Adult women's age differences in links between behavioral and physiological indicators of cognitive regulation.

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A Dissertation Presented

By

Jennifer D Christensen

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DEDICATION

To my husband, three daughters, and son-in-law.

They are my joy.

“A mother’s heart is a patchwork of love.”
-Author unknown

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ABSTRACT
ADULT WOMEN'S AGE DIFFERENCES IN LINKS BETWEEN BEHAVIORAL AND PHYSIOLOGICAL INDICATORS OF COGNITIVE REGULATION.

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The Neurovisceral Integration (NVI) model has demonstrated that there are many associations between physiological and behavioral indicators of cognitive regulation including among respiratory sinus arrythmia (RSA), frontoparietal coherence (FPc), and executive function (EF). EF is associated with both RSA and FPc, and each shows a developmental pattern that could be explained within the Selection, Optimization, and Compensation (SOC) model. Specifically, all develop in childhood, become more efficient in adulthood, and decline in old age. But how *all three* of these variables interact – especially in adult women – is still unknown. This dissertation consisted of three studies that tested the additive and interactive effects of age, RSA, and FPc on EF in adult women from both a variable- and person-centered approach. We expected to see age moderated interactions between RSA and FPc that predicted EF. Indeed, the Study 1 results indicated age moderated an RSA and FPc interaction that predicted EF. And those results seemed to follow a SOC type pattern. However, Study 2 did not generally replicate those findings. In addition, there were no significant person-centered results from Study 3. Despite the non-replication and lack of person-centered significant results, we caution against concluding with confidence that there are no developmentally based patterns between these physiological and behavioral indicators of cognitive regulation. We acknowledge that there are limitations in this series of studies that might account for the findings.

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I. CHAPTER 1: BACKGROUND

Cognitive regulation is a foundational feature of complex mental processing that allows for attending to and utilizing salient and non-salient information which can then lead to optimal use of psychophysiological resources. Despite the extensive prior work on cognitive regulation across the lifespan, women are a distinct population who are overlooked in many areas of psychological and neuroscience research. There are meaningful psychological and physiological constructs that make it important to conduct scientific investigations of cognitive regulation that focus on women. Interest in this line of inquiry is fundamental given that cognitive regulation is a foundational aspect of mental and physical health (e.g., Smith et al., 2017; Thayer & Ruiz-Padial, 2002 and 2006; Thayer et al., 2009), and women sometimes exhibit distinct patterns of health outcomes related to cognitive regulation indicators that compromise their capacities and quality of life (e.g., de Zambotti et al., 2018; Tada et al., 2017). In addition, it is widely accepted that pregnancy and early motherhood leads have a myriad of physiological and behavioral changes associated with cognition. Some of the changes are transient, but some are long-lasting (e.g. Duarte-Guterman et al., 2019; Orchard et al., 2023). According to a recent National Health Statistics report, it is estimated that 13.2% of women in the United States between the ages of 15 and 24 had at least one biological child. This number drastically jumps to 84.3% of women ages 40-49. All of the individuals in our research are mothers (Martinez and Daniels, 2023).

Sensitive and responsive parenting requires efficient and flexible cognitive regulation, which is a foundational feature of complex mental processing. Cognitive regulation allows

for attending to salient information (and disregarding irrelevant information) and is reflected in optimized corresponding activity among various psychophysiological mechanisms in the central and peripheral nervous systems (Han et al., 2019; McCurdy et al., 2022; Moilanen & Manuel, 2017; Tellegen et al., 2022; Yu et al., 2020). Poorer cognitive regulation is associated with negative emotional states and reactive as well as dysregulated behavior and thoughts in parents themselves and their children (Bridgett et al., 2015; Evans et al., 2020; Giuliano et al., 2015; Lisitsa et al., 2021). Thus, it is important to understand the whole system of parental cognitive regulation involving the nervous system, cognitions, emotions, and behaviors.

Parents' cognitive regulation varies between individuals from day to day, but overall, these individual differences are quite stable across time and situations (Bridgett et al., 2015; Deater-Deckard, 2004). What is not yet known is whether these individual differences in neurobiological and behavioral components of cognitive regulation work together differently through development and aging across mid-life when parents are doing the bulk of their "heavy lifting" of childrearing. If found, such shifts may reflect aging-based shifts from optimization of neurocognitive resources to compensatory processes (derived from Selection, Optimization, Compensation Theory or SOC; Baltes, 1997; see also Amodio, 2010; Cabeza et al. 2018; Reuter-Lorenz, 2010). Given this major gap in knowledge regarding adult development, we conducted a novel exploration of potential maternal age differences between two physiological indicators and cognitive regulation in mothers aged 21-49 years. [This and the previous paragraph were originally written as part of the

Introduction section of Chapter 2. The information there is pertinent to the broader introduction, so they are duplicated verbatim here.]

It is important to our work to also consider various viewpoints regarding who is considered a woman and how this classification is defined (e.g., Lannon, 2015; Schiappa, 2022) Henceforth, we emphasize that all the participants in our research self-identified as women. In addition, although not a part of our line of inquiry, it is worth noting that all of our participants were also mothers or female primary caregivers of children.

Cognitive regulation allows an individual to tune out some aspects of situations that require high cognitive load or that distract from a task at hand, so that they can then focus on what they deem meaningful in order to perform more effectively in cognitive processes like making decisions, planning actions, modifying emotional responses, and pursuing or updating goals. Thus, cognitive regulation is important in adaptive responding to the environment as well as achieving target behaviors and emotional responses (e.g. Kakhki et al., 2022; Saarikallio, 2010; Turner & Husman, 2008; Zimmerman & Iwanski, 2014).

Three commonly studied constructs used to operationalize cognitive regulation are executive functioning, effortful control, and cognitive reappraisal (e.g., Mohammed et al., 2022; Calkins & Marcovitch, 2010). Executive function encompasses at least three related classes of processes involving working memory, inhibitory control, and attention/set shifting that together result in controlled responses to the environment (Miyake & Friedman, 2012). Effortful control is similar to executive function and refers to those

aspects of temperament that represent the capacity to shift focus and inhibit impulsive responses as needed. Cognitive reappraisal is the capacity to accurately reframe salient negative events and internal sensations to reduce their aversiveness (Evans & Rothbart, 2007). All three constructs are clearly important to assessing the complex processes that are part of an integrated systems of psychophysiology and cognitive regulation.

