Change of Heart:
Parenting Stress and Distress, and Associations with Heart Rate Variability

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ABSTRACT

Change of heart: Parenting stress and distress, and associations with heart rate variability

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Challenging parent-child interactions can be an important source of stress and distress for parents. Family stress models highlight that there is substantial individual variability in adjustment to parenting stressors. The current dissertation sought to examine whether parent self-regulation, as indexed by heart rate variability (HRV), is an inter-individual factor that predicts variability in the association between parenting stressors and parent mood disturbances. The first study tested whether there was an association between parenting stressors and parent's self-regulatory capacities (i.e., parent HRV), and whether the marital context within which these stressful parent-child interactions occurred moderated this effect. Findings from this study, derived from a sample of 80 cohabiting heterosexual couples with preschool children, suggested that parenting stressors are associated with reduced parent self-regulation capacities and that fathers are especially vulnerable to the marital context within which this occurs. In the second study, using a daily diary design and the same sample as in the first study, we tested whether parent HRV moderated the association between parenting stressors and mood disturbances. Between- and within-person analyses indicated that the strength of the positive association between daily parenting stress and negative mood increased with decreasing HRV, suggesting that depleted parent self-regulatory capacities may index vulnerability to stress-related disturbances in negative mood. In the third and final study, sleep reactivity was identified as a potential pathway through which lower HRV confers greater risk for stress-related mood disturbances. The results from the moderated mediation model with 125 mothers of adolescents with developmental disorders and 97 mothers of typically developing adolescents, suggested that lower HRV is a potential biomarker of increased sleep reactivity which in turn increases the risk for elevated parent depressive symptoms associated with parenting stress. Taken together, these studies suggest that parent self-regulation capacities, as indexed by HRV, is a resource that may help identify which parents adapt, and which parents have difficulty adapting to, parenting stressors.

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Dedication

This dissertation is dedicated to my *vavó*, Maria do Céu "Tété" Benevides.

Thank you for teaching me the power of perseverance & the true meaning of unconditional love.

Contribution of Authors

This thesis consists of three manuscripts. The relative contributions of my colleagues to each manuscript is outlined below:

Study 1: da Estrela, C., MacNeil, S., Caldwell, W., & Gouin, J.P. (Under review). Child and marital stress are associated with a psychophysiological index of parent self-regulatory capacities. *Family Relations.*

JPG designed the study. SM and WC prepared the RSA data for analysis. JPG and CdE analyzed and interpreted the data. CdE wrote the first draft of the manuscript. All authors edited subsequent versions of the manuscript and approved the final manuscript.

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JPG designed the study. JPG and CdE analyzed and interpreted the data. CdE wrote the first draft of the manuscript. All authors edited subsequent versions of the manuscript and approved the final manuscript.

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JPG, JM, and LB designed the study. JPG and CdE analyzed and interpreted the data. CdE wrote the first draft of the manuscript. All authors edited subsequent versions of the manuscript and approved the final manuscript.

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Chapter 1: General Introduction

Parental depression is a robust predictor of a range of poor child outcomes (Goodman et al., 2011). Yet, transactional models of development highlight that children are not passive recipients of their environments. Instead, they impact and shape their environments, including the family system, in long-term and meaningful ways (Sameroff, 1975). Indeed, children can actually be an important source of stress and distress for parents. Child-related stressors, including challenging child behaviours that are disruptive and inappropriate given the child's age, developmental status and psychosocial context, share a bidirectional association with parental depression. Specifically, while parental depression predicts frequency of child-related stressors, there is also a substantial body of empirical evidence supporting child-driven pathways. That is, child-related stressors also predict increased parental depression over time (Bagner, Pettit, Lewinsohn, Seeley, & Jaccard, 2013; Ciciolla, Gerstein, & Crnic, 2014; Gross, Shaw, Burwell, & Nagin, 2009). However, not all parents will respond to child-related stressors in the same way. Some adapt, while others have difficulty adapting to, these parenting challenges.

The overarching goal of this dissertation is to investigate whether high-frequency heart rate variability (HRV), a psychophysiological index of self-regulation capacities, may help explain individual differences in parent adaptation to child-related stressors. To do so, this dissertation used data from two large studies examining the psychobiological correlates of parenting stress amongst parents of children with and without neurodevelopmental disorders. The two studies included two distinct samples of parents, each associated with key developmental transition periods: parents of preschool-aged children (i.e., as parents entered parenthood) and mothers of children who recently entered or graduated from high school (i.e., the transition in or out of adolescence). Across a series of three studies, this thesis focused on the following three research questions: (1) are child-related stressors associated with parent HRV, (2) does parent HRV moderate the association between child-related stressors and parent negative mood, and (3) does HRV moderate the daily psychological processes that mediate the association between parenting stress and parental depression? The purpose of this introductory chapter is to review the current parenting stress literature and to provide a rationale of why HRV is important to consider when examining the association between parenting stress and parent mood disturbances.

Family stress models: When does stress lead to distress?

Family stress theories provide theoretical frameworks to understand how child-related stressors may impact parental wellbeing, including the development of parental depressive symptoms. Generally, theories within the family stress literature posit that (a) stressors are normative and likely inevitable, (b) some stressors can place extraordinary demands on families, (c) most families will adapt well to stress, but (d) those who do not adapt well will experience negative outcomes (Hobfoll & Spielberger, 1992). Within these frameworks, child-related stressors are events that have the potential to challenge a family's resources and can yield important consequences on the wellbeing of members within that family.

The ABC-X model of family stress (Hill, 1949; 1958) is one of the earliest models developed to explain why some families adjust, while other families have difficulty adjusting to stressors. In this model, the family is conceptualized as a homeostatic system. Stressors are events that disrupt this equilibrium (variable A; e.g., challenging child behaviours). In addition to the way families interpret stressors (variable C), the resources available to family members both inside and outside of the nuclear family system (variable B) determines how families will respond (variable X). That is, the impact of a stressor on the individual is influenced by the resources available to them, which refers to their capacity – psychological, social, or economical – to meet the demands the stressor places on the family system. Psychological distress, including depressive symptoms, arises when there is an imbalance between the demands of a stressor and the family's capacity to meet its demands.

The Family Adjustment and Adaptation Response Model (FAAR; McCubbin & Patterson, 1983) builds upon the ABCX model by further elaborating on the processes by which individuals respond to stressors. The FAAR model posits that when families encounter a stressor, individual members will strive to maintain balance within the family system by using their resources to either adjust or adapt to the demands of the stressor. At first, individuals try to adjust to a stressor by resisting making changes and employing coping strategies to minimize the impact of stressors on themselves and the family system. However, prolonged or multiple stressors call for members to adapt to these demands, requiring them to recruit additional resources and adaptive coping strategies. Both in response to minor or prolonged stressors, the ability of family members to adjust or adapt to those demands determines the long-term wellbeing of the system and that of the individuals within the system. As in the ABCX model,

resources again appear to be a key factor in understanding who is most at risk of developing maladaptive outcomes such as depressive symptoms. In particular, this model highlights individuals' flexibility and employment of helpful coping strategies as predictors of positive outcomes.

Perry's integrative model of stress of families of children with developmental disabilities (Perry, 2004) specifically outlines how child-related characteristics can yield parent distress. The proposed model posits that both objective and subjective child characteristics (e.g., child behavioural problems, parent-child interactions perceived as stressful) create demands on parents by generating everyday frustrations and hassles that can begin to pileup. When these demands exceed parents' resources, they are at risk for poor outcomes such as the development of parental depressive symptoms. Traditionally, resources have been conceptualized as personality traits, coping behaviours, and social support that may buffer the stress-distress association. Further, the model posits that it is the inter-individual variability in these resources that helps explain the heterogeneity of negative outcomes (e.g., depressive symptoms) amongst parents of children with developmental disabilities. Although this model was designed with families of children with developmental disabilities in mind, it can easily be translated to families facing other parenting challenges.

Finally, the Family Stress Model (FSM; Masarik & Conger, 2017) draws links between family stressors and child developmental outcomes through the impact stressors can have on parental depression. Although this model was originally developed to understand how economic stressors could influence child outcomes via parent psychological distress, when applied in the context of child-related stressors it highlights the potential for bidirectional associations between child and parent functioning. Specifically, consistent with the family stress literature, the model posits that stressors place parents at risk for negative psychological outcomes including depressive symptoms. Negative psychological outcomes then increase the risk for conflict between parents and may disrupt adaptive parenting practices, both of which would place children at increased risk for adjustment problems. This model also emphasizes the importance of considering risk and protective factors that may moderate different aspects of the stress process, including which parents will experience depressive symptoms in response to stress.

Taken together, the family stress literature highlights that the association between family stressors and negative parent psychological outcomes, such as depressive symptoms, is

heterogeneous given the presence of moderating factors that may exacerbate or buffer the preceding association. Another common theme echoed throughout the different family stress models is an understanding of negative parent outcomes as the result of an imbalance between the demands created by a stressor and the resources a parent has available to address those demands. Self-regulation, the ability to adjust one's emotions, thoughts, and behaviours to achieve a goal (Baumeister, Muraven, & Tice, 2000), may be a resource that is particularly important in the context of parenting stress and depressive symptoms.

Parenting: An exercise in self-regulation

Self-regulation is an effortful and goal-directed process by which one attempts to adjust and adapt one's emotions, behaviours, and thoughts to a particular context, and is a resource that can be depleted (Baumeister et al., 2000). Parenting may be understood as an exercise in self-regulation. Parenting elicits both positive and negative emotions that place self-regulatory demands on parents to respond in ways that promote healthy child development (Bariola, Gullone, & Hughes, 2011; Dix, 1991). For example, the experience of a child throwing a temper tantrum in the grocery store requires a parent to regulate his or her own frustration to be able to effectively enact helpful parenting practices to deescalate the child's behaviour. Indeed, effective parent self-regulation has been associated with an increased use of positive parenting behaviours (Shaffer & Obradović, 2017). In a study with parents of young children, parents' own ability to inhibit unwanted behaviours and ignore distractions (i.e., inhibitory control) predicted greater sensitive and responsive parenting practices, whereas self-reported self-regulation difficulties were associated with the use of less positive and collaborative parenting behaviours during a parent-child interaction task. Additionnally, disrupted maternal self-regulation has been associated with over-reactive disciplining and harsher responses to children's negative emotions (Lorber, 2012; Mazursky-Horowitz et al., 2015).

In addition to the link between parent self-regulation and adaptive parenting behaviours, a parent's ability to self-regulate effectively may be a protective factor that buffers the association between parenting stressors and parental depression. Individuals who self-report stronger self-regulation capacities are more likely to implement adaptive coping strategies in response to laboratory-based and naturalistic stressors, can better predict the threat of a stressor and act accordingly before it occurs, and overall perceive less stress than individuals with poorer self-regulation (Aspinwall, 2004; Aspinwall & Taylor, 1997; Rosenbaum, 1989). Further,

interventions aimed at improving self-regulation have also been successful in reducing perceived stress (e.g., Gollwitzer, Mayer, Frick, & Oettingen, 2018). However, despite consistent links drawn between stress and self-regulation, and parent self-regulation and parenting behaviours, parent self-regulation has not yet been incorporated in contemporary family stress models.

The aforementioned gap in the literature is complicated by reliance on self-report measures of self-regulation. In general, using scores on one self-report measure to predict scores on another self-report measure introduces the potential for common method bias (i.e., inflated correlations resulting from completing measures using similar methodology at the same time; Podsakoff, MacKenzie, Podsakoff, & Lee, 2003). Further, the accuracy of self-report measures of self-regulation is unclear especially as a person's ability to self-regulate may influence the way they interpret and answer questions probing their use of self-regulation strategies. This is particularly problematic when trying to disentangle reactivity from regulation. Finally, self-regulation is a multifaceted construct that involves both explicit and implicit processes. While self-report measures may be helpful in capturing explicit aspects of self-regulation (aspects within the person's awareness), there is a call for objective measures of self-regulation to better comment on the implicit and underlying processes that support self-regulation (Calkins, 2010; Lewis, Zinbarg, & Durbin, 2010). As such, a psychophysiological objective measure of self-regulation such as HRV would help address the various methodological challenges inherent to self-reported measures, while also expanding current family stress models.

HRV as a biomarker of self-regulation capacities

HRV is a biomarker of self-regulation capacities (Beauchaine, 2015; Holzman & Bridgett, 2017; Smith, Deits-Lebehn, Williams, Baucom, & Uchino, 2020). HRV refers to the fluctuation of time between consecutive heartbeats. At rest, both the parasympathetic and sympathetic branches of the autonomic nervous system influence cardiac activity. The sympathetic nervous system is involved in acceleration of heart rate while its deceleration is under parasympathetic control. While both branches of the autonomic nervous system influence the low-frequency component of HRV, the high-frequency component of HRV reflects mostly the inhibitory influence of the parasympathetic system on the sinoatrial node of the heart via the vagal nerve. (Berntson et al., 1997).

The Neurovisceral Integration Model highlights that high-frequency HRV is an index of top-down inhibition from the prefrontal cortex to the heart (Thayer & Lane, 2009). Specifically,

HRV reflects a set of neural networks through which the prefrontal cortex sends inhibitory signals to the amygdala, whose inhibition maintains stronger vagal activity on the heart. According to this model, higher HRV promotes the affective, behavioural, and cognitive responses that are required for adaptive self-regulation. Processes required for effective self-regulation, such as greater attentional control, behavioural persistence, and affective and cognitive processing during low mood, have all been associated with greater HRV (Balzarotti, Biassoni, Colombo, & Ciceri, 2017; Hofmann, Schmeichel, & Baddeley, 2012; Thayer, Hansen, Saus-Rose, & Johnsen, 2009; Zahn et al., 2016). As such, HRV has been conceptualized as a proxy of general self-regulation capacities (Beauchaine, 2015; Holzman & Bridgett, 2017; Smith et al., 2020).

Accordingly, low HRV has been associated with correlates of disrupted self-regulation across the lifespan. Lower HRV has been linked to higher levels of depressive symptoms (Koch, Wilhelm, Salzmann, Rief, & Euteneuer, 2019; Schiweck, Piette, Berckmans, Claes, & Vrieze, 2018), negative affect (Sloan et al., 2017), and suicidality (Wilson et al., 2016). Individuals with lower HRV also report greater day-to-day difficulties in self-regulation and are more likely to engage in maladaptive mood repair and self-regulation strategies than individuals with higher HRV (Fabes & Eisenberg, 1997; Kovacs et al., 2016; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009; Williams et al., 2015; Yaroslavsky et al., 2016). Individuals with lower HRV also consistently show poorer emotional and physiological arousal regulation in response to stress compared to individuals with higher HRV (Gouin, Deschenes, & Dugas, 2014; Thayer & Lane, 2009; Weber et al., 2010), suggesting a trait-like vulnerability to stress.

The heart of parenting: HRV and parenting behaviours

Currently, there is preliminary support that HRV may moderate individual differences in adaptability to parenting stressors. In a study with mother-adolescent dyads, higher maternal HRV buffered the association between maternal depression and reduced emotional flexibility within dyads. That is, in the context of elevated maternal depression, mothers with higher HRV showed greater ability to adapt their emotions to contextual demands when interacting with their children as compared to mothers with lower HRV (Connell, Hughes-Scalise, Klostermann, & Azem, 2011). In a study with mothers of preschool-aged children, higher maternal HRV buffered the association between maternal anxiety and overprotective parenting behaviours (Root, Hastings, & Rubin, 2016). As such, higher parental HRV may buffer the association between

various maternal risk factors and unhelpful parenting practices while lower parental HRV may be predictive of behavioural and cognitive indices of disrupted parent self-regulation. Indeed, amongst mother-adolescent dyads, lower maternal HRV predicted greater escalation of negative parent-child interactions during mother-adolescent discussion tasks (Connell, McKillip, Patton, Klostermann, & Hughes-Scalise, 2015). In a study with mothers of preschool-aged children, mothers with lower HRV were more likely to attribute hostile intentions to child behavioural problems as compared to mothers with higher HRV (Wang, Deater-Deckard, & Bell, 2016). Overall, the preceding studies suggest that maternal self-regulation capacities, as indexed by HRV, may play an important role in understanding individual variability in parenting behaviours.

Similarly, there is evidence suggesting that higher HRV is linked to the effective deployment of adaptive parenting practices. Amongst mothers of newborns, greater maternal HRV was associated with greater maternal sensitivity such that these mothers were better able to recognize their infants' cues as compared to mothers with lower HRV (Joosen et al., 2013). In a study with fathers of young children, higher paternal HRV predicted greater validation of children's negative emotions (Blandon, 2015). From a self-regulation perspective, parents with higher HRV may be better equipped to respond and adapt to child-related demands as compared to parents with lower HRV.

Taken together, the emerging literature suggests there are indirect and direct associations between parental HRV and individual differences in parenting behaviours. However, an important gap in the literature remains in understanding how parental HRV may predict parental psychological outcomes, including the experience of depressive symptoms. That is, parental HRV has been linked to both behavioural and cognitive aspects of parenting (i.e., how parents act and think), but the role of parental HRV in understanding parents' own psychological functioning (i.e., how parents feel) has been overlooked. When considering both the HRV and stress literature, along with the HRV and parenting literature, parental HRV as an index of self-regulation capacities may be particularly important to consider in the context of how parents adapt – or have difficulty adapting to – parenting stressors.

Further, the pathways through which parents with lower HRV may be more vulnerable to experiencing stress-related mood disturbances are unclear. Prior studies indicate that individuals with lower HRV may experience increased emotional and physiological arousal in response to stress. Further, individuals with lower HRV may also have greater difficulty deploying and

benefiting from adaptive self-regulation strategies to decrease this heightened arousal, as compared to individuals with higher HRV (Ellis et al., 2016; Fabes & Eisenberg, 1997; Gouin et al., 2014; Weber et al., 2010; Yaroslavsky et al., 2016). As such, it is possible that this increased arousal may promote sleep disturbances, a key risk factor for depressive symptoms (Baglioni et al., 2010). Low HRV may be associated with greater sleep reactivity, i.e., stress-related sleep disturbances (Bonnet & Arand, 2003; Gouin et al., 2015; MacNeil et al., 2017). Specifically, the increased emotional and physiological arousal experienced in response to a stressor by individuals with lower HRV may interfere with sleep onset and maintenance (Riemann et al., 2010). Poorer sleep quality may then be a mechanism linking exposure to child-related stress with parent depressive symptoms.

Rationale and goals of the current dissertation

It is well established that there is a bidirectional association between parenting stressors and parental depression. Family stress models provide helpful theoretical frameworks for understanding how child-related stressors impact parental depression. These theories posit that stressors are events that place demands on families and that distress arises when these demands exceed the resources individuals can recruit to address them. Traditionally, these resources have been conceptualized as psychological, social, or economical characteristics; however, contemporary stress theories suggest that individual differences in psychophysiological functioning are also important to consider (Epel et al., 2018). Self-regulation may be a resource, with psychophysiological underpinnings as indexed by HRV, which could buffer the association between parenting stress and distress. As illustrated in Figure 1, parents with stronger self-regulation capacities may be better positioned to employ parenting practices that reduce the occurrence of child-related stressors and also minimize the impact of these stressors on their own psychological wellbeing. However, parent self-regulation has not been explicitly studied in the family stress process and the current data available on parent self-regulation has relied mostly on self-report data.

Using HRV as a psychophysiological index of self-regulation capacities can help address methodological challenges associated with using self-report data, while also serving to expand current family stress models to incorporate individual differences in psychophysiological functioning. Emerging evidence suggests that HRV both directly and indirectly predicts variability in parenting practices. However, the role of HRV in the context of family stress

processes, particularly in understanding the link between parenting stress and depression, remains unclear. As such the overarching goal of the dissertation is to understand whether HRV, as a marker of self-regulation capacities, can help explain which parents are at greatest risk for depressive symptoms in the context of parenting stress.

In Chapter 2 (Study 1) we sought to first establish an association between parenting stressors and parent HRV. Specifically, we tested whether increased child behavioural problems was associated with depleted parent self-regulation capacities, as indexed by lower parent HRV. We also tested whether the marital context within which these stressful parent-child interactions were occurring further moderated this association. To do so, 80 cohabitating heterosexual couples with preschool-aged children had their HRV recorded and completed a measure of child behavioural problems and marital stress. Parents of preschool-aged children were selected because the early parenting years are associated with normative increases in childcare demands and marital stress (Umberson, Pudrovska, & Reczek, 2010; Mitnick, Heyman, & Slep, 2009), providing a unique opportunity to examine the interaction between child-related and marital stressors with a parent's capacity for self-regulation. Further, by sampling both mothers and fathers, the moderating role of parent gender could also be tested.

In Chapter 3 (Study 2) we used a daily diary design to test whether parent self-regulation capacities moderated the association between daily child-related stress and parent negative mood. This design allowed us to disentangle both between-person and within-person variability in child-related stress and parent negative mood. Using an overlapping sample of participants from Study 1, Study 2 had 84 parents of preschool-aged children complete measures of child-related stress and negative mood for six consecutive days. We tested whether individual differences in HRV moderated the association between average child-related stress (i.e., between-person differences) and negative mood, as well as daily fluctuations in child-related stress (i.e., within-person differences) and negative mood. Three-way interactions with gender were also tested.

Finally, in Chapter 4 (Study 3) we used a moderated-mediation model to test whether lower HRV increased parents' vulnerability to the daily mechanisms processes that mediate the stress and depression association in the context of chronic parenting stress. Specifically, we tested whether poorer parent self-regulation capacities, as indexed by lower HRV, increased parents' vulnerability to stress-related sleep disturbances, thereby putting them at an increased risk of

experiencing depressive symptoms. In this study we used a chronic parenting stress model (Quittner, Glueckauf, & Jackson, 1990) by comparing two group of mothers: 125 mothers of adolescents with developmental disorders and 97 mothers of typically developing adolescents. In addition to completing a six-day sleep diary to assess their sleep quality, participants had their HRV recorded in the laboratory and completed a self-report measure of depressive symptoms.

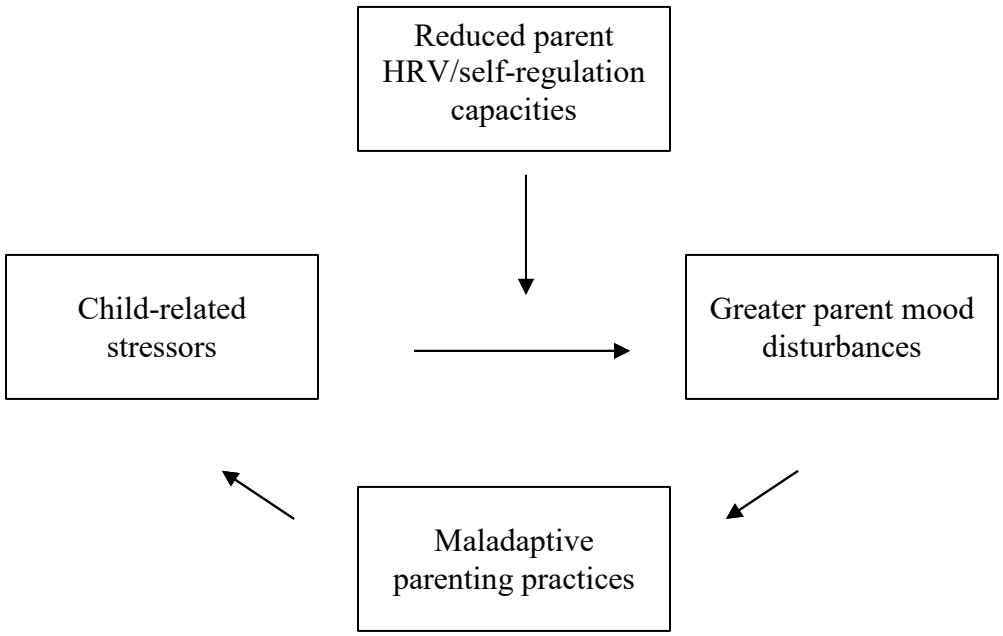

Figure 1. Proposed association between HRV, child-related stress, and parent mood disturbances. Poor self-regulation capacities are hypothesized to exacerbate the association between child-related stress and parent mood disturbances leading to the adoption of maladaptive parenting practices which may further increase the occurrence of child-related stressors.

Chapter 2: Child and marital stress are associated with a psychophysiological index of parent self-regulatory capacities

da Estrela, C., MacNeil, S., Caldwell, W., & Gouin, J.P. (Under review). Child and marital stress are associated with a psychophysiological index of parent self-regulatory capacities. *Family Relations.*

Abstract

Objective A parent's ability to self-regulate influences parenting practices. Child-related stressors may deplete parent's self-regulatory capacities. However this effect may be moderated by the marital context within which stressful parent-child interactions are occurring. The aim of the current study was to investigate the association between child behavioral problems and parent respiratory sinus arrhythmia (RSA), a psychophysiological index of self-regulatory capacities, and to test whether marital stress moderated this effect. **Methods** Eighty cohabiting heterosexual couples with preschool children had their RSA recorded during a laboratory session and completed a measure of child behavioral problems and marital stress. Partner's ratings of child behavioral problems and marital stress were used to predict participant's RSA. **Results** After adjusting for participant's age, gender, and ethnicity, greater child behavioral problems was associated with lower parent RSA. Marital stress exacerbated the association between child behavioral problems and parent RSA. This association was further moderated by parent's gender. Specifically, marital stress exacerbated the association between child behavioral problems and parent RSA for fathers, but not for mothers. **Conclusion** Child-related stressors are associated with reduced self-regulatory capacities amongst parents of preschool children as assessed by RSA. Fathers are especially vulnerable to the marital context within which these stressors are occurring. Poor self-regulation capacities during the early parenting years may place both parents and children at risk for long-term maladaptive outcomes.

