves (Joormann, Eugene, & Gotlib, 2008; Turner, Beidel, & Epstein, 1991). Children of parents who display symptoms of anxiety and/or depression show greater internalizing (e.g. withdrawal, shyness) and externalizing (e.g.,

overactivity, aggression) symptoms in early childhood (Goodman et al., 2011). Many pathways have been proposed between parent to child mental health problems (Goodman & Gotlib, 1999). One way that symptoms may be transferred is through disruptions in parent-child interactions, especially in infancy and early childhood (Tronick, 1989; Tronick & Beeghly, 2011). When parents are depressed, parent-child interactions are characterized by greater sadness, conflict, and hostility (Lovejoy et al., 2000). Parents with anxiety symptoms may respond to threats in their children's environment more strongly, modeling this anxious processing style to their children (Field & Lester, 2010).

Furthermore, depressive symptoms are linked to a lack of attention towards children and withdrawal from parent-child interactions, while anxiety symptoms are linked to hypervigilant attention and over-involvement, both of which can thwart children's developing ER abilities (Beebe & Lachman, 1998; Root, Hastings, & Rubin, 2016; Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). Parents with higher levels of depressive or anxiety symptoms may also have difficulty regulating their own emotions, presenting poor examples of ER strategies for children, which in turn could perpetuate the development of internalizing and externalizing symptoms (Han, Lei, Qian, Li, & Wang, 2016; Kerns, Pincus, McLaughlin, & Comer, 2017). Similarly, children tend to coordinate their RSA with their parent, so a parent with mental health symptoms and poor RSA regulation may pass those difficulties on to their children through the process of matching RSA over time (Lunkenheimer, et al., 2018; Suveg, Shaffer, & Davis, 2016). Taken together, this work suggests that there are behavioral and physiological mechanisms through which parents' depressive and anxiety symptoms may affect children's ER and the development of internalizing and externalizing symptoms. However, it is unclear how such mechanisms

organize within parents, and whether different risk profiles emerge when considering RSA regulation alongside mental health symptoms from a person-centered perspective.

**Fathers.** Much of the research in the field has focused on how symptoms of anxiety and depression affect parenting and child outcomes for mothers. However, in two-parent households, it is important to consider how symptoms of each parent may affect children's outcomes (Cox & Paley, 1997). Research on fathers suggests that fathers' depressive and anxiety symptoms have a similar negative effect on parenting practices and child emotional outcomes as do mothers' symptoms (Bögels & Phares, 2008; Sweeney & MacBeth, 2016; Wilson & Durbin, 2010). While fathers show similar deficits in parenting associated with mental health symptoms as mothers, the way that fathers parent their children is different than mothers (i.e., fathers engage in more *triadic* engagement with children, directing attention outward to the environment; Feldman, 2007). Moreover, the experience of symptoms of depression and anxiety is different for men than it is than it is for women (Angst et al., 2002). For example, women with depression experience more somatic symptoms than men do (e.g., heart palpitations, gastrointestinal distress; Dekker et al., 2008). Women are more likely than men to experience symptoms of anxiety or depression or both, however the gender gap is wider for anxiety than it is for depression (Simonds & Whiffen, 2003). Women also experience anxiety as more debilitating than do men (McLean, Asnaani, Litz, & Hofmann, 2011). Coping with anxiety and depressive symptoms is also different between men and women, with men more frequently turning to alcohol than women, and women more likely to cope through emotional release (Angst et al., 2002). Given the novelty of understanding profiles of mental health and physiological responding in parents, differences between mothers and fathers need to be explored, as well as potential differences of these profiles on children's behavioral adjustment.

## Current Study

The first goal of this study is to examine how symptoms of anxiety and depression and physiological reactivity cluster within individuals in a community sample of parents of young children using latent profile analysis (LPA). LPA allows for the differentiation of subgroups of individuals who differ based on their responses on a variety of indicators. In this case, indicators will include resting RSA, RSA response to stress, and symptoms of anxiety and depression. It is especially important to examine these processes in parents of young children, since they have the potential to either perpetuate symptoms in their children or protect against developing symptoms. As such, the second goal of this study is to examine whether parent profiles of mental health risk predict differences in parent-reported emotional and behavioral adjustment for children one year later. Mother and father models will be run separately to examine the unique mental health profiles that emerge for each parent and examine relationships between these profiles and children's symptoms and emotion regulation ability.

We expect distinct profiles to emerge. However, the analysis remains exploratory in terms of the expected constitution of the distinct profiles. One possibility based on theoretical and empirical work suggesting the possibility of a general distress factor is that three profiles will emerge. First, we may expect to observe a "typical" profile, characterized by low levels of symptoms, high resting RSA, and moderate RSA withdrawal. If RSA reflects a general biomarker of psychopathology (Beauchaine & Thayer, 2015), then we will not observe distinct anxious versus depressive profiles. Instead, we may see a profile emerge which indicates moderate risk, for example, lower levels of anxiety and/or depressive symptoms and differences in RSA regulation to stress. Third, we may observe a high-risk profile for which parents possess

both physiological dysregulation and experience higher levels symptoms of anxiety and/or depression.

A second option is that we may observe four distinct profiles. Specifically, based on prior research findings differentiating the relationship between anxiety and depressive symptoms with different patterns of RSA activation (e.g., Greaves-Lord et al., 2007), we may observe distinct depressive and anxious profiles. The former might be characterized by heightened self-reported depressive symptoms, low RSA, and blunted RSA stress response, and the latter characterized by heightened self-reported anxiety symptoms, low RSA, and excessive RSA response to stress. Additionally, we may still expect to observe both a "well-regulated" profile with low mental health risk, as well as a profile in which individuals possess high risk, with both physiological dysregulation and elevated symptoms of both depression and anxiety, for a total of four profiles.

## Methods

### Participants

One hundred and fifty families with young children were recruited to participate in a longitudinal study of parent-child biobehavioral dynamics and risk for child maltreatment. Ninety-four fathers also participated in the study. Participants were selectively recruited to be lower income and higher-risk for child maltreatment using multiple criteria that captured diverse risk indices. We asked about family income, history of involvement with Child Protective Services (CPS), utilization of government assistance programs such as Women, Infants, and Children (WIC), and major life events in the past year using the Social Readjustment Rating Scale (e.g. loss of job, change of address, changes to the child's school or home schedules; Holmes & Rahe, 1967). Families were recruited from the Fort Collins, CO area by distributing flyers in local preschools, daycares, community events, and WIC centers. Flyers were also distributed to families who qualified by CPS caseworkers. Participants were excluded from the

study if they could not read or write in English, if children had any pervasive developmental delays, or if mother or child had a heart condition that caused irregular heartbeat.

The sample consisted of 53% male children and 47% female children. At the first wave of data collection, mothers reported their race as 81.3% Caucasian, 7.3% Multi-racial, 2.7% African American, 2.7% Native American, 0.6% Asian, 3.3% other, and 3.3% did not wish to respond. Additionally, 16% self-reported their ethnicity as Hispanic or Latina, 80% non-Hispanic or Latina, and 4% did not wish to respond. Mothers reported children's race as 76% Caucasian, %13.7 Multiracial, 2.7% African American, 1.3% Native American, and 6.2% other race. Mothers reported children's ethnicity as 23.6% Hispanic or Latinx, 75% non-Hispanic or Latinx, and 1.4% were unknown or did not wish to respond. Fathers reported their race as 78% Caucasian, 9% Multiracial, 2.2% African American, 2.2% Native American, 1.1% Asian, and 6.7% other race. Fathers reported their ethnicity as 36.2% Hispanic or Latino, 62.5% non-Hispanic or Latino, and 1.3% were unknown or did not wish to respond.

Most parents were married (66.7%), while 12.7% were living together, 11.3% were single, 8.7% were separated or divorced. Average annual household income was between $30,000 to $39,000 (income ranged from less than $5,000 to over $90,000 annually). Mothers' education levels varied, with 1.3% completing junior high school, 5.3% having completed some high school, 7.3% with high school degrees, 26.0% having completed some college, 15.3% completing an associate degree, 29.3% with 4-year college degrees, 14.7% having completed some graduate level education. Fathers' education also varied, with 5.3% finishing some high school, 18% with high school degrees, 30.8% completing some college, 14.9% with an associate degree, 20.2% with a 4-year college degree, and 10.6% having completed some graduate level education.

## Procedure

Families participated in two laboratory sessions. The first session (Time 1) was completed when children were 3 years old (M=3.03 years, Min=2.83 years, Max=3.42 years), and the second session (Time 2) when children were 4 years old (M=4.00 years, Min=3.75 years, Max=4.42 years). Each laboratory session was approximately 2.5 hours in duration. Upon arrival to the laboratory, electrodes and respiratory belts were applied to parents and children to collect physiological data. Next, parents and children participated in a 3-minute-long resting period where they were asked to sit quietly and watch a calming video of dolphins swimming in the ocean. Next, parents and children participated in three dyadic tasks: free play (7 minutes), clean up (4 minutes), and the Parent-Child Challenge Task (10 minutes), described in detail below. Children also participated in several individual tasks that assessed their vocabulary, spatial reasoning, effortful control, and executive functioning. Parents filled out several questionnaires about their psychopathology symptoms, parenting practices, and child behavior. For families where both mothers and fathers participated, sessions were scheduled on different days (when possible) so that children would not become overwhelmed and minimize practice effects. Toys and puzzles for dyadic tasks were counterbalanced across parents so that children encountered novel things each time. For families with only one parent participating, a total of $135 could be earned if all procedures were performed. Families where both the mother and father participated required additional tasks, and thus families could be compensated up to $210 if all procedures were completed. Children were given a small toy at the end of each laboratory session. Attrition was relatively low between the lab sessions for both parents: 83% for mothers and 78% for fathers.

**Parent-Child Challenge Task.** At both laboratory sessions, parents and children participated in the Parent-Child Challenge Task (PCCT; Lunkenheimer et al., 2016), which is a dyadic task designed to be both collaborative and challenging. Parents and children were given a challenging puzzle and asked to complete three designs that should be just above the child's ability level based on age. Parents were asked to guide their children to assemble the puzzle using only their words and were told not to physically assist their children. They were told that the child will be awarded a prize if they are able to complete all three designs. The task consisted of three phases. The "baseline" phase consisted of the first 4 minutes after the experimenter gave instructions, where parents and children attempted to assemble to puzzles. Next, the experimenter entered the room to tell them that they only had two minutes left to complete the puzzles. The "stressor" phase consisted of the following 3 minutes where parents and children continued to assemble the puzzles, now under a time constraint. Finally, the experimenter re-entered the room and told the dyad that they were not given enough time and the child was given a toy as a prize regardless of whether they completed the puzzles. The "repair" phase lasted 3 minutes and consisted of the parent and child playing together with the new toy. The total duration of the PCCT is approximately 10 minutes. However, if the child was able to complete all three puzzles before the allotted time, the task was ended early.

## Measures

**Anxiety and depressive symptoms.** Parents filled out the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) at each time point. The BSI consists of 53 items, measures 9 distinct psychopathological constructs, and provides a general distress measure. Respondents indicate on a scale of 0-4 the severity of each symptom (i.e., the level to which they are affected by it), with 0=not at all and 4=extremely. The current study utilized the depression and anxiety

subscales, which consist of 6 items each. The depressive subscale reflects symptoms such as dysphoric mood, lack of interest in activities, and feelings of hopelessness. The anxiety subscale encompasses symptoms of restlessness, nervousness, tension, and feelings of panic. Each subscale has been shown to be internally consistent (depression $\alpha$=0.85; anxiety $\alpha$=0.81) and reliable across time (depression r=0.84; anxiety r=0.79; Derogatis & Melisaratos, 1983). Factor analysis confirmed that each subscale represents distinct measures of depressive and anxiety symptoms (Hayes, 1997). Parents' symptom scores reflect the total score on each subscale out of a possible score of 24.

**RSA.** Respiration and electrocardiograph (ECG) was recorded from parents and children throughout each laboratory visit. Data was transmitted to a computer through wireless devices worn by participants. Interbeat interval data was processed and cleaned offline by graduate research assistants using Mindware Heart Rate Variability software. ECG data was sectioned in to 30-second segments and RSA magnitude was calculated for each segment as the natural logarithm of the variance of heart period within the frequency related to respiration (0.24-1.04 Hz for children and 0.12-0.40 Hz for adults; Fracasso, Porges, Lamb & Rosenberg, 1994). RSA data was not included for segments that contained greater than 10% noise interference or if the signal was dropped at any point during the segment. Parent resting RSA will be measured as the average RSA across the 3-minute resting period. Parent RSA withdrawal to stress will be measured as the difference in average RSA between the resting period and the 3-minute long stressor phase of the PCCT.

**Child emotion regulation.** Parents reported on children's emotion regulation using the Emotion Regulation Checklist (ERC; Shields & Cicchetti, 1997). The ERC is a 24-item checklist that examines regulation of emotion and affective lability, intensity, valence and the

appropriateness of emotional displays. The ERC consists of two subscales: one examines emotion regulation (i.e., the ability to modulate emotion to facilitate engagement with the environment) and the other examines negativity//lability (i.e., lability of emotions and dysregulated negative emotions). Parents rated children on each item using a 4-point Likert scale where 1=never, 2=sometimes, 3=often, and 4=almost always. Example items include "Can recover from stress" for the emotion regulation scale, and "Is prone to angry outbursts" for the negativity/lability. Chronbach's alpha was 0.81 for the negativity/lability scale and 0.73 for the emotion regulation scale for this sample, indicating adequate internal reliability.

**Child internalizing and externalizing.** Mothers reported symptoms of psychopathology for children using the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). Mothers responded to each of 100 items about their children's behavior on a three-point scale, where 0=not true of my child, 1=somewhat or sometimes true, 2=very true. Internalizing was assessed using the total score on a subscale of items reflecting anxiety, depression, and withdrawal (e.g. 'Too fearful or anxious,' 'Too shy or timid,' 'Unhappy, sad, or depressed'). Externalizing was assessed using the total score on a subscale of items reflecting attention problems and aggressive behavior (e.g., 'Gets into many fights', 'Temper tantrums or hot temper,' 'Can't sit still, restless, or hyperactive). Chronbach's alpha was 0.89 for the internalizing subscale and 0.92 for the externalizing scale for the current sample indicating good internal consistency.

## Data Analytic Plan

Person-centered profiles of parents' mental health risk were examined using latent profile analysis (LPA; Lazarsfeld & Henry, 1968). The concept behind LPA is that individuals can be divided into subgroups based on an unobservable construct, which in this case would be risk for anxiety and or depressive disorders. LPA can reduce large amounts of data to a specific number

of subgroups based on patterns of responses on continuous variables, which in this case consisted of: resting RSA, RSA withdrawal, BSI anxiety, and BSI depression. LPA also allows for the groups to be uncertain, as in this case where it was difficult to predict how many distinct mental health profiles would emerge a priori and what patterns of responses would characterize each profile.

LPA was used to determine the optimal profile solution (i.e., how many distinct mental health profiles emerge and what patterns of RSA and anxiety and depressive symptom levels constitute them) through examining model fit indices of multi-solution models fitted in MPlus and weighting the most parsimonious solution that explained the greatest amount of individual variability. A model was selected once the AIC and BIC reached their smallest levels and began to increase with the addition of profiles, and once the BLRT became non-significant, indicating that the addition of another profile is not necessary. Four indicators were used to determine profiles. Parents' resting RSA and RSA change to stress were measured as continuous variables while parents' anxiety and depressive symptoms were categorical (described in detail below), making this a mixed indicator model. As such, class means for RSA variables were compared against the sample mean and symptoms variables were interpreted based on item-response probabilities. The latter provides information on the likelihood of a response on a categorical indicator given membership in a particular class. Since our symptom variables had three levels, observed probabilities were compared against the "chance" probability of 0.33. As an example, for individuals who belong to a hypothetical "high-risk" mental health profile, we would expect the class-specific mean of resting RSA to be lower than the sample mean, and that individuals will be most likely to report higher levels of anxiety and depressive symptoms.

Next, class membership was used to predict children's internalizing, externalizing, emotion regulation, and negativity/lability using the BCH method (Bakk & Vermunt, 2016). The BCH method is an alternative to the traditional classify-analyze approach where posterior probabilities are used to assign individuals to a class, and then treat class membership as a known variable in other models. This traditional approach flattens the variability associated with posterior probabilities across individuals, attenuating effects and increasing the chance of type II errors. Conversely, the BCH method applies weights to class membership values which helps to account for individual differences in posterior probabilities within a given class (Bakk & Vermunt, 2016).

## Results

### Preliminary Analyses

There was fairly minimal missing data for mothers at T2. 24 mothers did not participate, another 21 had no RSA data, and 3 had no BSI data. Fathers had more missingness because fewer fathers participated at T2, with a total of 63 not participating. In addition, 5 had no RSA data, and 5 had no BSI data. At T3, 32 were missing for maternal report of child internalizing and externalizing on the CBCL, and 33 missing for child emotion regulation and negativity/lability on the ERC. Analyses were run in MPlus version 8.1 using maximum likelihood estimation, so individuals were not removed from the analysis unless they were missing on all variables (i.e., did not participate), making the valid N=126 for the mother model and N=87 for the father model.

Means, standard deviations, and maximum and minimum values are displayed in Table 1.1. Symptoms of anxiety and depression were skewed for both mothers (anxiety subscale: skewness =1.06, SE =0.22; depressive subscale: skewness =2.35, SE =0.22) and fathers (anxiety

subscale: skewness =2.11, SE =0.27; depressive subscale: skewness =2.41, SE =0.27), so these variables were transformed into categorical values with three levels: no symptoms, lower symptoms, and higher symptoms. The "no symptoms" group consisted of individuals who did not report any symptoms of either anxiety and/or depression. The "lower symptoms" and "higher symptoms" groups were differentiated using sample-based percentile variables. Cut-off values were determined such that the top 25% of the sample would be classified as reporting "higher symptoms" and individuals with values greater than zero but below that cut-off were classified as reporting "lower symptoms." For mothers, cut-off values for both anxiety and depression were the same and equivalent to endorsing 3 of the 6 symptoms on the subscale. Using this criterion, the sample of mothers was split according to depressive symptoms such that 46% were classified as "no symptoms," 28% were classified as "lower symptoms," and 26% were classified as "higher symptoms." For mothers' anxiety, the sample was split such that 36% were classified as "no symptoms," 31% were classified as "lower symptoms," and 32% were classified as "higher symptoms." For fathers, cut-off values were also the same for both anxiety and depression and were equivalent to endorsing 2 of the 6 symptoms on the subscale. For depressive symptoms, the sample of fathers was split such that 55% were classified as "no symptoms," 21% were classified as "lower symptoms," and 24% individuals classified as "higher symptoms." For anxiety symptoms, the sample of fathers was split such that 46% were classified as "no symptoms," 21% were classified as "lower symptoms," and 22% were classified as "higher symptoms."