In line with addressing the complexity of cognitive regulation and the factors associated with it, the Neurovisceral Integration (NVI) model was developed and has evolved to incorporate the growing evidence informing that model. One aspect of this model postulates that autonomic regulation of the heart is causally interrelated with neocortical regulation of cognitive functions (Thayer & Ruiz-Padial, 2002 and 2006; Thayer et al., 2009; Thayer et al., 2012); these neocortical regions are described in detail below. The most widely used operational measure for autonomic regulation of the heart is respiratory sinus arrhythmia (RSA) collected from continuous electrocardiography (ECG or EKG). RSA represents an aspect of heart rate variability that is maintained by the vagus nerve (e.g., Balzarotti et al., 2017; Borges et al., 2020; Capuana et al., 2014; de Oliveira Matos et al., 2020; Geisler et al., 2013; Holzman & Bridgett, 2017; Wang et al., 2013). According to the NVI model, as the regulator of RSA the vagus nerve is hypothesized to be a mechanistic feature of the complex integrated system of physiology that functions to optimize neocortical cognitive regulation. Thus, efficient autonomic regulation of the heart – operationalized as higher RSA – is a part of efficient cognitive regulation.

In addition, the NVI model states that another part of the complex integrated system of cognitive regulation includes specific brain regions associated with cardiac regulation. Their activity is *also* causally interrelated with cognitive regulation. Most of those brain regions are in the prefrontal and frontal cortices; however, there is evidence that the parietal lobe also is important in cognitive regulation, as part of the frontoparietal or central executive network (Han, 2004). Relevant research findings pertaining to the parietal lobe and cognitive regulation have not been incorporated into the NVI model yet (Thayer & Lane, 2000; Thayer & Ruiz-Padial, 2002 and 2006; Smith et al., 2017). However, based on the NVI model and the separate literature related to the role of frontoparietal network activity in cognitive regulation, we determined that one of the best measures for relevant neocortical brain activity is frontoparietal alpha-power coherence (FPc) derived from continuous electroencephalography (EEG)(EEG e.g., Basar & Güntekin, 2012; Sadaghiani et al., 2012; Sauseng et al., 2005; van der Helden et al., 2010). FPc not only measures frontoparietal connectivity between the neocortical regions of the two lobes, but this specific measure has also been shown to be associated with cardiac and cognitive regulation measures (e.g., Cuevas et al., 2012; Fleck et al., 2016; Polich, 1997; Ponomareva et al., 2022; Solis et al., 2021; Thorpe et al., 2016).

One useful framework for our research hypotheses is a developmental model called the Selection, Optimization, Compensation (SOC) model (Baltes, 1997; see elaborations by Amodio, 2010; Cabeza et al., 2018; Reuter-Lorenz, 2010). This theory suggests describes Selection as an initial stage of development in childhood, adolescence, and early adulthood when the body is fine tuning its mechanisms toward its most efficient functioning. This is

followed by an Optimization stage in mid-life when the system is utilizing the selections earlier in development to continue to function in an optimal state. Lastly, during the Compensation stage as individuals enter late adulthood and old age and must face decreasing efficacy in its functioning, the body adapts by compensating for these declines by readjusting its mechanisms.

Furthermore, as with all aspects of human psychology and physiology – cognitive regulation is subject to developmental changes across a lifetime (i.e., ontogeny, species-typical average functioning) and is highly variable between individuals at any given point in the lifespan (i.e., individual differences). For this reason, it is essential to understand the age-related shifts in the relevant neurobiological systems that pertain to individual differences in cognitive regulation. Broadly, when using valid and reliable measurements, individual differences represent meaningful variation that indicate differential adaptations, and are not measurement noise. Focusing only on average age differences without consideration of individual differences at each age point would limit the interpretation of findings, which can lead to incomplete or poorly informed hypotheses and theories. Prior developmental research examining RSA, FPc, and cognitive regulation, indicates that there are age differences in RSA, but there are conflicting results regarding frontal and parietal lobe activity(e.g., for RSA Alemeida-Santos et al., 2016; Fernandes et al., 2005; Giardino et al., 2013; Hellman & Stacy, 1976; Hrushesky et al., 1984; Masi et al., 2007; Patriquin et al., 2015; Ribeiro et al., 2001; e.g. for FP connectivity Cuevas et al., 2012; Fleck et al., 2016; Thorpe et al.,2016; Polich, 1997; Ponomareva et al., 2022; Solis et al., 2021). Additionally, it is plausible that there are individual age differences in the patterns of

covariation of RSA and FPc with respect to cognitive regulation; this essential idea has never been examined.

We already know from the extant literature that research on specific isolated aspects of the cognitive regulation integrative system follow developmental patterns that seem consistent with the SOC model. For example, the association between variations in cognitive performance and individual differences in frontoparietal coherence observed from infancy in EEG studies starts to decrease in childhood (Bell, 2012; Bell & Wolfe, 2007). This gradual decline in the association continues across adolescence into early adulthood based on evidence from EEG and fMRI studies (Campbell et al., 2012; Cuevas et al., 2012; Solis et al., 2021; Thorpe et al., 2016). We also see a similar pattern of a developmentally decreasing association between RSA and cognitive performance. Children show increasing RSA efficiency related to cognitive regulation (e.g., Calkins & Keane, 2004; Graziano & Derefinko, 2013; Patriquin et al., 2015) and high efficiency in health adults (e.g., Balzarotti et al., 2017; Capuana et al., 2014; Geisler et al., 2013), followed by an age-related decline (e.g., Hrushesky et al., 1984; Masi et al, 2007). These patterns between the physiological and behavioral indicators then suggests there might be developmental selection, optimization, and compensation patterns occurring across the transition from early to late adulthood. However, to our knowledge, there is no previous research exploring the developmental patterns between RSA, FPc, and cognitive regulation, let alone research focusing specifically on women.

Cognitive regulation relies on an integrated system in which cardiac regulation and brain activity are important components in optimization of the process. For this reason, comprehensive analyses that are integrative of both the neocortical and cardiac components of this system are needed, to provide more valid tests of the theorized interactions between RSA and FPc in cognitive regulation. However, this has not yet been done in the extant literature; prior studies have examined either FPc *or* RSA, but not both together, in examining the etiology of cognitive regulation. Therefore, the main objective of the current research was to explore and quantify the correlation between RSA and FPc, and the potential statistical interactions between RSA and FPc, in the statistical prediction of cognitive regulation (specifically, executive function, effortful control, and cognitive reappraisal). To our knowledge, the current studies are the first set to do this—a major and surprising gap in the literature, given that RSA and FPc are both heavily implicated in prior empirical research and theories including the NVI model.

To address this gap in developmental patterns of the physiological and behavioral indicators of cognitive regulation in adult women within the context of the NVI model and SOC model, in the current dissertation and associated studies, examined cross-sectional age differences in the associations between individual differences in RSA, FPc, and various measures of cognitive regulation from a variable and person-centered approach. We expected to see age-based variable-centered and person-centered differences in the additive and interactive effects of RSA and FPc predicting cognitive regulation.