Keywords*: self-regulation, child behavioral problems, marital stress, respiratory sinus arrhythmia, heart rate variability*

Self-regulation is an effortful process whereby individuals attempt to control and adapt their emotions, behaviors, and thoughts to act in ways to help them achieve their goals (Baumeister, Muraven, & Tice, 2000). Parenting is one experience which elicits many positive and negative emotions that place self-regulatory demands to respond in ways that promote healthy child development (Bariola, Gullone, & Hughes, 2011; Dix, 1991). According to the resource model of self-regulation, the ability to self-regulate is not fixed and, much like a muscle, can become fatigued over time (Baumeister et al., 2000). As such, stressful family environments that demand greater self-regulation could paradoxically deplete an individual's ability to engage in this process. The early parenting years, as a period associated with increased childcare demands and marital stress (Umberson, Pudrovska, & Reczek, 2010; Mitnick, Heyman, & Slep, 2009), provides a unique opportunity to examine how the interaction between child-related and marital stressors impact a parent's capacity for self-regulation.

Parent's self-regulatory capacity can be indexed by respiratory sinus arrhythmia (RSA). RSA is the variability in time intervals between consecutive heart beats associated with the respiration cycle (Bernston et al., 1997). At rest, cardiac activity is regulated by both branches of the autonomic nervous system; the parasympathetic system exerts a tonic inhibitory influence on the sinoatrial node of the heart via the vagal nerve, while the sympathetic systems exerts a slower excitatory influence on cardiac activity. The fast acting parasympathetic inhibitory signal is temporarily lifted during expiration contributing to the beat-to-beat variability associated with the respiration cycle. RSA is thus conceptualized as a marker of vagal-dependent parasympathetic activity.

The Neurovisceral Integration Model describes that RSA indexes a set of neural networks through which the prefrontal cortex sends inhibitory signals to the amygdala, whose inhibition maintains stronger vagal activity on the heart (Thayer & Lane, 2009). According to this model, greater top-down inhibition from the prefrontal cortex to the heart, indexed by higher RSA, promotes better affective, behavioral, and cognitive responses required for adaptive self-regulation. Indeed, higher RSA has been associated with different processes supporting effective self-regulation such as greater attentional control, behavioral persistence, as well as affective and cognitive processing during mood induction tasks (Balzarotti, Biassoni, Colombo, & Ciceri, 2017; Hofmann, Schmeichel, & Baddeley, 2012; Thayer, Hansen, Saus-Rose, & Johnsen, 2009;

Zahn et al., 2016). Resting baseline RSA can thus be conceptualized as a proxy of general self-regulation capacities.

As a psychophysiological index of self-regulation capacities in a parenting context, individual differences in RSA moderate the association between various maternal risk factors such as maternal anxiety and depression, and unhelpful parenting practices (Connell, Hughes-Scalise, Klostermann, & Azem, 2011; Root, Hastings, & Rubin, 2016). Further, lower RSA has been associated with behavioral and cognitive correlates of disrupted parent self-regulation, including greater escalation of negative parent-child interactions during mother-child discussion tasks (Connell, McKillip, Patton, Klostermann, & Hughes-Scalise, 2015), and greater parental inferences of children's hostile intentions (Wang, Deater-Deckard, & Bell, 2016). Furthermore, emerging evidence suggests that RSA is associated with a number of characteristics related to the effective deployment of adaptive parenting practices, such as greater maternal sensitivity (Joosen et al., 2013) and the validation of children's negative emotions among fathers (Blandon, 2015).

Consistent with the resource model of self-regulation, increased stress is associated with decreased RSA in the general population. Indeed, indicators of psychosocial stress predict lower RSA in healthy community samples of both pediatric and adult populations (Dishman et al., 2000; Gouin, Deschenes, & Dugas, 2014; Horsten et al., 1999; Lampert et al., 2016). However, how stressors within the context of the early family environment influence parent RSA is not well-known. Further, the majority of what is known about RSA in the context of parent-child relationships is limited to mothers. Gaining a better understanding of psychophysiological markers of self-regulatory capacities among fathers is particularly relevant to consider as previous studies have suggested that fathers' parenting practices are particularly vulnerable to the impact of stress (Belsky, Gilstrap, & Rovine, 1984; Elam, Chassin, Eisenberg, & Spinrad, 2017).

Child behavioral problems are one of the key sources of stress for parents. Specifically, child behavioral problems are a robust predictor of parent emotional distress. A number of cross-sectional and longitudinal analyses demonstrate how child behavioral problems predict parental depressive symptoms (Bagner, Pettit, Lewinsohn, Seeley, & Jaccard, 2013; Ciciolla, Gerstein, & Crnic, 2014; Gross, Shaw, Burwell, & Nagin, 2009*).* Even when considering parents of children with developmental delays or other chronic medical conditions, child behavioral problems predict parental emotional distress over and above diagnosis-specific caregiving challenges (Herring et al., 2006; Hilliard, Monaghan, Cogen, & Streisand, 2011; Quittner et al., 2010).

Therefore, child behavioral problems may place demands on parents which over time could diminish their ability to effectively self-regulate and may thus decrease parent RSA.

Further, the family context within which these child behavioral problems are occurring may also be important to consider. The ABC-X model of family stress and coping explains how some families adjust, while others fail to adjust, to stressors (Hill, 1949). Within this framework, child behavioral problems will be associated with greater maladjustment when the family system is disrupted. Indeed, stress within the couples' marital relationship often spills over into parent-child interactions which, in turn, may generate more child-related stressors (Almeida, Wethington, & Chandler, 1999; Hartley, Papp, & Bolt, 2018; Goetz, Rodriguez, & Hartley, 2019; Stroud, Meyers, Wilson, & Durbin, 2015).

This combination of child-related and marital stress can be particularly detrimental for parents and has been associated with a range of parental outcomes across pediatric populations. Both parenting stressors and marital stress have independent and mutual effects on parent's sense of competence in their parenting (Sevigny & Loutzenhiser, 2010), on parenting behaviors across childhood and into adolescence (Elam et al., 2017), and on parent's self-reported depressive symptoms (Lin et al., 2017). Further, low marital satisfaction has been shown to moderate the association between child behavioral problems and parents' social functioning and negative perceptions of parenting (Suárez & Baker, 1997). As such, the increased marital stress typical of the early parenting years may further exacerbate the deleterious impact of child-related stressors on parents' self-regulatory capacities.

Taken together, the aim of the current study is to investigate the associations between child-related and marital stress, and parent self-regulatory capacity as indexed by RSA, amongst both mothers and fathers. Couples with preschool-aged children completed measures of child behavioral problems and daily marital stress, and had their RSA assessed. It was hypothesized that child behavioral problems would be associated with poorer self-regulation capacities amongst parents as indexed by lower RSA (Hypothesis 1). Further, it was hypothesized that marital stress would moderate this association. Specifically, it was predicted that frequency of daily negative marital interactions would exacerbate the association between greater child behavioral problems and lower parent RSA (Hypothesis 2). Gender was also tested as an additional moderator. It was hypothesized the moderating effect of increased daily marital stress would be particularly pronounced for fathers as compared to mothers (Hypothesis 3).

Method

Participants

Participants were 84 cohabitating heterosexual couples with young children (*M* = 3 years, 3 months; *SD* = 23 months; *range* = 5 months to 7 years, 5 months). Most couples had either one or two children (44% and 48.8% respectively; *range* = 1 to 4) and 18 couples (21.4%) had a child with special needs (e.g., intellectual disability, autism spectrum disorder, or a chronic medical condition such as diabetes and rheumatoid arthritis). In this ethnically diverse sample, 55.36% self-identified as White, followed by 15.47% self-identifying as Latin American and 8.33% as Middle Eastern. About 36.9% had a high school degree or less, 41.7% a technical degree, and 21.5% a university degree. The average household income was 55,000 (*SD* = 8,900) CAN$. About 56.5% of the participants were employed full time. Couples had been on average 9.94 (*SD* = 4.32) years together. Four couples were dropped from analyses because of missing child behavioral problem data (final *N* = 80).

Participants were recruited from online ads, community centers, and flyers posted around a large metropolitan city. To be included in the study, participants had to be in a romantic relationship and cohabiting with their children, and all their children had to be under the age of 7 years old. Exclusion criteria included being pregnant, nursing, or having a chronic medical condition, as these factors could influence RSA (Bernston et al., 1997). Participants were compensated $40 CAD for their participation. This study was approved by the Concordia University Human Research Ethics Committee.

Procedure

Couples attended a laboratory session to measure their RSA. During the visit, a consent form was reviewed and informed consent was obtained. Partners were then seated side-by-side and were each fitted with snap electrodes in a lead II configuration for electrocardiogram (ECG) recording. The session included a five minute rest period, the completion of a child functioning questionnaire, a fourteen minute marital discussion task about challenges related to raising young children, and another five minute recovery period (Caldwell, da Estrela, MacNeil, & Gouin, 2019).

Following the laboratory session, participants completed a series of short online questionnaires to assess their typical level of marital stress throughout the week. Participants were prompted to report on their negative marital interactions four times throughout the day

(before lunch, before leaving work, after putting the children to bed, and at bedtime) for six consecutive days using online surveys. On the last survey entry of the day (i.e., bedtime), participants answered three additional questions about their general perception of negative marital interactions with their partner during that day.

Measures

Child behavioral problems. The preschool version of the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000) was used to assess child behavioral problems of the participant's most demanding child within the past two months. The CBCL is a 100-item list of behavioral problems that participants must rate on a three-point Likert scale reflecting how true a particular behavior is of their child (0 = *not true*, 2 = *very true*). A total score was computed by summing the responses on all items and T-scores were derived based on the child's gender and age group, with higher T-scores suggesting greater problem behavior. Consistent with previous studies of parenting stress, child behavioral problems were used as a proxy and objective measure of child-related stressors (e.g., Brobst et al., 2009; da Estrela, Barker, Lantagne, Gouin, 2017).

Marital stress. Daily negative marital interactions were used to assess marital stress (Laurenceau & Bolger, 2005). As described above, participants rated the extent to which their partner argued or avoided them throughout the day. At the end of the day, participants also rated the extent to which their partner behaved selfishly, ignored them, and rejected them on that day. An average of these repeated daily measures was used as a proxy of the general levels of marital stress experienced by each participant (Bolger, Davis, & Rafaeli, 2003). The Cronbach's alpha for this composite index was .89.

RSA. An ECG recording was used to measure RSA as part of a 60-minute protocol. The ECG data was collected by an ECG amplifier module via an 8-slot BioNex chassis from MindWare and analyzed using MindWare HRV Analysis Software, Version 3.1 (MindWare Technologies Ltd, Gahanna, OH). Artifacts were detected using an automated algorithm and visual inspection. Less than 1% of data for each participant had to be corrected. RSA was extracted using spectral analysis with a Fast Fourier Transformation to compute the natural log of the .15-.40 Hz frequency band of each 30-s epoch to isolate vagal-dependent parasympathetic influences on the heart. RSA was calculated for each task by averaging the RSA value for each

30-s epoch across the task-period (Berntson et al., 1997). The mean of all task-level RSA values were used in analysis.

Analytic Plan

To account for the dyadic and dependent nature of the couples' data in the current study, hierarchical linear modeling was used to test the main study hypotheses. Within this framework individuals are considered level 1 variables nested within dyads at level 2 (Kenny, Kashy, & Cook, 2006). In order to minimize the possibility that the participant's own RSA might influence appraisals of child and marital stress, partner's ratings of both child behavioral problems and daily marital stress were used to predict each participant RSA. To test Hypothesis 1, a main effect model was tested with partner's ratings of child behavioral problems and of marital stress to predict each participant RSA. An interaction term was computed between child behavioral problems and marital stress and added to the model to test Hypothesis 2. Finally, a three-way interaction term was computed between child behavioral problems, marital stress, and gender to test Hypothesis 3. Participant's age, gender (coded as -1 for men and +1 for women), and ethnicity (coded as -1 for participants who self-identified as Non-White and +1 for White) were included as covariates in all of the models as these variables can influence RSA (Sloan et al. 2008). Child's age and gender were not entered in the model as they were already accounted for in the calculation of the CBCL T-scores. An alpha level of .05 was used for the present study. Analyses were conducted in SPSS Version 25.

Results

Descriptive statistics and bivariate correlations

On average, participants reported non-clinical levels of child behavioral problems (M = 47.39, SD = 27.80). Pearson correlations revealed that child behavioral problems were negatively correlated with RSA such that greater frequency of child-related stressors reported was associated with lower parent RSA (r = -.19, p = .014). There were no statistically significantly correlations between marital stress and child behavioral problems (r = .005, p = .953), or RSA (r = -.09, p = .232). Intra-class correlations (ICC) for each study variable were computed to assess the magnitude of the within-couple correlations. The ICC for child behavioral problems (r = .71, p < .01) suggests high agreement within couples, but moderate within couple agreement was observed for marital stress (r = .47, p < .01), and the within-couple correlation for RSA was not statistically significant (r = -.002, p = .983).

Summary statistics

Model 1 tested the main effects of child and marital stress on RSA. As shown in Table 1, greater child behavioral problems significantly predicted lower RSA. However, marital stress was not a statistically significant predictor of RSA. This model accounted for 21.42% of the variance in RSA.

To investigate whether marital stress moderated the association between child behavioral problems and RSA, an interaction term between CBCL scores and marital stress was added to the model. As reported in Table 1, the interaction term was statistically significant. Simple slopes analysis revealed that at 1SD above the mean of marital stress, there was a significant negative association between child behavioral problems and RSA, $t(153) = -3.25$, $p = .001$. In contrast, the conditional effect of child behavioral problems on RSA was no longer statistically significant at 1SD below the mean of marital stress, $t(153) = -.09$, $p = .926$. Model 2 accounted for 24.21% of the variance in RSA.

Model 3 included a three-way interaction testing whether parent's gender moderated the interaction between child and marital stress in predicting RSA. As shown in Table 1, this interaction term was statistically significant. As depicted in Figure 1, marital stress exacerbated the association between child behavioral problems and RSA for fathers, $t(74) = -3.04$, $p = .003$, but not for mothers, $t(74) = -.72$, $p = .477$.

Discussion

The purpose of the current study was to examine the interplay between child-related and marital stress with a psychophysiological index of parent self-regulation capacity during the early parenting years. Findings from the current investigation illustrated that child behavioral problems was associated with lower parent RSA. Further, this association was exacerbated by marital stress for fathers, but not for mothers. These findings suggest that child-related stressors are associated with depleted self-regulatory capacities amongst parents of preschool children and that fathers are especially vulnerable to the marital context within which these stressors are occurring.

Consistent with the resource model of self-regulation, findings from the current study suggest that repeated exposure to challenging child behaviors may deplete parents' self-regulatory capacities as indexed by lower RSA. That is, self-regulation is an effortful process involving the deployment of limited resources, and the demands associated with challenging

child behaviors may lead to a reduced capacity to sustain such effortful processes to be able to optimally cope with these parenting challenges over time (Baumeister et al., 2000). In particular, challenging child characteristics have been associated with self-reported parent fatigue (Loutzenhiser, McAuslan, & Sharpe, 2015) and may contribute to parental burnout (i.e., exhaustion, depersonalization, and inefficacy associated with parenting demands; Roskam, Raes, & Mikolajczak, 2017). Furthermore, prior studies in the general population indicate that psychosocial stress exposure is associated with reduced RSA (Dishman et al., 2000; Gouin et al., 2014; Horsten et al., 1999; Lampert et al., 2015; Michels et al., 2015). The present findings thus extend prior work by highlighting how child behavioral problems, a key source of parenting stress, is associated with a psychophysiological marker of self-regulation capacities among parents of young children.

Low RSA, as an index of depleted self-regulatory capacity, may increase parents' vulnerability to emotional distress. Indeed, low RSA is associated with increased negative emotional arousal, greater depressive symptoms and fatigue, and the use of less adaptive coping strategies in the context of stress (Fabes & Eisenberg, 1997; Fagundes et al., 2011; Gouin et al., 2014; Switzer, Caldwell, da Estrela, Barker, & Gouin, 2018). When considering the parent-child relationship, parent RSA has been linked with a wide range of parenting practices (Blandon, 2015; Connell et al., 2015; Joosen et al., 2013; Wang et al., 2016), and buffers the risk of maternal risk factors on parenting behaviors (Connell et al., 2011; Root et al., 2016). However, how RSA may moderate the association between parenting stressors and parenting distress is not well-understood. Future research investigating the moderating role of RSA on the association among child-related stressors, parent emotional distress and parenting behaviors are still warranted to test this hypothesis.

Marital stress moderated the association between child behavioral problems and parent RSA. Specifically, individuals exposed to heightened levels of child-related stressors and marital stress had the lowest RSA. This finding is in line with the broader family stress and coping literature which emphasizes that the association between stressors and (mal)adjustment is moderated by the context within which these stressors are occurring (Hill, 1949). Marital stress may exacerbate the impact of parenting challenges through stress spillover effects. Specifically, child-related and marital stress tends to co-occur on the same day and stressful partner interactions may impact parent-child interactions to further generate more child-related stressors

(Almeida et al. 1999; Hartley et al., 2018; Goetz et al., 2019; Stroud et al., 2015). Further, in addition to acting as a stressor in and of itself, marital stress may also reduce parents' ability to use each other or the relationship as a source of social support when confronted with parenting stressors (Kiecolt-Glaser & Wilson, 2017).

Prior studies suggest that this effect may be particularly pronounced for men (Nelson, O'Brien, Blankson, Calkins, & Keane, 2009) and that marital stress may disproportionately affect parenting practices amongst fathers compared to mothers (Elam et al., 2017). The present study extends these findings by showing that marital stress exacerbated the association between child behavioral problems and RSA amongst new fathers but not new mothers. According to the fathering-vulnerability hypothesis, the father-child relationship may be more sensitive to external stressors such as marital conflict because fathers' roles are traditionally less defined (Belsky et al., 1984). Further, this effect may be especially salient in early childhood when gender roles in parenting may be particularly distinct. However, this effect is not always replicated (Ponnet et al., 2013). Furthermore, the literature on marital functioning and individual wellbeing highlight gender as an important moderator, with men typically showing greater health benefits from marriage than women (Kaplan & Kronick, 2006; Kiecolt-Glaser & Newton, 2017). One explanation for why the consequences of marital stress may be more pronounced for husbands as compared to wives is that women typically have other sources of informal social support that they can draw on as resources (Umberson, Chen, House, Hopkins, & Slaten, 1996).

Examining the association between challenging child behavioral problems and both mothers and fathers' biomarker of self-regulatory capacities is one of the strengths of the current study. In the early parent-child literature, the focus is typically on mothers with fathers' perspectives being largely ignored. However, as demonstrated in the current study, fathers are also impacted by family functioning in different but important ways. Further, using multiple assessment methods adds to the current body of literature on parenting stress and self-regulation over and above studies exclusively relying on self-report measures which are subject to common method bias, i.e., inflated correlations because of shared variance due to using similar assessment methods.

Despite the aforementioned strengths, the cross-sectional design of the current study limits the inferences that can be made. Specifically, the directionality of the results cannot be confirmed. That is, it is possible that parents with lower RSA elicit greater child-related and

marital stressors. The design of the current study sought to address this limitation by using partner's scores and by measuring marital stress multiple times. Still, longitudinal studies wherein family functioning is examined prospectively are warranted, especially as changes in RSA are likely the result of chronic stress over time. Further, the current study did not assess self-regulation per se but instead used RSA which is linked to self-regulation capacities. As such, future studies should evaluate self-regulation in a parenting context more directly.

Findings from the current investigation indicate that child-related stressors in the early parenting years are significantly associated with parent RSA. Specifically, child behavioral problems were associated with poorer parent self-regulatory capacities, as indexed by lower RSA. Further, this effect was exacerbated by marital stress for fathers; highlighting how the context within which child-related stressors occur must be considered. As such, early interventions and prevention efforts targeting the marital relationship amongst families facing exceptional parenting stressors, such as parents of children with recurring behavioral problems, may be a way to support both child and parent well-being. Further, future studies should examine how child stress related changes in RSA predict both child and parental outcomes over time.

Table 1. *Hierarchical linear model of partner-rated child and marital stress in predicting RSA*

	Model 1	Model 2	Model 3
	β (*SE)*	*β* (*SE)*	*β* (*SE)*
Intercept	8.06 (.54)**	7.96 (.53)**	7.95 (.53)**
Gender	.17 (.07)*	.18 (.07)*	.17 (.07)*
Age	-.05 (.02)**	-.05 (.02)**	-.05 (.02)**
Ethnicity	.09 (.07)	.06 (.07)	.07 (.07)
Child behavioral problems	-.005 (.002)*	-.006 (.002)*	-.005 (.002)*
Negative marital interactions	-.01 (.01)	-.01 (.01)	-.01 (.01)
Child behavioral problems x Negative marital interactions		-.001 (.0004)*	-.001 (.0004)
Gender x Child behavioral problems			-.0001 (.002)
Gender x Negative marital interactions			-.002 (.01)
Gender x Child behavioral problems x Negative marital interactions			.001 (.0004)*

Note. * p <.05, ** p < .001. Gender was coded as -1 for men and +1 for women. Ethnicity was coded as -1 and +1 for participants who self-identified as Non-White and White, respectively

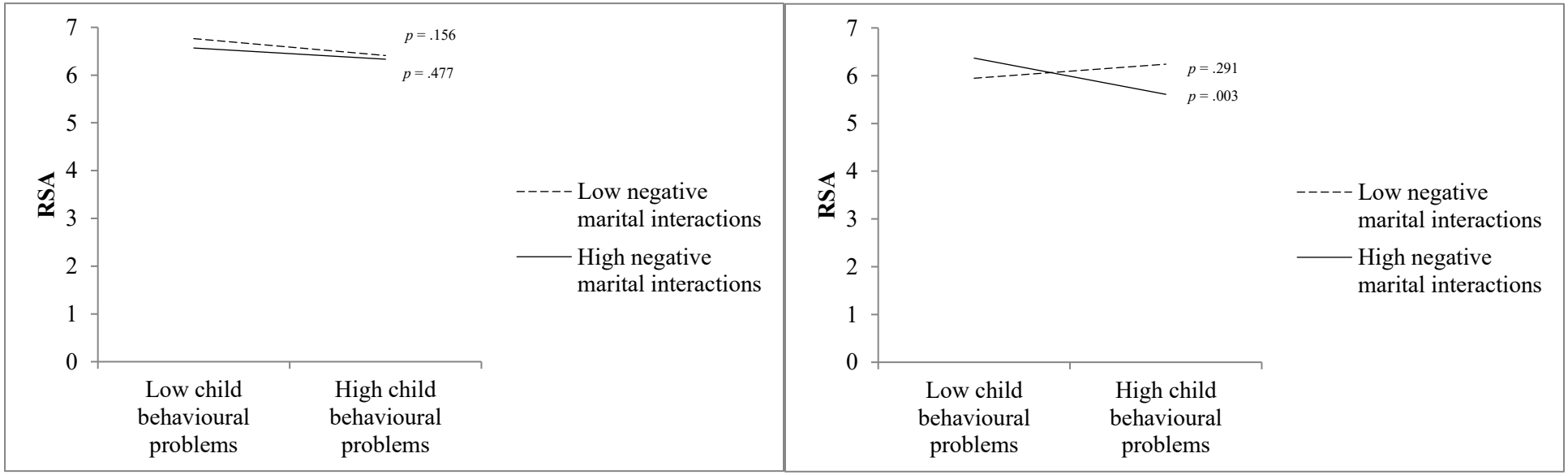

Figure 1. The left panel is a visual depiction of the interaction between child behavioral problems and mean negative marital interactions for mothers, the right panel is a visual depiction of the same interaction for fathers. The lines depicted represent ± 1 SD around the mean of child behavioral problems and mean negative marital interactions.

Conclusion Study 1

Findings from this first dissertation study demonstrated an association between parenting stressors and parent self-regulation as indexed by HRV (referred to as RSA throughout Chapter 2). Specifically, greater child behavioural problems were associated with lower parent HRV. This effect was exacerbated in the context of heightened marital stress, especially amongst new fathers. Capitalizing on the dyadic design, this study used one partner's report of child behavioral problems and marital stress to predict the other partner's HRV, thereby minimizing the likelihood that HRV influenced the perception of child-related and marital stress. Although the findings from Study 1 remain cross-sectional by nature, they suggest that child-related stressors are associated with reduced parent self-regulatory capacities.

In the discussion of Study 1, it was hypothesized that the increased regulatory demands parenting stressors place on parents paradoxically decreases their ability to self-regulate in response to future stressors. That is, lower HRV would be associated with increased vulnerability to stress-related increases in negative mood amongst parents. Study 2 was designed to test this hypothesis. Using the same sample of parents as in Study 1, a daily diary design was used to test whether parent HRV moderated the association between between-person and within-person differences in daily child-related stress and parent negative mood. As an index of self-regulatory capacities, it was hypothesized that lower parent HRV would exacerbate the association between heightened and transient increases in daily child-related stress and parent negative mood.