For mothers, resting RSA ranged from 1.91 to 9.75, with a mean of 6.10; for fathers, resting RSA ranged from 1.68 to 9.29, with a mean of 6.13. Mothers' mean RSA change score was -0.13, indicating RSA withdrawal to stress on average. Values ranged from -2.14 to 1.57,

indicating that some individuals withdrew RSA from baseline to challenge, while others did not have profound RSA change, and still others who augmented RSA. Similarly, fathers had a mean RSA change score of 0.02, indicating slight augmentation on average. Values ranged from -0.95 to 1.76, indicating that some individuals withdrew RSA from baseline to challenge, while others did not have profound RSA change, and still others who augmented RSA. Resting RSA was not correlated with the change in RSA between the baseline and challenge conditions of the PCCT for either mothers ($r$=.04, $p$=.71) or fathers ($r$=-.17, $p$=.14). Values for resting RSA and RSA change were standardized before entering into the latent class models so that class means could be easily compared against the sample mean of zero.

Bivariate correlation analyses indicated that mothers' change in RSA from baseline to challenge was associated with mothers' anxiety score, such that greater RSA withdrawal (i.e., more negative RSA change) was associated with greater symptoms of anxiety for mothers ($r$=-.27, $p$<.01). Mothers' and fathers' levels of resting RSA were positively correlated ($r$=.26, $p$<.05) suggesting that higher RSA in mothers was associated with higher RSA in fathers, too. There were significant correlations between mothers' depressive symptoms at T2 and child outcomes at T3 such that higher depressive symptoms in mothers was associated with greater internalizing ($r$=.19, $p$<.05), externalizing ($r$=.29, $p$<.01), and negativity/lability ($r$=.25, $p$=.01) when children were 4 years old. Mothers' depressive symptoms were also positively correlated with children's concurrent externalizing at T2 ($r$=.23, $p$=.01) such that greater depressive symptoms were associated with greater externalizing when children were 3 years old. Fathers' anxiety symptoms were related to children's emotion regulation at T3 such that higher anxiety symptoms were associated with lower emotion regulation ability in children at 4 years old ($r$=-.23, $p$<.05). Fathers' change in RSA from baseline to challenge was positively correlated with

children's externalizing score at both T2 and T3 (both $r$=.24, $p$<.05), indicating that greater augmentation of RSA in fathers was associated with high child externalizing at age 3 and age 4. Mothers' change in RSA from baseline to challenge was negatively related to children's emotion regulation at T2 ($r$=-.24, $p$=.01) such that greater RSA withdrawal was associated with higher emotion regulation ability in children.

**Primary Analyses**

**Model selection.** LPAs with 2 to 5 classes were run and fit indices were compared to determine the optimal number of classes for both mothers and fathers. For mothers, a 4-class solution was chosen (see Table 1.2). The BIC and $G^2$ decreased between the 2- and 3-class models and again between the 3- and 4-class models but began to increase between the 4- and 5-class models. Thus, indices were lowest for the 4-class model indicating best model fit. In addition, the BLRT was significant for the 2- and 3-class models but was not significant for the 4-class model, indicating that the addition of a fifth class was not necessary. The AIC and SSBIC continued to decrease indefinitely, which may be expected when continuous indicators are used (Collins & Lanza, 2010).

For fathers, a 3-class solution was chosen (see Table 1.3), however, indices did not provide such a cut-and-dry solution as was found with mothers. The BLRT was significant for the 2-class model but was not significant for the 3-class model, indicating that a fourth class may not be necessary. However, the AIC, BIC, and SSBIC continued to *increase* indefinitely, which may indicate that model fit did not improve from the 2- to 3-class model or with the continued addition of classes. Furthermore, the $G^2$ continued to increase between the 2- and 4-class solutions but decreased between the 4- and 5-class solutions, which would indicate that model fit improved between 4- and 5-class models.

Based on these results, a 3-class model was chosen for fathers for a few reasons. First, the 5-class model included two classes that only consisted of 2 individuals, which poses problems with further analyses and questions the validity of the results. Second, prior work suggests that in cases where continuous indicators are used, there are limitations to the interpretability of fit indices such as the AIC, BIC, SSBIC, and $G^2$ (Lanza & Collins, 2010), however, the BLRT is more robust to these limitations. Finally, the 3-class solution was hypothesized based on prior research on this topic and therefore fits in with extant findings with mental health symptoms and RSA in adults. It has been suggested that in cases where the indices are unclear, choosing the solution that is most interpretable and expected based on prior work is acceptable (Bray, Foti, Thompson, & Wills, 2014).

**Mother profiles.** The means and item-response probabilities for the mother model are displayed in Table 1.4. The first class, labeled "Typical", encompassed 62.7% of the sample. This class of individuals was given the label "typical" because they were the largest group, had the greatest likelihood of having neither depressive or anxiety symptoms (versus lower or higher symptoms), and had resting RSA and RSA change very close to the standardized group means of zero, which indicated slight withdrawal on average.

The second class, labeled "Moderate Risk/Sensitive", encompassed 9.5% of the sample and were the smallest class. These individuals had the greatest likelihood of showing lower levels of depressive and anxiety symptoms (versus higher symptoms or no symptoms), conveying some risk for mental health problems. These individuals also had resting RSA that was approximately 1½ SD higher than the sample mean and RSA change about equal to the sample mean. They were labeled "Sensitive" because of the higher RSA values which could

reflect greater sensitivity to stimuli including challenges or stressors (Berntson, Cacioppo, & Quigley, 1993; Porges, 2007).

The third class, labeled "High Risk/Anxious," encompassed 12.7% of the sample. These individuals had a high probability of reporting higher levels of anxiety symptoms and also a smaller but significant probability of reporting higher levels of depressive symptoms (versus no symptoms or lower symptoms). In addition, these individuals showed resting RSA about equal to the sample mean and RSA change that was around 1½ SDs lower than the mean, indicating RSA withdrawal to stress that was more pronounced than any of the other 3 classes.

Finally, the fourth class, labeled "High Risk/Depressive", encompassed 15.1% of the sample. This class was characterized by a high probability of reporting higher levels of depressive symptoms and a tendency toward reporting higher anxiety symptoms that was greater than the chance level of 0.33. This class of individuals also showed average levels of resting RSA and RSA change that was approximately ¾ SD above the sample mean, reflecting augmentation of RSA during stress. Classes 3 and 4 were labeled "High Risk/Anxious" and "High Risk/Depressive", respectively, because of the probabilities characterizing greater symptoms of one versus the other as well as differences in RSA change that have been shown in previous work to differentiate biological risk for anxiety (i.e., excessive withdrawal) versus depression (i.e., augmentation; Kircanski et al., 2016).

***Relations between mother profile membership and child outcomes.*** Figure 1.1 displays means of child internalizing, externalizing, negativity/lability, and emotion regulation at age four as a function of mothers' class membership one year prior. Results indicated that mothers' class membership predicted differences in children's internalizing (Overall $G^2$=14.50, $p$=.002), externalizing (Overall $G^2$=12.68, $p$=.005), and negativity/lability (Overall $G^2$=8.15, $p$=.04).

Patterns were such that children of mothers who belonged to one of the three moderate or high risk classes at T2 had higher levels of emotional and behavioral problems at T3 than children of mothers in the "Typical" class. With regard to child internalizing, significant differences were observed between the "Typical" class and the "Moderate Risk/Sensitive" ($G^2$=8.31, $p$=.004), "High Risk/Anxious" ($G^2$=4.42, $p$=.04), and "High Risk/Depressive" classes ($G^2$=6.91, $p$=.009) such that internalizing problems were higher when mothers belonged to one of these three higher-risk classes versus the low-risk "Typical" class With regard to externalizing, significant differences were observed between the "Typical" class and the "Moderate Risk/Sensitive" ($G^2$=6.98, $p$=.008) and the "High Risk/Depressive" classes ($G^2$=5.84, $p$=.02), but not the "High Risk/Anxious" class ($G^2$=1.67, $p$=.20) such that children displayed greater externalizing symptoms when mothers belonged to either the "Moderate Risk/Sensitive" or "High Risk/Depressive" class versus belonging to the "Typical" class. With regard to negativity/lability, only the difference between the "Typical" class and the "Moderate Risk/Sensitive" class was significant ($G^2$=6.52, $p$=.01) such that mothers' membership in the "Moderate Risk/Sensitive" class predicted greater negativity/lability for children when they were 4 years old. No significant differences were detected in means across classes for child emotion regulation (Overall $G^2$=0.44, $p$=.80). In addition, no significant differences were observed among any of the three moderate to higher risk classes.

**Father profiles.** The item response probabilities for the father model are displayed in Table 1.5. As with mothers, the first class of fathers was the largest, encompassing 47.1% of the sample. This class was labeled "Typical" because of a high likelihood of reporting neither anxiety nor depressive symptoms. These individuals also showed slightly lower-than-average

resting RSA, approximately ¼ SD below the sample mean, and RSA change very close to the sample mean.

The second class was labeled "Moderate Risk/Anxious" and encompassed 36.8% of the sample. These individuals did not appear to be differentiated on depressive symptoms, displaying equal probabilities across the three levels, however, they showed a higher likelihood of reporting lower levels of anxiety symptoms (versus no symptoms or higher symptoms). In addition, this group had the highest levels of resting RSA, approximately ½ SD above the sample mean, and negative RSA change approximately ¼ SD below the sample mean, indicating RSA withdrawal to stress.

Finally, the third class was labeled "High Risk" and encompassed 16.1% of the sample, making it the smallest of the three classes. These individuals had a very high probability of reporting higher symptoms of both depression and anxiety (0.97 and 1, respectively). In addition, these individuals were differentiated from the other two classes by showing higher-than-average RSA change, approximately ¾ SD above the mean, indicating a tendency toward RSA augmentation to stress. Resting RSA was approximately ¼ SD below the group mean, however, it did not differ from resting RSA for the "Typical" class.

**Relations between father profile membership and child outcomes.** Figure 1.2 displays the means of child internalizing, externalizing, negativity/lability, and emotion regulation at age four as a function of fathers' class membership one year prior. Trends were such that children of fathers in the "High Risk" class at T2 had higher levels of behavioral and emotional symptoms and lower levels of emotion regulation at T3 versus children of fathers in either the "Typical" or "Moderate Risk/Anxious" classes. However, none of the overall models reached significance, although the model for externalizing was marginally significant (Overall

$G^2$=5.80, *p*=.055). With regard to internalizing, there was a marginally significant difference between the "Moderate Risk/Anxious" class and the "High Risk" class ($G^2$=3.82, *p*=.051), such that children's internalizing problems were greater when fathers belonged to the "High Risk" class versus the "Moderate Risk/Anxious" class (however, not when compared to the "Typical" class). With regard to externalizing, there was a significant difference between the "Typical" class and the "High Risk" class ($G^2$=5.74, *p*=.02), and a marginally significant difference between the "Moderate Risk/Anxious" class and the "High Risk" class ($G^2$=3.62, *p*=.057), such that children had greater externalizing when fathers belonged to the "High Risk" class versus the "Typical" class and the "Moderate Risk/Anxious" class (marginally). No differences between classes were observed for child negativity/lability. Finally, child emotion regulation was marginally lower for the "High Risk" class compared with the "Typical" class ($G^2$=3.59, *p*=.058), such that children had lower emotion regulation ability when their fathers belonged to the "High Risk" class versus the "Typical" class.

## Discussion

This study is the first study to examine how symptoms of depression and anxiety and parasympathetic processes cluster within parents using a person-centered approach, and whether belonging to a low, moderate, or high-risk profile predicts children's emotional and behavioral adjustment. Findings suggest that there are distinct higher-risk profiles of parents that differ from the typical profile by nature of elevated anxiety and/or depression symptoms and differences in resting levels of RSA and change in RSA to stress. Distinct profile solutions were obtained for mothers versus fathers, however, they both had a typical, low-risk profile along with profiles of moderate and high risk. Children's emotional and behavioral problems were worse when either mothers or fathers belonged to one of the higher-risk profiles versus the typical class, however,

results were more robust for mothers. Overall, this suggests mental health risk may be signified by clusters of both behavioral and physiological factors. Using a person-centered approach to analyze the relationship between parents' mental health risk and children's behavioral and emotional adjustment may help fine tune the process of intervening with parents to prevent mental health problems in children.

**Mothers' Risk Classes**

**Moderate Risk/Sensitive.** Mothers showed three distinct risk classes in total, encompassing 37.3% of the sample. The "Moderate Risk/Sensitive" class was the most surprising based on prior research. We hypothesized a moderate risk class but did not expect higher RSA to be associated with anxiety or depressive symptoms, even only mild symptoms as was observed in this study. Higher resting RSA is typically thought to be adaptive, however, it may be that there is a threshold beyond which values convey biological risk for anxiety and depression. Resting RSA is thought to reflect an individual's readiness to respond to stimuli, including challenges or stressful situations (Porges, 2007). Thus, those with very high resting RSA may be characterized by overreactivity due to greater sensitivity to environmental challenges. We did not observe any differences in RSA change to stress among this group that would signal overreactivity, however, it is possible that while the parasympathetic nervous system did not show a strong response, activity of the sympathetic nervous system or the hypothalamic-pituitary axis reflected overreactivity, as activation of these systems is not always linked (Berntson, Caccioppo, & Quigley, 1993).

Sensitivity may serve adaptive purposes for parents in some contexts, but not others, a concept known as 'differential susceptibility' (Belsky & Pluess, 2009). Young children have been characterized as belonging to one of two subsets: orchids or dandelions. 'Dandelions' are

not as sensitive to their circumstances, for example, their academic performance does not hinge strongly on classroom context (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2011). However, for those more sensitive individuals (i.e., 'orchids'), they will thrive in environments which are nurturing but will struggle when the context is not supportive (Belsky & Pluess, 2009). This pattern has been observed in studies of infants' RSA, for instance, one study found that infants with higher resting RSA showed greater negative reactivity, but also greater positive affect and emotional lability (Stifter & Fox, 1990). In adults, some research has suggested that lower RSA may be adaptive for those who are suffering with major depressive disorder, predicting lower instances of depressive episodes compared to higher RSA (Rottenberg et al., 2002). This similarly could be explained by nature of sensitivity: lower RSA may reflect lower sensitivity to negative stressors, protecting from triggering another depressive episode.

Higher average RSA is typically associated with greater flexibility and adaptiveness to the environment (Porges, 2007), as well as with lower anxiety and depressive symptoms in adults. However, sensitivity to the environment, while adaptive in some situations (e.g., attending to children's needs), may also make individuals more susceptible to the impacts of chronic stress, as may be the case with mothers in the Moderate Risk/Sensitive Class (Belsky & Pluess, 2009). Supporting this idea, Lunkenheimer and colleagues (2019) found that maltreating parents have higher RSA when interactions are more child-initiated, reflecting a lack of engagement on behalf of parents. Mothers in the Moderate Risk/Sensitive group also had children who displayed greater internalizing and externalizing symptoms and negativity/lability at a one-year follow up. Our findings suggest directional effects such that mothers' risk class predicts children's emotional and behavioral adjustment, but it could also be that parenting a

child with such problems poses a challenge to sensitive mothers, raising their anxiety and depressive symptoms.

**High Risk/Anxious and High Risk/Depressive.** Two additional risk classes were observed for mothers, both of which displayed elevated depression and anxiety symptoms, but were differentiated by patterns of RSA change to stress, and whether anxiety versus depressive symptoms were more likely. The "High Risk/Anxious" class showed a mean RSA change that was lower than the sample mean and much lower than the other three classes, which could reflect RSA withdrawal that is more pronounced than expected. This is in line with prior research has shown that individuals with either clinical anxiety or subclinical symptoms may show "excessive" RSA withdrawal to stress, which could be associated with an overactive fight-flight response (e.g., Beauchaine, 2001; 2015). The "High Risk/Depressive" class showed a mean RSA change that was above the sample mean, indicating that RSA augmentation was the norm among this class. Again, this is in line with prior work showing a link between depressive symptoms and either no RSA change to stress or augmented RSA (Rottenberg et al., 2007). Taken together, these results support the notion that RSA change may be differentially related to greater anxiety and depressive symptoms respectively, with anxiety symptoms more strongly linked with "excessive" RSA withdrawal and depressive symptoms linked with RSA augmentation (Kircanski et al., 2016).

Our pattern of results lends support to the Tripartite Model of anxiety and depression (Clark & Watson, 1991) where the two share a general distress factor but are differentiated based on other criteria. We did not observe a clean divide in profiles such that either exclusively depressive versus anxiety symptoms were present. This may be expected using the BSI as a measurement instrument, which has a strong general distress component (Derogatis &

Melisaratos, 1983). However, we observed differences in autonomic activation reflecting some ideas of Clark & Watson (1991). Individuals with anxiety are expected to show signs of autonomic hyperarousal, characterized by physical symptoms such as heart palpitations and muscle tension. We found hyperreactivity in the form of an excessive RSA withdrawal, which has been linked with increased activation of the SNS and may be responsible for such symptoms. For individuals with depression, parasympathetic response to stress was blunted, an effect which has been observed with individuals with depression across physiological and neuroendocrine systems (Grisson & Bhatnagar, 2009). Hyporeactivity may be observed when an individual has been faced with chronic stress; it is a form of self-protection from the damaging effects of continued autonomic response (McEwen, 2000). Symptoms of depression, such as anhedonia, hopelessness, and worthlessness, may reflect hyporeactivity of the body's normative response to stress or challenges (i.e., failing to mobilize resources or coping strategies).

**Mother versus Father Profiles**

This study also tested whether differences in mothers' and fathers' mental health and parasympathetic activity translated into different profile solutions of mental health risk for mothers versus fathers. We found that in some ways, the profiles mapped well across groups. For example, both groups showed a typical class who did not report symptoms of depression or anxiety and showed well-regulated RSA from resting to stress. Fathers also had a "Moderate Risk" class and "High Risk" class which mirrored mothers' profiles in that the moderate risk group showed slight elevation of anxiety symptoms and resting RSA that was above the sample mean, and individuals in the High Risk group reported higher levels of both anxiety and depressive symptoms (see Figure 1.3). One key difference was that with fathers, we did not find differentiation between anxious and depressive higher risk profiles. The High Risk profile of

fathers expressed elevated symptoms of both anxiety and depression, but with no distinction in probabilities of one or another, and RSA augmentation to stress rather than withdrawal. This may be explained by differences in the way that men and women experience mental health symptoms (McClean et al., 2011). Men more typically display fewer somatic symptoms than women, which include increased heart palpitations (Dekker et al., 2008). This may also mean that fathers are less prone to physiological hyperreactivity as are mothers, and therefore parenting deficits among fathers may tend to fall into a disengaged, unresponsive category than an intrusive and hypervigilant category.