1.1 RESEARCH STRATEGY

The current dissertation includes three studies, each with two aims and two parts: Study 1a and 1b; Study 2a and 2b; Study 3a and 3b. The ordering of the studies is based on their chronological order in which the work was conducted, starting with an initial Study 1, followed by a direct replication in Study 2 and then a novel and distinct analysis of data in Study 3. This series of studies investigates whether there are cross-sectional age differences in the associations between specific brain and heart physiological measures predicting cognitive regulation in adult women.

1.1.1 Chapter II: Study 1 – Variable-Centered Analysis

This study of adult women had two aims, hereafter referred to as **Study 1a** and **1b.** The first aim **1a)** explored age differences in the covariation between RSA and FPc, and the second **1b)** tested whether age, RSA and FPc interacted in a meaningful way in the statistical prediction of cognitive regulation (See Figure 1.2.1). Each of three distinct cognitive regulation variables (executive function or EF, effortful control or EC, and cognitive reappraisal or CR) were analyzed in separate multiple regression equations using RSA, FPc, and age as predictors.

1.1.2 Chapter III: Study 2 – Replication of Study 1, Variable-Centered Analysis

The second study replicated **Study 1** (both aims a and b) and its variable-centered analyses using a demographically similar dataset. This replication consisted of the same two parts as found in Study 1. **2a)** assessed the age differences in covariation of RSA and FPc as well

as **2b)** estimated the statistical interactions between age, RSA and FPc predicting the same three measures of cognitive regulation (EF) (See Figure 1.2.2).

1.1.3 Chapter IV: Study 3 – Extension, Person-Centered Analysis

The final study used the dataset from Study 2 and extend the work in both prior studies by using a person-centered approach rather than a variable-centered approach. Study 3 also had two aims that reflect the general ideas of the aims of Studies 1 and 2. Study 3a examined age differences in the person-centered pattern of covariation in individual differences in RSA and FPc, and 3b tested for potential interactions between age and person-centered RSA-FPc covariation in the statistical prediction of the same three measures of cognitive regulation (EF) (See Figure 1.2.3). This study identified whether developmental differences in cognitive regulation were due to qualitatively distinct patterns of covariation between RSA and FPc across a broad range of RSA-FPc covariation—the level of this covariation will be distinct to each woman, and these analyses will address that (variable-centered analyses ignore this information). The person-centered estimation of RSA-FPc covariation was calculated using the "Q-correlation" method described briefly above, (see 4.3.3 Data Analysis for more detail).

Considering the substantial gaps in knowledge and challenges in psychophysiological research pertaining to cognitive regulation and development in women, we focused on the associations between RSA, FPc, and cognitive regulation using two distinct analytic approaches: variable-centered analysis, and person-centered analysis. Beginning with Study 1 and 2, we used a classic variable-centered analysis approach—with Study 2

replicating of Study 1 using a demographically similar dataset. The replication addressed the problem defined as the replication crisis in psychology research (see 3.1 Introduction for more details).

For Study 3, we used the same dataset used for Study 2. The essential difference was that we used the underutilized Q-correlation estimation method to calculate a within-person intraclass correlation—a type of person-centered analysis approach. The person-centered approach is an equally essential method for assessing individual differences yet is rarely used. Variable-centered analyses calculate the associations between two or more variables and reflect the "relationship between" those variables based on the assumption that that association is indicative of how the variables covary for all individuals in the population. In contrast, a person-centered approach "define(s) individuals in… a holistic fashion" (D'Alonzo, 2004; Miyazaki & Maier, 2005; Watts & Stenner, 2007). It does this by transforming the data matrix so that the covariation between variables is estimated using an array of variables based on the *within-person variation*, rather than estimating a correlation based on just two variables (at a time) using only the *between-person variation* in those two variables. Although this transformation of the data matrix might seem counterintuitive at first, it is through this transformation that the researcher produces scores across an *array of many variables* (and their overall correlation together) within each individual. Computationally, the calculation of the Q-correlation is done in the same way we would calculate a correlation between only two variables across an *array of individuals* (the traditional variable-centered approach).

In summary, Study 1 is an exploration of age differences in the correlations between RSA and FPc as well as the age differences in the interactions between RSA and FPc in the prediction of cognitive regulation in women. Study 2 is a replication of Study 1, to determine the validity of the significant correlations and interaction effects detected in Study 1. Finally, as a critically important extension of the variable-centered approach in Studies 1 and 2, Study 3 used a person-centered analytic approach instead of variable centered approach as an additional method for fully exploring individual differences related to age among women.

This dissertation research expands our understanding of developmental differences in neocortical and vagal regulatory factors that may influence cognitive regulation in women. Our approach is novel, systematic, and comprehensive. By assessing and including measures of the known constructs in this comprehensive analysis, we hope to move the field forward in identifying statistical additive and interactive patterns in predicting cognitive regulation. Gaining this knowledge is essential for optimizing women's cognitive functioning and health by advancing our understanding of age as a key factor in the system(s) of cardiac and brain functioning that pertains to cognitive regulation. This series of studies also add valuable information to the NVI model, by moving beyond the frontal lobe to examine FP connectivity. The following chapters describe and explain the characteristic of our research in each of the three studies including background, methodology, analysis, and results. The dissertation is completed with a concise integrative chapter.

1.2 FIGURES

Figure 1.2.1 Studies 1 and 2 Aim 1
Aim 1a) Age moderating FPc predicting RSA
Aim 1b) Age moderating RSA predicting FPc

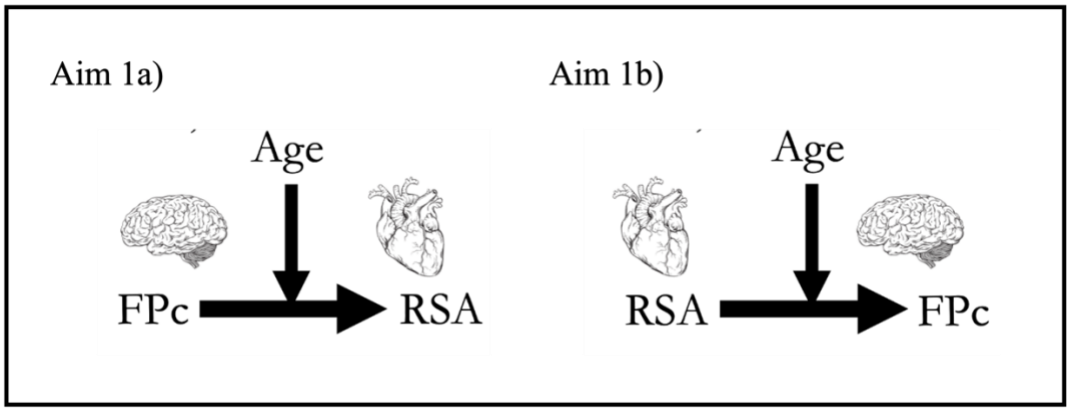


Figure 1.2.2 Studies 1 and 2 Aim 2
Aim 2a) Age moderation of RSA moderating FPc predicting Cognitive regulation
Aim 2b) Age moderation of FPc moderating RSA predicting Cognitive regulation