Chapter 3: Heart rate variability moderates the between- and within-person associations between daily stress and negative affect

da Estrela, C., & Gouin, J.P. (Revise & resubmit). Heart rate variability moderates the between- and within-person associations between daily stress and negative affect. *International Journal of Psychophysiology*.

Abstract

Stress exposure increases risk for depressive symptoms. However, there are substantial individual differences in affective responses to stress. High-frequency heart rate variability (HF-HRV), a marker of vagally-mediated parasympathetic activity, has been conceptualized as a psychophysiological index of emotion regulation that may moderate individuals' responses to stress. Using a daily diary design, we tested whether individual differences in HF-HRV moderated the association between daily child-related stress and negative affect amongst a sample of 84 heterosexual couples with preschool-aged children. After controlling for participants age, gender, socioeconomic status, and ethnicity, hierarchical linear modeling revealed that HF-HRV moderated both the between-person and within-person associations between self-reported child-related stress and daily negative affect. Between-person analyses indicated that the strength of the positive association between mean daily child stress and negative affect across the daily diary period increased with decreasing HF-HRV. Similarly, within-person analyses indicated that on days when participants reported more child-related stress than usual, the magnitude of the increase in negative affect on that day was inversely related to HF-HRV. Taken together, these findings suggest that lower HF-HRV may index vulnerability to stress-related disturbances in negative affect. This increased negative affective response to daily stress may be one pathway through which individuals with lower HF-HRV are at increased risk for depressive symptoms over time.

Keywords: daily stress, heart rate variability, respiratory sinus arrhythmia, negative affect, depression

1. Introduction

Stress is a risk factor for depressive symptoms. While there is a cumulative impact of chronic stress on depression, even everyday stressors are associated with significant changes in affect (Hammen, 2005). However, there exists significant individual variability in affective responses to stressors. Traditionally, cognitive and behavioral factors, such as appraisals and coping styles, have been used to understand these individual differences (Lazarus & Folkman, 1984). However, contemporary stress models argue that psychophysiological functioning should also be considered to understand individual differences in affective response to stress (Epel et al., 2018).

High-frequency heart rate variability (HF-HRV), as an index of emotion regulation (Beauchaine, 2015; Holzman & Bridgett, 2017; Smith, Deits-Lebehn, Williams, Baucom, & Uchino, 2020), may be one psychophysiological factor that moderates affective responses to stress. HRV refers to the variability in time intervals between consecutive heartbeats (Bernston et al., 1997). At rest, cardiac activity is influenced by the interplay between parasympathetic and sympathetic activation. While both branches of the autonomic nervous system influence the low-frequency component of HRV, the high-frequency component of HRV reflects mostly the inhibitory influence of the parasympathetic system on the sinoatrial node of the heart via the vagal nerve. The Neurovisceral Integration Model (Thayer & Lane, 2009) highlights the connection between prefrontal cortex inhibitory activity and vagally-mediated parasympathetic activity on the heart. HF-HRV is regulated by the integration of afferent projections connecting the peripheral nervous system to the central nervous system and efferent projections connecting the prefrontal cortex to the limbic system and the brain stem where parasympathetic output to the sinoatrial node are gated. According to this model, higher HF-HRV at rest is thought to index greater inhibition from the prefrontal cortex to the heart, which has been linked to better physiological and emotional arousal regulation.

Lower HF-HRV has been associated with greater emotional arousal and difficulties with emotion regulation. Individuals with lower HF-HRV experience greater increases in negative affect and negative facial expressions in response to emotion inductions (Demaree, Pu, Robinson, Schmeichel, & Everhart, 2006; Demaree, Robinson, Erik Everhart, & Schmeichel, 2004; Ellis, Shumake, & Beevers, 2016). Low HF-HRV is also associated with self-reported greater day-to-day difficulties in emotion regulation and individuals with lower HF-HRV are more likely to engage in maladaptive mood repair strategies in response to laboratory mood

induction tasks, as compared to individuals with higher HF-HRV (Kovacs et al., 2016; Vasilev, Crowell, Beauchaine, Mead, & Gatzke-Kopp, 2009; Visted et al., 2017; Williams et al., 2015; Williams et al., 2019 Yaroslavsky et al., 2016). Further, individuals with lower HF-HRV are less likely to spontaneously employ adaptive emotion regulation strategies relative to individuals with higher HF-HRV (Geisler, Vennewald, Kubiak, & Weber, 2010; Volokhov & Demaree, 2010). The difficulties with emotion regulation observed in individuals with lower HF-HRV may increase risk for elevated negative affect and depressive symptoms.

Individuals with lower HF-HRV report greater negative affectivity (Sloan et al., 2017). Similarly, meta-analyses indicate that lower HF-HRV is associated with higher depressive symptoms. However, the effect size is small and heterogeneous across studies, suggesting the presence of moderating factors (Kemp et al., 2010; Koch, Wilhelm, Salzmann, Rief, & Euteneuer, 2019; Schiweck, Piette, Berckmans, Claes, & Vrieze, 2018). Stress exposure may be one of those key factors. Across a range of psychosocial stressors, cross-sectional and longitudinal studies demonstrate that the association between stress and depressive symptoms varies as a function of HF-HRV. Lower HF-HRV exacerbates the association between psychosocial stressor exposure and negative affect, across pediatric and adult populations (El-Sheikh, Harger, & Whitson, 2001; Gouin, Deschênes, & Dugas, 2014; McLaughlin, Rith-Najarian, Dirks, & Sheridan, 2015). In the context of stressful family interactions, higher HF-HRV buffers the association between these negative social interactions and self-reported depressive symptoms prospectively (Mezulis, Crystal, Ahles, & Crowell, 2015; Switzer, Caldwell, da Estrela, Barker, & Gouin, 2018). This suggests that individuals with lower HF-HRV may experience larger increases in negative affect in response to stressors than their counterparts with higher HF-HRV.

Most of the studies to date have examined between-person associations among HF-HRV, stress, and negative affect. These between-person associations refer to whether individuals who experience greater stress exposure than others also report more negative affect, and that this association is more pronounced among individuals with lower HF-HRV. In contrast, within-person associations represent the extent to which individuals report increases in negative affect when they experience greater stress exposure than usual according to their own personal baseline (Bolger, Davis, & Rafaeli, 2003; Laurenceau & Bolger, 2005). It is an ecological fallacy to assume that between-person and within-person effects are equivalent (Bolger et al., 2003). Daily

diary studies, which involve repeated assessment of the same person over the course of several consecutive days, provide the opportunity to isolate between-person (i.e., inter-individual differences) and within-person (i.e., intra-individual differences). These daily stress processes are important to understand given that affective responses to daily stressors is predictive of future depressive symptoms (Charles, Piazza, Mogle, Sliwinski, & Almeida, 2013; Cohen, Gunthert, Butler, O'Neill, & Tolpin, 2005; Parrish, Cohen, & Laurenceau, 2011), over and above responses to major life events (Kanner, Coyne, Schaefer, & Lazarus, 1981).

The overarching goal of the current study is to explore the role of HF-HRV in moderating both the between- and within-person associations between daily stress and negative affect. In a prior study with college students, HF-HRV moderated the affective response to high, but not low, intensity daily stressors (Fabes and Eisenberg, 1997). However, this study did not simultaneously explore within- and between-effects of stress on negative affect. In the current study, the role of HF-HRV in daily stress processes was examined among parents of young children, a developmental period associated with increased psychosocial stress. Indeed, parenting young children is associated with increased caregiving responsibilities, time and financial constraints, difficulty maintain work-family balance, and in particular increased child-related stress. There is also significant individual variability, and gender differences, in adjustment during this transition period (Evenson & Simon, 2005; Nomaguchi & Milkie, 2003; Scharlarch, 2001; Umberson, Pudrovska, Reczek, 2010). As such, the early parenting years provides a unique opportunity to investigate the interplay between daily stress and negative affect using an ecologically valid model of psychosocial stress. In the current study, heterosexual couples of preschool-aged children had their baseline HF-HRV measured in the laboratory, and completed repeated measures of perceived child-related stress and negative affect using a daily diary design. Gender differences were also tested. As an index of poor emotion regulation, it was hypothesized that lower HF-HRV would exacerbate both the between-person and within-person associations in child-related stress and negative affect.

2. Method

2.1. Participants

Eighty-four heterosexual couples with preschool-aged children were recruited via online ads, community centers, and posters in a large metropolitan city. In order to be included in the study, participants had to be in a romantic heterosexual relationship and cohabiting with their

children, and all of their children had to be younger than seven years old, (*M* = 3 years, 3 months, *SD* = 23 months). Exclusion criteria included being pregnant, nursing, or having a chronic medical condition, as these factors could influence HF-HRV (Bernston et al., 1997).

On average, participants were 34.60 years old (SD = 4.70 years), had been with their current romantic partner for 9.94 years (*SD* = 4.32), and most couples had either one (44%) or two children (48%; *range* = 1 to 4). The sample was ethnically diverse such that 55.36% self-identified as White, followed by 15.47% self-identifying as Latin American and 8.33% as Middle Eastern. The majority of the participants were employed full-time (56.5%) and the average household income was 55,000 (*SD* = 8,900) CAN$.

2.2. Procedure

Participants attended a laboratory session to measure their HF-HRV. During this session, participants were sat side-by-side and fitted with snap electrodes in a lead II configuration for an electrocardiograph (ECG) recording. Participants completed a five-minute rest period during which their resting HF-HRV was recorded. During this resting period, participants were instructed to stay seated, try to breathe normally and relax as much as possible for five minutes. Subsequently, participated completed some cognitive and social tasks unrelated to the present study.

Following the laboratory session, participants reported on their negative affect at four times throughout the day (e.g., before lunch, before leaving work, after putting the children to bed, and at bedtime) for six consecutive days using a survey application installed on their phone. The last survey entry of each day (i.e., bedtime) also asked participants to provide a rating of child-related stress experienced within the last 24 hours. On average, participants completed 6.54 (*SD* = 1.50) daily diaries.

2.3. Measures

2.3.1. Daily negative affect. Participants used a 5-point Likert scale to rate the extent to which they felt anxious/tense, sad/down, and frustrated/angry since their previous questionnaire entry (1 = *Not at all,* 5 = *A great deal*). An average of these three items was computed for each entry and then averaged across the day to yield a single measure of daily negative affect, with higher scores indicating greater negative affect. Cronbach's alpha for this composite score was .84.

2.3.2. Daily child-related stress. A single item measure was used as a measure of child-related stress during the past 24 hours. Using a 5-point Likert scale, participants were asked to rate the extent to which they experienced stress in the relationship with their child (1 = *Not at all,* 5 = *A great deal*).

2.3.3. HF-HRV. To obtain the HF-HRV data, participants were fitted with snap electrodes in a lead II configuration and data was collected using an ECG amplifier module within a Mindware NioNex 8-slot BioNex chassis (MindWare Technologies Ltd, Gahanna, OH). Interbeat intervals were recorded using a sampling rate of 1000 Hz. An automated algorithm and visual inspection were used to detect and correct artefacts, with less than 1% of the data for each participant needing to be corrected. The data was then analyzed using MindWare HRV Analysis Software, Version 3.1 (MindWare Technologies Ltd, Gahanna, OH). To isolate the vagal-dependent parasympathetic influence on the heart, HF-HRV was extracted with a Fast Fourier Transform using a .15-.40 Hz frequency band. Resting HF-HRV was calculated by averaging 60-second HF-HRV epochs across the 5-minute resting period. HF-HRV values were log transformed.

2.4. Statistical Analyses

Hierarchical linear modeling was used to account for the dependent nature of the data. Specifically, participants' daily data were nested within individuals within dyads (Kenny, Kashy, & Cook, 2006). In order to isolate the within- and between-person effects of child-related stress, the daily data was person mean centered and grand mean centered respectively (Bolger & Laurenceau, 2013). To test the main study hypotheses, same-level interaction terms were computed with HF-HRV, gender, and between-person child-related stress, as well as cross-level interaction terms with HF-HRV and within-person child-related stress. The random effects of the intercept and slope of within-person child-related stress were estimated. Participants' age, socioeconomic status (computed as a composite of the standardized means of participant's income and education level), and ethnicity were included as covariates in all models tested. The data were analyzed using the PROC MIXED function in SAS Version 9.4.

3. Results

3.1. Descriptive Statistics and Bivariate Correlations

Female participants had higher HF-HRV, $t(166) = 2.68$, $p = .008$, and reported greater child-related stress on average, $t(162) = 2.12$, $p = .028$, than male participants. There were no

gender differences for mean daily negative affect, $t(162) = 0.73$, $p = .466$. Bivariate Pearson's correlations were computed using the mean values across the daily diary to examine between-person associations. Greater mean daily child-related stress was positively correlated with greater mean daily negative affect, $r(164) = .63$, $p < .01$. HF-HRV was not significantly correlated with mean child-related stress, $r(164) = -.02$, $p = .835$, or mean negative affect $r(164) = -.07$, $p = .393$.

3.2. Within- and Between-Person Analyses

3.2.1. Within-person fluctuations in daily child-related and negative affect. Intraclass correlations (ICC) were computed to estimate the between and within-person variability in daily child-related stress and negative affect. The ICC for child-related stress (*ICC* = .50) and negative affect (*ICC* = .63) indicated significant between- and within-person variability in both variables across the daily diary period. This indicates that some key moderators may explain variability in the between- and within- associations between daily child-related stress and negative affect.

3.2.2. Daily child-related stress, negative affect, and HF-HRV. Model 1 tested the main effects of participant HF-HRV, gender, and the between-person and within-person effects of daily child-related stress on negative affect. As shown in Table 1, there were no significant main effects of participant HF-HRV or gender in predicting negative affect. In contrast, both between-person and within-person differences in child-related stress were significantly associated with negative affect. Specifically, participants reporting on average greater child-related stress experienced greater negative affect across the assessment period. Further, on days in which participants experienced more child-related stress than usual, they also reported greater daily negative affect. There was also a significant random effect (i.e., slope) for the within-person effect[1] of child-related stress, such that there was statistically significant variability in the within-person association between daily child-related stress and negative affect.

Model 2 tested the hypothesis that HF-HRV moderates the between- and within-person associations between child-related stress and negative affect. As shown in Table 1, HF-HRV moderated the between-person associations between child-related stress and negative affect. As illustrated in Figure 1, the positive association between child-related stress and negative affect increased with decreasing HF-HRV. HF-HRV also moderated the within-person associations in child-related stress and negative affect. Simple slope analyses revealed that participants with

[1] Alternative models that included the random effect of the between-person effect of child-related stress were tested. However, this model did not converge and the effect could not be estimated. As such, only models that included the random effect of the within-person effect of child-related stress are presented.

lower HF-HRV showed a significant increase in negative affect on days when they experienced more child-related stress than usual, while this association was not statistically significant among participants with higher HF-HRV (Figure 2). Adding these interactions reduced the random effects of the association between within-person differences in child-related stress and negative affect by 10.06%.

In Model 3, participant gender was considered as an additional moderator to test whether the moderating role of HF-HRV between child-related stress and negative affect would vary as a function of parent gender. As shown in Table 1 (Model 3), the three-way interaction terms between participant gender, HF-HRV, and child-related stress were not statistically significant. That is, the two-way interaction between HF-HRV and child-related stress in predicting negative affect was equivalent across genders.

4. Discussion

The purpose of the current study was to examine whether HF-HRV moderated within- and between-person differences in affective responses to daily stress using a daily diary design amongst parents of preschool-aged children. Results suggested that lower HF-HRV exacerbated both the between-person and within-person associations between daily stress on negative affect. Specifically, in the context of heightened child-related stress, individuals with lower HF-HRV experienced greater negative affect across the daily diary period, relative to individuals with higher HF-HRV. Further, on days when they reported greater child-related stress than usual, participants with lower HF-HRV experienced larger increases in negative affect, compared to individuals with higher HF-HRV. These associations did not vary as a function of participant gender. These findings suggest that lower HF-HRV may index vulnerability to daily stress-related disturbances in negative affect.

Prior studies indicate that individuals with lower HF-HRV experience larger emotional and physiological response to stress. In both experimental and longitudinal studies, individuals with lower HF-HRV experience greater emotional arousal in response to stress, as compared to individuals with higher HF-HRV (Ellis et al., 2016; Gouin et al., 2014). Further, lower HF-HRV is associated with greater startle response to threat (Gorka et al., 2013; Melzig, Weike, Hamm, & Thayer, 2009). Similarly, individuals with lower HF-HRV also experience greater increased blood pressure and cortisol in response to stress relative to those with higher HF-HRV (Weber et al., 2010). These findings are also consistent with neuroimaging data that show that lower HF-

HRV is associated decreased activation in the prefrontal cortex and subcortical areas of the brain that have been linked to inhibition of emotional and physiological arousal (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). Individuals with lower HF-HRV may thus be more likely to respond with increased emotional and physiological arousal in response to daily stress exposure as compared to individuals with higher HF-HRV.

As a biomarker of emotion regulation (Beauchaine, 2015; Holzman & Bridgett, 2017), the link between lower HF-HRV and stress-related negative affect may also be explained by the decreased use and effectiveness of adaptive emotion regulation strategies. In a previous study with undergraduate students, higher HF-HRV was associated with self-reported greater use of adaptive emotion regulation strategies (Geisler et al., 2010; Geisler, Kubiak, Siewert, & Weber, 2013). In emotion induction studies, individuals with lower HF-HRV are more likely to spontaneously use maladaptive mood repair strategies in response to sadness induction tasks and benefit less from adaptive mood repair strategies (Yaroslavsky et al., 2016). Further, there is also evidence that it is the increased emotional arousal that individuals with lower HF-HRV report in response to stress that may interfere with their ability to use constructive strategies to regulate this arousal (Fabes & Eisenberg, 1997). That is, not only would individuals with lower HF-HRV experience greater emotional and physiological arousal in response to daily stress as compared to individuals with higher HF-HRV, but they may also have greater difficulty employing strategies that would be helpful in regulating this increased arousal, and even once they use these strategies they may actually be less effective.

The extant literature suggests a number of other psychobiological mechanisms that may underlie the association between lower HF-HRV and increased affective reactivity to stress. Individuals with lower HRV are more likely to develop sleep disturbance in response to stress, which may in turn increase risk for depressive symptoms (Gouin et al., 2015; da Estrela, McGrath, Booij, & Gouin, 2020). Also, lower HF-HRV is associated with greater rumination that increases stress-related depressive symptoms (Carnevali, Thayer, Brosschot, & Ottaviani, 2018). Moreover, individuals with lower HF-HRV may experience a greater spillover of their negative affect into their social interactions (Diamond et al., 2011; Ong et al., 2019; Switzer et al., 2018), and may benefit less from positive social interactions (Gouin, Caldwell, MacNeil, & Roddick, 2018; Hopp et al., 2013; Kok & Fredrickson, 2010). Additional research is needed to these potential mediating mechanisms.

Although there was an interaction between HF-HRV and daily stress, there was no main effect of HF-HRV in predicting negative affect. This is similar to another daily diary study in which HF-HRV moderates the within-person association between interpersonal stress and negative affect, despite the lack of bivariate association between HF-HRV and negative affect at the between-person level (Diamond, Hicks, & Otter-Henderson, 2011). Moreover, this finding is consistent with previous studies demonstrating that the association between HF-HRV and negative affect may be enhanced in conditions of stress or adversity. The daily diary study by Fabes and Eisenberg (1997) found that the association between HF-HRV and negative affect differed as a function of stressor intensity, with HF-HRV moderating the association between stress and negative affect for stressors rated high or moderate, but not minor, in intensity. Similarly, in a longitudinal study following undergraduate students as they transitioned from a low stress to high stress academic period, the association between HF-HRV and negative affect was most pronounced in the context of heightened stress (Gouin et al., 2014). While depressive symptoms were not assessed in the current study, this pattern of results may help understand the association between lower HF-HRV and risk for depression. Specifically, stress exposure may be a moderating factor that explains some of the variability observed in the association between HF-HRV and depressive symptoms across studies (Kemp et al, 2010; Koch et al., 2019; Schiweck et al., 2018).

The use of a daily diary design to test the association between stress and negative affect, and the moderating role of HF-HRV, is one of the main strengths of this study. Daily diary studies allow for the differentiation of between-person and within-person effects (Bolger & Laurenceau, 2013). However, the cross-sectional nature of the current study design limits the ability to infer directionality. For instance, it is possible that individuals with lower HF-HRV perceived or elicited greater stress than individuals with higher HF-HRV. Indeed, some longitudinal studies observed that increased parent negative affect led to increases in child-related stress (Goodman et al., 2011). It is also possible that ratings of perceived child stress might have been biased by participant mood as these measures were assessed at the same point in time. Future longitudinal studies, assessing both negative affect and stress at multiple time points throughout the day could help address these limitations. Further, while parenting stress models are ecologically valid models of stress (Quittner, Glueckauf, & Jackson, 1990), this does limit the generalizability of the current findings, especially as only one type of stress (child-related)

was assessed using a single-item measure in the present study. Additional research replicating these results with different populations facing different psychosocial stressors and using more comprehensive stress measures are warranted.

Findings from the current study suggest that lower HF-HRV may index vulnerability to stress-related disturbances in negative affect. As such, HF-HRV may be used to identify individuals most vulnerable to stress. However, clinical thresholds for risk for increased affective reactivity to stress have not yet been identified. One challenge is that although HF-HRV is generally stable over time (Bertsch, Hagemann, Naumann, Schächinger, & Schulz, 2012; Kleiger et al., 1991), it also fluctuates as a function of stress and social context (e.g., Gouin, Zhou, & Fitzpatrick, 2015; Kim, Cheon, Bai, Lee, & Koo, 2018). Further, to our knowledge, there are no studies that have tailored treatments or tested treatment effectiveness as a function of HF-HRV, therefore this remains an important future direction of research. Alternatively, interventions that can improve HF-HRV may be helpful in promoting resilience and adaptation to stress amongst individuals with lower HF-HRV. For example, exercise training increases both HF-HRV and resilience to stress (Hives et al., 2020; Raffin et al., 2019). There is also emerging, albeit preliminary, evidence that psychotherapies such as CBT may be effective in increasing HF-HRV and that targeting HF-HRV using biofeedback may enhance the effectiveness of traditional psychotherapies (Caldwell & Steffen, 2018; Garakani et al., 2008).

Overall, results from the current study suggest that lower HF-HRV is associated with increased negative affect in the context of both between- and within-person differences in daily stress. As affective responses to daily stressors are a predictor of future depressive symptoms (Charles et al., 2013), the results from the current study could suggest that affective vulnerability to daily stressors may be one pathway through which low HF-HRV confers risk for depression over time. Longitudinal studies examining possible mediating mechanisms are warranted to better understand how low HF-HRV increases affective reactivity to stress.

Table 1. Hierarchical linear modeling evaluating the between- and within- effects of child-related stress and their interactions with HF-HRV in predicting daily negative affect.

	Model 1 *Estimate (SE)*	**Model 2** *Estimate (SE)*	**Model 3** *Estimate (SE)*
Fixed effects			
Intercept	1.72 (.44)**	1.63 (.44)**	1.56 (.43)**
Day	-.02 (.01)*	-.02 (.01)*	-.02 (.01)*
Gender	-.03 (.04)	-.04 (.04)	-.21 (.23)
Age	.01 (.01)	.01 (.01)	.01 (.01)
Ethnicity	-.03 (.04)	-.03 (.04)	-.03 (.04)
Socioeconomic status	-.08 (.05)’	-.10 (.05)*	-.10 (.05)*
HF-HRV	.004 (.04)	.02 (.04)	.02 (.04)
Between-person differences in child-related stress	.44 (.04)**	.97 (.27)**	.86 (.28)*
Within-person differences in child-related stress	.09 (.02)**	.34 (.12)*	.37 (.12)*
Between-person differences in child-related stress x HF-HRV		-.08 (.04)*	-.06 (.04)
Within-person differences in child-related stress x HF-HRV		-.04 (.02)*	-.04 (.02)*
Between-person differences in child-related stress x HF-HRV x Gender			-.02 (.04)
Within-person differences in child-related stress x HF-HRV x Gender			.02 (.02)
Random effects			
Intercept	.20 (.02)**	.19 (.02)**	.19 (.02)**
Within-person differences in child-related stress (slope)	.014 (.01)*	.012 (.01)*	.011 (.01)'
Residuals	.17 (.01)**	.16 (.01)**	.17 (.01)**

*Note. ** p <.01, * p <.05, ‘ p < .10.* HF-HRV = High-frequency heart rate variability

Figure 1.