**Effects of Profile Membership on Child Emotional and Behavioral Adjustment**

Regarding child emotional and behavioral adjustment, parents' belonging to a higher-risk class predicted greater problems for children in some cases, but not others. The highest levels of child internalizing, child externalizing, and child negativity/lability were found for children whose mothers belonged to the Moderate Risk/Sensitive class. One explanation is that for parent-child relationships with preschool-aged children, level of resting RSA is more of a risk factor than elevated symptoms, at least for mothers. Levels of resting RSA measured in the lab may reflect a chronic elevation in RSA, and therefore greater sensitivity during parent-child interactions. Some evidence has been found supporting the relation between average RSA measured in the lab and chronically low RSA (Lunkenheimer et al., 2019). While sensitivity is characteristic of positive parenting, it may come at an expense to supporting children's autonomy, which becomes more important as children's needs change over the period of early childhood (Cicchetti & Schneider-Rosen, 1986).

For fathers, no differences were observed in emotional or behavioral problems between children of fathers in the Moderate Risk/Anxious class and the Typical class. Sensitivity may not

be as great of a risk factor for father-child interactions, and moderate levels of symptoms may not affect day-to-day relations between preschool-aged children and their fathers. In our sample, mothers predominantly reported being the child's primary caregiver, which means that fathers' time with their child was more limited. When instances of interaction are fewer, sensitive responsiveness may serve more as a protective factor for children than a hindrance to their independence. By contrast, fathers who belonged to the High Risk class had children who showed elevated externalizing symptoms and showed trends towards higher internalizing and lower emotion regulation ability. Interestingly, levels of children's behavioral and emotional problems were almost identical when fathers belonged to the High Risk class and when mothers belonged to the High Risk/Depressive class. In both cases, symptoms were elevated, and RSA augmentation was observed, suggesting hyporeactivity and in turn, lack of interest and disengagement (Rottenberg, 2007).

Mothers' membership in either the High Risk/Anxious or High Risk/Depressive groups were negatively associated with children's emotional and behavioral adjustment one year later. This is consistent with prior work associating mothers' depressive and anxiety symptoms with children's symptoms of internalizing and externalizing (Graziano & Derefinko, 2013). Differences in RSA patterns between the groups may also translate into different pathways from group membership to child symptoms (i.e., different means to the same ends). The overreactivity associated with the anxious group may translate to hypervigilance and intrusion during parent-child interactions (Beebe et al., 2011), whereas the blunted reactivity of the depressive group may lead to disengagement (Tronick, 1989). While both have detrimental outcomes on children's well-being, it is important to distinguish between the types of parents in order to know how to best direct intervention efforts for parents that can translate into preventative measures

for children's well-being. In this case, while symptoms of anxiety and depression were present in both groups, parasympathetic activation to stress was quite different, casting new light on understanding the defining features of mental health risk for parent and child.

**Strengths and Limitations**

This study has many strengths, including the use of person-centered methodology to disentangle relations between parasympathetic activity and mental health symptoms as well as differences between men and women. Using LPA, we were able to determine that unique groups of individuals with mental health symptoms are present, each with different ways of responding to the environment. For parents, this may affect the ways in which they interact with their children. It is important to know whether heterogeneity exists within parents who are at risk for mental health problems, and subsequently impacting their children's emotional well-being. Variable-centered models tend to be the norm in this field of research, which provide useful information about the individual contributions of many different risk factors that can be assessed from parents and children. Person-centered approaches offer a more nuanced account of the ways in which such factors organize together to form profiles of risk. To our knowledge, this is the first study to examine how symptoms of anxiety and depression and activation of RSA cluster within parents and in turn predict different outcomes for young children.

Limitations of the study include the use of a community sample in which a low incidence of clinical anxiety and depressive disorders is the norm. We also utilized a measurement tool, the BSI, that is more commonly used to measure general distress rather than differentiate between anxiety and depression (although the subscales have been shown to be meaningful, XX). In addition, the BSI does not provide a metric for assessing clinical cases, therefore, it is unknown what percentage of the given sample may meet criteria for a mental health disorder. Another

limitation of the current study was the use of self-report questionnaires to assess anxiety and depressive symptoms and mothers' report to assess behavioral and emotional adjustment in children. In the mother models, this might have led to an inflation of relations between mothers' symptoms and children's outcomes, however, this likely did not affect the father models.

## Conclusions

Relations between symptoms of anxiety and depression and underlying physiological regulation are likely complex. The current study attempted to disentangle these relations using a person-centered approach to understand how indices of mental health risk cluster within parents of young children. Our results suggest that relations between parents' mental health risk and children's emotional and behavioral adjustment are also complicated, and do not necessarily reflect a one-to-one relation between higher symptoms for parents and worse outcomes for children. Results of this study encourage continued research and information gathering to form more holistic profiles of risk, which are more likely to map on to real processes, potentially improving the fit of intervention efforts catered towards unique groups of individuals.

## Tables and Figures

**Table 1.1** Means, standard deviations, minimum, and maximum values for each of the key study variables.

| | **Mean** | **SD** | **Min** | **Max** |
|---|---|---|---|---|
| **Mothers' Anxiety Symptoms** | 1.74 | 1.92 | 0 | 7.98 |
| **Fathers' Anxiety Symptoms** | 1.60 | 2.33 | 0 | 12 |
| **Mothers' Depressive Symptoms** | 1.80 | 2.58 | 0 | 16.02 |
| **Fathers' Depressive Symptoms** | 1.66 | 2.89 | 0 | 13.98 |
| **Mothers' Resting RSA** | 6.10 | 1.32 | 1.91 | 9.75 |
| **Fathers' Resting RSA** | 6.13 | 1.33 | 1.68 | 9.29 |
| **Children's Internalizing** | 7.12 | 5.78 | 0 | 32 |
| **Child Externalizing** | 11.81 | 7.77 | 0 | 36 |
| **Child Negativity/Lability** | 26.56 | 5.71 | 17 | 43 |
| **Child Emotion Regulation** | 27.44 | 3.22 | 16 | 32 |

Table 1.2 Model fit statistics for mother LPA.

| Classes | AIC | BIC | SSBIC | BLRT | $G^2$ |
|---|---|---|---|---|---|
| **2** | 1107.5 | 1150.04 | 1102.61 | 0 | 8.33 |
| **3** | 1103.26 | 1165.66 | 1096.08 | 0 | 2.85 |
| **4** | 1101.35 | 1183.60 | 1091.89 | 0.2 | 0.40 |
| **5** | 1098.38 | 1200.48 | 1086.64 | 0.1 | 0.43 |

Table 1.3 Model fit statistics for father LPA.

| Classes | AIC | BIC | SSBIC | BLRT | $G^2$ |
|---|---|---|---|---|---|
| **2** | 748.871 | 785.860 | 1102.61 | 0 | 0.908 |
| **3** | 749.736 | 803.986 | 734.569 | 0.2 | 0.110 |
| **4** | 755.595 | 827.106 | 735.602 | 0.667 | 0.015 |
| **5** | 757.824 | 846.597 | 733.005 | 0.0 | 0.157 |

Table 1.4 Item-response probabilities for mother LPA.

| | Class 1 "Typical" (62.7%) | Class 2 "Moderate Risk Sensitive" (9.5%) | Class 3 "Higher Risk Anxious" (12.7%) | Class 4 "Higher Risk Depressive" (15.1%) |
|---|---|---|---|---|
| Depressive Symptoms | | | | |
| No symptoms | **0.74** | 0 | 0 | 0 |
| Lower Severity | 0.23 | **0.84** | 0.32 | 0.20 |
| Higher Severity | 0.04 | 0.16 | **0.68** | **0.80** |
| Anxiety Symptoms | | | | |
| No symptoms | **0.59** | 0 | 0.09 | 0 |
| Lower Severity | 0.29 | **1** | 0 | 0.33 |
| Higher Severity | 0.15 | 0 | **0.91** | **0.67** |
| Resting RSA | | | | |
| Class Mean | -0.10 | **1.56** | -0.16 | -0.24 |
| RSA Change | | | | |
| Class Mean | 0.13 | -0.20 | **-1.37** | **0.73** |

Table 1.5 Item-response probabilities for father LPA.

| | "Typical" (47.1%) | "Moderate Risk Anxious" (36.8%) | "High Risk" (16.1%) |
|---|---|---|---|
| Depressive Symptoms | | | |
| No symptoms | **0.79** | 0.47 | 0.00 |
| Lower Severity | 0.21 | 0.29 | 0.03 |
| Higher Severity | 0.00 | 0.24 | **0.97** |
| Anxiety Symptoms | | | |
| No symptoms | **0.97** | 0.00 | 0.00 |
| Lower Severity | 0.03 | **0.84** | 0.00 |
| Higher Severity | 0.00 | 0.16 | **1.00** |
| Resting RSA | | | |
| Mean | -0.26 | **0.45** | -0.27 |
| RSA Withdrawal | | | |
| Mean | -0.04 | -0.22 | **0.74** |

Figure 1.1 Children's internalizing, externalizing, emotion regulation, and negativity/lability at age 4 as a function of mother's class membership one year prior. Y-axis reflects standardized scores.

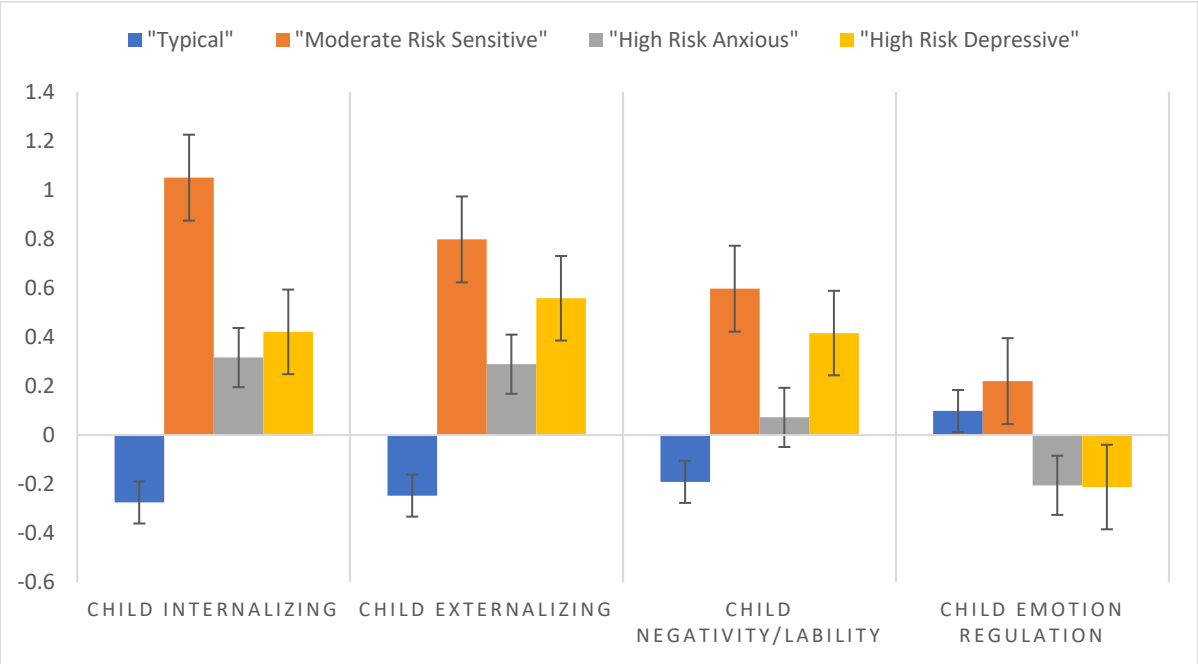


Figure 1.2 Children's internalizing, externalizing, emotion regulation, and negativity/lability at age 4 as a function of father's class membership one year prior. Y-axis reflects standardized scores.

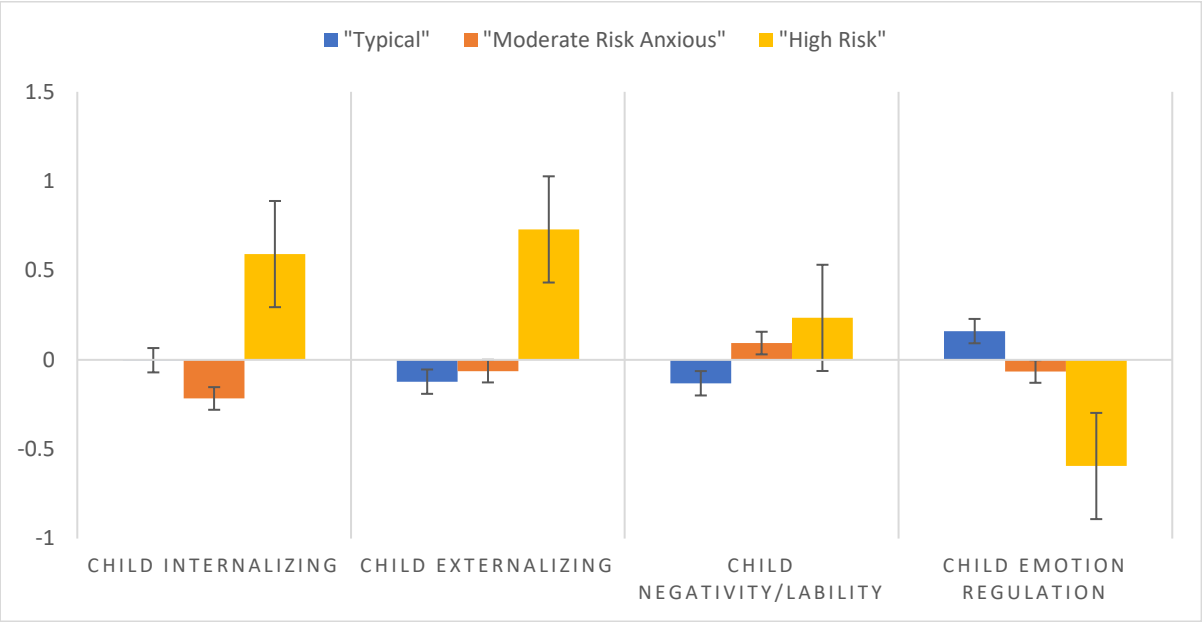
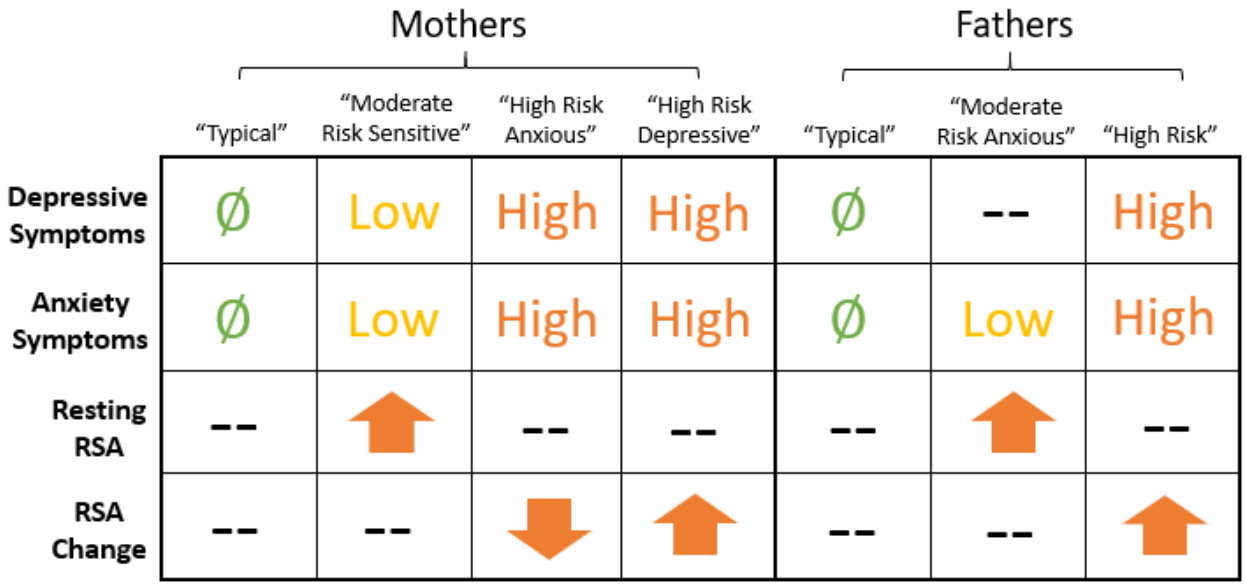


Figure 1.3 Visual comparison of mental health and stress physiology profiles of mothers versus fathers.



## STUDY II: EXAMINING THE IMPACT OF MOTHERS' AND FATHERS' MENTAL HEALTH PROFILES ON PARENT-CHILD PHYSIOLOGICAL COREGULATION

The ability to manage one's emotions through the down-regulation of psychophysiological processes is crucial for developing effective coping mechanisms with which to handle life's challenges. Developing these emotion regulation skills is an important task for children during the phase of early childhood (Calkins, 2011). Throughout infancy, children rely heavily on interactions with their parents for external regulation of emotion and physiological arousal (Calkins, 2011; Feldman, 2012). This can be facilitated through the coordination of emotional and physiological states between parents and children in real time, a process termed *coregulation* (Olsen & Lunkenheimer, 2009). Through coregulatory processes, parents and children match their physiological and emotional states, providing support for children's developing regulatory systems, a process known as *attunement* (Tronick, 1989). Through this matching, parents serve as unconscious guides for children's organization of their own regulatory processes (Feldman, 2012). Greater attunement has been found to occur more often when parents and children are more engaged in the interaction, and when parents are more sensitive to the needs of their child and are able to coordinate their behaviors with their children.

Research has shown that coregulation at the physiological level occurs in general for typical parents and their preschool-aged children (Davis et al., 2018), and poor coregulation is associated with parent and child mental health risk factors (Lunkenheimer et al., 2018). Specifically, attunement in physiological systems is adaptive for typical parents and children, however, may be less so for dyads who are at higher risk by nature of elevated psychopathology symptoms and/or dysregulated stress physiology in parents (Suveg et al., 2016). In some of these cases, it appears that attunement is disrupted such that parent-child physiological states become

negatively coordinated (Lunkenheimer et al., 2018), interrupting this crucial process and reducing the opportunity for children's developing physiological systems to align with the better-regulated systems of their parents. In other cases, the dyad may remain attuned, but coordination with parents' dysregulated states may convey additional risk for children instead of offering support (Suveg et al., 2016). Thus, the benefits of attunement may be disrupted in one of two ways when parents experience mental health symptoms: 1) they may lack the sensitivity to establish and maintain attunement with their children, and 2) they may convey their maladaptive regulatory patterns to their children through the process of attunement.