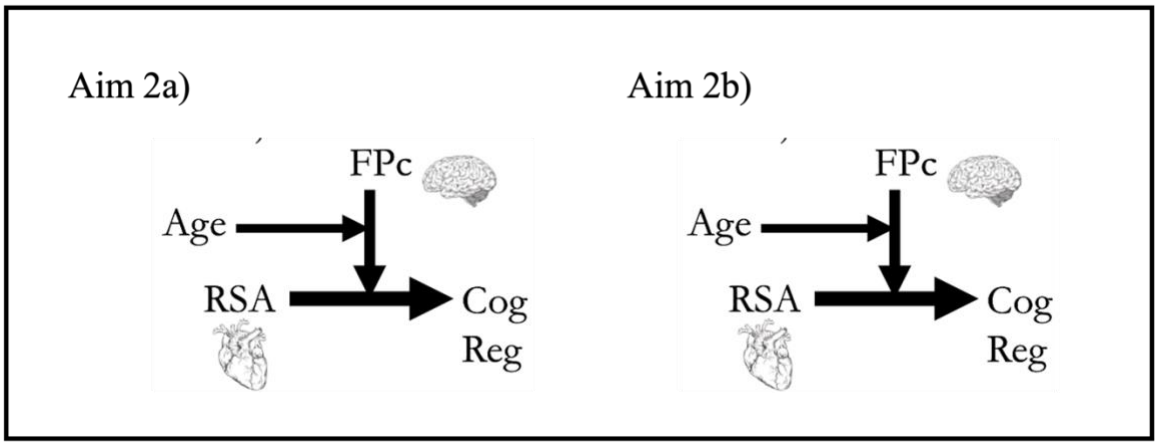
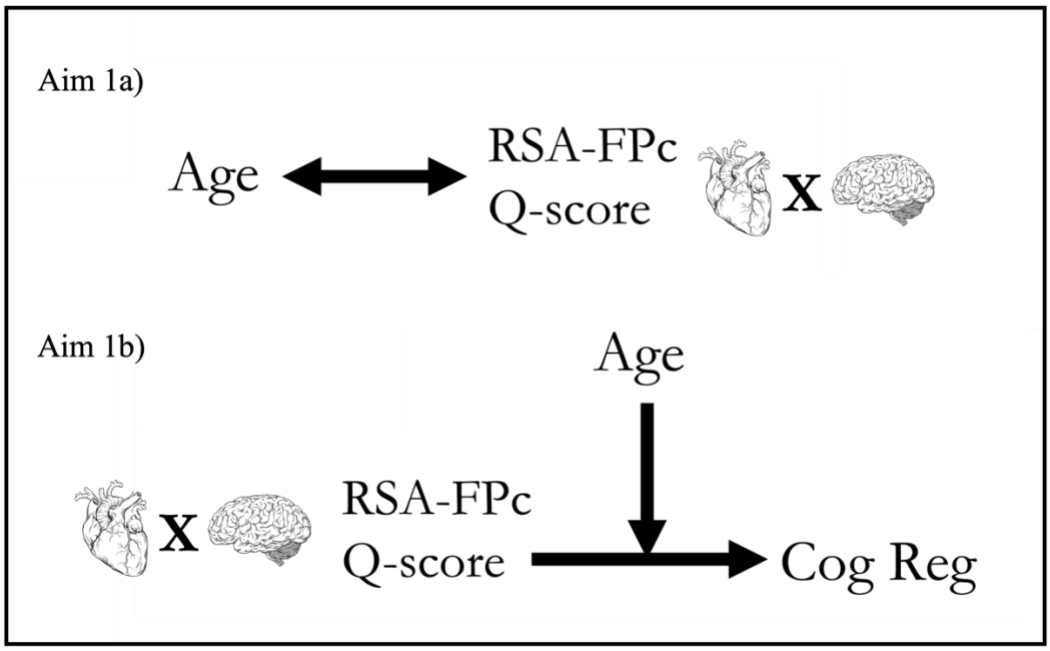


Figure 1.2.3 Study 3 Aims 1 and 2

Aim 1) Age and RSA-FPc Q-score association
Aim 2) Age moderating RSA-FPc Q-score predicting Cognitive regulation



II. CHAPTER 2: STUDY 1 – VARIABLE-CENTERED ANALYSIS

2.1 INTRODUCTION

Sensitive and responsive parenting requires efficient and flexible cognitive regulation, which is a foundational feature of complex mental processing. Cognitive regulation allows for attending to salient information (and disregarding irrelevant information) and is reflected in optimized corresponding activity among various psychophysiological mechanisms in the central and peripheral nervous systems (Han et al., 2019; McCurdy et al., 2022; Moilanen & Manuel, 2017; Tellegen et al., 2022; Yu et al., 2020). Poorer cognitive regulation is associated with negative emotional states and reactive as well as dysregulated behavior and thoughts in parents themselves and their children (Bridgett et al., 2015; Evans et al., 2020; Giuliano et al., 2015; Lisitsa et al., 2021). Thus, it is important to understand the whole system of parental cognitive regulation involving the nervous system, cognitions, emotions, and behaviors.

Parents' cognitive regulation varies between individuals from day to day, but overall, these individual differences are quite stable across time and situations (Bridgett et al., 2015; Deater-Deckard, 2004). What is not yet known is whether these individual differences in neurobiological and behavioral components of cognitive regulation work together differently through development and aging across mid-life when parents are doing the bulk of their "heavy lifting" of childrearing. If found, such shifts may reflect aging-based shifts from optimization of neurocognitive resources to compensatory processes (derived from

Selection, Optimization, Compensation Theory or SOC; Baltes, 1997; see also Amodio, 2010; Cabeza et al. 2018; Reuter-Lorenz, 2010). Given this major gap in knowledge regarding adult development, we conducted a novel exploration of potential maternal age differences between two physiological indicators and cognitive regulation in mothers aged 21-49 years.

Cognitive regulation is a key aspect of behavioral and emotion regulation (Kakhki et al., 2022; Saarikallio, 2010; Turner & Husman, 2008; Zimmerman & Iwanski, 2014), and is defined broadly as attending to and utilizing salient and non-salient information which can then lead to optimal use of cognitive and physiological resources. In the current study, we operationalize cognitive regulation using three of the most widely used constructs that also have been shown to be pertinent to maternal and paternal caregiving behavior: executive function, effortful control, and cognitive reappraisal (Crandall et al., 2015; Lin et al., 2022; Zeytinoglu et al., 2017). Executive function encompasses at least three related classes of processes involving working memory, inhibitory control, and attention/set shifting that together result in controlled responses to the environment (Marcovitch et al., 2010; Miyake & Friedman, 2012). Effortful control is a similar construct that refers to the shifting focus and inhibiting impulsive responses as needed (Evans & Rothbart, 2007). Cognitive reappraisal involves reframing salient negative events and internal sensations in order to reduce their aversiveness (Gross, 1998).