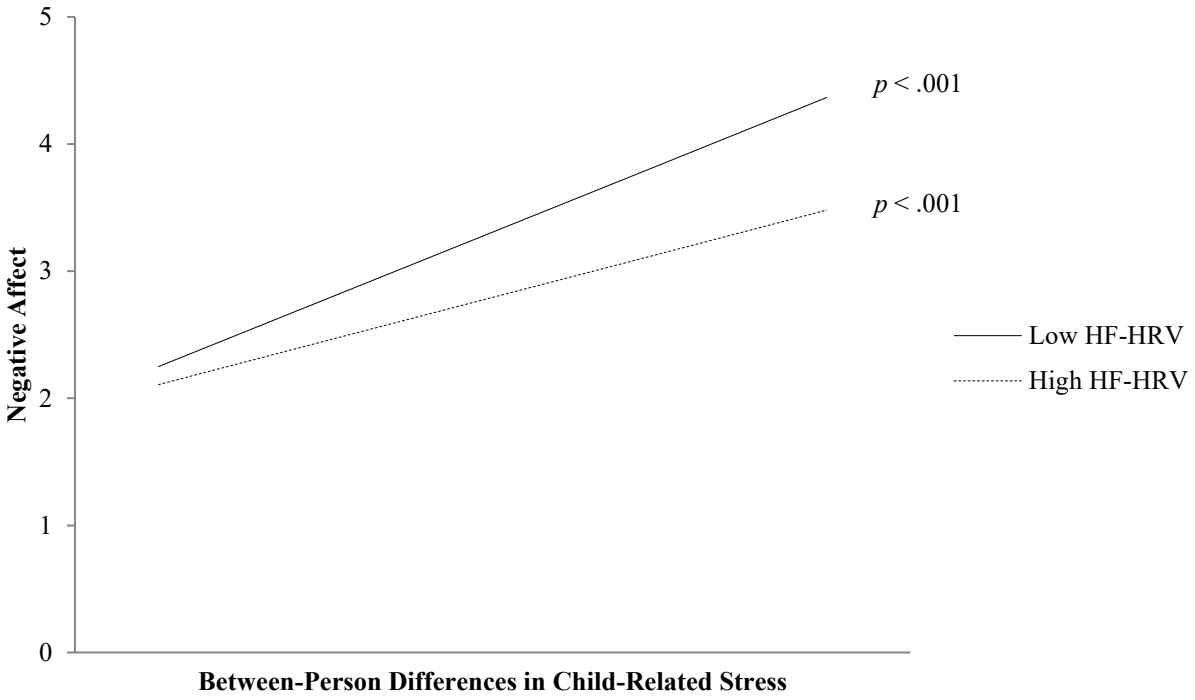

Figure 1. HF-HRV moderates the association between between-person differences in child-related stress and daily negative affect. This figure displays a two-way interaction between between-person differences in child-related stress and HF-HRV in daily negative affect, plotted as a function of ± 1 SD around the mean of HF-HRV. HF-HRV = High-frequency heart rate variability. *p* values refer to the statistical significance of the simple slopes.

Figure 2.

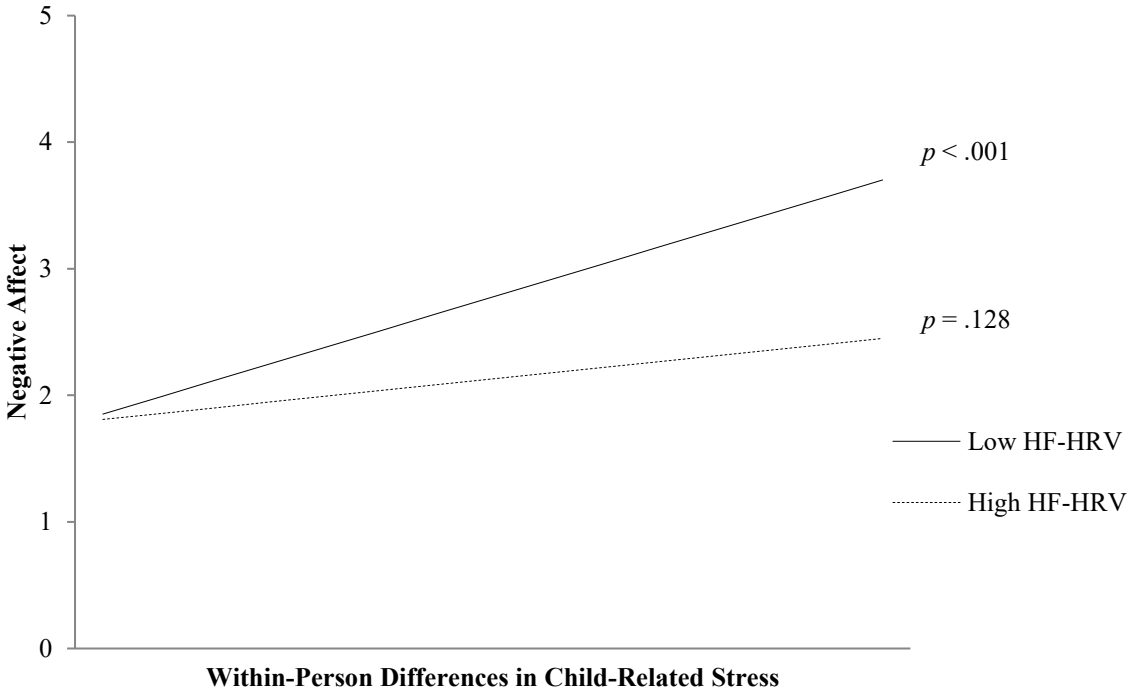

Figure 2. HF-HRV moderates the association between within-person differences in child-related stress and daily negative affect. This figures displays a two-way interaction between within-person differences in child-related stress and HF-HRV in predicting daily negative affect, plotted as a function of ± 1 SD around the mean of HF-HRV. HF-HRV = High-frequency heart rate variability. *p* values refer to the statistical significance of the simple slope.

Conclusion Study 2

Using a daily diary design, this second dissertation study tested whether parent HRV moderated the association between daily parenting stress and negative affect. Findings from this study suggest that lower self-regulation capacities, as indexed by lower HRV, is associated with increased parent negative affect in the context of greater between- and within-person differences in child-related stress. These findings suggest that this vulnerability to stress-related increases in daily negative affect may be a pathway through which parents with lower HRV are at greater risk for depression over time.

Prior studies indicate that parents with lower HRV may experience increased emotional and physiological arousal in response to child-related stress. Further, parents with lower HRV may also have greater difficulty deploying and benefiting from adaptive self-regulation strategies to decrease this heightened arousal, as compared to parents with higher HRV (Ellis et al., 2016; Fabes & Eisenberg, 1997; Gouin et al., 2014; Weber et al., 2010; Yaroslavsky et al., 2016). This increased arousal may interfere with psychobiological mechanisms that mediate the association between chronic stress and depression such as sleep. Specifically, the increased emotional and physiological arousal experienced in response to a stressor by individuals with lower HRV may interfere with sleep onset and maintenance (Riemann et al., 2010).

As such, in Study 3 we tested whether disrupted sleep quality is a pathway through which lower HRV increases vulnerability to stress-related mood disturbances using a moderated-mediation model. Specifically, we tested whether daily sleep quality mediated the association between chronic parenting stress exposure and depressive symptoms, and whether HRV moderated the association between chronic parenting stress exposure and sleep quality. Using a chronic parenting stress model comparing mothers of adolescents with and without neurodevelopmental disorders, it was hypothesized that lower HRV would render parents more vulnerable to stress-related decreases in sleep quality thereby increasing their risk for depressive symptoms.

Chapter 4: Heart rate variability, sleep quality, and depression in the context of chronic stress

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Abstract

Background: Disrupted sleep quality is one of the proposed mechanisms through which chronic stress may lead to depression. However, there exist significant individual differences in sleep reactivity, which is the extent to which one experiences sleep disturbances in response to stress. **Purpose:** The aim of the current study was to investigate whether low high-frequency heart rate variability (HRV), as a psychophysiological marker of poor emotional and physiological arousal regulation, predicts stress-related sleep disturbances associated with greater risk of depression symptoms. **Methods:** Using a chronic caregiving stress model, 125 mothers of adolescents with developmental disorders and 97 mothers of typically developing adolescents had their resting HRV and HRV reactivity recorded, completed a measure of depressive symptoms, as well as a seven-day sleep diary to assess their sleep quality. A moderated mediation model tested whether sleep quality mediated the association between chronic stress exposure and depressive symptoms, and whether HRV moderated this mediation. **Results:** After controlling for participant age, body mass index, ethnicity, socioeconomic status, and employment status, poor sleep quality mediated the association between chronic stress and depressive symptoms. Resting HRV moderated this indirect effect such that individuals with lower HRV were more likely to report poorer sleep quality in the context of chronic stressor exposure, which in turn was related to greater depressive symptoms. **Conclusions:** Lower HRV, a potential biomarker of increased sleep reactivity to stress, is associated with greater vulnerability to stress-related sleep disturbances, which in turn increases risk for elevated depressive symptoms in response to chronic stress.

Keywords: *chronic stress, depression, sleep reactivity, high-frequency heart rate variability, respiratory sinus arrhythmia*

Heart rate variability, sleep quality, and depression in the context of chronic stress

Chronic stress is a well-established risk factor for depression. Empirical evidence supports a causal relationship between chronic stressor exposure and the development of depressive symptoms (Hammen, 2005). Disrupted sleep quality is one of the key mechanisms that have been posited to explain how stress leads to depression (Drake, Pillai, & Roth, 2014; Riemann, Krone, Wulf, & Niseen, 2019, Vargas, Friedman, & Drake, 2015). However, there exist important individual differences in vulnerability to stress-related sleep disturbances, a construct known as sleep reactivity (Drake et al., 2004).

When considering the effects of transient sleep disturbances, experimental studies demonstrate how acute sleep deprivation negatively impacts daily mood amongst healthy individuals (Babson, Trainor, Feldner, & Blumenthal, 2010; Minkel et al., 2012; Scott, McNaughton, & Polman, 2006). Transient reductions in sleep quality have also been associated with increased negative mood in response to daily stressors (da Estrela, Barker, Lantagne, & Gouin, 2018). Moreover, increased insomnia severity prospectively predicts the onset, maintenance, and risk of recurrence of major depressive disorder (Baglioni et al., 2011), and interventions targeting insomnia symptoms also yield improvements in depressive symptoms (Taylor & Pruiksma, 2014). Thus, sleep disturbances appear to play a role in both the diathesis and maintenance of depressive symptoms over time.

Cross-sectional, longitudinal, and experimental studies suggest that stressor exposure is a precipitating factor for the development of sleep disturbances (Drake, Roehrs, & Roth, 2003; Pillai, Roth, Mullins, Drake, 2014). The association between stressor exposure and increased subjective and objective measures of sleep quality has been shown across a range of psychosocial stressors (Åkerstedt , 2006; Hall et al., 2008; Hall et al., 2007; Kalimo, Tenkanen, Härmä, Poppius & Heinsalmi, 2000; Kim & Dimsdale, 2007; Taylor et al., 2016; Verlander, Benedict, & Hanson, 1999). Daily diary studies also indicate that perceived stress on a given day predicts lower sleep efficiency and sleep satisfaction that night (Åkerstedt et al., 2012; Winzeler et al., 2014). Longitudinal studies highlight that chronic stressor exposure is a robust predictor of disrupted sleep quality over time (de Lange et al., 2009; Hall et al., 2015; Linton, 2010). However, not all individuals will develop sleep disturbances in response to stressor exposure (Drake et al., 2004).

Sleep reactivity to stress appears to be a stable characteristic that is influenced by both genetic and environmental factors (Bonnet & Arand, 2003; Drake, Friedman, Wright, & Roth, 2011). In particular, individual differences in physiological and emotional arousal have been proposed to underlie vulnerability to stress-related sleep disturbances (Kalmbach, Anderson, & Drake, 2018; Riemann et al., 2010). Within this context, high-frequency heart rate variability (HRV), as a marker of vagally-mediated parasympathetic activity, has recently been identified as a putative marker of vulnerability to sleep reactivity (Bonnet & Arand, 2003; Gouin et al., 2015; MacNeil et al., 2017). HRV refers to the fluctuation in time intervals between consecutive heartbeats. At rest, both branches of the autonomic nervous system influence cardiac activity; sympathetic activation accelerates heart rate while parasympathetic activation is primarily responsible for its deceleration. While the low-frequency component of HRV reflects the influence of both the sympathetic and parasympathetic nervous system, the high-frequency component is predominantly modulated by parasympathetic output to the heart through the vagal nerve. More specifically, HRV is regulated by the integration of afferent projections from the periphery to the brain via the vagal nerve, and efferent projections connecting the prefrontal cortex with the amygdala and the brain stem where parasympathetic output to the sinoatrial node of the heart is gated (Berntson et al., 1997). Poorer physiological and emotional arousal regulation, indexed by lower resting HRV, has been associated with increased physiological and emotion arousal in response to stress (Thayer & Lane, 2009; Weber et al., 2010).

HRV is also associated with individual differences in objective and subjective assessments of sleep quality. Some studies suggest that both nocturnal and diurnal HRV differs between good sleepers and individuals with insomnia, but these results have not been replicated in all studies (Dodds, Miller, Kyle, Marshall, & Gordon, 2017). In some studies, diurnal HRV was more strongly associated with objective and self-reported measures of sleep quality than nocturnal HRV (Werner et al., 2015). Higher HRV during resting wakefulness has been associated with higher actigraphy-based assessments of sleep efficiency and sleep duration (Elmore-Staton, El-Sheikh, Vaugh, & Arsiwalla, 2012; Castro-Diehl et al., 2016) and negatively correlated with self-reported sleep onset latency and sleep disturbances (Hovland et al., 2013).

There is emerging evidence that individual differences in diurnal HRV may also moderate the association between stressor exposure and sleep quality. In a study examining predictors of sleep reactivity, resting HRV predicted differences in sleep quality in response to a range of

situational stressors (Bonnet & Arand, 2003). Further, individual differences in HRV reactivity, changes in HRV in response to a laboratory stressor, were associated with actigraphy-based and self-report measures of sleep quality (El-Sheikh, Erath, & Bagley, 2013; Bylsma, Salomon, Taylor-Clift, Morris, & Rottenberg, 2014) and moderated the association between psychosocial risk factors and the development of sleep disturbances (El-Sheikh, Hinnant, & Erath, 2015; Keller, Kouros, Erath, Dahl, & El-Sheikh, 2014). In longitudinal studies of academic stress, greater HRV reactivity predicted changes in self-reported sleep disturbances as participants transitioned from periods of lower stress to higher stress (Gouin et al, 2015; MacNeil et al., 2017). These studies provide preliminary evidence that HRV may be a psychophysiological marker of sleep reactivity. However, prior studies have focused on HRV within the context of laboratory stressors or ecologically valid stressors that were time-limited and relatively predictable. Whether HRV is associated with increased sleep reactivity in response to more chronic and uncontrollable stressors has yet to be explicitly tested.

Prior studies indicate that sleep reactivity is an independent predictor of both depressive symptoms and of insomnia symptoms, which then further increases the risk of major depressive disorder (Drake et al., 2014; Riemann et al., 2019). In parallel, HRV has been associated with both elevated depressive symptoms and insomnia severity (Koch, Wilhelm, Salzmann, Rief, & Euteneuer, 2019; Schiweck, Piette, Berckmans, Claes, & Vrieze, 2018) and emerging evidence indicates that it may be a biomarker of sleep reactivity. This suggests that HRV may be associated with higher depression risk through greater stress-related sleep disturbances.

The goal of the current study was thus to test whether HRV moderates the impact of chronic stressor exposure on sleep quality and the mediating effect of poor sleep quality on the association between chronic stressor exposure and depressive symptoms using a moderated mediation model. Using a chronic parenting stress model, an ecologically valid model of chronic stressor exposure that is both ongoing and largely uncontrollable (Quittner, Gluekauf, & Jackson, 1990), this study compared mothers of adolescents with developmental disorders and mothers of typically developing adolescents. Parents of children with developmental disorders tend to report poorer sleep quality and more depressive symptoms than other parenting populations (Karst & Van Hecke, 2012; Lopez-Wagner, Hoffman, Sweeney, Hodge, & Gilliam, 2008**)** and therefore provide a unique opportunity to study the interplay between chronic stressor exposure, sleep quality, and depression.

It was hypothesized that the chronic caregiving stress group would report poorer sleep quality and greater depressive symptoms as compared to the control group. Further, it was hypothesized that sleep quality would mediate the association between chronic stressor exposure and depressive symptoms, and that HRV would moderate the association between chronic stressor exposure and sleep quality. Specifically, it was predicted that individuals with lower resting HRV and increased HRV reactivity would be particularly vulnerable to experiencing poorer sleep quality in the context of chronic stressor exposure, putting them at increased risk of developing depressive symptoms.

Method

Participants

The total sample (N = 222) comprised of two distinct groups, the chronic caregiving stress group (n = 125) comprising mothers of adolescents with an autism spectrum disorder (n = 94) or intellectual disability (n = 31) and the comparison group of mothers of typically developing adolescents (n = 97). Participants were the biological or legal mothers cohabiting with their child, and were in the process of transitioning their child in or out of the high school system. The exclusion criteria included being pregnant or nursing, having a chronic medical condition or acute infection, regular use of anti-inflammatory medication, or major mental illness (e.g., schizophrenia, bipolar disorder, or substance abuse). Participants were recruited via posters and meetings at schools, community groups, and social service centers, and ads in the local newspapers in the Greater Montreal Area. Participants were compensated $60 CAD for their participation in the study.

Procedure

Participants first completed an online questionnaire assessing sociodemographic information and depressive symptoms over the past week. For seven consecutive days prior to the study visit, participants completed a sleep diary assessing their sleep quality. Specifically, participants reported on their previous night's sleep and functioning throughout the day before going to bed each night. Participants then attended a home or laboratory session to measure their HRV using a digital interbeat interval recorder (Polar RS800CX; Finland: Kempele). To obtain a measure of resting HRV, participants were seated and instructed to breathe normally and to try to relax as much as possible for five minutes. Following the resting period, the experimenter administered a worry induction task. Specifically, participants were asked to identify a topic they

tended to worry about the most and then worry about that topic for the 5-minute period (Hofmann, Schulz, Heering, Muench, & Bufka, 2010). This task elicited significant HRV reactivity in prior studies (Gouin et al., 2015; MacNeil et al., 2017; Gouin, Deschênes, & Dugas, 2014). Anthropometric measurements were also taken to calculate body mass index (BMI). To account for diurnal variations in HRV, testing sessions were completed in the morning before noon while participants were fasting (Bonnemeier et al., 2003).

Measures

HRV. At the beginning of the laboratory session, participants were fitted with a digital interbeat interval recorder (Polar RS800CX; Finland: Kempele). This device recorded the timing between consecutive R-peaks of QRS complexes (interbeat intervals) at a sampling rate of 1000Hz. Interbeat intervals were visually screened for recording artifacts, and manually corrected in CardioEdit. HRV values were calculated with CardioBatch from corrected IBI files according to the Porges-Bohrer method (Porges et al., 1980). First, a moving polynomial is applied to the IBI time-series to remove heart-rate variability associated with slower periodic or aperiodic processes. Then, a bandpass filter is applied to the detrended time-series to isolate heart-rate variability attributable to the typical respiratory cycle frequency in adults (between 0.12 and 0.40Hz) to assess respiratory sinus arrhythmia. Variance scores were calculated from 30s epochs of the detrended and filtered IBI time-series. These HRV values were log-transformed, and averaged across the different epochs within each task. HRV reactivity was computed as the difference between participant's HRV during the resting period and the 5-minute worry induction task.

Depressive symptoms. The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) is a 20-item self-report scale that was used to assess frequency of depressive symptoms during the past week. Participants were asked to rate how many days during the past week they felt or behaved a certain way on a four-point Likert scale (0 = *less than one day* and 3 = *five to seven days*). Responses on each item were summed, with higher scores indicating more depressive symptoms. Scores range from 0 to 60, any score above 16 is considered to be a clinically significant level of depressive symptoms (Radloff, 1977). The sample specific Cronbach's α was .91.

Sleep quality. Sleep quality was evaluated over seven consecutive days using sleep diaries adapted from the Consensus Sleep Diary (Carney et al., 2012). Specifically, the sleep

diary assessed total time spent in bed, total sleep time, sleep onset latency, wake time after initial sleep onset, and early morning awakenings. Participants also rated their sleep satisfaction on a 4-point Likert scale (1 = *Very bad* and 4 = *Very good*), and were asked to rate their level of fatigue throughout the day (0 = *Not at all fatigued* and 5 = *As fatigued as I could be; reverse coded)*. Sleep efficiency was calculated as a ratio of total sleep time to total time spent in bed, with a higher ratio indicative of better sleep efficiency (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006). Total time spent in bed was calculated by adding the time between when the participant went to bed at night and got out of bed in the morning. Total sleep time was calculated by subtracting sleep onset latency, nocturnal awakenings, and early morning awakenings from the total time in bed. Sleep efficiency, sleep duration (i.e., total sleep time), sleep satisfaction, and fatigue were recorded for each participant on each day. A mean value across the entire sleep diary period was also computed. A global sleep quality composite was then calculated by averaging the standardized means of all four indices such that higher scores represented better sleep quality. This provided a unified measure of sleep quality, that accounts for multiple dimensions of subjective sleep quality, including waking variables, important to both good sleepers and individuals with insomnia (Harvey, Stinson, Whitaker, Moskovitz, & Virk, 2008) and that is consistent with sleep health recommendations (Buysse, 2014). The Cronbach's α for this composite was .75.

Statistical Analysis

A moderated mediation model was used to test the main study hypothesis that sleep quality (M) mediates the association between chronic stressor exposure (X) and depressive symptoms (Y), and that HRV (W) moderates the association between chronic stressor exposure (coded as -1 for participants in the comparison group and +1 for participants in the chronic caregiving stress group) and sleep quality. This model simultaneously tests the effect of chronic stressor exposure on sleep quality (path *a*) and sleep quality on depressive symptoms (path *b*) as well as the total (path *c*), direct (path *c'*), and indirect effect of chronic stressor exposure on depressive symptoms (path *ab*) using a series of regression models. Further, it tests the interaction of chronic stressor exposure and HRV in predicting sleep quality, and the conditional indirect effect of chronic stressor exposure on depressive symptoms. This model also computes an index of the moderated mediation. Using bootstrapping procedures, a 5,000 bootstrapped

sample was used to compute 95% bias corrected confidence intervals for each effect. Data was analyzed using the PROCESS macro (Model 7; Hayes, 2013) in SPSS version 25.

Participant socioeconomic status (SES) was calculated as a composite of the standardized means of their education level and reported annual household income. This SES variable was included along with participant age, BMI, ethnicity (coded as 0 for those who self-identified as White and 1 for those who did not self-identify as White), and employment status (coded as 1 for employed and 0 for unemployed), as covariates in the model. These covariates were selected given their known associations with HRV and sleep or depressive symptoms in prior studies (Sloan et al., 2008; Bhattacharyya, Whitehead, Rakhit, & Steptoe, 2008; Grandner et al., 2010). Inclusion of these covariates did not change the pattern of results for the moderated mediation models, therefore only fully adjusted models are presented in the manuscript.

Two outliers, defined as a score three SDs away from the sample mean, were identified on the sleep duration variable; these scores were then winsorized to three SDs above the sample mean. One participant had missing data for sleep efficiency and sleep duration, so only their self-reported fatigue and sleep satisfaction were used to compute their sleep quality score. For the main statistical analyses, scores on the CES-D were log transformed to correct for positive skewness in this variable. An alpha level of .05 was used in the current study.

Results

Descriptive Statistics and Intercorrelations

Descriptive statistics and group comparisons of participants' sociodemographic characteristics are presented in Table 1. On average, participants completed 6.05 out of 7 daily diaries (*SD* = 1.44). In total, 35.6% of participants self-reported difficulty falling sleep, staying asleep, or early morning awakenings lasting longer than 30 minutes at least three times during the sleep diary period, which is suggestive of clinically significant insomnia symptoms. Further, 28.2% of the sample reported an average sleep efficiency ratio of less than .85. In terms of depressive symptoms, 41.0% of the sample had a score on the CESD above the clinical cut-off suggesting the presence of clinically significant depressive symptoms.

Spearman's rho correlations were computed amongst the main study variables. There was a marginally significant negative correlation between resting HRV and depressive symptoms $r(220) = -.13$, $p = .06$. Frequency of depressive symptoms was significantly negatively correlated with sleep quality, $r(220) = -.42$, $p < .001$, including shorter sleep duration, $r(218) = -.15$, $p = .03$,

lower sleep efficiency, $r(220) = -.30$, $p < .001$, lower sleep satisfaction, $r(219) = -.44$, $p < .001$, and greater daytime fatigue, $r(220) = -.41$, $p < .001$. HRV reactivity was not significantly correlated with depressive symptoms or any of the sleep quality parameters.

Group Comparisons Amongst the Main Study Variables

As reported in Table 1, independent samples t-tests were run to test group differences on the main study variables. Overall, the chronic caregiving stress group was older, $t(220) = 2.51$, $p = .013$, $d = .34$, and had older children, $t(219) = 3.91$, $p < .01$, $d = .56$, than the comparison group. They also reported greater depressive symptoms, $t(220) = 2.40$, $p = .017$, $d = .33$, and poorer overall sleep quality, $t(220) = -3.13$, $p < .001$, $d = .43$. Specifically, the chronic caregiving stress group slept less hours, $t(218) = -2.46$, $p = .015$, $d = .35$, and were more fatigued, $t(220) = 3.93$ $p < .01$, $d = .53$, than the comparison group. Group differences in sleep efficiency were marginally significant, with the chronic stress group reporting poorer sleep efficiency, $t(218) = -1.92$, $p = .056$, $d = .33$. Sleep satisfaction did not differ as a function of group, $t(219) = 1.12$, $p = -.266$, $d = .14$, and neither did resting HRV, $t(220) = -1.26$, $p = .209$, $d = .17$ or HRV reactivity, $t(214) = -.82$, $p = .412$, $d = .11$.