In the event that parent-child states become chronically miscoordinated, detriments in ER may arise in children, which can become more severe over time and possibly translate to symptoms of anxiety and depression later on in development (Beauchaine 2001; 2015). Despite the importance of the crucial process of coregulation, the nuances of the relations between parent psychopathology and parent-child coregulation are not well understood. However, parenting research suggests that there may be different detrimental ways that parents with anxiety symptoms interact with their children versus parents with depression (Tronick, 1989; Beebe & Lachman, 1998), including differences in parent-child coregulation (Tronick, 1989). Similarly, coregulation may appear different among parent-child dyads where parents show dysregulated physiology and may also serve different (potentially maladaptive) functions (Gray et al., 2017; Smith et al., 2016). These dynamic processes are important but complex, and therefore require different methodological considerations than have been employed in research in this field to date. Questions remain as to how each of these components (i.e., depressive symptoms, anxiety symptoms, regulation of stress physiological) independently and in combination influences coregulation processes. The current study aims to address these gaps by using person-centered

methodology to capture the organization of mental health risk in parents, accounting for both expressed symptoms of anxiety and depression as well as physiological regulation. In turn, person-centered profiles will be used to determine whether different patterns of parent-child coregulation are associated with different forms of mental health risk.

**Emotion Regulation**

Emotion regulation (ER) refers to the ability to the ability to monitor, evaluate, and modify emotional responses in accordance with one's goals (Thompson, 1994). Developing ER skill is thought to be a crucial developmental task for children in early childhood (Diamond & Aspinwall, 2003). ER ability is associated with social and emotional adjustment in children and adolescents and can have implications for individuals throughout the lifespan. In adults, difficulty with ER may make individuals vulnerable to developing a mental health problem such as anxiety or depression (Beauchaine, 2015).

While ER involves behavioral strategies, it is also thought that ER has a physiological basis in the form of activation of the parasympathetic nervous system (PNS). Activity of the PNS can be measured by assessing respiratory sinus arrythmia (RSA), which is the variability in heart rate that can be attributed to breathing (Porges, 1991). RSA is determined by activation of the PNS, so RSA should increase during times of rest or relaxation and decrease during times of activity, stress, or challenge (Porges, 1991; 2007). The withdrawal of RSA is thought to facilitate the activation of the sympathetic nervous system and the hypothalamic-pituitary axis which prepare the individuals to deal with stress or challenge (Bernston et al., 1993). RSA is also thought to play a role in supporting social communication by helping to regulate emotional expressions during interpersonal interactions (Porges, 2003). Theories have proposed that RSA is

part of a neuro-visceral network that also includes the expression of emotion and cognitive control, both of which are associated with ER (Porges, 2007; Thayer & Lane, 2000).

RSA appears to provide support for ER as infancy, continuing into early childhood (Beauchaine, 2001). By adulthood, RSA is related to the presence of mental health symptoms. Lower levels of resting or average RSA have been shown to relate to symptoms of both anxiety (Watkins et al., 1995) and depression (Rottenberg et al., 2007) among both patient samples and community samples with sub-clinical levels of symptoms. Similarly, differences in the RSA response to stress have also been observed between individuals with depressive and/or anxiety and controls (). However, individuals with anxiety versus depressive symptoms may show different patterns of RSA withdrawal during stress. While depressive symptoms have been linked with either no change or increasing RSA to stress (i.e., hyporeactivity; Rottenberg et al., 2007), anxiety symptoms have been linked with greater withdrawal of RSA to stress (i.e., hyperreactivity; Kircanski et al., 2016).

**Parent-Child Coregulation**

Across the developmental period of early childhood, children move from using primarily external means of regulating their emotions to developing internal ER skills (Calkins, 2011). In infancy, attunement occurs through shared gaze and emotional expressions during face-to-face interactions. This matching requires that the parent perceive, understand, and respond sensitively to the child's emotional states (Feldman, 2012; Tronick & Beeghly, 2011). Attunement in parent-child behavior is characterized by parental behaviors that are well-coordinated with infants' social cues so that the interaction is not overstimulating, but still provides the child with useful input for forming and understanding social relationships (Feldman, 2012).

Beyond infancy, parents still play a crucial role in supporting children's ER development. Coregulation of emotional and physiological states during parent-child interactions still occurs, but behaviors look different as children age and their developmental needs change (Cicchetti & Schneider-Rosen, 1986). As parents support children's growing autonomy in these early childhood years, attunement could occur when a parent provides positive reinforcement when their child performs a difficult task on their own (Lunkenheimer et al., 2016). As parents and children match and contingently reinforce each other's emotional expressions and behaviors, attunement also occurs in their complementary biological systems, including autonomic arousal, hormonal release, and brain activation (Feldman, 2012).

Research has shown that in typical parent-child dyads, parents and their preschool-aged children coregulate their physiological states just as they do in infancy (Davis et al., 2018). Recent studies have found that in low-risk community samples, RSA is coordinated between parents and preschool-aged children such that parent RSA predicts concurrent child RSA, and vice versa (e.g., Lunkenheimer et al., 2015). The coordination of RSA activation between parents and children may serve a particularly important role for children's ER, considering the close link between RSA and ER (Beauchaine, 2015). Given the importance of parent-child coregulation for the development of children's ER, understanding parent factors that compromise coregulation becomes particularly important when parents show symptoms of psychopathology.

## Anxiety and Depressive Symptoms

**Parenting.** The presence of depressive and anxiety symptoms in parents impacts the parenting behaviors and emotions that are expressed during parent-child interactions. When parents experience elevated symptoms of depression, parent-child interactions are characterized by greater sadness, conflict, and hostility (Lovejoy et al., 2000). Anhedonia, a key symptom of

depression, may cause parents to withdraw from social engagement, causing relationships with their children to suffer (Tronick, 1989). Rumination, which refers to the unproductive rehearsal of thoughts related to negative mood or experiences, may distract parents from attending to children's needs (Goodman & Gotlib, 1999; Joorman & Gotlib, 2010). This lack of attention towards children may cause parents with depressive symptoms to over- or underestimate their children's abilities (Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). As such, parents with depressive symptoms were found to place unrealistic expectations on their young children, thus overwhelming their ER capacity (Zahn-Waxler et al., 1990).

Parent-child interactions are also impacted by parents' symptoms of anxiety. Parents with anxiety may also become withdrawn during parent-child interactions, offering less assistance, acknowledgement, or praise to their children while completing difficult tasks (Woodruff-Borden, Morrow, Bourland, & Cambron, 2002). Mothers with anxiety symptoms may also possess a parenting style characterized by hypervigilant attention, overprotectiveness, and intrusive behaviors; for example, attempting to help a child complete a task when they are capable of doing it alone (Feldman et al, 2009; Root, Hastings, & Rubin, 2016). Parents with anxiety symptoms may respond to threats in their children's environment more strongly, modeling this anxious processing style to their children (Field & Lester, 2010). Parents with higher levels of anxiety symptoms may also have difficulty regulating their own emotions, which in turn could perpetuate the development of internalizing symptoms in children (Han, Lei, Qian, Li, & Wang, 2016; Kerns, Pincus, McLaughlin, & Comer, 2017).

**Parent-child coregulation.** Patterns of parent-child coregulation differ when parents display heightened symptoms of anxiety and depression (Tronick, 1989). Parents with more anxiety and/or depressive symptoms find it more difficult to achieve and maintain attunement

with their children (Tronick & Beeghly, 2011). One explanation is that since withdrawal from interactions is characteristic for parents with both depression and anxiety, these parents may lack the presence of mind or energy to attune themselves with children's emotions and needs (Feldman et al., 2009). Depressive symptoms have been associated with a disengaged, nonresponsive style of parenting, which limits opportunities for coregulation between parent and child (Tronick, 1989; Tronick & Beeghly, 2011). By contrast, parents with anxiety tend to be hypervigilant and overinvolved, which may lead to intrusion (Beebe et al., 2011). For example, mothers with anxiety may engage in patterns of excessive "chase" behavior, attempting to regain their infant's attention when they turn away from the parent to engage in self-soothing behavior (Beebe & Lachmann, 1998).

Symptoms of anxiety and depression in parents seem to also disrupt or impair physiological attunement between parents and children. For instance, one study examined RSA attunement in mothers and adolescent children among patients with MDD and control participants. They found that parent-child dyads where mothers had MDD were characterized by *negative* attunement in RSA, such that increases in RSA for mothers were met with decreases in RSA for children, and vice versa (Woody, Feurer, Sosoo, Hastings, & Gibb, 2016). Disruption in RSA coregulation may be a result of the detrimental effects of parental depressive symptoms on individuals parent and child RSA (Lunkenheimer et al., 2017). RSA attunement is not always disrupted in contexts of risk, in some cases, dysfunction in dyadic relationships may result in greater attunement of physiological states, which may also convey risk through excessive coupling and transmission of poor regulatory strategies (Gray, Lipschutz, & Scheeringa, 2017; Smith, Woodhouse, Clark, & Skowron, 2016). Another study found that high risk parents and their children showed greater levels of physiological attunement, however, this was negatively

associated with children's self-regulation abilities, suggesting that coordination of physiological systems might actually be conferring additional risk in the already high risk group (Suveg, Shaffer, & Davis, 2016). While disruption of physiological attunement may be expected when parents display depressive symptoms, and therefore more likely to become withdrawn and disengaged (Woody et al., 2016), parents with anxiety symptoms may be more likely to maintain attunement with their children (i.e, through hypervigilant attention; Beebe & Lachman, 1998), therefore transmitting their poor regulation patterns to their children.

**Modeling parent psychophysiological functioning.** Research has shown thus far that physiological attunement between parents and their preschool-aged children can be impaired when parents experience symptoms of depression and/or anxiety. It is still unknown whether depressive and anxiety symptoms may lead to differing coregulatory patterns (e.g., disengaged and unresponsive versus hypervigilant and intrusive). In considering implications for etiology and family intervention, it is important to determine whether parent-child attunement is affected in different ways depending on whether parents show a more depressogenic or anxiogenic style. In addition, prior studies have not parsed whether disrupted parent-child physiological coregulation is a function of parents' dysregulated stress physiology, their mental health symptoms, or some combination of both. Given that RSA is differentially related to anxiety and depressive symptoms in adults (Kircanski et al., 2016; Rottenberg et al., 2007; Watkins et al., 1995), understanding the interface of stress physiology and mental health symptoms is an essential next step in the empirical research literature. To account for these factors, new statistical techniques must be employed. Person-centered approaches allow for the differentiation of subgroups of individuals based on their responses on a variety of indicators. For example, this approach has been used to analyze the joint effect of different forms of parent emotion

socialization on children's problem behaviors (Hernandez, Smith, Day, Neal, & Dunsmore 2018; Miller et al., 2015), and to determine profiles of emotion regulation which included physiological factors as well as observed emotion expression (Turpyn, Chapllin, Cook, & Martelli, 2015). The resulting subgroups represent the organization of various risk factors, thus condensing the information while continuing to account for multiple facets of risk (Larsen & Hoff, 2006). As such, person-centered methodological approaches may shed light on how multiple risk factors organize within parents, improving our understanding of how these factors underscore differences in physiological coregulation between parent and child.

**Current Study**

The current study aims to between understand the relations between parents' mental health risk and parent-child coregulation of RSA through combining a person-centered method for identifying profiles of parents and a fine-grained time series method for capturing attunement in RSA between parents and children over time. Mental health profiles will be determined using a latent profile analysis (LPA) with four indices: anxiety symptoms, depressive symptoms, resting RSA, and RSA withdrawal to stress. Attunement between parents' and children's RSA will be modeled over time using multilevel coupled autoregressive modeling (MCAM), with posterior probabilities from the LPA entered as the level 2 predictor. We expect that on average, attunement will be observed in parents' and children's RSA (i.e., parent RSA will predict concurrent child RSA and vice versa). We also expect that compared to no-risk or lower-risk profiles, belonging to a higher-risk mental health profile will be associated with either a reduction in the strength of attunement, or disruption of attunement such that parents' and children's RSA are unrelated, or negatively correlated with one another. The overwhelming majority of work on physiological attunement between parents and young children has been

investigated with only mothers. In the given study, we assessed fathers' RSA attunement with children in a separate set of models to examine differences in mothers' and fathers' coregulation patterns with their children.

## Methods

### Participants

One hundred and fifty families with young children were recruited to participate in a longitudinal study of parent-child biobehavioral dynamics and risk for child maltreatment. Ninety-four fathers also participated in the study. Participants were selectively recruited to be lower income and higher-risk for child maltreatment using multiple criteria that captured diverse risk indices. We asked about family income, history of involvement with Child Protective Services (CPS), utilization of government assistance programs such as Women, Infants, and Children (WIC), and major life events in the past year using the Social Readjustment Rating Scale (e.g. loss of job, change of address, changes to the child's school or home schedules; Holmes & Rahe, 1967). Families were recruited from the Fort Collins, CO area by distributing flyers in local preschools, daycares, community events, and WIC centers. Flyers were also distributed to families who qualified by CPS caseworkers. Participants were excluded from the study if they could not read or write in English, if children had any pervasive developmental delays, or if mother or child had a heart condition that caused irregular heartbeat.

The sample consisted of 53% male children and 47% female children. At the first wave of data collection, mothers reported their race as 81.3% Caucasian, 7.3% Multi-racial, 2.7% African American, 2.7% Native American, 0.6% Asian, 3.3% other, and 3.3% did not wish to respond. Additionally, 16% self-reported their ethnicity as Hispanic or Latina, 80% non-Hispanic or Latina, and 4% did not wish to respond. Mothers reported children's race as 76% Caucasian,

%13.7 Multiracial, 2.7% African American, 1.3% Native American, and 6.2% other race. Mothers reported children's ethnicity as 23.6% Hispanic or Latinx, 75% non-Hispanic or Latinx, and 1.4% were unknown or did not wish to respond. Fathers reported their race as 78% Caucasian, 9% Multiracial, 2.2% African American, 2.2% Native American, 1.1% Asian, and 6.7% other race. Fathers reported their ethnicity as 36.2% Hispanic or Latino, 62.5% non-Hispanic or Latino, and 1.3% were unknown or did not wish to respond.

Most parents were married (66.7%), while 12.7% were living together, 11.3% were single, 8.7% were separated or divorced. Average annual household income was between $30,000 to $39,000 (income ranged from less than $5,000 to over $90,000 annually). Mothers' education levels varied, with 1.3% completing junior high school, 5.3% having completed some high school, 7.3% with high school degrees, 26.0% having completed some college, 15.3% completing an associate degree, 29.3% with 4-year college degrees, 14.7% having completed some graduate level education. Fathers' education also varied, with 5.3% finishing some high school, 18% with high school degrees, 30.8% completing some college, 14.9% with an associate degree, 20.2% with a 4-year college degree, and 10.6% having completed some graduate level education.

**Procedure**

Families participated in two laboratory sessions as a part of a larger study. Each laboratory session was approximately 2.5 hours in duration. Upon arrival to the laboratory, electrodes and respiratory belts were applied to parents and children to collect physiological data. Next, parents and children participated in a 3-minute-long resting period where they were asked to sit quietly and watch a calming video of dolphins swimming in the ocean. Next, parents and children participated in three dyadic tasks: free play (7 minutes), clean up (4 minutes), and the

Parent-Child Challenge Task (10 minutes), described in detail below, for a total of 14 minutes of dyadic interaction. The first session (Time 1) was completed when children were 3 years old (M=3.03 years, Min=2.83 years, Max=3.42 years), and the second session (Time 2) when children were 4 years old (M=4.00 years, Min=3.75 years, Max=4.42 years. Parents filled out several questionnaires about their psychopathology symptoms, parenting practices, and child behavior. For families where both mothers and fathers participated, sessions were scheduled on different days (when possible) so that children would not become overwhelmed and minimize practice effects. Toys and puzzles for dyadic tasks were counterbalanced across parents so that children encountered novel things each time. For families with only one parent participating, a total of $135 could be earned if all procedures were performed. Families where both the mother and father participated could be compensated up to $210 if all procedures were completed. Children were given a small toy at the end of each laboratory session. Attrition was good between the lab sessions for both parents; 83% for mothers and 78% for fathers.

**Parent-Child Challenge Task.** At both laboratory sessions, parents and children participated in the Parent-Child Challenge Task (PCCT; Lunkenheimer et al., 2016), which is a dyadic task designed to be both collaborative and challenging. Parents and children were given a challenging puzzle and asked to complete three designs that should be just above the child's ability level based on age. Parents were asked to guide their children to assemble the puzzle using only their words and were told not to physically assist their children. They were told that the child will be awarded a prize if they are able to complete all three designs. The task consisted of three phases. The "baseline" phase consisted of the first 4 minutes after the experimenter gave instructions, where parents and children attempted to assemble to puzzles. Next, the experimenter entered the room to tell them that they only had two minutes left to complete the

puzzles. The "stressor" phase consisted of the following 3 minutes where parents and children continued to assemble the puzzles, now under a time constraint. Finally, the experimenter re-entered the room and told the dyad that they were not given enough time and the child was given a toy as a prize regardless of whether they completed the puzzles. The "repair" phase lasted 3 minutes and consisted of the parent and child playing together with the new toy. The total duration of the PCCT is approximately 10 minutes. However, if the child was able to complete all three puzzles before the allotted time, the task was ended early.

**Measures**

**Anxiety and depressive symptoms.** Parents filled out the Brief Symptom Inventory (BSI; Derogatis & Melisaratos, 1983) at each time point. The BSI consists of 53 items, measures 9 distinct psychopathological constructs, and provides a general distress measure. Respondents indicate on a scale of 0-4 how much they experience each symptom, with 0=not at all and 4=extremely. The current study will examine the depression and anxiety subscales, which consist of 6 items each. The depressive subscale reflects symptoms such as dysphoric mood, lack of interest in activities, and feelings of hopelessness. The anxiety subscale encompasses symptoms of restlessness, nervousness, tension, and feelings of panic. Each subscale has been shown to be internally consistent (depression $\alpha$=0.85; anxiety $\alpha$=0.81) and reliable across time (depression r=0.84; anxiety r=0.79; Derogatis & Melisaratos, 1983). Factor analysis confirmed that each subscale represents distinct measures of depressive and anxiety symptoms (Hayes, 1997). Three different measures of symptoms can be obtained using the BSI: The Global Severity Index (GSI), the Positive Symptom Total (PST), and the Positive Symptom Distress Index (PSDI; Derogatis & Melisaratos, 1983). In this case, we utilized the PST score, which is the count of the number of symptoms endorsed by the individual for each subscale. The PST was chosen because

the majority of individuals in our sample did not report elevated severity, however, a few individuals reported scores greater than 3 SD above the mean, creating a skewed distribution. This skew was diminished when examining the PST for our sample. Thus, scores reported in this paper reflect the number of symptoms each individual endorsed out of 6.