Together, these variables capture related, distinct aspects of cognitive regulation that aid in responsive and supportive (versus reactive and punitive) parenting behaviors in the many

situations that require flexibility in maternal attention and cognition, impulse control, and active updating of appraisals (Evans et al., 2020; Tellegen et al. 2022). It is well – versus poorly – regulated caregiving behaviors that is of concern given their well-established effects on children's and adolescent's well-being and psychological health (Lisita et al. 2021; Yu et al., 2020).

Regarding developmental changes, the cognitive components of executive function and effortful control (i.e., inhibitory control, attentional control, working memory) show rapid growth across early childhood with continued, very gradual improvement across adolescence into young adulthood, followed by a gradual decrease across adulthood and accelerated decreases in old age (LaPlume et al., 2022; Rhodes, 2004; Rodriguez-Aranda & Martinussen, 2006). The developmental literature regarding cognitive reappraisal shows evidence of improvements with age across childhood and adolescence into early adulthood, but the findings regarding age-based changes across adulthood and into old age are mixed as to whether gradual decreases occur or not (Hamilton & Allard, 2021; McRae et al., 2012; Opitz et al., 2012). And like all cognitively demanding tasks, executive function, effortful control, and cognitive reappraisal show very substantial individual differences at all ages, even among the very young and very old.

Cognitive regulation is supported by cerebral and cardiovascular mechanisms including respiratory sinus arrhythmia (RSA) measured using electrocardiography (ECG) (Balzarotti et al., 2017; Borges et al., 2020; Capuana et al., 2014; Wang et al., 2013) as well as frontal and parietal brain region activity measured via alpha frequency power using

encephalography (EEG) (Basar & Güntekin, 2012; Sadaghiani et al., 2012; Sauseng et al., 2005; van der Helden et al., 2010). RSA is a key indicator of autonomic regulation of the heart via the vagus nerve (Porges, 2007; Yasuma, 2004). Higher RSA is indicative of better autonomic regulation. Developmentally, it increases in early childhood, plateaus across childhood through young adulthood, then gradually starts to decrease across adulthood as part of normative aging (Giardino et al., 2013; Hrushesky et al., 1984; Patriquin et al., 2015). There is robust support of the association between RSA and many aspects of cognitive regulation. For example, research indicates a positive correlation between higher RSA and better cognitive regulation (Capuana, 2014; Geisler et al., 2013). Higher RSA indicates more adaptability and efficient cardio physiological responsiveness to cognitive demands, in addition to more efficient and effective regulation of emotional states (Balzarotti et al., 2017).

Turning to the brain, EEG alpha power is one key indicator of brain activity patterns associated with cognitive regulation because it provides information about the degree of active effortful processing (versus disengagement and passivity) in wakeful cognitive states. We utilized correlated activation and deactivation measurements in frontal and parietal regions – operationalized using EEG frontoparietal alpha-power coherence, which is the squared correlation of alpha-power values between frontal and parietal sites (Basar & Güntekin, 2012; Babaeeghazini, 2021; Heugal et al., 2022; Warbrick, 2022). Covarying individual differences in power values representing activation and deactivation is thought to indicate the functional co-activation and co-deactivation of those brain regions; for the purposes of the current study, frontoparietal alpha-power coherence is thought to represent

activity of the frontoparietal (also known as the central executive) network (Heugel et al., 2022; Laufs et al., 2003).

EEG frontoparietal alpha-power coherence is evident in early infancy but begins to decrease in childhood (Bell & Wolfe, 2007). The association between cognitive regulation and frontoparietal alpha-power coherence in early childhood indicates that there are cognitive function benefits to frontoparietal regions being synchronized (Bell, 2012). Beyond the few studies of infancy and childhood, little is known about age-related developmental changes in frontoparietal alpha-power coherence in adolescence and adulthood. By comparison, there is more research in those age periods that has examined fMRI coherence. Given that fMRI coherence and alpha-power coherence are inversely correlated in adolescence and adulthood (Goldman et, al 2002 and Laufs et al. 2003), the fMRI coherence literature can provide some insights. fMRI frontoparietal coherence gradually increases across adolescence and early adulthood (Campbell et al., 2012; Cuevas et al., 2012; Solis et al., 2021; Thorpe et al., 2016). Its average levels across adulthood are not established, but some research indicates that fMRI coherence decreases in old age (Campbell et al., 2012). Thus, it stands to reason that EEG frontoparietal alpha-power coherence may decrease across adolescence and through early adulthood and increase among aging adults. These developmental age-related changes are thought to reflect maturation and learning, whereby cognitively demanding tasks become more automated as the frontoparietal network becomes more efficient at processing, but then become more correlated again to reflect increasing effortful processing to compensate for aging-related degradation in the system. Regarding individual differences, at any given age point, higher

EEG frontoparietal alpha-power coherence likely reflects greater effort, and lower frontoparietal alpha-power coherence reflects recruitment of more physiological resources (Bell 2001 and Bell & Wolfe 2007).

Of particular importance for the current study, major portions of the frontoparietal network play a key role in the neocortical regulation of the vagus nerve and cardiac activity that together comprehensively represent the central and peripheral nervous systems' roles in cognitive regulation (Balzarotti et al., 2017; Holzman & Bridgett, 2017). The prefrontal cortex regions encompass – but are not limited to – portions of the insular cortex, the anterior cingulate, and the amygdala (Smith et al., 2017; Thayer & Lane, 2000; Thayer & Ruiz-Padial, 2002 and 2006). The parietal regions we assessed in the current study have been identified as important regions involved in cognitive processing of the environment (Han, 2004). These regions are integral to self-regulation (Thayer and Lane, 2000; Thayer et al., 2009).

In the current study, we focused specifically on the most relevant neurophysiological, behavioral, and self-reported indicators of the cognitive regulation system for mothers caring for young children. Women's transitions into parenthood and the hour by hour, day after day caregiving role place chronic and acute demands on their cognitive and physiological regulation (along with many other systems) for decades, typically spanning the 20s through the 50s (Deater-Deckard & Panneton, 2017; Mehta et al., 2020). This occurs within the context of normative developmental changes across adulthood in key aspects of cognitive regulation and physiology described above.