Moderated Mediation Analyses

As illustrated in Figure 1, a moderated mediation model was used to the test the main study hypothesis. When controlling for participant age, ($\beta = -.001$, $SE = .004$, $p = .752$), BMI ($\beta = .002$, $SE = .004$, $p = .604$), ethnicity ($\beta = .08$, $SE = .05$, $p = .156$), SES ($\beta = -.02$, $SE = .05$, $p = .420$), employment status ($\beta = -.06$, $SE = .06$, $p = .274$), and HRV ($\beta = -.03$, $SE = .02$, $p = .112$), the total effect of chronic stressor exposure (i.e., having an adolescent with an autism spectrum disorder or intellectual disability) on depressive symptoms was statistically significant (path c in Figure 1; $\beta = .05$, $SE = .02$, $p = .047$, 95% CI [.001, .09]) such that chronic stressor exposure was associated with greater depressive symptoms.

When adjusting for the effects of participant age, ($\beta = -.008$, $SE = .01$, $p = .337$), BMI ($\beta = -.01$, $SE = .008$, $p = .073$), ethnicity ($\beta = -.35$, $SE = .11$, $p = .002$), SES ($\beta = -.04$, $SE = .06$, $p = .548$), and employment status ($\beta = .16$, $SE = .11$, $p = .164$), the effect of chronic stress on sleep quality was statistically significant, such that chronic stressor exposure predicted poorer sleep quality (path a; $\beta = -.15$, $SE = .05$, $p = .002$, 95% CI [-.25, -.06]). Further, when controlling for participant age, ($\beta = -.001$, $SE = .004$, $p = .717$), BMI ($\beta = .0002$, $SE = .004$, $p = .948$), ethnicity ($\beta = .002$, $SE = .05$, $p = .961$), SES ($\beta = -.03$, $SE = .03$, $p = .305$), and employment status ($\beta = -$

.04, *SE* = .05, *p* = .417), the effect of sleep quality on depressive symptoms was also statistically significant, whereby poorer sleep quality was associated with greater depressive symptoms (path b; β = -.18, *SE* = .03, *p* < .01, 95% CI [-.24, -.12]).

After controlling for sleep quality, the direct effect of chronic stressor exposure on depressive symptoms was no longer statistically significant and reduced compared to the total effect (path c'; β = .02, *SE* = .02, *p* = .369, 95% CI [-.02, .06]). The indirect effect of chronic stressor exposure on depressive symptoms was statistically significant (path *ab*; β = .02, *SE* = .01, 95% CI [.01, .04]). Taken together, this pattern of results suggests that sleep quality partially mediated the association between chronic stressor exposure and frequency of depressive symptoms. This model accounted for 18.18% of the variance in depressive symptoms.

As part of the moderated mediation hypothesis, the interaction between chronic stressor exposure and resting HRV in predicting sleep quality was tested. There was a statistically significant interaction between chronic stressor exposure and resting HRV in predicting sleep quality (β = .08, *SE* = .04, *p* = .036, 95% CI [.005, .15], R^2= .02). As illustrated in Figure 2, there was a significant association between chronic stressor exposure and poor sleep quality at lower resting HRV levels (*Effect* = -.26, *SE* = .07, *p* < .001, 95% CI [-.39, -.12]), but not at higher resting HRV levels (*Effect* = -.05, *SE* = .07, *p* = .450, 95% CI [-.17, .11]). Further, the 95% confidence interval around the index of moderated mediation suggests that resting HRV moderated the indirect effect of sleep quality on the association between chronic stressor exposure and depressive symptoms (*Index* = -.01, *Boot SE* = .01, 95% CI [-.03, -.001]). Specifically, the conditional indirect effect of chronic stress on depressive symptoms was statistically significant at lower resting HRV (*Effect* = .05, *Boot SE* = .01, 95% CI [.02, .08]), but not higher resting HRV (*Effect* = .009, *Boot SE* = .01, 95% CI [-.01, .04]). Given that the CES-D included a sleep-related item ("My sleep was restless"), analyses were repeated while omitting this item. The pattern of results remained the same such that the moderated mediation remained statistically significant (*Index* = -.01, *Boot SE* = .006, 95% CI [-.03 -.003]). As excessive fatigue is a symptom of depression, analyses were also repeated using a sleep quality variable excluding the daily fatigue component. Again, the pattern of results remained the same; the moderated mediation remained statistically significant, *Index* = -.01, *Boot SE* = .006, 95% CI [-.03, -.001].

In contrast, HRV reactivity did not moderate the association between chronic stressor exposure and sleep quality, β = -.05, *SE* = .07, *p* = .494, 95% CI [-.19, .09], R^2= .002. The index

of moderated mediation was also not significant for HRV reactivity (*Index* = .009, *Boot SE* = .01, 95% CI [-.01, .04]). These analyses were repeated using residualized change scores as an alternative way to calculate HRV reactivity, the pattern of results remained the same; the moderated mediation was not statistically significant; *Index* = -.005, *Boot SE* = .01, 95% CI [-.03, .02].

Discussion

The goals of the current study were to investigate whether sleep quality mediated the association between chronic stressor exposure and depressive symptoms, and whether HRV moderated this association. As hypothesized, chronic stressor exposure was associated with decreased sleep quality, which then predicted greater depressive symptoms. Moreover, this mediation effect was moderated by HRV. Specifically, individuals with lower resting HRV were more likely to report poorer sleep quality in the context of chronic stressor exposure than their counterparts with higher HRV. These findings suggest that lower HRV may be a biomarker of sleep reactivity to stress and that vulnerability to stress-related sleep disturbances may be a key pathway through which individuals with lower HRV are at greater risk for stress-related depressive symptoms.

In the current study, individuals with lower resting HRV were more susceptible to experiencing poorer sleep quality in the context of chronic stressor exposure than individuals with higher HRV. Contemporary models of insomnia posit that emotional and physiological hyperarousal are key factors involved in the development and maintenance of sleep disturbances (Riemann et al., 2010). It is well-known that stress increases arousal, and individuals with lower HRV are more likely to exhibit increased arousal in response to stress. Specifically, in addition to increased emotional arousal (Thayer & Lane, 2009; Gouin et al., 2014), lower HRV is also associated with increased physiological arousal as indicated by increased blood pressure and cortisol (Weber et al., 2010). This stress-induced hyperarousal may interfere with sleep onset and maintenance among individuals with lower resting HRV.

Sleep reactivity, the tendency to experience sleep disturbances in response to stress exposure, has been associated with greater risk of depression, over and above the effects of insomnia severity (Drake et al., 2014; Vargas et al., 2015). Prior work indicates that lower HRV is associated with greater sleep disturbances in response to acute laboratory challenges as well as predictable and time-limited naturalistic stressors (Bonnet & Arand, 2003; Gouin et al., 2015;

MacNeil et al., 2017). The present findings provide further evidence that lower resting HRV may be a biomarker of sleep reactivity. Specifically, the current study extended previous findings by examining the link between HRV and sleep reactivity in the context of a chronic stressor.

Sleep quality mediated the association between chronic stressor exposure and depressive symptoms in the current study. This is consistent with previous research that highlights disrupted sleep quality as a key mechanism linking stress and depression (Drake et al., 2014; Riemann et al., 2019; Harvey, 2011). The present findings suggest that this greater sleep reactivity may be one of the pathways through which individuals with lower HRV are at greater risk of developing depressive symptoms in response to chronic stressor exposure. The literature on the link between HRV and depressive symptoms has previously been characterized by modest and heterogeneous effect sizes (Koch et al., 2019). Recent findings suggest that accounting for sleep disturbances may explain some of the heterogeneity in the association between low HRV and depression (Bylsma et al., 2014; Hamilton, Stange, Burke, Franzen, & Alloy, 2019; Werner et al., 2017).

However, contrary to what was hypothesized, the moderated mediation effect with HRV reactivity was not significant. This is consistent with a recent daily diary study that demonstrated that individual differences in resting HRV, but not HRV reactivity, moderated the association between sleep quality and depressive symptoms amongst individuals with a prior history of depression (Hamilton et al., 2019). Furthermore, while resting levels of HRV is a reliable predictor of psychopathology, the predictive utility of HRV reactivity has been limited by methodological challenges. Specifically, findings from a recent meta-analysis suggest that null findings can partially be attributed to systematic differences in the baseline and reactivity procedures used, as well as participants' sex (Beauchaine et al., 2019). The current study included only females and used a worry induction task as the reactivity procedure. Future studies should include both sexes and test whether different reactivity procedures modify the association between HRV reactivity and stress-related sleep reactivity.

The chronic caregiving stress model used in the present study included mothers of adolescents with an autism spectrum disorder or intellectual disability. Although mothers of children with special needs report more depressive symptoms than mothers of typically-developing children (Olson & Hwang, 2001), prior research indicates that mothers of children with autism report more parenting stress than mothers of children with other developmental disabilities (Hayes & Watson, 2013). In the present study, additional follow up analyses

indicated that mothers of adolescents with ASD showed the highest levels of depressive symptoms and poorest sleep quality, followed by mothers of adolescents with intellectual disabilities, and then mothers of typically developing adolescents. However, significant group differences in depressive symptoms, $t(189) = 2.25$, $p = .025$, $d = .33$, and sleep quality, $t(189) = -3.36$, $p = .001$, $d = .49$, were observed between mothers of adolescents with ASD and mothers of typically developing adolescents, but not between mothers of adolescents with ASD and mothers of adolescents with other developmental disabilities (depressive symptoms, $t(123) = -.085$, $p = .932$, $d = .02$; sleep quality, $t(123) = 1.06$, $p = .294$, $d = d = .22$). Of note, although the chronic caregiving stress group displayed worst overall sleep quality, the overall differences were of small magnitude. These results dovetail with prior studies highlighting individual differences in sleep reactivity to stress (Drake et al., 2004). Further, while it is often assumed that caregivers and non-caregiving populations differ on health outcomes, the association between caregiver status and health is heterogeneous and influenced by other individual differences (Vitaliano, Zhang, & Scanlan, 2003). Low resting HRV may be one inter-individual factor that places certain caregivers at an increased risk for stress-related sleep disturbances.

One of the key limitations of this study is its cross-sectional design, precluding inferences of directionality among the associations observed. Depressive symptoms may have preceded sleep disturbances (Jansson-Fröjmark & Lindblom, 2008). An alternative model wherein lower HRV is related to increased vulnerability to the effects of sleep deprivation on mood is also possible (Hamilton et al., 2019). The cognitive-energy model (Zohar, Tzischinsky, Epstein, & Lavie, 2005) posits that sleep loss deplete the cognitive resources required for adaptive self-regulation, which may be enhanced among individuals with lower HRV who may have less self-regulatory capacity (Thayer & Lane, 2009). Although alternative moderated mediation models wherein depressive symptoms was the mediator or HRV moderated the association between sleep quality and depressive symptoms were not significant (data not shown), these alternative conceptualizations cannot be ruled out in this cross-sectional study. As such, longitudinal studies are required to better comment on the directionality of the observed associations. Further, chronic stressor exposure was inferred by caregiver status, future studies should include continuous measure of stress severity (e.g. child's symptoms severity) rather than relying on a dichotomous caregiving variable (Pastor-Cerezuela, Fernández-Andrés, Tárraga-Mínguez, Navarro-Peña, 2016). This is important to consider given that different individuals with the same

diagnosis of ASD or intellectual disability may exhibit a wide range of functioning that is directly impacting the caregiving stress experienced by the mother. Moreover, the generalizability of the sample is limited such that future studies should include men, as sex and gender differences have been found in relation to HRV (Koenig & Thayer, 2016), insomnia (Zhang & Wing, 2006), and depression (Nolen-Hoeksema, 2001). In the current study, participant age also differed across groups and may represent an important confound as age is independently associated with both HRV and sleep disturbances (Martin, Shochat, & Ancoli-Israel, 2000; Sloan et al., 2008). However, the associations observed in the current study were found over and above the effects of age. Moreover, a longer assessment of the sleep quality variables (e.g., two weeks instead of one week) would better capture the instability of sleep quality characteristic of poor sleepers (Wohlgemuth, Edinger, Fins, & Sullivan, 1999). Further, the inclusion of objective measures of sleep quality are also warranted.

Meta-analytic studies suggest that lower HRV is associated with elevated depressive symptoms (Koch et al., 2019; Beauchaine & Thayer, 2015). The present findings indicate that individuals with lower HRV are more likely to experience stress-related impairment in sleep quality and, in turn, poor sleep explains part of the association between chronic stressor exposure and depressive symptoms for these individuals. This suggests that sleep-focused treatment could play an important role in preventing depressive symptoms in the context of chronic stressor exposure, particularly among individuals with lower HRV. Cognitive-behavioural therapy for insomnia is the current gold standard treatment for chronic insomnia (Morin et al., 2019). Even when it includes only sleep-related interventions, CBT for insomnia also reduces depression (Cunningham & Shapiro, 2018). This intervention strategy, that has already been successfully adapted to caregiving populations, may be particularly helpful for individuals who are facing exposure to uncontrollable and unpredictable stressors (Swanson, Flynn, Adams-Mundy, Armitage, & Arnedt, 2013), such as parents of children with developmental disabilities. Individuals with lower HRV may reap larger benefits from such sleep-focused intervention. However, at this time, a clinical HRV threshold value has not been identified to select individuals who would benefit most from such treatment. Future studies should investigate the clinical utility of HRV measures in tailoring treatments for individuals facing chronic stressor exposure. Furthermore, there is also research indicating that successful CBT for depression is associated with increases in HRV (Carney et al., 2000; Taylor et al., 2009) and that HRV

biofeedback enhances the efficacy of CBT for depression (Caldwell & Steffen, 2018). It is possible that interventions that increase HRV may then reduce sleep reactivity. Further empirical work is needed to test this hypothesis.

Taken together, the results from the current study suggest that low resting HRV moderates the impact of chronic stressor exposure on sleep quality. Specifically, low HRV appears to be a psychophysiological marker of sleep reactivity that is associated with risk for elevated depressive symptoms in response to chronic stressor exposure. Future studies should replicate these findings in longitudinal studies and evaluate whether difficulties with physiological and emotional arousal regulation explain the risk for poor sleep and depression associated with lower HRV.

Table 1. *Comparison of participants' sociodemographic characteristics and the main study variables as a function of group*

	Chronic Stress Group	**Comparison Group**	
	M (SD)	*M (SD)*	*t (d)*
Participant age (years)	47.71 (6.15)	45.69 (5.69)	-2.51* (.34)
Child age (years)	16.45 (2.82)	15.16 (1.80)	-3.91** (.56)
BMI	28.01 (5.86)	27.58 (6.71)	-.50 (.07)
Income (CAN$)	64,919.31 (41,667.45)	66,822.91 (42,414.76)	.33 (.05)
HRV ln(ms^2)	5.55 (1.30)	5.77 (1.31)	1.26 (.17)
HRV reactivity ln(ms^2)	-.23 (.72)	-.16 (.55)	.82 (.11)
Depressive symptoms	17.89 (10.93)	14.40 (10.44)	-2.40* (.33)
Sleep quality	-.14 (.77)	.18 (.68)	3.13** (.43)
Sleep efficiency	.86 (.10)	.89 (.08)	1.92' (.35)
Sleep duration (hours)	6.72 (1.02)	7.04 (.89)	2.46* (.33)
Sleep satisfaction	2.88 (.55)	2.95 (.46)	1.12 (.14)
Fatigue	2.61 (.88)	2.16 (.83)	-3.93** (.53)
	%	*%*	χ^2
Relationship status (% in a current romantic relationship)	75.2	77.3	.14
Ethnicity (% self-identified as White)	80.8	74.2	1.37
Employment status (% employed)	72.8	76.3	.35
Education level (% graduated from university)	46.4	47.4	8.76

Note. ** $p < .01$*, * $p < .05$*, ' $p < .10$.* BMI = body mass index. ln = Logarithmic transformation of power spectral measures.

Figure 1.

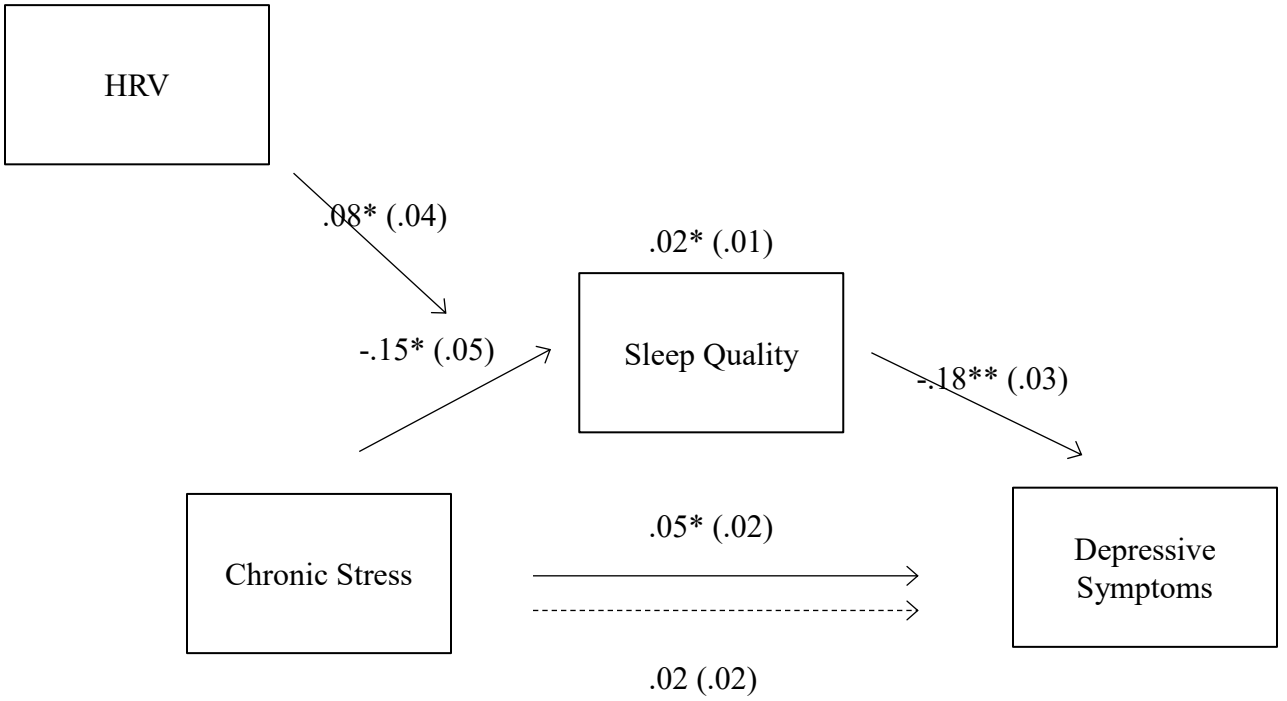

Figure 1. Visual depiction of the moderated mediation model tested. Beta coefficients and corresponding standard errors (in parentheses) for each effect are also presented. The dotted arrow refers to the direct effect of chronic stress predicting depressive symptoms when controlling for the effect of sleep quality. ** $p < .01$, * $p < .05$. HRV = Baseline high-frequency heart rate variability.

Figure 2.

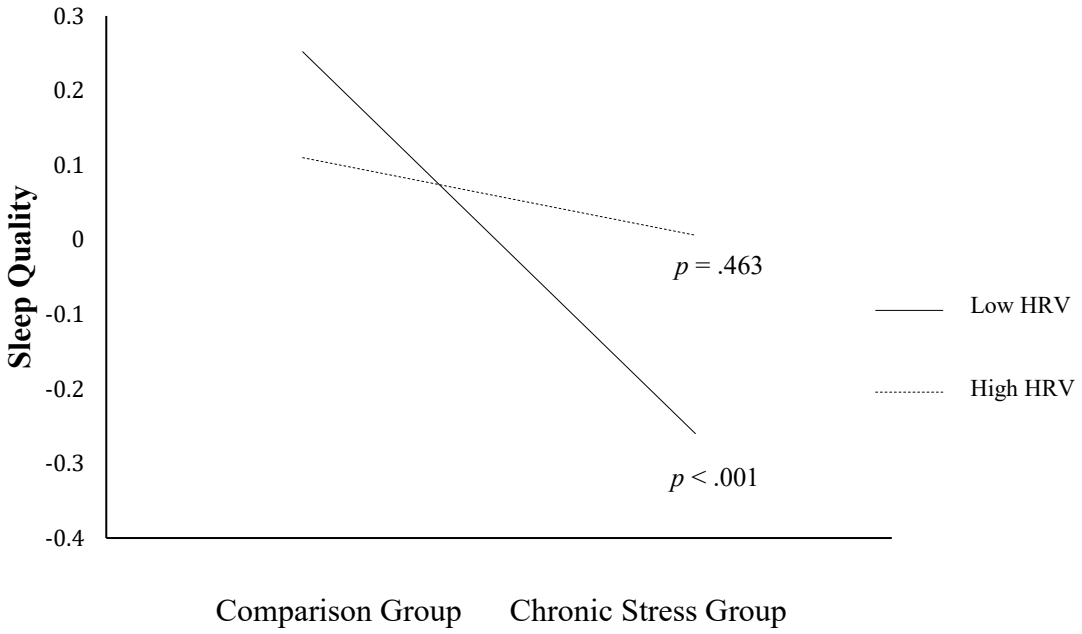

Figure 2. Interaction between chronic stress and HRV in predicting sleep quality, plotted as a function of ± 1 SD around the mean of HRV. HRV = Resting high-frequency heart rate variability. *p* values refer to the statistical significance of the simple slopes.

Chapter 5: General Discussion

Child-related stressors are a robust predictor of parental depression (Bagner et al., 2013; Ciciolla et al., 2014; Gross et al., 2009). Family stress models such as the ABC-X model of family stress (Hill 1949; 1958), the Family Adjustment and Adaptation Response Model (McCubbin & Patterson, 1983), Perry's integrative model of stress (Perry, 2004), and the Family Stress Model (Masarik & Conger, 2017), each emphasize that not all parents will develop negative outcomes, such as depressive symptoms, in response to parenting stressors. Instead, parenting stressors are likely to lead to parental depression when the demands that these stressors place on parents exceed the resources parents can recruit to address those demands. The goal of this dissertation was to study whether parent self-regulatory capacities, as indexed by HRV, is a resource that can explain individual differences in adaptation to parenting stressors. Findings across the three studies support the hypothesis that parent self-regulatory capacities, as indexed by HRV, plays a role in understanding the link between parenting stress and parental depressive symptoms.

In Study 1 (Chapter 2), we first established that there is an association between child-related stressors and parent HRV. Specifically, increased child behavioural problems were associated with lower parent HRV. It is well-known that stressor exposure has a negative impact on physical health outcomes. The allostatic load model provides a conceptual framework to understand how stress gets "under the skin" to impact physical health (McEwen 2006; 2012; Seeman et al. 1997). Stressor exposure initiates a cascade of neuroendocrine, immunological, and autonomic responses that in the short term promote adaptation to stress (allostasis). However, over time, prolonged exposure to stress leads to chronic activation of these stress response systems, which leads to wear and tear (allostatic load) conferring risk for a variety of negative health outcomes such as reduced HRV (Dishman et al., 2000; Horsten et al., 1999; Gouin et al., 2014; Lampert et al., 2016). Our findings suggest that recurring child-related stressors may have the same physiological effects as other stressors.

Findings from Study 1 thus add to an emerging body of literature on the impact of challenging child functioning on parent neurophysiology. Parent cortisol, a biomarker of the stress response system, has been associated with child-related stressors. Amongst working mothers of preschoolers, self-reported parenting stress and job strain interacted to predict suboptimal cortisol awakening responses (Hibel, Mercado, & Trumbell, 2012). Further, when

looking at preschooler's self-regulation capacities, preschoolers' effortful control moderates the association between parenting behaviours and parents' cortisol awakening responses (Merwin, Smith, & Doughtery, 2015), highlighting that child's characteristics influences parents' stress responses. Similarly, in a study with parents of children with conduct disorder, self-reported parenting stress was associated with self-reported maternal physical health status, and this association was explained by increased exposure to daily child-related stressors (BeLue, Halgunseth, Abiero, & Bediako, 2015). Taken together, this data shows the need to expand our current family stress models to consider not only the psychological impact of child-related stressors on parent functioning, but also on their physiological stress responses.

To date, the link between stress exposure and HRV specifically has been studied mostly outside of the context of child-related stressors. Within this broader literature, exposure to psychosocial stress has been consistently associated with decreased HRV. Greater perceived stress prospectively predicts lower HRV amongst healthy adult (Dishman et al., 2000). A similar pattern of results has been observed when considering exposure to chronic stress and other adverse life events, as well as social isolation (Horsten et al., 1999; Lampert et al., 2016). Further, during periods of increased stress, greater psychosocial stress predicts reductions in HRV, especially for individuals who are not well socially integrated (Gouin et al., 2015). Consistent with both the resource model of self-regulation (Baumeister et al., 2000) and the allostatic load model (Seeman et al. 1997; McEwen 2006; 2012), it has been argued that individuals exposed to stress may initially experience increased HRV as they attempt to self-regulate in response to their environment (Katz, 2007; Lampert et al., 2016). Over time, frequent activation of this neurophysiologic system (i.e., increased allostatic load) leads to fatigue of this system. Findings from Study 1 expand this literature by showing that increased exposure to child-related stressors is also associated with lower HRV.