**RSA.** Respiration and electrocardiograph (ECG) was recorded from parents and children throughout each laboratory visit. Data was transmitted to a computer through wireless devices worn by participants. Interbeat interval data was processed and cleaned offline by graduate research assistants using Mindware Heart Rate Variability software. ECG data was sectioned in to 30-second segments and RSA magnitude was calculated for each segment as the natural logarithm of the variance of heart period within the frequency related to respiration (0.24-1.04 Hz for children and 0.12-0.40 Hz for adults; Fracasso, Porges, Lamb & Rosenberg, 1994). RSA data was not included for segments that contained greater than 10% noise interference or if the signal was dropped at any point during the segment. Parent resting RSA will be measured as the average RSA across the 3-minute resting period. Parent RSA withdrawal to stress will be measured as the difference in average RSA between the resting period and the 3-minute long stressor phase of the PCCT.

## Data Analytic Plan

**Latent Profile Analysis.** The latent profile models were fit using MPlus version 8 (Muthen & Muthen, 1998-2012). Models with 2 to 5 classes were run to determine the best fitting model for mothers and for fathers. Model selection was determined by values of the AIC, BIC, SSBIC, and chi square, in which lower values denote better model fit. The significance of the bootlegged log ratio test (BLRT) was also used as a criterion for model fit, with non-significant values indicating that the addition of another class will not improve model fit. Once

an optimal model was selected, the item response probabilities and item response means were used to define each class. Response probabilities were used to compare categorical variables (i.e., anxiety and depressive symptoms) and response means were compared for continuous variables (i.e., resting RSA and RSA response to stress). Models were labeled according to this information regarding how each class was set apart from the others. Finally, the posterior probabilities, which are the probabilities that a given individual could belong to each class given their responses to the four indices (anxiety symptoms, depressive symptoms, resting RSA, and RSA response to stress), were exported and saved so that they could be used in the multilevel coupled autoregressive models, described below. LPAs were fit using indicators measured at T2, and posterior probabilities were used to predict differences in parent-child coregulation at T3.

**Multilevel Coupled Autoregressive Modeling.** Patterns of coordination of RSA between parents and children were examined as the extent to which parent and child RSA were predicted by the other's concurrent RSA while controlling for intra-individual variability in RSA using multilevel coupled autoregressive modeling (MCAM) fit using MPlus version 8 (Muthen & Muthen, 1998-2012). We examined whether parent mental health profile membership predicted differences in the strength of coregulation between parent and child. Level 1 models examined parent RSA over time as a function of their prior RSA (to control for intraindividual fluctuations) and concurrent child RSA (a measure of coregulation). Similarly, child RSA was modeled as a function of their prior RSA and concurrent parent RSA so that differential influences between parent and child may be assessed. Level 1 within-dyad equations are based on prior published work (Lunkenheimer et al., 2015; Lunkenheimer et al., 2018; Skoranski et al., 2018) and are displayed below.

$$mRSA_{i,t} = \mu_{mi} + \beta_{m,self1} mRSA_{i,t-1} + \beta_{m,co} cRSA_{i,t} + \varepsilon_{mi,t}$$

$$cRSA_{i,t} = \mu_{ci} + \beta_{c,self1} cRSA_{i,t-1} + \beta_{c,co} mRSA_{i,t} + \varepsilon_{ci,t}$$

In these equations, $mRSA_{i,t}$ and $cRSA_{i,t}$ represent the $i^{th}$ mother's and $i^{th}$ child's RSA at epoch $t$, respectively. The entirety of the parent-child dyadic interaction was modeled, a total of 48 time points, each representing RSA measured over 30-second epochs. Overall mean RSA, denoted as $\mu_{mi}$ for mothers and $\mu_{ci}$ for children, was included in the models to account for the effects that individual differences in mean RSA across all tasks might have on coregulation. Fixed effects for mothers' and children's own prior RSA at a 30-second time lag, $\beta_{m/c,self1}$, was included in the models to account for intraindividual variability in each individual's own fluctuations in RSA over time. Finally, the effect of the partner's concurrent RSA, $\beta_{m,co}$ and $\beta_{c,co,}$ on mother or child RSA were estimated as a measure of coregulation between mothers and children.

Level 2 between-dyad models used the posterior probabilities exported from the LPA to examine whether parents' probability of membership to either the typical class or one of the risk classes would predict differences in parent-child coregulation over the course of the dyadic interaction tasks. Although true class membership for each individual is unknown, posterior probabilities reflect the likelihood that an individual belongs to each of the classes based on the levels of their indicators. The equations for the Level 2 models are displayed below. (Note that fixed effects denoted as $\beta_{m/c}$ in the Level 1 equations are denoted as $\gamma_{m/c}$ in the Level 2 equations.)

$$\mu_{mi} = \mu_{mi} + \gamma_m + \alpha_{m,Mean} Posterior\ Prob$$

$$\beta_{m,self1} = \gamma_{m,self1} + \alpha_{m,self1} Posterior\ Prob$$

$$\beta_{m,co} = \gamma_{m,co} + \alpha_{c,co} Posterior\ Prob$$

We will focus on the coefficient for coregulation ($\beta_{m,co}$) which will give a measure of the change in the strength of attunement based on probability of belonging to a particular class.

## Results

### Preliminary Analyses

At T2, 24 mothers did not participate, another 21 had no RSA data, and 3 had no BSI data. Fathers had more missingness because fewer fathers participated at T2, with a total of 63 not participating. In addition, 5 had no RSA data, and 5 had no BSI data. Analyses were run in MPlus version 8.1 using maximum likelihood estimation, so individuals were not removed from the analysis unless they were missing on all variables (i.e., did not participate), making the valid N=126 for the mother model and N=87 for the father model.

Symptoms of anxiety and depression were skewed for both mothers (anxiety subscale: skewness =1.06, SE =0.22; depressive subscale: skewness =2.35, SE =0.22) and fathers (anxiety subscale: skewness =2.11, SE =0.27; depressive subscale: skewness =2.41, SE =0.27), so these variables were transformed into categorical values with three levels: no symptoms, lower symptoms, and higher symptoms. The "lower symptoms" and "higher symptoms" groups were differentiated using sample-based percentile variables. Cut-off values were determined such that the top 25% of the sample would be classified as reporting "higher symptoms" and individuals with values greater than zero but below that cut-off were classified as reporting "lower symptoms.

For mothers, resting RSA ranged from 1.91 to 9.75, with a mean of 6.10; for fathers, resting RSA ranged from 1.68 to 9.29, with a mean of 6.13. Mothers' mean RSA change score was -0.13, indicating RSA withdrawal to stress on average. Values ranged from -2.14 to 1.57, indicating that some individuals withdrew RSA from baseline to challenge, while others did not

have profound RSA change, and still others who augmented RSA. Similarly, fathers had a mean RSA change score of 0.02, indicating slight augmentation on average. Values ranged from -0.95 to 1.76, indicating that some individuals withdrew RSA from baseline to challenge, while others did not have profound RSA change, and still others who augmented RSA. Resting RSA was not correlated with the change in RSA between the baseline and challenge conditions of the PCCT for either mothers ($r$=.04, $p$=.71) or fathers ($r$=-.17, $p$=.14). Values for resting RSA and RSA change were standardized before entering into the latent class models so that class means could be easily compared against the sample mean of zero.

Bivariate correlation analyses indicated that mothers' change in RSA from baseline to challenge was associated with mothers' anxiety score, such that greater RSA withdrawal (i.e., more negative RSA change) was associated with greater symptoms of anxiety for mothers ($r$=-.27, $p$<.01). Mothers' and fathers' levels of resting RSA were positively correlated ($r$=.26, $p$<.05) suggesting that higher RSA in mothers was associated with higher RSA in fathers, too. At T3, mothers' RSA and children's RSA were positively correlated ($r$=.045, $p$<.001) when broken down into 30-second intervals for each person. The same pattern of results was observed with fathers and children at T3, with RSA positively correlated when broken down into 30-second intervals ($r$=.066, $p$<.001).

**Primary Analyses**

**Latent profile analysis of parent mental health and stress physiology.** LPAs with 2 to 5 classes were run and fit indices were compared to determine the optimal number of classes for both mothers and fathers. For mothers, a 4-class solution was chosen (see Table 1.2), and for fathers, a 3-class solution was chosen (see Table 1.3). For mothers, the BLRT was significant for the 2- and 3-class models but was not significant for the 4-class model, indicating that the

addition of a fifth class was not necessary. The AIC and SSBIC continued to decrease indefinitely, which may be expected when continuous indicators are used (Collins & Lanza, 2010). For fathers, some conflicting information was found between the model fit indices. The BLRT was significant for the 2-class model but was not significant for the 3-class model, indicating that a 4$^{th}$ class was not necessary. Based on these results, a 3-class model was chosen. It has been suggested that in cases where the indices are unclear, choosing the solution that is most interpretable and expected based on prior work is acceptable (Bray et al., 2014).

***Mothers.*** The means and item-response probabilities for the mother model are displayed in Table 1.4. Four distinct classes emerged: 1) the "Typical" class where RSA means were close to the sample mean and no symptoms were reported; 2) the "Moderate Risk/Sensitive" class where mild levels of depressive and anxiety symptoms were endorsed and resting RSA was approximated 1 ½ SD higher than the sample mean; 3) the "High Risk/Anxious" class where anxiety and depressive symptoms were reported to be higher and RSA displayed a more anxious style of responding ("excessive" withdrawal); and 4) the "High Risk/Depressive class where anxiety and depressive symptoms were reported to be higher and RSA displayed a more depressive style of responding (augmentation).

***Fathers.*** The item response probabilities for the father model are displayed in Table 1.5. Three distinct classes emerged for fathers: 1) the "Typical" class, which again reported no symptoms of anxiety or depression and had RSA values close to the sample mean; 2) the "Moderate Risk/Anxious" class where individuals reported mild anxiety symptoms and had resting RSA approximately ½ SD above the sample mean; and 3) the "High Risk" class where individuals endorsed higher levels of both anxiety and depression and displayed RSA

augmentation to stress. As with mothers, the first class of fathers was the largest, encompassing 47.1% of the sample.

**Effects of parent psychophysiological profiles on parent-child coregulation.** Parent and child RSA were modeled using the MCAM procedure in which each partners' RSA was a function of the other's over time, accounting for intraindividual variation in RSA. A significant positive coefficient would suggest attunement (i.e., parents and children tend to increase or decrease their RSA simultaneously), whereas a significant negative coefficient would indicate negative attunement (i.e., parents' increases in RSA are met with decreases in children's RSA and vice versa). Non-significant coefficients represent miscoordination wherein parent-child RSA is not dependent on their partner's RSA.

Parents' probabilities of belonging to a given mental health risk class were entered into the models as level 2 predictors. For mothers, there were posterior probabilities for each of the four classes, and thus four pairs (mother and child) of models, and three posterior probabilities for fathers, offering 3 pairs of models. This set of 7 coupled models (total of 14) represented the prediction of parent-child coregulation at T3 based on the likelihood that parents belonged to a given class at T2. An alpha adjustment following the methods of Benjamini & Hochberg (1995) was applied to the effects of parents' profile membership on parent-child coregulation. See table 2.3 for the adjusted alphas for significant and marginally significant effects.

***Mothers' class membership and coregulation.*** Coefficients for the mean RSA, self-regulation of RSA (prediction of current RSA by the individual's prior RSA), parent-child coregulation of RSA, and the effects of mothers' profile membership on the level 2 predictors are displayed in Table 2.1. Mothers' average RSA ranged from 6.15-6.16 across the course of the interaction and children's RSA ranged from 5.22-5.23, approximately one unit lower than their

mothers. Average self-regulation (i.e., the extent to which an individual's prior RSA predicted their current RSA) was significant in all models (*B*= 0.17 for mothers and 0.36 for children). Across all 8 models, including both mother RSA and child RSA models, mother-child coregulation was significant and positive (*B* ranged from 0.041-0.043 for mothers and 0.051 and 0.053 for children) , indicating attunement in mothers' and children's RSA was occurring across the sample on average. The probability of mothers' membership in the typical class did not affect attunement in the mother RSA model, but there was a marginal negative effect in the child RSA model (*B*=-0.06, *p*=.09) indicating that as the probability that mothers belonged to the typical class increased, the extent to which mother's RSA predicted child RSA was reduced. As the average coregulation coefficient was 0.052 and was decreased by 0.06 with a one-unit increase in likelihood of mothers belonging to the typical class, it is likely that when mothers had a higher probability of belonging to the typical class, parent-child RSA coregulation was negatively coordinated (i.e., inversely related) rather than positively coordinated.

The probability that mothers belonged to the Moderate/Sensitive class again did not affect coregulation in the mother model, but there was a marginally significant positive effect in the child model (*B*= 0.13, *p*=.09) indicating that as the probability that mothers belonged to the Moderate Risk/Sensitive class increased, the extent to which mothers' RSA predicted children's RSA was greater, reflecting greater attunement between these mothers in their children. As the average coregulation coefficient was 0.051 in this model, and the effect of mothers' posterior probability was 0.13, attunement more than doubled as the likelihood of belonging to the Moderate Risk/Sensitive class increased by one unit. In addition, mothers who had a higher likelihood of belonging to the Moderate Risk/Sensitive class showed higher mean RSA over the course of the interaction with their child. The probability that mothers belonged to the High

Risk/Anxious class was again not related to mother-child coregulation in the mother model, however, there was a marginally significant effect in the child model (*B*= 0.08, *p*=.08) indicating that as the probability that mothers belonged to the High Risk/Anxious class increased, the extent to which mother's RSA predicted children's RSA increased, again strengthening attunement by a factor of 2.5. There were no effects of posterior probability of belonging to the High Risk/Depressive class on coregulation.

***Fathers class membership and coregulation.*** Coefficients for the mean RSA, self-regulation of RSA (prediction of current RSA by the individual's prior RSA), parent-child coregulation of RSA, and the effects of fathers' profile membership on the level 2 predictors are displayed in Table 2.2. Fathers' average RSA ranged from 5.92-5.93 across the course of the interaction and children's RSA was 5.08 across the three models, approximately one unit lower than their fathers. Average self-regulation (i.e., the extent to which an individual's prior RSA predicted their current RSA) was significant in all models for both fathers and children (0.11 for fathers and 0.31 for children). We did not observe significant coregulation between fathers and children in any of the 6 child-with-father models. In addition, no significant effects on coregulation were found as a function of the probability that fathers belonged to either the Typical class or the High Risk class. Fathers who had a greater likelihood of belonging to the Moderate Risk/Anxious class showed higher mean RSA across the dyadic interaction tasks with their children.

***Alpha Adjustment.*** Results of the alpha adjustment for the effects of parent profile membership on parent-child coregulation are displayed in Table 2.3. Although there were no significant effects of parent class membership on parent-child coregulation, there were marginal effects in the child-with-mother models. After adjustment, the effects of mothers' posterior

probability for the Typical, Moderate Risk/Sensitive, and High Risk/Anxious classes were no longer of marginal significance (adjusted $\alpha$ = .014, .021, and .007, respectively).

## Discussion

The current study examined person-centered profiles of mental health risk in parents and whether belonging to different risk classes (i.e., typical, Moderate Risk, and High Risk) has an impact on RSA coregulation between parents and their children. We found that mothers and their children displayed positive attunement on average, however, no attunement was observed for fathers and their children. Mothers' mental health risk class had a marginal impact on coregulation with their children. Higher probabilities of belonging to the Moderate Risk/Sensitive class or the High Risk/Anxious class predicted strengthening of attunement while higher probabilities of belonging to the Typical class predicted the disruption of attunement between mothers and their children. However, these effects were rendered non-significant when adjusting alphas for the number of MCAM models run in this study. The present findings suggest that there was a potential relation between mothers' class membership and mother-child attunement, but these effects were not robust. More research may be needed to better understand how parents' mental health risk and parent-child coregulation are related and whether they are moderated by other factors.

### Coregulation with Mothers versus Fathers

We observed that while mothers and their four-year-old children tended to display attunement in their RSA activity during laboratory dyadic tasks, fathers and their children did not appear to coregulate their RSA activity in the same contexts. Across all 6 of the father-child MCAM models, the coregulation coefficients were non-significant and close to zero, which would suggest the absence of coordination or miscoordination (i.e., RSA of one person is not

contingent on the RSA of another). This is an important finding because much of the research on parent-child coregulation of physiology from infancy to adolescence has been conducted with mothers and their children (Davis et al., 2018). Thus, much of what we have discovered on the topic of parent-child physiological coregulation has been between mothers and their children. It seems that coregulation of physiological states is common for mothers and their preschool aged children in the absence of risk factors. For example, one study found that on average, mothers' RSA during a challenging dyadic task positively predicted their children's RSA and vice versa, indicating that attunement is observed between mothers and their 3-year-old children (Lunkenheimer et al., 2015).

Such findings are not ubiquitous, however, and RSA coregulation seems to depend on characteristics of the parent and the child. Lunkenheimer and colleagues (2015) discovered that RSA attunement between mothers and children was disrupted when children displayed symptoms of externalizing (e.g., aggression, difficulty controlling impulses). Further, coregulation at the physiological level is thought to be reciprocally associated with coregulation at the behavioral levels. For example, Feldman and colleagues (2009) found that attunement between mothers' and infants' heart rate became stronger during instances of shared gaze, smiles, etc. Similarly, another study found that parental engagement in a dyadic interaction task with their children was associated with the level of RSA attunement between mothers and their 3-year-old children (Skoranski et al., 2018). When mothers showed greater engagement with their children and accomplishing the task (e.g., using teaching behaviors to guide children), attunement in RSA was strengthened. However, when mothers showed more disengaged behaviors (e.g., ignoring children's bids for attention), attunement in RSA was weakened, and disrupted at the highest levels of disengagement (Skoranski, et al., 2018).

The fact that fathers did not show significant attunement with the same children in the same contexts as did mothers may have to do with differences in engagement style between the parents. Some research suggests that the way that fathers interact with their children is different than the way that mothers interact with their children. Mothers tend to engage in more face-to-face interactions during infancy, which in turn has been shown to boost attunement between mothers and children at the physiological level (Feldman et al., 2009). In these interactions, mothers and children seem to exist in a sort of bubble, with little attention paid to stimuli outside of the dyad (Feldman, 2012). As children grow older, this type of interaction may take other forms, such as a focus on children's needs and support of those needs using interpersonal mechanisms (e.g., teaching, emotional support; Lunkenheimer et al., 2016). Conversely, fathers tend to employ more *triadic* engagement while interacting with their children (Feldman, 2007). This type of engagement involves directing attention toward the environment, for example, moving from face-to-face play toward attending to objects in the room (e.g., toys), encouraging engagement with the environment (Feldman, 2007). The fact that face-to-face attention is interrupted during this form of interaction, it may follow that attunement in physiological systems is less common between fathers and children than it is between mothers and children. Another, more simple, explanation may be that mothers and their children tend to attune to one another more readily because they spend more time together than do fathers and their children, as the majority of mothers reported being the child's primary caregiver.