It is worth noting that in the current analysis, we do not include measures of parenting behavior. Some members of the current authorship team have previously published findings from the current study dataset, showing the patterns of covariation between individual differences in maternal harsh parenting behaviors (self-reported and observed), executive function, heart rate and EEG alpha-power reactivity values (Deater-Deckard & Bell, 2017). Specifically, modest to moderate increases in heart rate along with larger decreases in alpha-power (when going from a resting state to an active cognitive task state) yielded the strongest association between the highest executive function levels and lowest harsh parenting levels. However, that prior research did not consider maternal age differences, nor did it consider the roles of frontoparietal alpha-power coherence and RSA in accounting for the key indicators of self-regulation that are fundamental to nonreactive, regulated parenting behavior (e.g., executive function, effortful control, and cognitive reappraisal).

As all women with children develop and age across adulthood, they are experiencing developmental changes amidst parenting challenges to many systems of the body – including those supporting cognitive regulation. According to Selection, Optimization and Compensation (SOC) theory (Baltes, 1997; Baltes et al., 1999), these changes lead to a gradual shift from *optimization* (i.e., maximal efficacy and efficiency) of available capacities and resources in young adulthood, to *compensation* (i.e., adapting utilization to sustain efficacy in response to decreasing capacity). This theorized shift is not related to changes in the role demands on women, but rather reflects an adaptive response to developmental changes in regulatory capacities and resources that optimize cognitive

regulation in young adult parents (i.e., in their 20s and 30s) then shifts gradually toward compensation for sustaining cognitive regulation among older parents (i.e., in their 40s and 50s). Reuter-Lorenz and Lustig (2008) suggested three potential mechanisms for compensation in cognitive neuroscience: increased activity in the previously active regions, recruitment of more brain regions, or engaging both hemispheres where only one was previously activated. In addition, Cappell, Gmeindl, and Reuter-Lorenz (2010) suggested that certain brain regions might engage sooner in older adults to compensate to meet demands even when cognitive load is low. While our study focuses on aspects of the parasympathetic cardio regulation, there is a good example of compensation in the sympathetic nervous system. Age-degradation leads to deficits in norepinephrine reuptake in the cardio sympathetic neuromuscular junction. When this occurs, there is an increase in expression of pre-junction catecholamines – specifically, an increase in norepinephrine spillover (Kaye & Esler 2008). The spillover is a shift in the system resulting in more norepinephrine produced within the cells to compensate for the loss at the junction.

In order to detect developmental changes in the association between RSA and cognitive regulation involving potential compensation among older mothers of young children, researchers need to consider not only age differences in average cognitive regulation (which as noted above, shows shifts toward decreases across middle age into old age), but also how various systems of the body supporting cognitive regulation changes in their covariation with each other and in their independent and interactive links with executive function (EF), effortful control (EC), and cognitive regulation (CR) As noted above, RSA and frontoparietal alpha-power coherence change with age. However, to our knowledge no

prior studies have tested for age differences in the covariation between RSA and frontoparietal alpha-power coherence, or age differences in RSA and frontoparietal alpha-power coherence statistical predictions of cognitive regulation phenotypes (i.e., EF, EC, CR) – let alone within the context of studying adult development of women raising children.

To address these two major gaps: (Aim 1) we tested whether the covariation between RSA and frontoparietal alpha-power coherence differed by maternal age, and then (Aim 2) tested whether there are additive or interactive statistical predictive effects of RSA and frontoparietal alpha-power coherence on variance in cognitive regulation (i.e., EF, EC, CR) differed by maternal age. Based on SOC theory, we hypothesized that the systems of the body supporting cognitive regulation would show a developmental shift toward compensation spanning early through middle adulthood (e.g., 20s to 50s). We expected to find significant maternal age statistical moderating effects on the association between RSA and frontoparietal alpha-power coherence (Aim 1), and on the additive or interactive statistical effect of RSA and frontoparietal alpha-power coherence on EF, EC, and CR (Aim 2). Given that the current study is the first of its kind, we did not have specific expected results regarding the exact age-based patterns in the results from Aim 1 and Aim 2 analyses. However, based on related research, we expect cognitive regulation to be somewhat automated in adulthood, and because of that, we expect cardiac and frontoparietal function to be specialized and not tightly coupled. This idea is based on shifts seen between early development – where cardiac regulation and frontoparietal coherence are tightly coupled when predicting good cognitive function – and young adults where

there is less coupling (Sauseng et al., 2005). We further anticipate that the system might be starting to decline in the older women, so there might be more coupling between the cardiac and frontoparietal functions as those systems work together to maintain optimum levels of cognitive regulation. This expectation is based on the decrease in RSA with age and the association between RSA and cognitive regulation as well as the association between frontoparietal coherence and cognitive regulation.

2.2 METHODS

2.2.1 Participants

The sample included 90 women (age, *M* = 32.35 years, *SD* = 5.86 years, *range* = 21-49 years). The original sample size was 127 women who had some physiological and behavioral data, but 26 participants were missing one or more of the key variables needed for our analyses, and another 11 were excluded because their ECG data showed an unexplainable decrease in heart rate when shifting from resting state to active cognitive task states (for more details see Deater-Deckard & Bell, 2017). When we compared the included and excluded sub-samples on key demographic measures (e.g., mother and father years of education), there were no significant differences (e.g., two-tailed t-test p-values greater than .10).

There are multiple viewpoints regarding the definition of "woman"; in the current study, we included self-identifying women. Families resided in rural, small town and small city areas of southwest Virginia on the eastern edge of the Appalachian region of the United States. The racial background of these individuals reflected the distribution in the region

(74% Caucasian, 13% African American, and 2% Asian, 6% multiracial, 5% other). Regarding the education level of the mothers, 22% had a high school diploma/graduate equivalent diploma (GED) or less; 28% had some college or an associate degree; 30% had a 4-year degree; and 20% had a postgraduate degree.

2.2.2 Procedures

One-third of the participants were recruited as part of a longitudinal study exploring mother-child interactions (children were 3-yrs old). The other two-thirds were recruited via advertisements and flyers distributed to community organizations also as a part of a family, community-based research project (children were 3 to 7-yrs old). Signed consent was obtained as part of an informed consent procedure during the laboratory visit. Participants also completed demographic and emotional regulation questionnaires which were submitted in person or by mail after the laboratory visit. Physiological measures were collected during resting conditions (eyes opened and eyes closed paradigm) for two minutes total, and continuously during task conditions involving executive function tasks. An honorarium was given for participation. IRB approval was granted through Virginia Polytechnic Institute and State University.

2.2.3 Measures

2.2.3.1 Executive Function (EF)

Four counterbalanced tasks that measured attentional control, inhibitory control, and working memory were administered on a computer or face to face.

2.2.3.1.1 *Tower of Hanoi*

This task was a computerized adaptation of the classic peg task or game. Three disks were all placed on one of three pegs in order from largest to smallest. The participants were required to end with all the pegs in the same order on one of the other pegs. While transferring the pegs, a disk placed on top can never be larger than the one below. The score was measured in seconds to finish with a 60 second maximum possible score (Davis & Keller, 1998).