In this first empirical study, we also showed that the association between child behavioural problems and parent HRV was most pronounced in the context of increased marital stress. This finding is consistent with family stress models like the ABC-X model (Hill, 1949; 1958), which emphasizes that the association between stressor exposure and individual outcomes is dependent on the family context within which these stressors are occurring. Within this framework, marital stress may be an additional stressor that places increased demands on the family system and may also reduce the likelihood that parents will be able to use each other as a

resource to address the demands that child-related stressors place on the family system (Kiecolt-Glaser & Wilson, 2017). The ABC-X model is a specific example of a contemporary family stress model that has traditionally been used to explain parent psychological outcomes. Our findings suggest that it would be appropriate to extend the application of this model to parent physiological stress responses as well.

The interaction between child-related stress and marital stress in predicting parent HRV was most pronounced for new fathers. Traditionally, it has been hypothesized that father-child relationships are more vulnerable to external stressors, such as marital stress, than mothers because their roles may be less defined especially during the early parenting years (Belsky et al., 1984). This three-way interaction also dovetails with findings regarding the association between marital status and health wherein men typically reap greater health benefits than women from being married (Kaplan & Kronick, 2006; Kiecolt-Glaser & Newton, 2017). The differences in outcomes might be explained by gender differences in availability of other forms of informal social support, with women traditionally having greater access to social support networks outside of the home (Umberson et al., 1996). Our findings suggest the effect of child-related stress on HRV is moderated by marital stress for fathers. For mothers, the marital context seems to matter less. However, these effects are not always replicated and are sometimes even more pronounced for women (e.g., Hartley et al, 2018; Smith et al., 2011).

Findings from Study 1 may have important implications for parent functioning given the association between self-regulation and HRV. In the seminal study by Segerstrom & Nes (2007), HRV was hypothesized to index self-regulation capacities because of the anatomical overlap between brain structures involved in self-regulation and the central autonomic network that governs HRV, such as the prefrontal cortex (Thayer & Lane, 2000). Using an experimental manipulation of self-regulation by having participants eat or resist eating a desirable but unhealthy food (e.g., chocolate or cookies), they showed that participates exhibited an increase in HRV during tasks involving increased self-regulation (resisting the desirable food) compared to the low self-regulation condition, suggesting that elevations in HRV indexed increased self-regulatory efforts. They then showed that individuals with higher HRV were more likely to persist on a challenging anagram task, suggesting that higher HRV is associated with greater self-regulatory strength. Similarly, a meta-analysis examining the association between HRV and

self-regulatory strength has shown a small, but consistent, positive association between HRV and self-regulation across laboratory tasks (Zahn et al., 2016).

As such, lower HRV has been posited to index poorer self-regulation capacities. Indeed, individuals with lower HRV self-report greater day-to-day difficulties with self-regulation as compared to individuals with higher HRV (Vasilev et al., 2009; Visted et al., 2017; Williams et al., 2015; Williams et al., 2019). They are also less likely to use adaptive self-regulation strategies relative to individuals with higher HRV (Geisler et al., 2010; Volokhov et al., 2010). A similar pattern of results has been replicated in laboratory settings, with individuals with lower HRV more likely to engage in maladaptive self-regulation strategies in response to negative mood induction tasks (Kovacs et al., 2016; Yaroslavsky et al., 2016). These self-regulation difficulties may be one pathway through which lower HRV is associated with an increased risk for mood disturbances.

Broadly, poor self-regulation is a risk factor for depression. Multiple theoretical frameworks have been proposed to explain why difficulties with self-regulation may increase the risk of experiencing depressive symptoms. For instance, it has been speculated that difficulties with self-regulating both negative and positive mood represents one of the core deficits observed in individuals with depression (Morris & Reilly, 1987). Similarly, Matthews's self-regulation model of depression (1977) emphasizes that self-regulation includes the ability to self-monitor, self-evaluate, and self-reinforce and that dysfunctions in any three of these components may lead to the cognitive and behavioural symptoms observed in depression such as low self-worth and anhedonia. Contemporary models of self-regulation and depression point towards similarities in the neurobiological substrates that are implicated in self-regulation and depression such as serotonergic functioning (Carver, Johnson, & Joormann, 2008). If HRV is an index of self-regulation, and poor self-regulation is a risk factor for depression, then it follows that individuals with lower HRV should be at an increased risk for developing mood disturbances.

Indeed, individuals with lower HRV do report greater negative mood on average and are more likely to engage in suicidal behaviour as compared to individuals with higher HRV (Sloan et al., 2017; Wilson et al. 2016). Meta-analyses on HRV and depression suggest an association between lower HRV and increased depressive symptoms, with small effect sizes observed (Kemp et al., 2010; Koch et al., 2019). However, heterogeneity in the association between HRV and depression has been observed across studies, which suggests the presence of moderating

factors. Stressor exposure may be one of those moderating factors. In line with stress-diathesis models of depression (Colodro-Conde et al., 2018), lower HRV may represent a diathesis, or vulnerability, to developing stress-related mood disturbances. Indeed, findings from cross-sectional and prospective studies suggest that the association between lower HRV and increased mood disturbances is most pronounced under conditions of heightened psychosocial stress (Fabes & Eisenberg, 1997; Diamond et al., 2011; Gouin et al., 2014). Study 2 (Chapter 3) was designed to extend these findings by testing whether parent HRV was associated with stress-related mood disturbances in the context of daily child-related stress.

Whereas previous research had focused mostly on between-person differences in stressor exposure and mood, Study 2 used a daily diary design to demonstrate that the association between both between- and within-person differences in daily child-related stress and parent negative mood increased with decreasing HRV. The daily diary design allowed for differentiation between between-person and within-person effects. Unlike between-person effects, which provide information about individuals who experience greater stress on average and trait-level differences, within-person effects provide information about situational and state-like responses (Bolger, Davis, & Rafaeli, 2003; Laurenceau & Bolger, 2005). Further, affective responses to daily stressors are a robust predictor of future depressive symptoms (Cohen, Gunthert, Butler, O'Neill, & Tolpin, 2005), over and above responses to major life events (Kanner, Coyne, Schaefer, & Lazarus, 1981). As such, it has also been suggested that examining affective responses to daily stressors can provide insight into a person's trait-like vulnerability to maladaptive long-term outcomes in response to stress (Epel et al., 2018). Findings from Study 2 showed that low parent HRV might be a trait-like individual factor that increases vulnerability to stress-related mood disturbances in response to both between- and within-person differences in stressor exposure.

In Study 2 we also observed that there was significant variability in the association between child-related stress and parent negative mood. This is in line with previous findings showing heterogeneity in parent adjustment during developmental periods of increased child-related stress (Evenson & Simon, 2005; Nomaguchi & Milkie, 2003; Scharlach, 2001; Umberson et al., 2010). Variability in response to stressor exposure is also very much consistent with the main tenant of family stress models, which emphasize that not all families will respond to the same stressors in the same way. These models are similar in that they posit that differences in

resources available to address the demands associated with a stressor moderate the association between stressor exposure and parent outcomes. In Study 2 we examined only one potential individual resource, which was parent self-regulation capacities. Our findings suggest that parent self-regulation, as indexed by HRV, is a resource that predicts individual adaptation to daily parenting stress.

In Study 3 (Chapter 4) we sought to replicate and extend findings from Study 2. Specifically, we tested whether lower HRV predicted stress-related mood disturbances in the context of a *chronic* parenting stressor. Literature on parents of children with neurodevelopmental disorders provides information on the association between ongoing and prolonged exposure to parenting stress and risk for parent depression. For instance, as a group, parents of children with neurodevelopmental disorders report more depressive symptoms than parents of typically developing children and other children with special needs (Brobst et al., 2009; Dumas et al., 1991; Hayes & Watson, 2013; Weiss, 2002). Exposure to child-related stressors, and in particularly challenging child behaviours, is a robust predictor of these group differences (Phetrasuwan & Miles, 2009; Rezendes & Scarpa, 2011; Walsh, Mulder, & Tudor, 2013; Weiss, Cappadocia, MacMullin, Viecili, & Lunsky, 2012). Yet despite these group differences, there remains heterogeneity in parent outcomes amongst parents of children with neurodevelopmental disorders, suggesting the presence of moderating factors and a need to identify these factors for better prevention and early intervention (Karst & Van Hecke, 2012). We propose that parent self-regulation, as indexed by HRV, is an individual resource, that could help explain variability in adaptation to both daily and chronic parenting stressors.

There are a number of different psychobiological mechanisms that have been proposed to underlie the association between lower HRV and vulnerability to stress-related mood disturbances. For instance, individuals with lower HRV experienced increased physiological arousal, including increased blood pressure and cortisol, in response to stress as compared to individuals with higher HRV (Weber et al., 2010). With regards to attention processes, lower HRV has been associated with less flexible attention, poorer attentional control and attentional inhibition (Park et al., 2012; 2013). Difficulties with disengaging from negative stimuli and redirecting attention may increase the likelihood that individuals with lower HRV are more likely to experience mood disturbances when exposed to a stressor. It is also possible that parents with lower HRV may be more vulnerable to experiencing stress-related mood disturbances

because of difficulty recruiting additional resources, such as social support, that could otherwise buffer against the negative effects associated with stress exposure (Kok & Fredrickson, 2010; Geisler et al., 2013; Gouin et al., 2018; Hopp et al., 2013). In Study 3 we tested whether disrupted sleep quality is a potential pathway through which lower HRV confers risk for stress-related mood disturbances.

Poor sleep quality was selected as it is a well-established mechanism linking stressor exposure and the development of depressive symptoms. Findings from experimental and naturalistic studies provide evidence that stress is a risk factor for sleep disturbances (Bkeland et al., 1968; Fortunato & Harsh, 2006; Gross & Borkovec, 1982; Hall et al., 2004; Ota et al., 2009; Taylor et al., 2016). Further, emotion regulation theories, neuroimaging, and experimental data suggest that sleep quality predicts emotional responses to stressors (Deliens et al., 2014; Goldstein & Walker, 2014; Hall et al., 2012; Meerlo, 2008; Prather et al., 2013; Yoo et al., 2007). Zohar and colleagues' cognitive-energy model (2005) has been used to explain how sleep quality may mediate the association between stressor exposure and depression. Specifically, it proposes that self-regulation in the face of even mild stressors requires cognitive resources that can be depleted by sleep loss. Findings from Study 3 add to this body of literature by suggesting that poor sleep may interfere with a parent's ability to cope with the demands associated with chronic parenting stress.

Indeed, disrupted sleep quality as a mechanism linking stress and depression is also consistent with findings from the broader parenting stress literature. For instance, increased self-reported parenting stress and both subjective and objective markers of poor sleep quality are associated with increased depressive symptoms amongst parents of children with neurodevelopmental disorders (Chu & Richdale, 2009; Meltzer, 2011). Studies with other populations of parents experiencing increased parenting stress (e.g., parents of children with chronic illnesses and new mothers) also show associations between poor sleep quality and self-reported mood disturbances (Calcagni, Bei, Milgrom, & Trinder, 2012; Meltzer & Booster, 2016). Further, in the context of chronic parenting stress, parents who on average report poorer sleep quality, or following nights when they report poorer sleep quality than their own personal baseline, are more likely to experience mood disturbances in response to daily child behavioural problems (da Estrela, Barker, Lantagne, & Gouin, 2018). The findings from Study 3 add to this body of literature by suggesting that parents with lower self-regulation capacities, as indexed by

lower HRV, may be particularly vulnerable to experiencing poor sleep in the context of heightened parenting stress.

The finding that disrupted sleep quality is a potential pathway through which low HRV confers risk for depressive symptoms in the context of stress is also in line with previous research examining the association between HRV and sleep reactivity. Sleep reactivity is a trait-like variable that refers to the development of sleep disturbances in response to stress (Bonnet & Arand, 2003; Drake et al., 2014). Cross-sectional and longitudinal studies show associations between HRV and differences in sleep reactivity such that lower HRV predicts greater vulnerability to developing stress-related sleep disturbances (Bonnet & Arand, 2003; Bylsma et al., 2014; El-Sheikh et al., 2013; Gouin et al., 2015; MacNeil et al., 2017) It has been hypothesized that the increased emotional and physiological arousal experienced in response to a stressor associated with lower HRV interferes with both sleep onset and maintenance (Ellis et al., 2016; Gouin et al., 2014; Riemann et al., 2010; Weber et al., 2010). Our findings suggest that sleep reactivity can also occur in the context of chronic parenting stress, especially for parents with lower HRV, and that vulnerability to stress-related sleep disturbances may help explain part of the variability in the association between HRV and depressive symptoms observed in the broader HRV literature (Bylsma et al., 2016; Hamilton, Stange, Burke, Franzen, & Alloy, 2019; Werner et al., 2017).

Is there a link between parenting HRV, vulnerability to stress-related mood disturbances, and parenting behaviours?

Not only may parents with lower HRV be at greater risk of developing stress-related mood disturbances, but emerging evidence also suggests that parents with lower HRV may parent differently than parents with higher HRV. Mothers with lower HRV are more likely to contribute to greater escalation of negative parent-child interactions and are more likely to hold negative hostile attribution biases about their children's problematic behaviours (Connell et al., 2015; Wang et al., 2016). Further, lower HRV has been associated with greater difficulty recognizing cues of distress and less validation of children's negative emotions (Joosten et al., 2013; Blandon et al., 2015). Our finding that low parent HRV is a risk factor for the development of stress-related mood disturbances draws attention to a potential mechanism that may help explain these differences in parenting behaviours. Specifically, it is possible that the increased mood disturbances parents with lower HRV may experience in response to challenging child

behaviours may impact their ability to engage in adaptive parenting practices. Whether the increased negative mood parents experience in response to challenging child behaviours mediates the association between parent HRV and parenting behaviours has yet to be empirically investigated.

The Family Stress Model (Masarik & Conger, 2017) provides a helpful framework to understand how stress-related mood disturbances in parents may impact parenting practices and in turn child developmental outcomes. Consistent with other family stress models, this model posits that stressors lead to increased parent psychological distress and that different factors moderate parents' susceptibility to experiencing depression in response to stressor exposure. We have proposed that HRV is one of these factors. The model then hypothesizes that parent depression disrupts adaptive parenting practices, which then leads to poor youth outcomes. This latter part of the model has received extensive empirical support. Figure 1 displays a model integrating findings from the current dissertation with the Family Stress Model. It illustrates how exposure to child-related stress is associated with decreased HRV, which then increases vulnerability to developing stress-related mood disturbances because of disrupted sleep quality. Following the Family Stress Model, these mood disturbances are then hypothesized to disrupt parenting practices and yield additional challenging child behaviours, which may then further deplete parent self-regulatory capacities. However, it is important to note that we did not study parenting practices nor child outcomes. As such, this integrated model remains theoretical and comprehensive longitudinal designs are warranted to best capture the directionality and implications of these associations.

As alluded to above, the association between parenting stress and depression symptoms is likely bidirectional. Empirical findings from research on parents of children with developmental disabilities highlights the interplay between parenting stressors and parent depression. Together, findings from this literature suggest that parenting stressors are a robust predictor of parent depressive symptoms and that parent depressive symptoms predict the use of harsher parenting behaviours in response to parenting stressors. These parenting behaviours are then likely to exacerbate challenging child behaviours, which then further increases the risk of parent depression (Neece & Chan, 2017). Although this process is likely relevant for all parents, as shown in Figure 1, our findings suggest that parents with poorer self-regulation capacities may be particularly vulnerable to these processes. Specifically, it is possible that parents with lower

HRV are more likely to develop mood disturbances in response to parenting stressors and also more likely to respond to stressors in ways that increase the occurrence of parenting stressors as compared to other parents. As per findings from Study 1, increased stressor exposure may then further deplete parents' ability to cope with those stressors. Prospective designs that carefully consider the pathways through which parenting stressors, parent self-regulation and depression interact are warranted to address these hypotheses.

As the studies presented in this dissertation were cross-sectional in nature, an alternative hypothesis wherein parents with greater mood disturbances elicited or perceived greater parenting stress than other parents is also possible. A longitudinal study following mothers through pregnancy and postnatally showed that parental depression prospectively predicts perceived parenting stress over and above baseline levels of parenting stress (Leigh & Milgrom, 2008). The stress generation hypothesis posits a transactional relationship between stressor exposure and depressive symptoms by highlighting the individual as an active participant in their environment. Specifically, it hypothesizes that depression is recurrent in part because individuals with a history of depression are more likely to encounter more interpersonal stressors due to personal and situational characteristics associated with depression (Hammen, 2006). In the context of parental mood disturbances, parents with depressive symptoms may be more likely engage in maladaptive parenting practices that increase the occurrence of challenging child behaviours. In a study with mothers of preschool-aged children, maternal depressive symptoms predicted child behavioural problems one year later. Decreased use of scaffolding behaviours (providing optimal levels of support to allow a child to succeed at a given task) amongst mothers with elevated depressive symptoms as compared to other mothers helped explain this pattern of results (Hoffman, Crnic, & Baker, 2006). As such, it is possible that the findings in the current dissertation reflect parent-driven and not exclusively child-driven pathways.

Another consideration is that parents with poorer self-regulation capacities are at increased risk for depression because they are more likely to be exposed to challenging child behaviours as they are more likely to have children with difficult temperament and self-regulation difficulties. Studies examining the intergenerational transmission of self-regulation suggest both environmental and genetic contributions (Bridgett, Burt, Edwards, & Deater-Deckard, 2015). Parents with poorer self-regulatory capacities are less likely to engage in parenting practices that promote the development of adaptive self-regulation than parents with

stronger self-regulatory capacities. For instance, parents with lower self-regulatory capacities are more likely to engage in negative parenting behaviours such as more hostile and aggressive parenting, which mediates the association between parent and child self-regulatory capacities across multiple generations (Pears, Capaldi, & Owen, 2007). Further, there is a heritable component to self-regulation and specifically to HRV as well. As reported in the qualitative review by Bridgett and colleagues (2015), twin studies suggest that 33% to 66% of the variance is HRV is heritable and 20% of the variance in HRV during childhood may be attributed to shared environmental factors. A further complication is that children with difficult temperaments are also more likely to become parents with poorer self-regulation due to their genetics. Taken together, it is possible that parents with lower HRV are at an increased risk of developing depressive symptoms because they are more likely to have children who also have difficulties with self-regulation, thereby increasing their likelihood of encountering child-related stressors.

Strengths and limitations

Overall, one of the novel contributions of this dissertation is incorporating parent self-regulation, as indexed by HRV, into current family stress models. Despite evidence that parent self-regulation has been associated with behavioural and cognitive aspects of parenting (Aspinwall, 2004; Aspinwall & Taylor, 1997; Lorber, 2012; Mazursky-Horowitz et al., 2015; Rosenbaum, 1989; Shaffer & Obradović, 2017), the role of parent self-regulation in supporting adaptation to parenting stress remained an important gap in the literature. Further, despite the well-documented impact of parent psychopathology on child developmental outcomes (Goodman et al., 2011), there is a general paucity of literature on antecedents of parent depression. An additional strength of this dissertation was the use of a variety of indicators of parenting stress across the different studies. Parenting stress was conceptualized as frequency of child behavioural problems in Study 1, as perceived child stress in Study 2, and inferred by caregiving status in Study 3. Although there may be limitations associated with each approach independently, being able to demonstrate meaningful associations between these different indicators of parenting stress and parent HRV across the three studies adds to the validity of the overall findings.

This dissertation also examined parent self-regulation from a psychophysiological perspective by using HRV. Using a psychophysiological measure addressed several

methodological challenges with self-report measures. Specifically, it minimized the possibility of inflated correlations as a result of common method bias. It also allowed for the study of the implicit processes involved in self-regulation, which otherwise could not be captured by self-report measures. Further, studying self-regulation from a psychophysiological perspective addresses a recent call from the broader stress literature to expand current stress models to include psychophysiological processes (Epel et al., 2018). However, while using HRV as a psychophysiological index of self-regulation is one of the main strengths of this dissertation, its use in the absence of other measures of self-regulation is a limitation. That is, self-regulation capacities were inferred by HRV and were not measured directly. Self-regulation is considered to be a multifaceted construct with multilevel processes that interact to predict future outcomes (Ciccheti & Dawson, 2002; Cole, Martin, & Dennis, 2004). Further, it has been suggested that different components of parent self-regulation predict different parenting behaviours (Shaffer, Whitehead, Davis, Morelen, & Suveg, 2017). Future studies should include a more comprehensive measure of self-regulation by including empirically validated self-report measures (see Enkavi et al., 2019 for a review), along with observational and collateral data.

Although different measures of child-related stress were used across the different studies, we did not use well-validated measures of stress in all studies. For instance, in Study 1 we used child behavioural problems, as reported on the CBCL (Achenbach & Rescorla, 2000), as a proxy for exposure to child-related stress. While the CBCL is a well-validated measure of child behavioural problems, and while child behavioural problems have been used a proxy of child-related stress in previous research (e.g., da Estrela et al., 2018), the CBCL was not designed to be a measure of child-related perceived stress per se. Similarly, in Study 2, child-related stress was assessed using a one-item measure of perceived child-related stress. The psychometric properties of which are not known. Further, when individuals report on their perceived stress it is possible that we are better capturing their stress reactivity and not stress exposure per se (Almeida, 2005; Epel et al., 2018). Finally, in Study 3, exposure to child-related stressors was inferred by caregiving status. Dichotomizing stressor exposure may have masked the heterogeneity in stress exposure within each caregiving group. It also rested on the assumption that parents of children with neurodevelopmental disorders encounter more child-related stressors than parents of typically developing children, however, there are parents of typically developing children who also report experiencing significant parenting stress (e.g., Neece, Green, & Baker, 2012). The

inclusion of more comprehensive and well-validated measures of parenting stress, including measures distinguishing stressor exposure from stressor reactivity within a given study, may help address the gaps associated with each approach independently.

One of the main inferences motivating the studies in the dissertation is that a better understanding of the antecedents of parent depression could have important prevention and early intervention implications for youth outcomes. Further, it was hypothesized that disrupted parenting practices is the pathway through which stress-related mood disturbances may impact youth outcomes. Although previous research has supported an association between parent mood disturbances, disrupted parenting practices, and maladaptive youth outcomes (Masarik & Conger, 2017), we did not assess youth outcomes or parenting behaviours. An independent but relevant body of literature also points towards an association between youth HRV and parent HRV and the concept of physiological synchrony and co-regulation (Suveg et al., 2019). The inclusion of youth HRV as either a covariate or outcome measure would be warranted. Finally, in order to fully address the model proposed in Figure 1, future studies may want to consider both the antecedents and consequences of parent stress-related mood disturbances. A longitudinal design would also help provide information regarding the directionality of these associations.

As discussed throughout, one major caveat is that the analyses described are cross-sectional and thus causality or directionality cannot be inferred for any our findings. As such, it remains possible that parents with lower HRV elicited greater parenting stress or are more likely to encounter parenting stress than parents with higher HRV. Further, the association between parenting stress and parent depressive symptoms is likely bidirectional with both parent- and child-driven pathways (Neece & Chan, 2017). However, these associations could not be captured by our cross-sectional designs. Prospective studies assessing HRV and stress at multiple time points are warranted to address these alternative hypotheses. Specifically, using a prospective design with at least three time points would allow researchers to address whether 1) changes in child-related stressor exposure predicts changes in parent HRV, 2) changes in parent HRV predicts the development of stress-related mood disturbances, and 3) whether changes in stress exposure predicts changes in sleep quality which then predicts changes in parent mood.

The findings that low HRV exacerbated the impact of parenting stress on parent mood disturbances was interpreted in the context of the diathesis-stress model. However, it is possible

that a differential susceptibility framework is more appropriate. The differential susceptibility hypothesis posits that individuals who are vulnerable to experiencing negative outcomes in response to features of their environment are also more likely to experience benefit from positive environments (Belsky, 2016). Without having considered positive parent-child interactions or positive mood indices in this dissertation, it is unclear if parents with lower HRV show enhanced benefits relative to parents with higher HRV. The broader HRV suggests that lower HRV is better conceptualized within a diathesis-stress framework as individuals with lower HRV have greater difficulty benefitting from the use of adaptive self-regulation strategies (Yaroslavsky et al., 2016) and are less likely to experience positive emotions in response to positive social interactions (Gouin et al., 2018; Hopp et al., 2013; Kok & Fredrickson, 2010). Still, it would be important for future research to include positive-valence predictors and outcomes to better address this alternative hypothesis, especially in terms of how it pertains to parent-child interactions.