**Effects of Parents' Profile Membership on Coregulation**

We observed some interesting trends with mothers' profile membership and mother-child coregulation, although they were of marginal significance and rendered non-significant when applying the alpha adjustment. When the probability of mothers' belonging to either the

"Moderate Risk/Sensitive" or "High Risk/Anxious" classes increased, coregulation of RSA between mothers and children also increased, reflecting a strengthening of attunement. Conversely, when the probability of mothers' belonging to the Typical class increased, coregulation of RSA between mothers and children decreased, reflecting negative attunement. Mothers who belonged to the "Moderate Risk/Sensitive" class were differentiated from the rest of the sample by highly elevated resting RSA and low levels of anxiety and depressive symptoms. Mothers who belonged to the "High Risk/Anxious" class were differentiated from the rest of the sample by higher levels of depression and anxiety symptoms, as well as excessive RSA withdrawal.

Mothers with heightened anxiety symptoms may be more prone to direct hypervigilant attention towards their children (Beebe et al., 2011), which in turn may lead to increased attunement with children by nature of high levels of engagement. The "Moderate Risk/Sensitive" class was so named due to the fact that extremely heightened resting RSA may reflect greater sensitivity to environmental stimulation among mothers (Porges, 2007). In the case of parent-child interactions, this could also translate to greater attentiveness and engagement for children, and in turn greater attunement of RSA (Skoranski et al., 2018). Although attunement is being facilitated, it does not necessarily serve as a buffering factor leading to the improvement of children's developing ER ability. RSA attunement with a parent who displays symptoms of anxiety and depression may not be adaptive for children, as they may develop similar patterns of physiological regulation as their parent (Suveg et al., 2016). When the chance of mothers' belonging to the Typical class increased, the opposite effect was observed where attunement between mother and child RSA was inversely related (i.e., when one increased, the other decreased). This may mean that this class is heterogeneous and may include other risk factors not

assessed in this study, for example, there may be parents who show low depressive and anxiety symptoms but are at risk in other ways (e.g., hostility).

The mostly null effects for parents' profile membership on parent-child coregulation may be explained in a number of ways. One explanation is that although parents' mental health status plays a role, coregulation is a dyadic construct and thus both parent and child factors need to be considered when examining individual differences in physiological attunement. This point has been demonstrated by Lunkenheimer and colleagues 2018 who found that when considering the effects of both parent psychopathology and child behavioral problems on attunement in parent-child RSA, child externalizing was a stronger predictor of weakening of attunement than parent depressive symptoms. Thus, considering child risk factors alongside parent symptoms and physiological regulation may be necessary to observe the full impact of mental health risk on parent-child coregulation. This may be particularly important as children move into the period of early childhood and beyond, when child autonomy is growing and thus the effects of children's dispositions may become more important than in infancy (Cicchetti & Schneider-Rosen, 1986).

Another explanation is that parent psychopathology plays a stronger role in parent-child coregulation in some interaction contexts but not others. Lunkenheimer and colleagues (2018) also found that parent and child risk factors had the greatest impact on attunement during unstructured dyadic tasks (i.e., free play) versus tasks with set guidelines and goals to accomplish (Lunkenheimer et al., 2018). The current study analyzed parent-child coregulation across a number of different tasks, each with differing levels of structure. Perhaps analyzing separately by task would reveal different results. However, it could be argued that the laboratory setting in and of itself is a more structured environment than is normally encountered by parents and their 4-year-old children.

Further, parent mental health risk may have a greater impact on expressed parenting behaviors than on the coordination of underlying physiology. Research has shown a link between parent symptoms of anxiety and depression and the behaviors that they display with their children, for example, type of engagement, and even hostility and aggression (Goodman & Gotlib, 1999; Lovejoy et al., 2000). Behaviors such as these were not assessed in the current study, nevertheless, may be more likely to lead to the disruption of coregulation at the physiological level. However, the examination of parent and child risk factors on coregulation of physiological activity is still in the early phases and requires greater attention in the field to determine what is typical and what is a factor of heightened mental health risk.

**Strengths and Limitations**

The combination of a person-centered approach to mental health risk and assessment of the moment-to-moment coordination of parent and child physiological states was a major strength of the current study. This approach allowed us to examine multiple facets of risk on a process-oriented outcome. The clustering of parent mental health symptoms and physiological regulation using LPA along with the time-series measurement of parent and child physiological states using MCAM allowed for greatly increased power in the model even with a relatively low sample size of 150. In addition, the current study assessed differences in mental health risk and parent-child coregulation between mothers and fathers. To our knowledge, this is the first study to examine the coregulation of RSA between fathers and their children during the period of early childhood, adding to a growing base of information on physiological coregulation patterns among parents and their children.

Despite these strengths, several limitations must be mentioned. We did not consider covariates in the current model, such as child sex or family income, which could have played a

role in parent-child coregulation. Future work should assess whether there are differences in the strength of coregulation for same-gender versus different-gender parent-child dyads, for example, fathers may have displayed significant physiological coregulation with their sons but not daughters, or vice versa. In addition, we did not utilize a clinical diagnostic tool to assess depressive and anxiety symptoms, and as such, it is difficult to know what percentage of mothers and fathers would meet criteria for a mental health disorder. This limits generalizability to families where parents display sub-clinical levels of depression and anxiety. It is possible that different trends would emerge when examining severe levels of mental health problems alongside RSA regulation and parent-child coregulation. Indeed, our lack of significant findings for the impact of parent mental health profile membership on parent-child coregulation may be due to the fact that we are examining mild levels of mental health risk versus more pervasive problems, which may have a greater impact on the way that parents and children interact with one another.

**Conclusions**

Understanding the ways in which parents influence ER skill building among children remains a key goal of research in developmental psychology. The current study contributes to that knowledge pool, suggesting that 1) parents' mental health risk is not homogeneous and involves the clustering of both behavioral and emotional symptoms as well as physiological regulation; and 2) parent-child coregulation of RSA is apparent for at least mothers and their preschool-aged children, which may be one way in which risk is transmitted from parents and their young children. Findings also suggest that it is important to consider parent gender when attempting to establish norms for the coregulation of physiology between parents and children, as we found that fathers' and their 4-year-old children's RSA was miscoordinated on average. This

may suggest that when it comes to fathers, coregulatory processes play a smaller role in conveying risk for mental health to their young children.

## Tables

Table 2.1 Coefficients for MCAM models of mother-child coregulation by mothers' class membership. A total of 8 models were run; two models for each mother profile including a model predicting mother RSA ("Mother Model") and a model predicting child RSA ("Child Model"). The coregulation effects are highlighted in red text. "Coregulation" refers to the effect of mothers' RSA on children's RSA and vice versa. "Class Prob x Coreg" refers to the effect that mothers belonging to a particular class had on mother-child RSA coregulation. Significant coefficients are bolded and noted as follows: * $p<.05$, ** $p<.01$, *** $p<.001$.

| | **Typical Class** | | | | **Moderate Risk/Sensitive Class** | | | | **High Risk/Anxious Class** | | | | **High Risk/Depressive Class** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Mother Model | | Child Model | | Mother Model | | Child Model | | Mother Model | | Child Model | | Mother Model | | Child Model | |
| | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE |
| **Self-Mean** | **6.15*** ** | 0.09 | **5.22*** ** | 0.06 | **6.16*** ** | 0.09 | **5.22*** ** | 0.06 | **6.16*** ** | 0.09 | **5.23*** ** | 0.06 | **6.16*** ** | 0.09 | **5.23*** ** | 0.06 |
| **Self-Reg 30 secs** | **0.17*** ** | 0.02 | **0.36*** ** | 0.02 | **0.17*** ** | 0.02 | **0.36*** ** | 0.02 | **0.17*** ** | 0.02 | **0.36*** ** | 0.02 | **0.17*** ** | 0.02 | **0.36*** ** | 0.02 |
| **Coregulation** | **0.042** ** | **0.01** | **0.052*** ** | 0.01 | **0.043** ** | 0.02 | **0.051** ** | 0.02 | **0.041** ** | 0.01 | **0.053*** ** | 0.02 | **0.042** ** | 0.01 | **0.054*** ** | 0.02 |
| **Class 1 Prob x Self-Mean** | -0.24 | 0.22 | -0.12 | 0.14 | **1.06*** ** | 0.32 | 0.17 | 0.28 | 0.29 | 0.19 | -0.11 | 0.14 | -0.50 | 0.56 | 0.23 | 0.21 |
| **Class 1 Prob x Self-Reg** | -0.07 | 0.05 | -0.01 | 0.05 | -0.03 | 0.06 | 0.04 | 0.10 | 0.06 | 0.08 | -0.01 | 0.07 | 0.10 | 0.07 | 0.00 | 0.08 |
| **Class 1 Prob x Coreg** | -0.04 | 0.03 | **-0.06^** | 0.04 | 0.09 | 0.10 | **0.13^** | 0.08 | 0.05 | 0.04 | **0.08^** | 0.04 | -0.05 | 0.04 | -0.06 | 0.04 |

Table 2.2 Coefficients for MCAM models of father-child coregulation by fathers' class membership. A total of 6 models were run; two models for each father profile including a model predicting father RSA ("Father Model") and a model predicting child RSA ("Child Model"). The coregulation effects are highlighted in red text. "Coregulation" refers to the average effect of fathers' RSA on children's RSA and vice versa. "Class Prob x Coreg" refers to the effect that fathers belonging to a particular class had on father-child RSA coregulation. Significant coefficients are bolded and noted as follows: * $p<.05$, ** $p<.01$, *** $p<.001$.

| | **Typical Class** | | | | **Moderate Risk/Anxious Class** | | | | **High Risk Class** | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Father Model | | Child Model | | Father Model | | Child Model | | Father Model | | Child Model | |
| | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE | Estimate | SE |
| **Self-Mean** | **5.92*** ** | 0.14 | **5.08*** ** | 0.12 | **5.93*** ** | 0.14 | **5.08*** ** | 0.09 | **5.92*** ** | 0.14 | **5.08*** ** | 0.09 |
| **Self-Reg 30 secs** | **0.11*** ** | 0.02 | **0.31*** ** | 0.03 | **0.11*** ** | 0.02 | **0.31*** ** | 0.03 | **0.11*** ** | 0.02 | **0.31*** ** | 0.03 |
| **Coregulation** | 0.017 | 0.02 | 0.017 | 0.02 | 0.018 | 0.02 | 0.017 | 0.02 | 0.017 | 0.02 | 0.014 | 0.02 |
| **Class Prob x Self-Mean** | -0.17 | 0.29 | 0.08 | 0.19 | **0.52*** | 0.30 | -0.18 | 0.22 | -0.55 | 0.34 | 0.11 | 0.28 |
| **Class Prob x Self-Reg** | 0.03 | 0.05 | -0.06 | 0.05 | -0.07 | 0.05 | -0.02 | 0.05 | 0.04 | 0.06 | 0.13 | 0.08 |
| **Class Prob x Coreg** | -0.01 | 0.04 | -0.03 | 0.04 | 0.02 | 0.04 | 0.04 | 0.04 | -0.01 | 0.08 | -0.02 | 0.11 |

Table 2.3 Table of adjusted alphas given 14 different models using the Benjamini & Hochberg (1995) alpha correction protocol. Coregulation coefficients were compared against the adjusted alphas for significance and marginal significance. Marginal effects in the original models are highlighted in red text; the effects are no longer marginal after adjusting the alphas.

| Coregulation Coefficient | *p*-value | Adjusted Alpha criterion | Adjusted Alpha (marginal effects) |
|---|---|---|---|
| 1. Mother Typical – Mother Model | .255 | .025 | .05 |
| **2. Mother Typical – Child Model** | **.094** | **.007** | **.014** |
| 3. Mother Mod Risk/Sensitive – Mother Model | .354 | .032 | .064 |
| **4. Mother Mod Risk/Sensitive – Child Model** | **.096** | **.010** | **.021** |
| 5. Mother High Risk/Anx – Mother Model | .185 | .017 | .038 |
| **6. Mother High Risk/Anx – Child Model** | **.083** | **.004** | **.007** |
| 7. Mother High Risk/Dep – Mother Model | .224 | .021 | .043 |
| 8. Mother High Risk/Dep – Child Model | .177 | .014 | .029 |
| 9. Father Typical – Father Model | .758 | .043 | .086 |
| 10. Father Typical – Child Model | .526 | .036 | .071 |
| 11. Father Mod Risk/Anx – Father Model | .616 | .039 | .079 |
| 12. Father Mod Risk/Anx – Child Model | .310 | .029 | .057 |
| 13. Father High Risk – Father Model | .953 | .05 | .10 |
| 14. Father High Risk – Child Model | .877 | .046 | .093 |

# GENERAL DISCUSSION

Symptoms of anxiety and depression continue to plague millions of Americans each year, and as such, remain a top priority for intervention (NIMH, 2017). One method that can be used to improve the well-being of the general population is to prevent the development of symptoms before they start. Symptoms of anxiety and depression can be observed as early as the preschool years, however, they continue to grow in prevalence and severity throughout childhood, reaching their peak during adolescence (APA, 2013). As such, targeting prevention efforts during the period of early childhood seems practical. Young children at risk for developing symptoms of anxiety and depression, and potentially clinical disorders, can be identified by assessing symptoms of their parents, as the presence of anxiety and depressive symptoms in parents is a key risk factor for the development of symptoms in children (Joorman et al., 2008). Further, mental health symptoms affect individuals' physiological processes as well as behavioral and emotional experiences, and as such, knowing how the presence of symptoms in parents impacts children is crucial for developing prevention efforts.

This set of two studies attempts to contribute to these efforts by first examining how symptoms of anxiety and depression as well as their physiological substrates cluster within parents of young children using a person-centered profile approach. Findings suggest that unique mental health profiles can be observed in parents, conveying different levels of risk for a mental health problem. In addition, the relationship between parent mental health risk profiles and children's behavioral and emotional adjustment was examined. In general, parents who belonged to mental health profiles conveying greater amounts of risk had children who showed greater levels of emotional and behavioral problems. In study II, the relationship between parents'

mental health profile membership was used to predict differences in the moment-to-moment coordination of physiological states between parents and children. This analysis produced null findings, however, it still provides potentially useful information about the mechanisms of transmission of mental health risk from parents to children.

**Person-Centered Approach**

One important contribution of this work is the utilization of a person-centered approach to untangle relationships between symptoms of anxiety and depression and the underlying physiological regulation processes that produce them. In both studies, a latent profile analysis was performed to determine whether different meaningful patterns of alignment between physiological and behavioral/emotional dimensions of mental health symptoms emerged. Symptoms of anxiety and depression are thought to arise from difficulties with emotion regulation (ER), which has a biological basis in parasympathetic stress reactivity (Beauchaine, 2015). We considered levels of resting RSA and RSA withdrawal to stress along with reported anxiety and depressive symptoms as part of a constellation of risk for mental health difficulties. Although it is widely accepted that RSA is related to difficulties with ER (e.g., Beauchaine, 2015; Beauchaine & Thayer, 2015) and symptoms of anxiety and depression in adults (e.g., Beauchaine, 2001; Thayer & Lane, 2001; 2009), it remains unclear whether RSA represents a general biomarker for psychopathology or displays different patterns of activity that can be attributed to a more depressive or anxious style. Our findings suggest that the latter may be true, at least for mothers of young children. We found that mothers displayed two "High Risk" groups which were distinguishable by patterns of "excessive" RSA withdrawal and higher probability of having heightened anxiety symptoms versus patterns of augmented RSA and higher probability of depressive symptoms.

Another interesting development from this person-centered approach was the observation of a "Moderate Risk/Sensitive" class among mothers, which was not anticipated based on prior research that had analyzed patterns of RSA and anxiety and depressive symptoms using a variable-centered approach. This group of individuals was differentiated from the other groups by their elevated levels of resting RSA as well as mild levels of depressive and anxiety symptoms. In prior work, higher resting RSA is thought to be a protective factor when it comes to mental health and is typically associated with lower levels of anxiety and depressive symptoms. However, higher resting RSA is also associated with greater sensitivity to environmental stimuli, which may also apply to social interactions. The observation of a mental health risk class with moderate levels of symptoms as well as heightened resting RSA points to the importance of the function of RSA in sensitivity, and that in some contexts, resting RSA may generate increased symptoms of depression and anxiety (e.g., in high-stress environments; Lunkenheimer et al., 2016). Thus, future studies examining resting RSA as a generally adaptive feature of mental health should be sure to consider the context in which the measurement is occurring.

Applying a person-centered approach toward understanding the transmission of mental health risk was also useful in that the highest levels of child emotional and behavioral problems were observed among children of mothers who belonged to the "Moderate Risk/Sensitive" class versus the two "High Risk" classes. In study I, we examined the relationship between parent mental health profiles and children's emotional and behavioral adjustment, including internalizing, externalizing, emotion regulation, and negativity/lability. In prior studies using a variable-centered approach, elevated symptoms of anxiety and depression among parents predicted greater internalizing and externalizing symptoms for children (e.g., Graziano &

Derefinko, 2010). Our results suggest that when examining relationships between parents' mental health risk using a person-centered approach, physiological risk factors (in this case, elevated RSA) may take precedence over mental health symptoms in predicting child outcomes. As has already been discussed, heightened RSA may reflect greater sensitivity among parents, which would be adaptive in some ways (e.g., being sensitive to children's needs), however, may also lead to worse outcomes depending on contexts (e.g., they may be more sensitive to the effects of parenting stress; Ellis et al., 2011). Another way that elevated RSA may convey risk is that it could reflect a disengaged style of interacting, as some level of parasympathetic withdrawal should be evident even in low stress, but active social interactions (Porges, 2003). Although the "High Risk" classes of mothers had children with higher levels of behavioral and emotional problems than mothers in the "Typical" class (as expected), the "Moderate Risk/Sensitive" class of mothers had children with the most problematic outcomes in terms of externalizing and internalizing symptoms. These results underscore the importance of incorporating person-centered approaches to examine nuances in the complex relationship between mental health symptoms, physiological biomarkers, and the transmission of mental health symptoms from parent to child.