2.2.3.1.2 *Backward digit span task*

Participants heard a set of digits (0-9) that they then repeated back in reverse order. After two practice trials (two-digit list), the test began with an increasing number of digits (two attempts permitted per series of digits). Each correct trial was followed by a subsequent trial increasing the number of digits by one digit. The final score was the longest series of correctly reported digits (i.e., span).

2.2.3.1.3 *Wisconsin Card Sorting Test (WCST)*

Participants were shown four cards with different symbols that varied in quantity, shape, and color. They then had to match a stack of 64 or 128 cards (depending on test laboratory site), first by detecting the matching rule (match on quantity, shape, or color) through trial and error, then continuing to match on that rule until the rule changed without warning. At a rule change, the participant would then need to try a new matching rule, and this continued with rule changes throughout the task. The score for analyses was the number of

perseveration errors (i.e., the number of continuing to use the old rule after a rule change) (Heaton & PAR Staff, 2003).

2.2.3.1.4 *Stroop color-word task*

This task was a computerized version of the classic Stroop color-word task (Stroop, 1935). The name of a color is written in either the matching color (congruent) or a different color (incongruent). The participant indicated the color of the letters using keyboard presses. After several practice trials, the participant completed 20 trials each of congruent, incongruent, and mixed congruent/incongruent. The score was the number of correct responses in the most difficult mixed congruent/incongruent trial block.

2.2.3.1.5 *Executive function composite score*

The individual task performance scores described above were compiled into a general EF composite to capture the most reliable general EF performance measure (Miyake & Friedman, 2012). The first principal component among the four task scores (with Stroop, Wisconsin Card Sorting Test, and Tower of Hanoi scores reversed so that higher scores represented better performance) explained 41% of the variance (loadings from .57 to .75). Indicators were standardized, averaged, and standardized again to compute a composite z-score.

2.2.3.2 Effortful Control (EC)

Participants completed the Adult Temperament Questionnaire Short Form (Evans & Rothbart, 2007), with items rated on a 7-point Likert scale (1=strongly disagree to

7=strongly agree). We used the Effortful Control Scale score that comprises inhibitory control, attention control, and activation control and represents self-reported cognitive regulatory capacity (α = . 68).

2.2.3.3 Cognitive Reappraisal (CR)

Mothers completed the Emotional Regulation Questionnaire (Gross & John, 2003) that includes items rated on a 7-point Likert scale. We used the Cognitive Reappraisal Scale (α = .81), which captures self-reported frequency of use of cognitive strategies for reappraising events and feelings to mitigate the effects of negative emotional states. This scale captures self-construed utilization of reappraisal, not accuracy or effectiveness of strategy use.

2.2.3.4 Respiratory sinus arrhythmia (RSA)

ECG was used to derive RSA. Two disposable ECG electrodes were placed by the participants themselves with help and instruction from research assistants on the right collarbone and lower left rib cage (Stern, Ray, & Guigley, 2001). A ground lead was positioned near the base of the scalp. Raw cardiac measures were amplified with a James Long Bioamp (Caroga Lake, NY) with a bandpass from 0.1 to 100Hz. The data were digitized at 512 samples per second with Snapshot-Snapstream analyzation software (HEM Data Corp.; Southfield, MI). ECG signals were manually checked to remove any potentially erroneous R peaks. A four-pass peak detection algorithm was used to identify the R-waves which were then calculated for inter-beat-interval (IBI). Respiratory sinus arrhythmia was derived from the IBI as the high-frequency heart rate variability.

Aggregated continuous physiological scores were derived within each condition state (e.g., resting, task 1, task 2, etc.). Principal components analysis (PCA) of these indicators showed that the first component accounted for 89% of the variance, with loadings from .92 to .95. The resting and task state indicators of RSA were averaged and then standardized to center the scores for subsequent statistical analyses.

2.2.3.5 Frontoparietal alpha-power coherence

Frontoparietal alpha-power coherence was collected using EEG. Alpha-power measures were acquired with the International 10-20 System consisting of 16 electrodes per hemisphere and were recorded with an Electro-Cap (Eaton, OH) following standard guidelines. The James Long Bioamp (Caroga Lake, NY) was used for the amplification of the electrical signals with pass from 1 to 100 Hz while impedance was kept below 10K ohms. The data was then analyzed with EEG Analysis System software (James Long Company; Caroga Lake, NY). Artifacts from eye and gross motor movements were eliminated from analysis. Cleaned data were converted to Hamming windows (1 second) with a 50% overlap and then were transformed with discrete Fourier processing. The alpha band power was computed for 8-13 Hz expressed as mean square microvolts. Transformation for normal distribution was achieved with a natural log transformation. Frontoparietal alpha-power coherence was calculated as the squared correlation between the frontal and parietal alpha-power F3, F4, P3, P4, P7, and P8 sites using an algorithm by Saltzberg et al. (1986, Equation 9).

As with RSA, aggregated continuous physiological scores were derived within each condition state (e.g., resting, task 1, task 2, etc.). PCA was again used to confirm the reliability of these composites. The first component for each composite: F3P3, explained 65% of the variance with loadings from .80 to .82; F3P7, 61%, loadings .78 to .80; F4P4, 71%, loadings .83 to .86; and F4P8, 66%, loadings from .79 to .83. A composite F3-P3/F3-P7 frontoparietal coherence value was derived from averaging the F3P3 and F3P7 sites, and a composite F4-P4/F4-P8 frontoparietal coherence value was derived from averaging F4P4 and F4P8 sites. These areas represent the dorsolateral and ventrolateral prefrontal cortexes as well as the posterior parietal cortex. Scores were then standardized to center variables for subsequent analyses.

2.2.4 Data Analysis

Descriptive statistics, bivariate correlations, and multiple regression analyses were computed using *IBM SPSS Statistics* (Version 26, 2019). Variables were centered for regression equations and estimation of potential statistical interaction effects. Post-hoc probing of significant interaction terms was conducted using analysis of simple slopes. For Aim 1, we sought to determine whether maternal age moderated the association between RSA and frontoparietal alpha-power coherence. To this end, we used multiple regression to test the statistical prediction of RSA from frontoparietal alpha-power coherence and maternal age (and their interaction); and the statistical prediction of frontoparietal alpha-power coherence from RSA and maternal age (and their interaction). For Aim 2, we used multiple regression to test whether maternal age moderated the independent or interactive

statistical predictive effects of RSA and frontoparietal alpha-power coherence on cognitive regulation measures (i.e., EF, EC, CR).