Finally, this dissertation focused mainly on resting HRV whereas there are the two additional components of HRV that are also relevant to consider in the context of adaptation to stress (Smith et al., 2020). HRV reactivity and recovery were either not well represented or considered across the three studies. HRV reactivity refers to the change in HRV in response to given a situation; increased HRV reactivity to a situation-specific stressor is hypothesized to index greater self-regulation exertion (Thayer & Lane, 2009). When considering adaptation to stress, interactions between resting HRV and HRV reactivity have also been observed (Gouin et al., 2014). HRV recovery refers to the time it tasks HRV to return to resting baseline following stressor exposure and is generally less studied than resting HRV and HRV reactivity. Faster HRV recovery is associated with stronger self-regulation (Stange, Hamilton, Fresco, & Elloy, 2017) and may have important implications for the long-term impact of stressor exposure on health. Future studies may want to include HRV reactivity and HRV recovery in response to child-related stressors or stressful parent-child interactions, as well the interaction with resting HRV, to better understand the association between parent HRV and vulnerability to stress-related mood disturbances.

Implications and future directions

Taken together, findings from this dissertation suggest that lower parent HRV may index vulnerability to developing stress-related mood disturbances. As such, HRV may be helpful in

identifying parents most at-risk for developing depressive symptoms. This would have broad implications for prevention and early intervention of poor parent mental health outcomes, and in turn also have implications for promoting positive child developmental outcomes. However, there are limitations to using HRV as a way of identifying at-risk populations. First, clinical thresholds have yet to be identified and the feasibility of incorporating HRV into clinical practice must also be considered. Further, while HRV remains generally stable over time (Bertsch, Hagemann, Naumann, Schächinger, & Schulz, 2012; Kleiger et al., 1991), it also fluctuates as a function of stress and social context (e.g., Gouin, Zhou, & Fitzpatrick, 2015; Kim, Cheon, Bai, Lee, & Koo, 2018). Thus, the clinical utility of HRV remains an important future direction of research.

Beyond HRV, if parent self-regulatory capacities are a resource that may facilitate adaptation to parenting stress, then interventions aimed at building self-regulation skills may be an important next step in supporting families facing exceptional caregiving challenges. Evidence-based intervention programs that target parent self-regulation skills, such as the Triple P-Positive Parenting program, have yielded positive parent and youth outcomes (Nowak & Heinrichs, 2008). Specifically, the Triple P program has been associated with improved parental wellbeing, including self-reported depressive symptoms, and reduced frequency of child behavioural problems. However, it is unclear whether changes in parental wellbeing preceded or followed changes in child behavioural problems. Further empirical work is needed to understand how parental HRV may change as a result of these parenting interventions and whether the efficacy of these interventions may vary as a function of parent HRV.

Further, if sleep reactivity is a key pathway through which lower HRV confers risk for mood disturbances then targeted sleep interventions could play an important role in preventing the development of depressive symptoms amongst this at-risk population. Indeed, cognitive-behavioural therapy for insomnia (CBT-i), the current gold standard treatment for insomnia, has been effective in reducing depressive symptoms (Cunningham & Shapiro, 2018) and is an intervention strategy that has already been successfully adapted to other caregiving populations (Swanson et al., 2013). It is possible that parents with lower HRV may reap additional benefits from these targeted sleep interventions, however, this hypothesis has not yet been tested.

Conclusions

Overall, the findings from this dissertation provide novel information examining parent self-regulatory capacities as a key resource in moderating parents' adaptation to parenting stressors. Specifically, parent self-regulation as indexed by HRV, is a resource that may help identify which parents adapt, and which parents have difficulty adapting to, parenting stressors. Taken together, our findings suggested that parents with lower HRV are more likely to experience mood disturbances in the context of stressor exposure, and that disrupted sleep quality may be one mechanism through which this vulnerability to stress-related mood disturbances occurs. Further, increased child-related stress was associated with lower parent HRV suggesting that increased exposure to stress may paradoxically deplete parent's ability to cope with future child-related stressors. The studies from this dissertation also show the value of expanding contemporary family stress models to incorporate individual differences in psychophysiological functioning, particularly for implicit processes such as self-regulation, in understanding individual responses to stress.

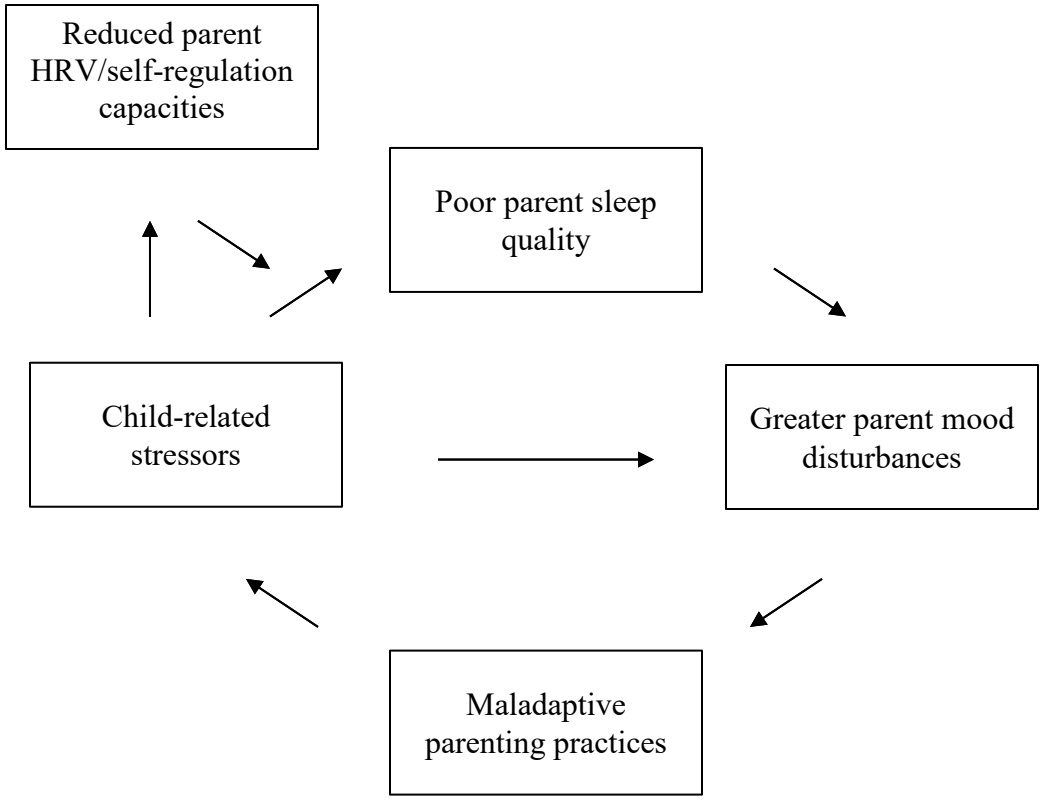

Figure 1. Proposed model explaining associations between parenting stress, HRV, and depression. Prolonged exposure to child-related stressors leads to reduced parent HRV (depleted parent self-regulation capacities). Reduced parent HRV increases vulnerability to stress-related mood disturbances via disrupted sleep quality. Parents who experience greater mood disturbances may then be more likely to engage in maladaptive parenting practices that further increase the occurrence of child-related stressors which may then in turn further deplete parent self-regulation capacities.

References

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA Preschool Forms & Profiles*. Burlington, VT: University of Vermont.

Åkerstedt, T. (2006). Psychosocial stress and impaired sleep. *Scand J Work Environ Health, 32*, 493-501.

Åkerstedt, T., Orsini, N., Petersen, H., Axelsson, J., Lekander, M., & Kecklund, G. (2012). Predicting sleep quality from stress and prior sleep – A study of day-to-day covariation across six weeks. *Sleep Medicine, 13*, 674-679.

Almeida, D.M. (2005). Resilience and vulnerability to daily stressors assessed via diary methods. *Current Directions in Psychological Science, 14*, 64-68.

Almeida, D. M., Wethington, E., & Chandler, A. L. (1999). Daily transmission of tensions between marital dyads and parent–child dyads. *Journal of Marriage and the Family, 61*(1), 49-61.

Aspinwall, L. G. (2004). Dealing with Adversity: Self-regulation, Coping, Adaptation, and Health. In M. B. Brewer & M. Hewstone (Eds.), *Perspectives on social psychology. Applied social psychology* (p. 3–27). Blackwell Publishing.

Aspinwall, L.G., & Taylor, S.E. (1997). A stitch in time: Self-regulation and proactive coping. *Psychological Bulletin, 121*, 417-436.

Babson, K.A., Trainor, C.D., Feldner, M.T., & Blumenthal, H. (2010). A test of the effects of acute sleep deprivation on general and specific self-reported anxiety and depressive symptoms: An experimental extension. *Journal of Behaviour Therapy and Experimental Psychiatry, 41*, 297-303.

Baglioni, C., Battagliese, G., Feige, B., Spiegelhalder, K., Nissen, C., Voderholzer, U., … Riemann, D. (2011). Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. *Journal of Affective Disorders*, *135*, 10-19.

Bagner, D.M., Pettit, J.W., Lewinsohn, P.M., Seeley, J.R., & Jaccard, J. (2013). Disentangling the temporal relationship between parental depressive symptoms and early child behaviour problems: A transactional framework. *Journal of Clinical Child & Adolescent Psychology, 42,* 78-90.

Balzarotti, S., Biassoni, F., Colombo, B., & Ciceri, M.R. (2017). Cardiac vagal control as a marker of emotion regulation in healthy adults: A review. *Biological Psychology, 130,* 54-66.

Bariola, E., Gullone, E., & Hughes, E.K. (2011). Child and adolescent emotion regulation: The role of parental emotion regulation and expression. *Clinical Child and Family Psychology Review, 14*, 198-212.

Bhattacharyya, M.R., Whitehead, D.L., Rakhit, R., & Steptoe, A. (2008). Depressed mood, positive affect, and heart rate variability in patients with suspected coronary artery disease. *Psychosomatic Medicine, 70*, 1020-1027.

Baumeister, R. F., Muraven, M., & Tice, D.M. (2000). Ego depletion: A resource model of volition, self-regulation, and controlled processing. *Social Cognition*, *18*, 130-150.

Beauchaine, T. (2015). Respiratory sinus arrhythmia: A transdiagnostic biomarker of emotion dysregulation and psychopathology. *Current Opinion in Psychology, 3*, 43-47.

Beauchaine, T.P., Bell, Z., Knapton, E., McDonough-Caplan, H., Shader, T., & Zisner, A. (2019). Respiratory sinus arrhythmia reactivity across empirically based structural dimensions of psychopathology: A meta-analysis. *Psychophysiology, 56*, e13329.

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

Belsy, J. (2016). The differential susceptibility hypothesis: Sensitivity to the environment for better and for worse. *JAMA Pediatrics, 170*.

Belsky, J., Gilstrap, B., & Rovine, M. (1984). The Pennsylvania Infant Family Development Project, I: Stability and change in mother-infant and father-infant interaction in a family setting at one, three, and nine months. *Child Development, 55*, 692-705.

Bernston, G. G., Bigger Jr., J. . T., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., ... van der Molen, M. W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34*, 623-648.

Bertsch, K., Hagemann, D., Naumann, E., Schächinger, H., & Schulz, A. (2012). Stability of heart rate variability indices reflecting parasympathetic activity. *Psychophysiology, 49*, 672-682.

Blandon, A.Y. (2015). Mothers' and fathers' responses to children's negative emotions: Family and physiological correlates. *Family Relations, 64*, 431-445.

Bolger, N., Davis, A., & Rafaeli, E. (2003). Diary methods: Capturing life as it is lived. *Annu. Rev. Psychol., 54*, 579-616.

Bolger, N., & Laurenceau, J.P. (2013). *Intensive longitudinal methods: An introduction diary and experience sampling research*. Guilford Press.

Bonnemeier, H., Wiegand, U.K.H., Brandes, A., Kluge, N., Katus, H.A., Richardt, G., & Potratz, J. (2003). Circadian profile of cardiac autonomic nervous modulation in healthy subjects: Differing effects of aging and gender on heart rate variability. *Journal of Cardiovascular Electrophysiology, 14*, 791-799.

Bonnet, M. H., & Arand, D. L. (2003). Situational insomnia: Consistency, predictors, and outcomes. *Sleep, 26*, 1029-1036.

Bridgett, D.J., Burt, N.M., Edwards, E.S., & Deater-Deckard, K. (2015). Intergenerational transmission of self-regulation: A multidisciplinary review and integrative conceptual framework. *Psychological Bulletin, 141*, 602-654.

Brobst, J. B., Clopton, J. R., & Hendrick, S. S. (2009). Parenting children with autism spectrum disorders: The couple's relationship. *Focus on Autism and Other Developmental Disabilities*, *24*(1), 38–50.

Buysse, D. J. (2014). Sleep health: Can we define it? Does it matter? *Sleep, 37*, 9-17.

Buysse, D. J., Ancoli-lsrael, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2006). Recommendations for a standard research assessment of insomnia. *Sleep, 29,* 1155-1173.

Bylsma, L. M., Salomon, K., Taylor-Clift, A., Morris, B. H., & Rottenberg, J. (2014). Respiratory sinus arrhythmia reactivity in current and remitted major depressive disorder. *Psychosomatic Medicine, 76*, 66-73.

Caldwell, W., da Estrela, C., MacNeil, S., & Gouin, J.P. (2019). Association between romantic partners' rumination and couples' conflict is moderated by respiratory

sinus arrhythmia: An actor-partner interdependence model. *Journal of Family Psychology.*

Caldwell, Y.T., & Steffen, P.R. (2018). Adding HRV biofeedback to psychotherapy increases heart rate variability and improves the treatment of major depressive disorder. *Journal of Psychophysiology, 131*, 96-101.

Calkins, S.D. (2010). Commentary: Conceptual and methodological challenges to the study of emotion regulation and psychopathology. *Journal of Psychopathology and Behavioural Assessment, 32,* 92-95.

Carnevali, L., Thayer, J.F., Brosschot, J.F., & Ottaviani, C. (2018). Heart rate variability mediates the link between rumination and depressive symptoms: A longitudinal study. *International Journal of Psychophysiology, 131*, 131-138.

Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The Consensus Sleep Diary: Standardizing prospective sleep self-monitoring. *Sleep, 35,* 287-302.

Carney, R.M., Freedland, K.E., Stein, P.K., Skala, J.A., Hoffman, P., & Jaffe, A.S. (2000). Change in heart rate variability during treatment for depression in patients with coronary heart disease. *Psychosomatic Medicine, 62*, 639-647.

Castro-Diehl, C., Diez Roux, A. V., Redline, S., Seeman, T., McKinley, P., Sloan, R., & Shea, S. (2016). Sleep duration and quality in relation to autonomic nervous system measures: The Multi-Ethnic Study of Atherosclerosis (MESA). *Sleep*, *39*, 1927-1940.

Charles, S.T., Piazza, J.R., Mogle, J., Sliwinski, M.J., & Almeida, D.M. (2013). The wear and tear of daily stressors on mental health. *Psychological Science, 24*, 733-741.

Cicchetti, D., & Dawson, G. (2002). Editorial: Multiple levels of analysis. *Development and Psychopathology, 14,* 417-420.

Ciciolla, L., Gerstein, E.D., & Crnic, K.A. (2014). Reciprocity among maternal distress, child behaviour, and parenting: Transactional processes and early childhood risk. *Journal of Clinical Child & Adolescent Psychology, 43*, 751-764.

Cohen, L.H., Gunthert, K.C., Butler, A.C., O'Neill, S.C., & Tolpin, L.H. (2005). Daily affective reactivity as a prospective predictor of depressive symptoms. *Journal of Personality, 73*, 1687-1714.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75,* 317-333.

Connell, A.M., Hughes-Scalise, A., Klostermann, S., & Azem, T. (2011). Maternal depression and the heart of parenting: Respiratory sinus arrhythmia and affective dynamics during parent–adolescent interactions. *Journal of Family Psychology, 25,* 653-662.

Connell, A.M., McKillop, H., Patton, E., Klostermann, S., & Hughes-Scalise, A. (2015). Actor–partner model of physiology, negative affect, and depressive symptoms in mother–child dyadic interactions. *Journal of Social and Personal Relationships, 32*, 1012-1033.

Cunningham, J., & Shapiro, C. M. (2018). Cognitive Behavioural Therapy for Insomnia (CBT-I) to treat depression: A systematic review. *Journal of Psychosomatic Research, 106,* 1-12.

da Estrela, C., Barker, E. T., Lantagne, S., & Gouin, J.-P. (2017). Chronic parenting stress and mood reactivity: The role of sleep quality. *Stress & Health.*

da Estrela, C., McGrath, J.J., Booij, L., & Gouin, J.P. (2020). Heart rate variability, sleep quality, and depression in the context of chronic stress. *Annals of Behavioural Medicine.*

de Lange, A.H., Kompier, M.A., Taris, T.W., Geurts, S.A., Beckers, D.G., Houtman, I.L., & Bongers, P.M. (2009). A hard day's night: A longitudinal study on the relationships among job demands and job control, sleep quality and fatigue. *Journal of Sleep Research, 18*, 374-383.

Demaree, H.A., Pu, J., Robinson, J.L., Schmeichel, B.J., & Everhart, D.E. (2006). Predicting facial valence to negative stimuli from resting RSA: Not a function of active emotion regulation. *Cognition and Emotion, 20*, 161-176.

Demaree, H.A., Robinson, J.L., Everhart, D.E., & Schmeichel, B.J. (2004). Resting RSA is associated with natural and self-regulated responses to negative emotional stimuli. *Brain and Cognition, 56*, 14-23.

Diamond, L.M., Hicks, A.M., & Otter-Henderson, K.D. (2011). Individual differences in vagal regulation moderate associations between daily affect and daily couple interactions. *Personality and Social Psychology Bulletin, 37*, 731-744.

Dishman, R. K., Nakamura, Y., Garcia, M.E., Thompson, R.W., Dunn, A.L., & Blair, S.N. (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *International Journal of Psychophysiology, 37*, 121-133.

Dix, T. (1991). The affective organization of parenting: Adaptive and maladaptive processes. *Psychol Bull, 110*, 3-25.

Dodds, K. L., Miller, C. B., Kyle, S. D., Marshall, N. S., & Gordon, C. J. (2017). Heart rate variability in insomnia patients: A critical review of the literature. *Sleep Medicine Reviews, 33,* 88-100.

Drake, C. L., Friedman, N. P., Wright, K. P., Jr., & Roth, T. (2011). Sleep reactivity and insomnia: Genetic and environmental influences. *Sleep, 34*, 1179-1188.

Drake, C.L., Pillai, V., & Roth, T. (2014). Stress and sleep reactivity: A prospective investigation of the stress-diathesis model of insomnia. *Sleep, 37,* 1295-1304.

Drake, C., Richardson, G., Roehrs, G., Scofield, T., & Roth, T. (2004). Vulnerability to stress-related sleep disturbance and hyperarousal. *Sleep, 27*, 285-291.

Drake, C.L., Roehrs, T., Roth, T. (2003). Insomnia causes, consequences, and therapeutics: An overview. *Depression & Anxiety, 18*, 163-176.

Elam, K.K., Chassin, L., Eisenberg, N., & Spinrad, T.L. (2017). Marital stress and children's externalizing behaviour as predictors of mothers' and fathers' parenting. *Development and Psychopathology, 29*, 1305-1318.

Ellis, A.J., Shumake, J., & Beevers, C.G. (2016). The effects of respiratory sinus arrhythmia on anger reactivity and persistence in major depression. *Psychophysiology, 53*, 1587-1599.

Elmore-Staton, L., El-Sheikh, M., Vaughn, B., & Arsiwalla, D. D. (2012). Preschoolers' daytime respiratory sinus arrhythmia and nighttime sleep. *Physiology & Behaviour, 107*, 414-417.

El-Sheikh, M., Erath, S.A., & Bagley, E.J. (2013). Parasympathetic nervous system activity and children's sleep. *Journal of Sleep Research, 22,* 282-288.

El-Sheikh, M., Hinnant, J.B., & Erath, S.A. (2015). VI. Marital conflict, vagal regulation, and children's sleep: A longitudinal investigation. *Monographs of the Society for Research in Child Development, 80*, 89-106.

Enkavi, A.Z., Eisenberg, I.W., Bissett, P.G., Mazza, G.L., MacKinnon, D.P.,… Poldrack, R.A. (2019). Large-scale analysis of test-retest reliabilities of self-regulation measures. *PNAS, 116*, 5472-5477.

Epel, E.S., Crosswell, A.D., Mayer, S.E., Prather, A.A., Slavich, G.M., Puteran, E., & Mendes, W.B. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology, 49*, 146-169.

Evenson, R.J., & Simon, R.W. (2005). Clarifying the relationship between parenthood and depression. *Journal of Health & Social Behaviour, 46*, 341-358.

Fabes, R.A, Eisenberg, N. (1997). Regulatory control and adults' stress related responses to daily life events. *Journal of Personality and Social Psychology, 73*, 1107-1117.

Fagundes, C.P., Murray, D.M., Seuk Hwang, B., Gouin, J.P., Thayer, J.F., Sollers III, … Kiecolt-Glaser, J.K. (2011). Sympathetic and parasympathetic activity in cancer-related fatigue: More evidence for a physiological substrate in cancer survivors. *Psychoneuroendocrinology, 36*, 1137-1147.

Garakani, A., Martinez, J.M., Aaronson, C.J., Voustianiouk, A., Kaufmann, H., & Gorman, J. (2008). Effect of medication and psychotherapy on heart rate variability in panic disorder. *Depression & Anxiety, 26,* 251-258.

Geisler, F.C.M., Kubiak, T., Siewert, K., & Weber, H. (2013). Cardiac vagal tone is associated with social engagement and self-regulation. *Biological Psychology, 93*, 279-286.

Geisler, F.C.M., Vennewald, N., Kubiak, T., & Weber, H. (2010). The impact of heart rate variability on subjective well-being is mediated by emotion regulation. *Personality and Individual Differences*, *49,* 723-728.

Goetz, G. L., Rodriguez, G., & Hartley, S. L. (2019). Actor-partner examination of daily parenting stress and couple interactions in the context of child autism. *Journal of Family Psychology, 33,* 554-564.

Gollwitzer, P.M., Mayer, D., Frick, & Oettingen, G. (2018). Promoting the self-regulation of stress in health care providers: An internet-based intervention. *Frontiers in Psychology, 9*.

Goodman, S.H., Rouse, M.H., Connell, A.M., Broth, M.R., Hall, C.M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child & Family Psychology Review, 14*, 1-27.

Gorka, S.M., Nelson, B.D., Sarapas, C., Campbell, M., Lewis, G.F., Bishop, J.R., Porges, S.W., & Shankman, S.A. (2013). Relation between respiratory sinus arrythymia and startle response during predictable and unpredictable threat. *Journal of Psychophysiology, 27*, 95-104

Gouin, J.P., Caldwell, W.C, MacNeil, S.L., & Roddick, C.M. (2018). Respiratory sinus arrhythmia reactivity moderates within-person associations of daily capitalization with positive affect and relationship quality. *Journal of Social and Personal Relationships, 36*, 2896-2917.

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

Gouin, J.-P., Wenzel, K., Boucetta, S., O'Byrne, J., Salimi, A., & Dang-Vu, T.T. (2015). High-frequency heart rate variability during worry predicts stress-related increases in sleep disturbances. *Sleep Medicine, 16*, 659-664.

Gouin, J.P., Zhou, B., & Fitzpatrick, S. (2015). Social integration prospectively predicts changes in heart rate variability among individuals undergoing migration stress. *Annals of Behavioural Medicine, 49*, 230-238.

Grandner, M. A., Patel, N. P., Gehrman, P. R., Xie, D., Sha, D., Weaver, T., & Gooneratne, N. (2010). Who gets the best sleep? Ethnic and socioeconomic factors related to sleep complaints. *Sleep Medicine, 11*, 470-478.

Gross, H.E., Shaw, D.S., Burwell, R.A., & Nagin, D.S. (2009). Transactional processes in child disruptive behaviour and maternal depression: A longitudinal study from early childhood to adolescence. *Developmental Psychopathology, 21*, 139-156.

Hall, M., Buysse, D. J., Nofzinger, E. A., Reynolds, C. F. III, Thompson, W., Mazumdar, S., & Monk, T. H. (2008). Financial strain is a significant correlate of sleep continuity disturbances in late-life. *Biological Psychology, 77*, 217-222.

Hall, M. H., Casement, M. D., Troxel, W. M., Matthews, K. A., Bromberger, J. T., Kravitz, H. M., . . . Buysse, D. J. (2015). Chronic stress is prospectively associated with sleep in midlife women: The SWAN sleep study. *Sleep, 38*, 1645-1654.

Hall, M., Thayer, J. F., Germain, A., Moul, D., Vasko, R., Puhl, M., . . . Buysse, D. J. (2007). Psychological stress is associated with heightened physiological arousal during NREM sleep in primary insomnia. *Behavioural Sleep Medicine, 5*, 178-193.

Hamilton, J.L., Stange, J.P., Burke, T.A., Franzen, P.L., & Alloy, L.B. (2019). Sleep disturbance and physiological regulation among young adults with prior depression. *Journal of Psychiatric Research, 115*, 75-81.

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology, 1,* 293-319.

Hammen, C. (2006). Stress generation in depression: Reflections on origins, research, and future directions. *Journal of Clinical Psychology, 62*, 1065-1082.

Hartley, S.L., Papp, L.M., & Bolt, D. (2018). Spillover of marital interactions and parenting stress in families of children with autism spectrum disorder. *J Clin Child Adolesc Psychol., 47*, S88-S89.