**Transmission of Mental Health Risk**

Understanding the processes through which mental health risk at both the behavioral and physiological levels is conveyed from parent to child is also important for determining the best way to direct preventative efforts. In study II, we attempted to contribute to this knowledge base by examining the relationship between parent mental health profiles and parent-child coregulation of RSA. Prior research has shown a link between risk factors in both the parent and child and their ability to coregulate their physiological states. Physiological coregulation is

thought to be one process by which poor regulatory strategies may be passed from parent to children early in development (Feldman, 2012). In typical parent-child dyads, being able to attune to the parents' physiological states is adaptive because it provides a means through which children's systems become regulated over time (Feldman, 2012; Calkins, 2011). However, this process can be disrupted when parents and/or children show behavioral problems or symptoms, impacting their ability to engage in interaction with one another (Lunkenheimer et al., 2018). Additionally, attunement may prove to be less beneficial for children when parents tend to be dysregulated themselves (Suveg et al., 2016). As such, developing interventions for higher risk parents and children that specifically targets the strengthening of physiological attunement will be helpful for some, but not all cases.

We found that using this person-centered approach to assess mental health risk did not yield significant differences in parent-child coregulation of RSA. Since this is a somewhat recent line of inquiry, it would be premature to suggest that physiological coregulation is not at all impacted by parents' mental health risk. Instead, the null findings may be better explained by the fact that we used a community sample of individuals, whose symptoms were too mild to have a detectable impact on parent-child coregulation. Another possibility to consider is that using a person-centered approach to analyze the impact of one individual's characteristics on a dyadic process may not be the optimal approach. In predicting dyadic outcomes, it might be more fruitful to examine mental health profiles that include both parent and child indicators of risk to understand the full picture.

**Differences between Mothers and Fathers**

Another important contribution of this work was to examine relationships among mental health risk, parent-child processes, and child behavioral and emotional adjustment among fathers

as well as mothers. In general, there is a paucity of research on fathers, which is a problem because most of what we can conclude about mental health, parenting, and transmission of mental health symptoms from parent to child can only be generalized to mothers (Goodman & Gotlib, 1999). Findings from both of the present studies indicate that this is a major oversight, because patterns observed with fathers are meaningfully distinct from what we observed with mothers. In study I, we found that while mothers could be divided into four mental health classes, with distinct "High Risk/Anxious" and "High Risk/Depressive" classes, fathers were divided into only three, showing a Typical class along with a Moderate Risk and High Risk class. This could mean that for fathers, differentiation between anxious and depressive symptoms may not be as meaningful as it is for mothers. Further, the "High Risk" class for fathers showed a pattern that suggested hyporeactivity in terms of RSA withdrawal to stress, which has been shown to be associated more with depressive symptoms than symptoms of anxiety (Rottenberg et al., 2007). In turn, this could lead to differences in characteristic parenting styles of high-risk fathers, with depressogenic styles being more common and leading to behavior that is more withdrawn from the environment and linked with lower engagement with children (Wilson & Durbin, 2010).

Another difference we observed was that for mothers, the "Moderate Risk/Sensitive" class of mothers seemed to have the most problematic outcomes for children, whereas for fathers, their "Moderate Risk/Anxious" class did not show significant effects on children's outcomes. The "High Risk" class of fathers had children with significantly higher externalizing symptoms than the "Typical" class, however. This may suggest that when it comes to fathers' influence on their young children, mental health risk is conveyed only when fathers display more elevated levels of depressive and anxious symptoms, whereas for mothers, risk may be conveyed

with only mild levels of symptoms. This may be explained simply by way of exposure. Since a large majority of mothers in our sample reported being the primary caregiver of the child, they would have spent more time with their children. Exposure to low levels of mental health symptoms (i.e., mild anxiety and depression) for extended periods of time may have the same effect on children as exposure to more heightened levels of symptoms only occasionally.

In study II, we found that while mothers and their 4-year-old children appeared to coregulate their RSA such that mothers' RSA predicted children's concurrent RSA over time and vice versa, indicating positive attunement between RSA states, the same could not be said regarding fathers and their children. We found that fathers' RSA had no effect on children's concurrent RSA and vice versa, which may indicate miscoordination in RSA states rather than attunement. To our knowledge, this is the first study to examine RSA coregulation among fathers and their preschool-aged children, establishing a precedent in the field. The fact that while many studies have found significant attunement among physiological states for mothers and their children (Davis et al., 2018), yet we did not find significant attunement for fathers, highlights again the importance of examine father-child interactions.

Although it is premature to say that fathers do not engage in physiological coregulation with their young children, our finding points to important differences between mother-child and father-child relationships. Previous research has suggested that fathers interact with their young children in ways that are different than mothers, for example, father-child play involves more attention to objects (i.e., toys) versus face-to-face interactions (Feldman, 2007). This fact alone may explain differences in physiological attunement between mothers and fathers, however, there is currently very little evidence base from which to draw conclusions. Findings from this study suggest an important route for future research: investigating father-child coregulation and

how it differs from and compliments mothers' contributions to children's developing ER. In addition, it may be useful to examine triadic coregulation; in other words, how mothers', fathers', and children's physiological states influence one another's, especially given that many social interactions at home involve more than two people (i.e., only mothers and children or fathers and children).

**Conclusions**

Major take-aways from this dissertation project include: 1) parents belong to unique profiles of mental health risk and stress physiology involving different combinations of anxiety and depressive symptoms, resting levels of RSA, and RSA response to stress; 2) parents' belonging to a higher-risk mental health profile predicts poorer behavioral and emotional health for young children; 3) important differences exist between mothers' and fathers' mental health profiles, as well as interactional dynamics between fathers and their young children. The finding that distinct mental health profiles may exist for parents, particularly that there may be differentiation between higher-risk profiles, is important when considering routes for intervention for parent mental health and therefore prevention of mental health problems for their children. For example, if it is known that a given parent belongs to a more anxious high-risk profile, characterized by hyperactivity, this response style can be selectively attended to in intervention versus a more depressive style of withdrawal and hyporeactivity.

The same may be said about differences between mothers and fathers. We found that fathers' "High Risk" profile was characterized by a more hyporeactive RSA response to stress, which may mean that fathers are more prone to withdrawal and disengagement, whereas mothers with heightened anxiety and depressive symptoms may show either withdrawal or a hyperreactive parenting style characterized by hypervigilance and intrusion. Further, knowing

that mothers' "Moderate Risk/Sensitive" class was most problematic for child outcomes helps to hone in on the types of behaviors and response styles that need to be intervened upon (i.e., in this case, sensitivity to environmental stressors) to prevent emotional and behavioral problems in children. Finally, being aware that fathers may show different patterns of coregulation with their young children than do mothers suggests that findings associated with predominantly mother-child dyads may not apply to father-child relationships. Therefore, interventions involving adjustment of parent-child behavioral patterns (e.g., Parent-Child Interaction Therapy) may need to be catered to suit the unique needs of father-child interactions.

## References


Achenbach, T. M. & Rescorla, L. A. (2001). *Manual for the ASEBA school-age forms & profiles.* Burlington, VA: University of Vermont, Research Center for Children, Youth, & Families.

Aktar, E., Van Bockstaele, B., Pérez-Edgar, K., Wiers, R. W., & Bögels, S. M. (2019). Intergenerational transmission of attentional bias and anxiety. *Developmental Science, 22*(3). doi:10.1111/desc.12772

American Psychological Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychological Association

Anderson, E. R. & Hope, D. A. (2008). A review of the tripartite model for understanding the link between anxiety and depression in youth. *Clinical Psychology Review, 28,* 275-287. doi:10.1016/j.cpr.2007.05.004

Angst, J., Gamma, A., Gastpar, M., Lépine, J. -., Mendlewicz, J., & Tylee, A. (2002). Gender differences in depression: Epidemiological findings from the european DEPRES I and II studies. *European Archives of Psychiatry and Clinical Neuroscience, 252*(5), 201-209. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1007/s00406-002-0381-6

Barnett, M. A., Deng, M., Mills-Koonce, W., Willoughby, M., & Cox, M. (2008). Interdependence of parenting of mothers and fathers of infants. *Journal of Family Psychology, 22(*4), 561-573. http://dx.doi.org/10.1037/0893-3200.22.3.561

Barrett, J., & Fleming, A. S. (2011). Annual research review: All mothers are not created equal: Neural and psychobiological perspectives on mothering and the importance of individual differences. *Journal of Child Psychology and Psychiatry, 52*(4), 368-397. http://dx.doi.org/10.1111/j.1469-7610.2010.02306.x

Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*(2), 183-214. doi: 10.1017/S0954579401002012

Beauchaine, T. P. (2015). Future directions in emotion dysregulation and youth psychopathology. *Journal of Clinical Child & Adolescent Psychology, 44(*5), 875-896. doi:10.1080/15374416.2015.1038827

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology, 98*, 338-350. doi:10.1016/j.ijpsycho.2015.08.004

Beebe, B. & Lachmann, F. M. (1998). Co-constructing inner and relational processes: Self- and mutual regulation in infant research and adult treatment. *Psychoanalytic Psychology, 15*(4), 480-516.

Beebe, B., Steele, M., Jaffe, J., Buck, K. A., Chen, H. … & Feldstein, S. (2011). Maternal anxiety symptoms and mother-infant self- and interactive contingency. *Infant Mental Health Journal, 32*(2), 174-206. doi:10.1002/imhj.20274

Berntson, G. G., Cacioppo, J. T. & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review, 98*(4), 459-487.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology, 30*(2), 183-196. doi: 10.1111/j.1469-8986.1993.tb01731.x

Blandon, A. Y., Calkins, S. D., Keane, S. P., & O'Brien, M. (2010). Contributions of child's physiology and maternal behavior to children's trajectories of temperamental reactivity. *Developmental Psychology, 46*(5), 1089-1102. doi: 10.1037/a0020678

Bögels, S., & Phares, V. (2008). Fathers' role in the etiology, prevention and treatment of child anxiety: A review and new model. *Clinical Psychology Review, 28*, 539-558. doi:10.1016/j.cpr.2007.07.011

Brooker, R. J., Buss, K. A., Lemery-Chalfant, K., Aksan, N., Davidson, R. J., & Goldsmith, H. H. (2013). The development of stranger fear in infancy and toddlerhood: Normative development, individual differences, antecedents, and outcomes. *Developmental Science, 16*(6), 864-878.

Calkins, S. D. (2011). Caregiving as coregulation: Psychobiological processes and child functioning. In A. Booth, S. McHale, N. Landale (Eds.), *Biosocial foundations of family processes* (pp. 49-59). New York, NY: Springer. doi:10.1007/978-1-4419-7361-0_3

Calkins, S. D., & Dedmon, S. E. (2000). Physiological and behavioral regulation in two-year-old children with aggressive/destructive behavior problems. *Journal of Abnormal Child Psychology, 28*(2), 103-118. doi: 10.1023/A:1005112912906

Calkins, S. D., Graziano, P. A., Berdan, L. E., Keane, S. P., & Degnan, K. A. (2008). Predicting cardiac vagal regulation in early childhood from maternal-child relationship quality during toddlerhood. *Developmental Psychobiology, 50*(8), 751-766. doi: 10.1002/dev.20344

Calkins, S. D., Graziano, P. A., & Keane, S. P. (2007). Cardiac vagal regulation differentiates among children at risk for behavior problems. *Biological Psychology, 74*(2), 144-153. doi: 10.1016/j.biopsycho.2006.09.005

Calkins, S. D., & Keane, S. P. (2004). Cardiac Vagal Regulation across the Preschool Period: Stability, Continuity, and Implications for Childhood Adjustment. *Developmental Psychobiology, 45*(3), 101-112. doi: 10.1002/dev.20020

Carthy, T., Horesh, N., Apter, A., Edge, M. D., & Gross, J. J. (2010). Emotional reactivity and cognitive regulation in anxious children. *Behaviour Research and Therapy, 48*(5), 384-393. doi:10.1016/j.brat.2009.12.013

Center for Disease Control (2019, April 19). *Children's mental health.* Retrieved from https://www.cdc.gov/childrensmentalhealth/data.html.

Cicchetti, D., & Schneider-Rosen, K. (1986). An organizational approach to childhood depression. In M. Rutter, C. Izard, & P. Read (Eds.), *Depression in Young People: Clinical and Developmental Perspectives* (pp. 71-134). New York: Guilford.

Clark, L. A. & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*(3), 316-336.

Cole, D. A., Truglio, R., & Peeke, L. G. (1997). Relation between symptoms of anxiety and depression in children: A multitrait-multimethod-multigroup assessment. *Journal of Consulting and Clinical Psychology, 65*, 110-119.

Cole, P. M., Luby, J., & Sullivan, M. W. (2008). Emotions and the development of childhood depression: Bridging the gap. *Child Development Perspectives, 2*(3), 141-148.

Cox, M. J., & Paley, B. (1997). Families as systems. *Annual Review of Psychology, 48*, 243-267. http://dx.doi.org/10.1146/annurev.psych.48.1.243

Dale, L. P., O'Hara, E. A., Schein, R., Inserra, L., Keen, J., Flores, M., & Porges, S. W. (2011). Measures of infant behavioral and physiological state regulation predict 54-month

behavior problems. *Infant Mental Health Journal, 32*(4), 473-486. doi: 10.1002/imhj.20306

Davis, M., West, K., Bilms, J., Morelen, D., & Suveg, C. (2018). A systematic review of parent-child synchrony: It is more than skin deep. *Developmental Psychobiology,* 1-18. doi:10.1002/dev.21743

Dekker, J., Koelen, J. A., Peen, J., Schoevers, R. A., & Gijsbers-Van Wijk, C. (2008). Gender differences in clinical features of depressed outpatients: Preliminary evidence for subtyping of depression? *Women & Health, 46*(4), 19-38. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1300/J013v46n04_02

Derogatis, L. R. & Melisaratos, N. (1983). The Brief Symptom Inventory: An introductory report. *Psychological Medicine, 13,* 595-605.

Ditto, B., Eclache, M., & Goldman, N. (2017). Short-term autonomic and cardiovascular effects of mindfulness body scan meditation. In *Mindfulness: Nonclinical applications of mindfulness: Adaptations for school, work, sports, health, and general well-being (vol. IV).* (pp. 341-357). New York, NY: Routledge/Taylor & Francis Group.

Dunbar, M., Ford, G., Hunt, K., & Der, G. (2000). A confirmatory factor analysis of the Hospital Anxiety and Depression scale: Comparing empirically and theoretically derived structures. *British Journal of Clinical Psychology, 39,* 79-94.

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M., & Van Ijzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary–neurodevelopmental theory. *Development and Psychopathology, 23*(1), 7-28. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1017/S0954579410000611

Feldman, R. (2007). Parent-infant synchrony and the construction of shared timing; Physiological precursors, developmental outcomes, and risk conditions. *Journal of Child Psychology and Psychiatry, 48*(3-4), 329-354. doi: 10.1111/j.1469-7610.2006.01701.x

Feldman, R. (2012). Bio-behavioral synchrony: A model for integrating biological and microsocial behavioral processes in the study of parenting. *Parenting: Science and Practice, 12*(2-3), 154-164. doi: 10.1080/15295192.2012.683342

Feldman, R., & Eidelman, A. I. (2007). Maternal postpartum behavior and the emergence of infant-mother and infant-father synchrony in preterm and full-term infants: the role of neonatal vagal tone. *Developmental Psychobiology, 49*(3), 290-302. doi: 10.1002/dev.20220

Feldman, R., Granat, A., Pariente, A., Kanety, H., Kuint, J., & Gilboa-Schechtman, E. (2009). Maternal depression and anxiety across the postpartum year and infant social engagement, fear regulation, and stress reactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*(9), 919-927. doi:10.1097/CHI.0b013e3181b21651

Feldman, R., Magori-Cohen, R., Galili, G., Singer, M., & Louzoun, Y. (2011). Mother and infant coordinate heart rhythms through episodes of interaction synchrony. *Infant Behavior & Development, 34*(4), 569-577. doi: 10.1016/j.infbeh.2011.06.008

Field, A. P., & Lester, K. L. (2010). Learning of information processing iases in anxious children and adolescents. In J. A. Hadwin & A. P. Field (Eds.), *Information processing biases and anxiety: A developmental perspective* (pp. 253-278). Chichester: John Wiley & Sons.

Field, T. M., Hossain, Z., & Malphurs, J. (1999). "Depressed" fathers' interactions with their infants. *Infant Mental Health Journal, 20*(3), 322-332.

Fox, N. A. (1989). Psychophysiological correlates of emotional reactivity during the first year of life. *Developmental Psychology, 25*(3), 364-372. doi: 10.1037/0012-1649.25.3.364

Fox, N. A., & Calkins, S. D. (2003). The development of self-control of emotion: Intrinsic and extrinsic influences. *Motivation and Emotion, 27*(1), 7-26.

Fracasso, M. P., Porges, S. W., Lamb, M. E., & Rosenberg, A. A. (1994). Cardiac activity in infancy: Reliability and stability of individual differences. *Infant Behavior & Development, 17*(3), 277-284. doi: 10.1016/0163-6383(94)90006-X

Friedman, B. (2006). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology, 74*, 185-199. doi:10.1016/j.biopsycho.2005.08.009

Gaynes BN, Gavin N, Meltzer-Brody S, et al. Perinatal Depression: Prevalence, Screening Accuracy, and Screening Outcomes: Summary. 2005 Feb. In: AHRQ Evidence Report Summaries. Rockville (MD): Agency for Healthcare Research and Quality (US); 1998-2005. 119. Available from: https://www.ncbi.nlm.nih.gov/books/NBK11838/

Goodman, J. H. (2008). Influences of maternal postpartum depression on fathers and on father-infant interaction. *Infant Mental Health Journal, 29*(6), 624-643. http://dx.doi.org /10.1002/imhj.20199

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106*(3), 458-490.

Goodman, S. H., Rouse, M. H., Connell, A. M., Robbins Broth, M., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical*

*Child and Family Psychology Review, 14*, 1-27. http://dx.doi.org/10.1007/s10567-010-0080-1

Gorka S. M., Nelson, B. D., Sarapas, C., Campbell, M., Lewis, G. F. … & Shankman, S. A. (2013). Relation between respiratory sinus arrhythmia and startle response during predictable and unpredictable threat. *Journal of Psychophysiology, 27*(2), 95-104. doi:10.1027/0269-8803/a000091

Gotlib, I. H., Joormann, J., & Foland-Ross, L. C. (2014). Understanding familial risk for depression: A 25-year perspective. *Perspectives on Psychological Science, 9*(1), 94-108. http://dx.doi.org/10.1177/1745691613513469

Gouin, J. P., Deschenes, S. S., & Dugas, M. J. (2014). Respiratory sinus arrhythmia during worry forecasts stress-related increases in psychological distress. *Stress, 17*(5), 416-422. doi:10.3109/10253890.2014.949666

Gray, S. O., Lipschutz, R. S., & Scheeringa, M. S. (2017). Young children's physiological reactivity during memory recall: Associations with posttraumatic stress and parent physiological synchrony. *Journal of Abnormal Child Psychology, 46*(4), 871-880. doi:10.1007/s10802-017-0326-1

Graziano, P. A., & Derefinko, K. (2013). Cardiac vagal control and children's adaptive functioning: A meta-analysis. *Biological Psychology, 94*(1), 22-37. doi: http://dx.doi.org/10.1016/j.biopsycho.2013.04.011

Graziano, P. A., Keane, S. P., & Calkins, S. D. (2007). Cardiac Vagal Regulation and Early Peer Status. *Child Development, 78*(1), 264-278. doi: 10.1111/j.1467-8624.2007.00996.x

Greaves-Lord, K., Ferdinand, R. F., Sondeijker, F., Dietrich, A. Oldehinkel, A. J. … & Verhulst, F. C. (2007). Testing the tripartite model in young adolescents: Is hyperarousal specific

for anxiety and not depression? *Journal of Affective Disorders, 102*, 55-63. doi:10.1016/j.jad.12.009

Grissom, N., & Bhatnagar, S. (2009). Habituation to repeated stress: Get used to it. Neurobiology of Learning and Memory, 92(2), 215-224. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1016/j.nlm.2008.07.001

Hammen, C. (2009). Children of depressed parents. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 275-297). New York, NY: Guilford Press.

Han, Z. R., Lei, X., Qian, J., Li, P., Wang, H., & Zhang, X. (2016). Parent and child psychopathological symptoms: The mediating role of parental emotion dysregulation. *Child and Adolescent Mental Health, 21*(3), 161-168. doi:10.1111/camh.12169

Hernandez, E., Smith, C. L., Day, K. L., Neal, A., & Dunsmore, J. C. (2018). Patterns of parental emotion-related discourse and links with children's problem behaviors: A person-centered approach. Developmental Psychology, 54(11), 2077-2089. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1037/dev0000602

Hoffman, C., Crnic, K. A., & Baker, J. K. (2006). Maternal depression and parenting: Implications for children's emergent emotion regulation and behavioral functioning. *Parenting: Science and Practice, 6*(4), 271-295.

Holmes, T. H., & Rahe, R. H. (1967). The Social Readjustment Rating Scale. *Journal of Psychosomatic Research, 11*(2), 213-218. http://dx.doi.org/10.1016/0022-3999(67)90010-4

Hu, M. X., Larners, F., de Geus, E. J. C., & Penninx, B. W. J. H. (2016). Differential autonomic nervous system reactivity in depression and anxiety during stress depending on type of stressor. *Psychosomatic Medicine, 78*, 562-572. doi:10.1097/PSY.0000000000000313

Huffman, L. C., Bryan, Y. E., del Carmen, R., Pedersen, F. A., Doussard-Roosevelt, J. A., & Porges, S. W. (1998). Infant temperament and cardiac vagal tone: Assessments at twelve weeks of age. *Child Development, 69*(3), 624-635. doi: 10.2307/1132194

Joormann, J., Eugène, F., & Gotlib, I. H. (2008). Parental depression: Impact on offspring and mechanisms underlying transmission of risk. In S. Nolen-Hoeksema (Ed.), *Handbook of adolescent depression* (pp. 441-471). New York, NY: Guilford Press.

Kerns, C. E., Pincus, D. B., McLaughlin, K. A., & Comer, J. S. (2017). Maternal emotion regulation during child distress, child anxiety accommodation, and links between maternal and child anxiety. *Journal of Anxiety Disorders, 50*, 52-59. doi:10.1016/j.janxdis.2017.05.002

Kircanski, K., Waugh, C. E., Camacho, M. C., & Gotlib, I. H. (2016). Aberrant parasympathetic stress responsivity in pure and co-occurring major depressive disorder and generalized anxiety disorder. *Journal of Psychopathology and Behavioral Assessment, 38*(1), 5-19. doi:10.1007/s10862-015-9493-y

Lamers, F., van Oppen, P., Comijs, H. C., Smit, J. H., Spinhoven, P., van Balkom, Anton J. L. M., . . . Penninx, B. W. J. H. (2011). Comorbidity patterns of anxiety and depressive disorders in a large cohort study: The netherlands study of depression and anxiety (NESDA). *The Journal of Clinical Psychiatry, 72*(3), 341-348. doi:10.4088/JCP.10m06176blu

Laursen, B., & Hoff, E. (2006). Person-centered and variable-centered approaches to longitudinal data. Merrill-Palmer Quarterly, 52(3), 377-389. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1353/mpq.2006.0029

Lazarsfeld, P. F., & Henry, N. W. (1968). *Latent structure analysis.* Boston, MA: Houghton Mifflin.

Licht, C. M. M., de Geus, E. J. C., van Dyck, R., & Penninx, B. W. J. H. (2009). Association between anxiety disorders and heart rate variability in the Netherlands Study of Depression and Anxiety (NESDA). *Psychosomatic Medicine, 71,* 508-518. doi:10.1097/PSY.0b013e3181a292a6

Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review, 20*(5), 561-592.

Lunkenheimer, E. S., Kemp, C. J., Lucas-Thompson, R. G., Cole, P. M., & Albrecht, E. C. (2016). Assessing Biobehavioural Self-Regulation and Coregulation in Early Childhood: The Parent-Child Challenge Task. *Infant and Child Development.* doi:10.1002/icd.1965

Lunkenheimer, E. S., Tiberio, S. S., Buss, K. A., Lucas-Thompson, R. G., Boker, S. M., & Timpe, Z. C. (2015). Coregulation of respiratory sinus arrhythmia between parents and preschoolers: Differences by children's externalizing problems. *Developmental Psychobiology, 57*(8), 994-1003. doi:10.1002/dev.21323

Lunkenheimer, E., Tiberio, S. S., Skoranski, A. M., Buss, K. A., & Cole, P. M. (2018). Parent-child coregulation of parasympathetic processes varies by social context and risk for psychopathology. *Psychophysiology, 55*(2), 1-16. doi:10.1111/psyp.12985

Malmberg, L., & Flouri, E. (2011). The comparison and interdependence of maternal and paternal influences on young children's behavior and resilience. *Journal of Clinical Child and Adolescent Psychology, 40*(3), 434-444. http://dx.doi.org/10.1080/15374416.2011.563469

Maxwell, S. E., & Delaney, H. D. (1990). *Designing experiments and analyzing data: A model comparison perspective.* Belmont, CA, US: Wadsworth/Thomson Learning.

McEwen, B. S. (2000). Allostasis and allostatic load: Implications for neuropsychopharmacology. Neuropsychopharmacology, 22(2), 108-124. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1016/S0893-133X(99)00129-3

McLean, C. P., Asnaani, A., Litz, B. T., & Hofmann, S. G. (2011). Gender differences in anxiety disorders: Prevalence, course of illness, comorbidity and burden of illness. Journal of Psychiatric Research, 45(8), 1027-1035. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1016/j.jpsychires.2011.03.006

Mezulis, A. H., Hyde, J. S., & Clark, R. (2004). Father involvement moderates the effect of maternal depression during a child's infancy on child behavior problems in kindergarten. *Journal of Family Psychology, 18*(4), 575-588. doi:10.1037/0893-3200.18.4.575

Miller, J. G., Chocol, C., Nuselovici, J. N., Utendale, W. T., Simard, M., & Hastings, P. D. (2013). Children's dynamic RSA change during anger and its relations with parenting, temperament, and control of aggression. *Biological Psychology, 92*(2), 417-425. doi:10.1016/j.biopsycho.2012.12.005

Mineka, S., Watson, D., & Clark, L. A. (1998). Comorbidity of anxiety and unipolar mood disorders. *Annual Review of Psychology, 49*, 377-412. doi:10.1146/annurev.psych.49.1.377

Moore, G. A. & Calkins, S. D. (2004). Infants' vagal regulation in the still-face paradigm is related to dyadic coordination of mother-infant interaction. *Developmental Psychology, 40*(6), 1068-1080. doi:10.1037/0012-1649.40.6.1068

Moore, G. A., Hill-Soderlund, A. L., Propper, C. B., Calkins, S. D., Mills-Koonce, W. R., & Cox, M. J. (2009). Mother—infant vagal regulation in the face-to-face still-face paradigm is moderated by maternal sensitivity. *Child Development, 80*(1), 209-223. doi:10.1111/j.1467-8624.2008.01255.x

Moser, M., Lehofer, M., Hoehn-Saric, R., McLeod, D. R., Hildebrandt, G. … & Zapotoczky, G. (1998). Increased heart rate in depressed subjects in spite of unchanged autonomic balance? *Journal of Affective Disorders, 48*, 115-124.

Muthén, L.K. and Muthén, B.O. (1998-2012). *Mplus user's guide.* Seventh Edition. Los Angeles, CA: Muthén & Muthén

Murray, L., Woolgar, M., Cooper, P., & Hipwell, A. (2001). Cognitive vulnerability to depression in 5-year-old children of depressed mothers. *Journal of Child Psychology and Psychiatry, 42*(7), 891-899.

National Institute of Mental Health. (2017, November). *Statistics: Any anxiety disorder.* Retrieved from https://www.nimh.nih.gov/health/statistics/any-anxiety-disorder.shtml.

Substance Abuse and Mental Health Services Administration (2017). *Results from the 2017 National Survey on Drug Use and Health: Detailed tables.* Retrieved from https://www.samhsa.gov/data/sites/default/files/cbhsq-reports/NSDUHDetailedTabs2017/NSDUHDetailedTabs2017.htm#tab8-56A.

Olson, S. L., & Lunkenheimer, E. S. (2009). Expanding concepts of self-regulation to social relationships: Transactional processes in the development of early behavioral adjustment. In A. Sameroff & A. Sameroff (Eds.), *The transactional model of development: How children and contexts shape each other.* (pp. 55-76). Washington, DC, US: American Psychological Association.

Patterson, G. R. (2002). The early development of coercive family processes. In J. B. Reid, G. R. Patterson, & J. Snyder (Eds.), *Antisocial behavior in children and adolescents: A developmental analysis and model for intervention* (pp. 25-44). Washington, DC: American Psychological Association.

doi:10.1016/j.brat.2017.05.017

Porges, S. W. (1991). Vagal tone: An autonomic mediator of affect. In J. Garber, K. A. Dodge, J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation.* (pp. 111-128). New York, NY, US: Cambridge University Press.

Porges, S. W. (2003). The Polyvagal Theory: Phylogenetic contributions to social behavior. *Physiology & Behavior, 79*(3), 503-513. doi: 10.1016/S0031-9384(03)00156-2

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology, 74*(2), 116-143. doi: 10.1016/j.biopsycho.2006.06.009

Porges, S. W., Doussard-Roosevelt, J. A., Portales, A. L., & Greenspan, S. I. (1996). Infant regulation of the vagal 'brake' predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology, 29*(8), 697-712. doi: 10.1002/(SICI)1098-2302(199612)29:8<697::AID-DEV5>3.0.CO;2-O

Psychogiou, L., & Parry, E. (2014). Why do depressed individuals have difficulties in their parenting role? *Psychological Medicine, 44,* 1345-1347. http://dx.doi.org/10.1017/S0033291713001931

Reinfjell, T., Karstad, S. B., Berg-Nielson, T. S., Luby, J. L., & Wichstrom, L. (2016). Predictors of change in depressive symptoms from preschool to first grade. *Development and Psychopathology, 28,* 1517-1530. doi:10.1017/S0954579415001170

Root, A. E., Hastings, P. D., & Rubin, K. H. (2016). The parenting behaviors of shy–anxious mothers: The moderating role of vagal tone. *Journal of Child and Family Studies, 25*(4), 1325-1333. doi:10.1007/s10826-015-0296-2

Rottenberg, J. (2007). Cardiac vagal control in depression: A critical analysis. *Biological Psychology, 74*(2), 200-211. doi:10.1016/j.biopsycho.2005.08.010

Rottenberg, J., Clift, A., Boden, S., & Salomon, K. (2007). RSA fluctuation in major depressive disorder. *Psychophysiology, 44,* 450-458. doi:10.1111/j.1469-8986.2007.00509.x

Rottenberg, J., Wilhelm, F. H., Gross, J. J., & Gotlib, I. H. (2002). Respiratory sinus arrhythmia as a predictor of outcome in major depressive disorder. *Journal of Affective Disorders, 71*, 265-272.

Shields, A., & Cicchetti, D. (1997). Emotion regulation among school-age children: The development and validation of a new criterion Q-sort scale. *Developmental Psychology, 33*(6), 906-916.

Simonds, V. M., & Whiffen, V. E. (2003). Are gender differences in depression explained by gender differences in co-morbid anxiety? Journal of Affective Disorders, 77(3), 197-202. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1016/S0165-0327(02)00113-1

Skoranski, A., Lunkenheimer, E., & Lucas-Thompson Graham, R. (2017). The effects of maternal respiratory sinus arrhythmia and behavioral engagement on mother-child physiological coregulation. *Developmental Psychobiology, 59*(7), 888-898.

Smith, J. D., Woodhouse, S. S., Clark, C. A. C., & Skowron, E. A. (2016). Attachment status and mother-preschooler parasympathetic response to the strange situation procedure. *Biological Psychology, 114*, 39-48. doi:10.1016/j.biopsycho.2015.12.008

Snyder, R., Shapiro, S., & Treleavan, D. (2012). Attachment theory and mindfulness. Journal of *Child and Family Studies, 21,* 709-717. doi:10.1007/s10826-011-9522-8

Suveg, C., Shaffer, A., & Davis, M. (2016). Family stress moderates relations between physiological and behavioral synchrony and child self-regulation in mother-preschooler dyads. *Developmental Psychobiology, 83-97*. doi:10.1002/dev21358

Suveg, C., & Zeman, J. (2004). Emotion regulation in children with anxiety disorders. Journal of *Clinical Child and Adolescent Psychology, 33*(4), 750-759. doi:10.1207/s15374424jccp3304_10

Sweeney, S., & MacBeth, A. (2016). The effects of paternal depression on child and adolescent outcomes: A systematic review. *Journal of Affective Disorders, 205,* 44-59. http://dx.doi.org/10.1016/j.jad.2016.05.073

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1995). Autonomic characteristics of generalized anxiety disorder. *Biological Psychiatry, 39,* 255-266.

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

Thayer, J. F. & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience and Biobehavioral Reviews, 33,* 81-88. doi:10.1016/j.neubiorev.2008.08.004

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*(2-3), 25-52, 250-283. doi:10.2307/1166137

Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist, 44*(2), 112-119. doi:10.1037/0003-066X.44.2.112

Tronick, E. & Beeghly, M. (2011). Infants' meaning-making and the development of mental health problems. *American Psychologist, 66*(2), 107-119. doi:10.1037/a0021631

Turner, S. M., Beidel, D. C., & Epstein, L. H. (1991). Vulnerability and risk for anxiety disorders. *Journal of Anxiety Disorders, 5*, 151-166. doi:10.1016/0887-6185(91)90026-P

Turpyn, C. C., Chaplin, T. M., Cook, E. C., & Martelli, A. M. (2015). A person-centered approach to adolescent emotion regulation: Associations with psychopathology and parenting. Journal of Experimental Child Psychology, 136, 1-16. doi:http://dx.doi.org.ezaccess.libraries.psu.edu/10.1016/j.jecp.2015.02.009

Verstraeten, K., Vasey, M. W., Raes, F., & Bijttebier, P. (2009). Temperament and risk for depressive symptoms in adolescence: Mediation by rumination and moderation by effortful control. *Journal of Abnormal Child Psychology, 37*(3), 349-361. doi:10.1007/s10802-008-9293-x

Watkins, L. L., Grossman, P., Krishnan, R., & Sherwood, A. (1998). Anxiety and vagal control of heart rate. *Psychosomatic Medicine, 60*, 498-502.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3-14.

Wilson, S., & Durbin, C. E. (2010). Effects of paternal depression on fathers' parenting behaviors: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 167-180. http://dx.doi.org/10.1016/j.cpr.2009.10.007

Woodruff-Borden, J., Morrow, C., Bourland, S., & Cambron, S. (2002). The behavior of anxious parents: Examining mechanisms of transmission of anxiety from parent to child. *Journal*

*of Clinical Child and Adolescent Psychology*, *31*(3), 364-374. doi:10.1207/153744202760082621

Woody, M. L., Feurer, C., Sosoo, E. E., Hastings, P. D., & Gibb, B. E. (2016). Synchrony of physiological activity during mother-child interaction: Moderation by maternal history of major depressive disorder. *Journal of Child Psychology and Psychiatry*, doi:10.1111/jcpp.12562

Zahn-Waxler, C., Iannotti, R. J., Cummings, E. M., & Denham, S. (1990). Antecedents of problem behaviors in children of depressed mothers. *Development and Psychopathology*, *2*(3), 271-291. http://dx.doi.org/10.1017/S0954579400000778

# Amanda M. Skoranski Vita

Department of Psychology, The Pennsylvania State University, University Park, PA 16801
302-668-6355 | axs1238@psu.edu

## Education

Ph.D. | Psychology | 2019
The Pennsylvania State University

M.S. | Human Development and Family Studies | 2017
Colorado State University

B.S | Psychology, with distinction | 2009
University of Delaware

## Selected Publications

1. **Skoranski, A.,** Coatsworth, J.D., & Lunkenheimer, E. (2019). A dynamic systems approach to understanding interpersonal mindfulness. *Journal of Child and Family Studies, 28*(10), 2659-2672.
2. **Skoranski, A.,** Lunkenheimer, E., & Lucas-Thompson Graham, R. (2017). The effects of maternal respiratory sinus arrhythmia and behavioral engagement on mother-child physiological coregulation. *Developmental Psychobiology, 59*(7), 888-898.
3. **Skoranski, A.,** Kelly, N.R., Radin, R.M., Thompson, K.A., Galescu, O., Demidowich, A.P., Brady, S.M., Chen, K.Y., … & Shomaker, L.B. (2018). Relationship of dispositional mindfulness to subjective distress and cortisol stress response in adolescent girls at risk for type 2 diabetes. *Journal of Child and Family Studies, 27*(7), 2254-2264.
4. Lunkenheimer, E., Tiberio, S., **Skoranski, A.,** Buss, K., & Cole, P. (2018). Parent-child coregulation of parasympathetic processes varies by social context and risk for psychopathology. *Psychophysiology, 55*(2), 1-16.

## Teaching Experience

Instructor | Fall 2019
PSYCH 462: Physiological Psychology
Pennsylvania State University

Co-Instructor | Fall 2017
HDFS 470: Mentoring At-Risk Youth
Colorado State University

Co-Instructor | Spring 2016
HDFS 470: Mentoring At-Risk Youth
Colorado State University