Harvey, C.J. (2011). Sleep and circadian functioning: Critical mechanisms in the mood disorders? *Annual Review of Clinical Psychology, 7*, 297-319.

Harvey, A. G., Stinson, K., Whitaker, K. L., Moskovitz, D., & Virk, H. (2008). The subjective meaning of sleep quality: A comparison of individuals with and without insomnia. *Sleep, 31*, 383-393.

Hayes, AF. (2013). *Methodology in the social sciences. Introduction to mediation, moderation, and conditional process analysis: A regression-based approach.* Guilford Press.

Herring, S., Gray, K., Taffe, J., Tonge, B., Sweeney, D., & Einfeld, S. (2006). Behaviour and emotional problems in toddlers with pervasive developmental disorders and developmental delay: Associations with parental mental health and family functioning. *Journal of Intellectual Disability Research, 50*, 874-882.

Hill, R. (1949). *Families under stress*. New York: Harper and Row.

Hilliard, M.E., Monaghan, M., Cogen, F.R., & Streisand, R. (2011). Parent stress and child behaviour among young children with type 1 diabetes. *Child: Care, Health and Development*, *37*, 224-232.

Hives, B.A., Buckler, E.J., Weiss, J., Schilf, S., Johansen, K.L., … Puterman, E. (2020). The effects of aerobic exercise on psychological functioning in family caregivers: Secondary analyses of a randomized controlled trial. *Annals of Behavioural Medicine,* kaaa031.

Hobfoll, S.E., & Spielberger, C.D. (1992). Family stress: Integrating theory and measurement. *Journal of Family Psychology, 6*, 99-112.

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

Hofmann, W., Schmeichel, B.J., & Baddeley, A.D. (2012). Executive functions and self-regulation. *Trends in Cognitive Science, 16*, 174-180.

Hoffmann, S. G., Schulz, S. M., Heering, S., Muench, F., & Bufka, L. F. (2010). Psychophysiological correlates of generalized anxiety disorder with or without comorbid depression. *International Journal of Psychophysiology, 78*, 35-41.

Holzman, J.B., & Bridgett, D.J. (2017). Heart rate variability indices as bio-markers of top-down self-regulatory mechanisms: A meta-analytic review. *Neuroscience & Biobehavioural Reviews,* 74.

Hopp, H., Shallcross, A.J., Ford, B.Q., Troy, A.S., Wilhelm, F.H., & Mauss, I.B. (2013). High cardiac vagal control protects against future depressive symptoms under conditions of high social support. *Biological Psychology, 93*, 143-149.

Horsten, M., Ericson, M., Perski, A., Wamala, S.P., Schenck-Gustaffson, K., & Orth-Gomér, K. (1999). Psychosocial factors and heart rate variability in health women. *Psychosomatic Medicine, 61*, 49-57.

Hovland, A., Pallesen, S., Hammar, Å., Hansen, A. L., Thayer, J. F., Sivertsen, B., . . . Nordhus, I. H. (2013). Subjective sleep quality in relation to inhibition and heart rate variability in patients with panic disorder. *Journal of Affective Disorders, 150*, 152-155.

Jansson-Fröjmark, M., & Lindblom, K. (2008). A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population. *Journal of Psychosomatic Research, 64,* 443-449.

Joosen, K.J., Mesman, J., Bakermans-Kranenburg, M.J., Pieper, S., Zesking, P.S., & van IJzendoorn, M.H. (2013). Physiological reactivity to infant crying and observed maternal sensitivity. *Infancy, 18*, 414-431.

Kalimo, R., Tenkanen, L., Härmä, M., Poppius, E., & Heinsalmi, P. (2000). Job stress and sleep disorders: Findings from the Helsinki Heart Study. *Stress Medicine, 16*, 65-75.

Kalmbach, D. A., Anderson, J. R., & Drake, C. L. (2018). The impact of stress on sleep: Pathogenic sleep reactivity as a vulnerability to insomnia and circadian disorders. *Journal of Sleep Research, 6*, 1-21.

Kanner, A.D., Coyne, J.C., Schaefer, C., & Lazarus, R.S. (1981). Comparison of two modes of stress measurement: Daily hassles and uplifts versus major life events. *Journal of Behavioural Medicine, 4*, 1-39.

Kaplan, R.M., & Kronick, R.G. (2006). Marital status and longevity in the United States population. *Journal of Epidemiology & Community Health, 60*, 760-765.

Karst, J. S., & Van Hecke, A. V. (2012). Parent and family impact of autism spectrum disorders: A review and proposed model for intervention evaluation. *Clinical Child and Family Psychology Review, 15*, 247-277.

Keller, P.S., Kouros, C.D., Erath, S.A., Dahl, R.E., & El-Sheikh, M. (2014). Longitudinal relations between maternal depressive symptoms and child sleep problems: The role of parasympathetic nervous system reactivity. *Journal of Child Psychology & Psychiatry, 55,* 172-179.

Kemp, A.H., Quintana, D.S., Gray, M.A., Felmingham, K.L., Brown, K., & Gatt, J.M. (2010). Impact of depression and antidepressant treatment on heart rate variability: A review and meta-analysis. *Biological Psychiatry, 67*, 1067-1074.

Kenny, D.A., Kashy, D.A., & Cook, W.L. (2006). *Dyadic data analysis.* New York: NY: Guilford Press.

Kiecolt-Glaser, J.K., & Wilson, S.J. (2017). Lovesick: How couples' relationships influence health. *Annu. Rev. Clin. Psychol., 13*, 421-443.

Kim, E.-J., & Dimsdale, J. E. (2007). The effect of psychosocial stress on sleep: A review of polysomnographic evidence. *Behavioural Sleep Medicine, 5*, 256-278.

Kim, H.G., Cheon, E.J., Bai, D.S., Lee, Y.H., & Koo, B.H. (2018). Stress and heart rate variability: A meta-analysis and review of the literature. *Psychiatry Investigation, 15*, 235-245.

Kleiger, R.E., Bigger, J.T., Bosner, M.S., Chung, M.K., Cook, J.R., Rolnitzky, L.M., … Fleiss, J.L. (1991). Stability over time of variables measuring heart rate variability in normal subjects. *The American Journal of Cardiology, 68*, 626-630.

Koch, C., Wilhelm, M., Salzmann, S., Rief, W., & Euteneuer, F. (2019). A meta-analysis of heart rate variability in major depression. *Psychological Medicine,* 1948-1957.

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

Kok, B.E., & Fredrickson, B.L. (2010). Upward spirals of the heart: Autonomic flexibility, as indexed by vagal tone, reciprocally and prospectively predicts positive emotions and social connectedness. *Biological Psychology, 85*, 432-436.

Kovacs, M., Yaroslavsky, I., Rottenberg, J., George, C.J., Kiss, E., Halas, K., … Kapornai, K. (2016). Maladaptive mood repair, atypical respiratory sinus arrhythmia, and risk of a recurrent major depressive episode among adolescents with prior major depression. *Psychological Medicine, 46*, 2109-2119.

Laurenceau, J.P., & Bolger, N. (2005). Using diary methods to study marital and family processs. *Journal of Family Psychology, 19*, 86-97.

Lampert, R., Tuit, K., Hong, K., Donovan, T., Lee, F., & Sinha, R. (2016). Cumulative stress and autonomic dysregulation in a community sample. *Stress, 19*, 269-279.

Laurenceau, J.P., & Bolger, N. (2005). Using diary methods to study marital and family processs. *Journal of Family Psychology, 19*, 86-97.

Lazarus R.S, Folkman S. (1984) *Stress, appraisal, and coping.* New York, NY: Springer.

Leigh, B., & Milgrom, J. (2008). Risk factors for antenatal depression, postnatal depression and parenting stress. *BMC Psychiatry, 8*.

Lewis, A.R., Zinbarg, R.E., & Durbin, C.E. (2010). Advances, problems, and challenges in the study of emotion regulation: A commentary. *Journal of Psychopathology and Behavioural Assessment, 32*, 83-91.

Lin, L.X., Zhang, Y., Chi, P., Ding, W., Heath, M.A., Fang, X., & Xu, S. (2017). The mutual effect of marital quality and parenting stress on child and parent depressive symptoms in families of children with oppositional defiant disorder. *Front. Psychol., 8*,

Linton, S.J. (2010). Does work stress predict insomnia? A prospective study. *British Journal of Health Psychology, 9*, 127-136.

Lopez-Wagner, M. C., Hoffman, C. D., Sweeney, D. P., Hodge, D., & Gilliam, J. E. (2008). Sleep problems of parents of typically developing children and parents of children with autism. *The Journal of Genetic Psychology, 169*, 245-259.

Lorber, M.F. (2012). The role of maternal emotion regulation in overactive and lax discipline. *Journal of Family Psychology, 26*, 642-647.

Loutzenhiser, L., McAuslan, P., & Sharpe, D.P. (2015). The trajectory of maternal and paternal fatigue and factors associated with fatigue across the transition to parenthood. *Clinical Psychologist, 19*, 15-27.

MacNeil, S., Deschênes, S.S., Caldwell, W., Brouillard, M., Dang-Vu, T.T., & Gouin, J.-P. (2017). High-frequency heart rate variability reactivity and trait worry interact to predict the development of sleep disturbances in response to natural stressor. *Annals of Behavioural Medicine, 51*, 912-924.

Martin, J., Shochat, T., & Ancoli-Israel, S. (2000). Assessment and treatment of sleep disturbances in older adults. *Clinical Psychology Review, 20*, 783-805.

Masarik, A.S., & Conger, R.D. (2017). Stress and child development: A review of the Family Stress Model. *Current Opinions in Psychology 13*, 85-90.

Mazursky-Horowitz, H., Felton, J.W., MacPherson, L., Ehrlich, K.B., Cassidy, J., Lejuez, C.W., & Chronis-Tuscano, A. (2015). Maternal emotion regulation mediates the association between adult attention-deficit/hyperactivity disorder symptoms and parenting. *Journal of Abnormal Child Psychology, 43*, 121-131.

McCubbin, H.I., & Patterson, J.M. (1983). The family stress process: The Double ABCX Model of adjustment and adaptation. *Marriage & Family Review, 6*, 7–37.

McLaughlin, K.A., Rith-Najarian, L., Dirks, M.A., & Sheridan, M.A. (2015). Low vagal tone magnifies the association between psychosocial stress exposure and

internalizing psychopathology in adolescents. *Journal of Clinical Child & Adolescent Psychology, 44*, 314-328.

Melzig, C.A., Weike, A.I., Hamm, A.O., & Thayer, J.F. (2009). Individual differences in fear-potentiated startle as a function of resting heart rate variability: Implications for panic disorder. International Journal of Psychophysiology, *71*, 109-117.

Mezulis, A.H., Crystal, S.I., Ahles, J.J., & Crowell, S.E. (2015). Examining biological vulnerability in environmental context: Parenting moderates effects of low resting respiratory sinus arrhythmia on adolescent depressive symptoms. *Developmental Psychobiology, 57*, 974-983.

Minkel, J. D., Banks, S., Htaik, O., Moreta, M. C., Jones, C. W., McGlinchey, E. L., . . . Dinges, D. F. (2012). Sleep deprivation and stressors: Evidence for elevated negative affect in response to mild stressors when sleep deprived. *Emotion, 12*, 1015-1020.

Mitnick, D.M., Heyman, R.E., & Slep, A.M.S. (2009). Changes in relationship satisfaction across the transition to parenthood: A meta-analysis. *J Fam Psychol., 23*, 848-852.

Morin, C. M., Vallières, A., Guay, B., Ivers, H., Savard, J., Mérette, C., … Baillargeon, L. (2009). Cognitive behavioural therapy, singly and combined with medication, for persistent insomnia: A randomized controlled trial. *Journal of the American Medical Association, 301*, 2005-2015.

Neece, C.L., & Chan, N. (2017). The Stress of Parenting Children with Developmental Disabilities. In K. Deater-Deckard & R. Panneton (Eds.). *Parental stress and early child development* (p. 107-124). Springer International Publishing.

Neece, C.L., Green, S.A., & Baker, B.L. (2012). Parenting stress and child behaviour problems: A transactional relationship across time. *Am J Intellect Dev Disabil, 117*, 48-66.

Nelson, J.A., O'Brien, M., Blankson, A.N., Calkins, S.D., & Keane, S.P. (2009). Family stress and parental responses to children's negative emotions: Tests of the spillover, crossover, and compensatory hypotheses. *Journal of Family Psychology, 23*, 671-679.

Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Science, 10*, 173-176.

Nomaguchi, K. M., & Milkie, M. A. (2003). Costs and rewards of children: The effects of becoming a parent on adults' lives. *Journal of Marriage and Family, 65,* 356-374.

Nowak, C. & Heinrichs, N. (2008). A comprehensive meta-analysis of Triple P-Positive Parenting Program using hierarchical linear modeling: Effectiveness and moderating variables. *Clinical Child & Family Psychology Review, 11*, 114-144.

Olsson, M. B., & Hwang, C. P. (2001). Depression in mothers and fathers of children with intellectual disability. *Journal of Intellectual Disability Research, 45,* 535-543.

Parrish, B.P., Cohen, L.H., & Laurenceau, J.P. (2011). Prospective relationship between negative affective reactivity to daily stress and depressive symptoms. *Journal of Social and Clinical Psychology, 30*, 270-296.

Pastor-Cerezuela, G., Fernández-Andrés, M. I., Tárraga-Mínguez, R., & Navarro-Peña, J. M. (2016). Parental stress and ASD: Relationship with autism symptom severity, IQ, and resilience. *Focus on Autism and Other Developmental Disabilities, 31*, 300-311.

Perry, A. (2004). A model of stress in families of children with developmental disabili*ties:* Clinical and research applications. *Journal of Developmental Disabilities, 11*, 1-15

Pillai, V., Roth, T., Mullins, H.M., & Drake, C.L. (2014). Moderators and mediators of the relationship between stress and insomnia: Stressor chronicity, cognitive intrusion, and coping. *Sleep, 37*, 1199-1208A.

Podsakoff, P. M., MacKenzie, S. B., Lee, J.-Y., & Podsakoff, N. P. (2003). Common method biases in behavioural research: A critical review of the literature and recommended remedies. *Journal of Applied Psychology, 88*, 879-903

Ponnet, K., Mortelmans, D., Wouters, E., Van Leeuwen, K., Bastaits, K., & Pasteels (2013). Parenting stress and marital relationship as determinants of mothers' and fathers' parenting. *Personal Relationships, 20*, 259-276.

Porges, S., Bohrer, RE., Cheung, MN., Drasgow, F., Mccabe, PM., & Keren, G. (1980). New time-series statistic for detecting rhythmic co-occurrence in the frequency-domain - The weighted coherence and its application to psychophysiological research. *Psychological Bulletin, 88,* 580-587.

Quittner, A. L., Glueckauf, R. L., & Jackson, D. N. (1990). Chronic parenting stress: Moderating versus mediating effects of social support. *Journal of Personality and Social Psychology, 59,* 1266-1278.

Quittner, A.L., Barker, D.H., Cruz, I., Snell, C., Grimley, M.E., Botteri, M., & CDaCI Investigative Team. (2010). Parenting stress among parents of deaf and hearing children: Associations with language delays and behaviour problems. *Parenting: Science and Practice, 10,* 136-155.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401.

Raffin, J., Barthélémy, J.C., Dupré, C., Pichot, V., Berger, M., … Hupin, D. (2019). Exercise frequency determines heart rate variability gains in older people: A meta-analysis and meta-regression. *Sports Medicine, 49*, 719-729.

Riemann, D., Krone, L.B., Wulff, K., & Nissen, C. (2019). Sleep, insomnia, and depression. *Neuropsychopharmacology*.

Riemann, Spiegelhalder, Feige, Voderholzer, Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews, 14*, 19-31.

Root, A.E., Hastings, P.D., & Rubin, K.H. (2016). The parenting behaviours of shy-anxious mothers: The moderating role of vagal tone. *Journal of Child and Family Studies, 25*, 1325-1333.

Rosenbaum, M. (1989). Self-control under stress: The role of learned resourcefulness. *Adv. Behav. Res. Ther. 11*, 249-258.

Roskam, I., Raes, M-E., & Mikolajczak, M. (2017). Exhausted parents: Development and preliminary validation of the Parental Burnout Inventory. *Frontiers in Psychology, 8.*

Rutherford, H.J.V., Wallace, N.S., Laurent, H.K., & Mayers, L.C. (2015). Emotion regulation in parenthood. *Developmental Review, 36*, 1-14.

Sameroff, A. (1975). Transactional models in early social relations. *Human Development, 18*, 65-79.

Sevigny, P.R., & Loutzenhiser, L. (2010). Predictors of parenting self-efficacy in mothers and fathers of toddlers. *Child: Care, Health and Development, 36*, 179-189.

Scharlach, A. E. (2001). Role strain among working parents: Implications for workplace and community. *Community, Work & Family, 4*, 215–230.

Schiweck, C., Piette, D., Berckmans, D., Claes, S., & Vrieze, E. (2018). Heart rate and high frequency heart rate variability during stress as biomarker for clinical depression. A systematic review. *Psychological Medicine,* 49, 200-211.

Scott, J.P.R., McNaughton, L.R., & Polman, R.C.J. (2006). Effects of sleep deprivation and exercise on cognitive, motor performance and mood. *Physiology & Behaviour, 87*, 396-408.

Segerstrom, S.C., & Nes, L.S. (2007). Heart rate variability reflects self-regulatory strength, effort, and fatigue. *Psychological Science, 18*, 275-281.

Shaffer, A., & Obradović, J. (2017). Unique contributions of emotion regulation and executive functions in predicting the quality of parent-child interactions behaviours. *Journal of Family Psychology,* 31*,* 150-159.

Shaffer, A., Whitehead, M., Davis, M., Morelen, D., & Suveg, C. (2017). A model-based cluster analysis of maternal emotion regulation and relations to parenting behaviour. *Family Process*, *57*, 707-718.

Sloan, R.P., Huang, M.-H., McCreath, H. Sidney, S., Liu, K., Williams, D., & Seeman, 2008. Cardiac autonomic control and the effects of age, race, and sex: The CARDIA study. *Autonomic Neuroscience, 139*, 78-85.

Smith, T.W., Deits-Lebehn, C., Williams, P.G., Baucom, B.R., & Uchino, B.N. (2020). Towards a social psychophysiology of vagally-mediated heart rate variability: Concepts and methods in self-regulation, emotion, and interpersonal processes. *Social and Personality Psychology Compass, 14*, e12516.

Suárez, L.M., & Baker, B.L. (1997). Child externalizing behaviour and parents' stress: The role of social support. *Family Relations, 46*, 373-381.

Suveg, C., Braunstein West, K., Davis, M., Caughy, M., Smith, E. P., & Oshri, A. (2019). Symptoms and synchrony: Mother and child internalizing problems moderate

respiratory sinus arrhythmia concordance in mother–preadolescent dyads. *Developmental Psychology, 55,* 366-376.

Stroud, C.B., Meyers, K.M., Wilson, S., & Durbin, C.E. (2015). Marital quality spillover and young children's adjustment: Evidence for dyadic and triadic parenting as mechanisms. *J Clin Child Adolesc Psychol., 44*, 800-813.

Swanson, L. M., Flynn, H., Adams-Mundy, J. D., Armitage, R., & Arnedt, J. T. (2013). An open pilot of cognitive-behavioural therapy for insomnia in women with postpartum depression. *Behavioural Sleep Medicine, 11*, 297-307.

Switzer, A., Caldwell, W., da Estrela, C., Barker, E.T., & Gouin, J.-P. (2018). Dyadic coping, respiratory sinus arrhythmia, and depressive symptoms among parents of preschool children. *Front Psychol., 16*, 1959.

Taylor, D.J. & Pruiksma, K.E. (2014). Cognitive and behavioural therapy for insomnia (CBT-I) in psychiatric populations: A systematic review. *International Review of Psychiatry*, *26*, 205-213.

Taylor, C.B., Conrad, A., Wilhelm, F.H., Strachowski, D., Khaylis, A., Neri, E., … Spiegal, D. (2009). Does improving mood in depressed patients alter factors that may affect cardiovascular disease risk? *Journal of Psychiatric Research, 43*, 1246-1252.

Taylor, D., Pruiksma, K., Hale, W., Kelly, K., Maurer, D., Peterson, A.L., … STRONG STAR Consortium. (2016). Prevalence, correlates, and predictors of insomnia in the US army prior to deployment. *Sleep, 39*, 1795-1806.

Thayer, J.F., Åhs, F., Fredrikson, M., Sollers III, J.J., & Wager, T.D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioural Reviews, 36*, 747-756.

Thayer, J.F., Hansen, A.L., Saus-Rose, E., & Johnsen, B.H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioural Medicine, 37*, 141-153.

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

Umberson, D., Chen, M.D., House, J.S., Hopkins, K., & Slaten, E. (1996). The effects of social relationships on psychological well-being: Are men and women really so different? *American Sociological Review, 61,* 837-857.

Umberson, D., Pudrovska, T., & Reczek, C. (2010). Parenthood, childlessness, and well-being: A life course perspective. *Journal of Marriage & Family, 72*, 612-629.

Vargas, I., Friedman, N.P., & Drake, C.L. (2015). Vulnerability to stress-related sleep disturbance and insomnia: Investigating the link with comorbid depressive symptoms. *Translational Issues in Psychological Science, 1*, 57-66.

Vasilev, C.A., Crowell, S.E., Beauchaine, T.P., Mead, H.K., & Gatzke-Kopp, L.M. (2009). Correspondence between physiological and self-report measures of emotion dysregulation: A longitudinal investigation of youth with and without psychopathology. *Journal of Child Psychology & Psychiatry, 50*, 1357-1364.

Verlander, L. A., Benedict, J. O., & Hanson, D. P. (1999). Stress and sleep patterns of college students. *Perceptual and Motor Skills, 88*, 893-898.

Visted, E., Sørensen, K., Osnes, B., Svendsen, J.L., Binder, P.E., & Schanche, E. (2017). The association between self-reported difficulties in emotion regulation and heart rate variability: The salient role of not accepting negative emotions. *Frontiers in Psychology, 8*. Page nr ?

Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin, 129*, 946-972.

Volokhov, R.N., & Demaree, H.A. (2010). Spontaneous emotion regulation to positive and negative stimuli. *Brain and Cognition, 73*, 1-6.

Wang, Z., Deater-Deckard, K., & Bell, M.A. (2016). The role of negative affect and physiological regulation in maternal attribution. *Parenting*, 206-218.

Weber, C.S., Thayer, J.F., Rudat, M., Wirtz, P.H., Zimmermann-Viehoff,F., Thomas, A., … Deter, H.C. (2010). Low vagal tone is associated with impaired post stress recovery of cardiovascular, endocrine, and immune markers. *European Journal of Applied Physiology, 109*, 201-211.

Werner, G. G., Ford, B. Q., Mauss, I. B., Schabus, M., Blechert, J., & Wilhelm, F. H. (2015). High cardiac vagal control is related to better subjective and objective sleep quality. *Biological Psychology, 106*, 79-85.

Werner, G. G., Ford, B. Q., Mauss, I. B., Schabus, M., Blechert, J., & Wilhelm, F. H. (2017). Cardiac vagal control and depressive symptoms: The moderating role of sleep quality. *Behavioural Sleep Medicine, 6*, 451-465.

Williams, D.P., Cash, C., Rankin, C., Bernardi, A., Koenig, J., & Thayer, J.F. (2015). Resting heart rate variability predicts self-reported difficulties in emotion regulation: A focus on different facets of emotion regulation. *Frontiers in Psychology, 6*.

Williams, D.P., Tracy, L.M., Gerardo, G.M., Rahman, T., Spangler, D.P., … Thayer, J.F. (2019). Sex moderates the relationship between resting heart rate variability and self-reported difficulties in emotion regulation. *Emotion, 19*, 992-1001.

Wilson, S.T., Chesin, M., Fertuck, E., Keilp, J., Brodsky, B., … Stanley, B. (2016). Heart rate variability and suicidal behaviour. *Psychiatry Research, 240*, 241-247.

Winzeler, K., Voellmin, A., Schäfer, V., Meyer, A. H., Cajochen, C., Wilhelm, F. H., & Bader, K. (2014). Daily stress, presleep arousal, and sleep in healthy young women: A daily life computerized sleep diary and actigraphy study. *Sleep Medicine, 15*, 359-366.

Wohlgemuth, W. K., Edinger, J. D., Fins, A. I., & Sullivan, R. J. (1999). How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. *Psychophysiology, 36*, 233-244.

Yaroslavsky, I., Rottenberg, J., Bylsma, L.M., Jennings, J.R., George, C., Baji, I., … Kovacs, M. (2016). Parasympathetic nervous system activity predicts mood repair use and is effectiveness among adolescents with and without histories of major depression. *Journal of Abnormal Psychology, 125*, 323-336.

Zahn, D., Adams, J., Krohn J., Wenzel, M., Mann, C.G., Gomille, L.K., … Kubiak, T. (2016). Heart rate variability and self-control – A meta-analysis. *Biological Psychology, 115*, 9-26.

Zhang, B., & Wing, Y.K. (2006). Sex differences in insomnia: A meta-analysis. *Sleep, 29*, 85-93.

Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: A cognitive-energy model. *Sleep*, *28*, 47-54.