2.3 RESULTS

Descriptive statistics and bivariate correlations (see Table 1) were calculated. All variables were normally distributed (skewness from .03 to .48) and showed modest or moderate kurtosis (-.51 to .97). Regarding frontoparietal alpha-power coherence, all frontoparietal sites had similar means and standard deviations. Turning to bivariate correlations, EC was positively associated with CR, as well as with lower frontoparietal alpha-power coherence at F3-P3/F3-P7. Frontoparietal alpha-power coherence of the two hemispheres (F3 and F4 sites) was positively correlated. Age was positively associated with EF, and negatively associated with RSA and frontoparietal alpha-power coherence at F4-P4/F4-P8.

For descriptive purposes, we also examined whether the age differences in EF replicated the cross-sectional pattern found in prior studies. That prior evidence indicates a positive linear association between age and general EF in community samples of adults from 20-60 years (e.g., Yao et al., 2020), as well as a negative parabolic (i.e., inverted "u") quadratic growth pattern spanning adulthood. We examined our data and found this pattern, as shown in Figure 1. Executive function task performance peaks in the 30s and then begins a gradual decline across the 40s (in the current study data) and, as noted in prior literature, continues to decline across the 50s and onward (e.g., LaPlume et al., 2022).

We also examined potential curvilinear age-based functions in EC and CR, but did not see significant nonlinear functions. For EC, $F(2,94) = 1.176$, $p = .313$, $R^2 = .024$; for CR, $F(2,91) = .025$, $p = .975$, $R^2 = .001$. We also estimated curvilinear age-based functions for RSA $F(2, 91) = 5.380$, $p = .006$, $R^2 = .106$; frontoparietal alpha-power coherence F3-P3/F3-P7 $F(2, 88) = 1.117$, $p = .332$, $R^2 = .025$; and frontoparietal alpha-power coherence F4-P4/F4-P8 $F(2, 88) = 4.503$, $p = .014$, $R^2 = .093$. Both RSA and F4-P4/F4-P8 showed an age-based decrease that decelerated (i.e., "flattened out") with age.

2.3.1 Aim 1 Age-RSA interaction predicting FPc & Age-FPc interaction predicting RSA

Our first aim was to test whether the covariation between RSA and frontoparietal alpha-power coherence differed by maternal age. We started by estimating four equations (one for each frontoparietal site combination) with the statistical predictors of age, frontoparietal alpha-power coherence, and their two-way interaction term and RSA as the dependent variable. For a thorough analysis, we estimated the four equations a second time, swapping the position of RSA and frontoparietal alpha-power coherence – that is, with the predictors of age, RSA, and their two-way interaction term and frontoparietal alpha-power coherence as the dependent variable.

2.3.1.1 F3-P3/F3-P7 and RSA

When we tested age and frontoparietal alpha-power coherence for F3-P3/F3-P7 as predictors of RSA, the equation was significant: $F(3, 86) = 4.378$, $p = .006$, $R^2 = .132$. However, when we tested age and RSA as predictors of frontoparietal alpha-power coherence for F3-P3/F3-P7, the equation was not significant: $F(3, 86) = 2.542$, $p = .062$,

R^2 = .081. Because this equation was not significant when estimated both ways, we did not proceed with an interpretation involving F3-P3/F3-P7 for Aim 1.

2.3.1.2 F4-P4/F4-P8 and RSA

When we tested age and frontoparietal alpha-power coherence for F4-P4/F4-P8 as predictors of RSA, the equation was significant. The equation also was significant when examining age and RSA as predictors of frontoparietal alpha-power coherence for F4-P4/F4-P8. Since this equation was significant when estimated both ways, we proceeded with interpretation. Full results are shown in Table 2.5.2.

The two-way interactions involving age were significant or marginally significant (depending on the equation). To interpret this age statistical moderation effect, we estimated simple slopes with age as the moderator of the association between frontoparietal alpha-power coherence for F4-P4/F4-P8 and RSA. With age as the moderator in the equation frontoparietal alpha-power coherence predicting RSA the results indicated -2 standard deviations below mean age, β = -.47, p < .01; -1 SD, β = -.26, p < .05; at mean age, β = -.06, n.s.; +1 SD above mean age, β = .15, n.s.; at +2 SD, β = .36, n.s. (see Figure 2.5.2). For completeness, we also examined age as the moderator in the equation RSA predicting frontoparietal alpha-power coherence and found a very similar pattern: -2 standard deviations below mean age, β = -.37, p < .06; -1 SD, β = -.20, n.s.; at mean age, β = -.06, n.s.; +1 SD above mean age, β = .15, n.s.; at +2 SD, β = .32, n.s. The only significant association observed between frontoparietal alpha-power coherence and RSA was a negative association that was observed only among younger mothers.

2.3.2 Aim 2 Age moderating RSA and FPc interactions predicting EF

Our second aim was to test for additive or interactive statistical predictive effects of RSA and frontoparietal alpha-power coherence on variance in EF, EC, and CR as a function of maternal age.

2.3.2.1 EF, RSA, and F3-P3/F3-P7

The equation for EF with the additive and interactive effects of age, RSA, and frontoparietal alpha-power coherence for the F3-P3/F3-P7 composite was not significant: F (7, 82) = 1.648, p = .134. We did not proceed with further analysis involving F3-P3/F3-P7.

2.3.2.2 EF, RSA, and F4-P4/F4-P8

The equation for EF with additive and interactive effects of age, RSA, and frontoparietal alpha-power coherence for the F4-P4/F4-P8 composite was significant. Results are shown in Table 2.5.3. To interpret the age differences in RSA and frontoparietal alpha-power coherence moderation effects in the statistical prediction of EF, we estimated simple slopes separately for younger and older women (median split on age), then computed simples slopes to interpret 1) the association between frontoparietal alpha-power coherence for F4-P4/F4-P8 and EF with RSA as a moderator, and 2) the association between RSA and EF with frontoparietal alpha-power coherence as the moderator.

A clear age-difference pattern emerged in the simple slope estimates. For the younger half of the sample, there was not a single significant simple slope for the association between

EF and either RSA or frontoparietal alpha-power coherence in either equation (i.e., with RSA as moderator of frontoparietal alpha-power coherence predicting EF, or with frontoparietal alpha-power coherence as moderator of RSA predicting EF). In contrast, there was emergence of significant simple slope estimates among the older half of women in the sample. For RSA as moderator of frontoparietal alpha-power coherence predicting EF the results indicated -2 standard deviations below mean RSA, β = .80, p < .05; -1 SD, β = .35, n.s.; at mean RSA, β = -.09, n.s.; +1 SD above mean RSA, β = -.54, p < .05; at +2 SD, β = -.98, p < .05 (see Figure 3). For frontoparietal coherence as moderator of RSA predicting EF: -2 standard deviations below mean frontoparietal coherence, β = .59, p < .05; -1 SD, β = .25, n.s.; at mean RSA, β = -.09, n.s.; +1 SD above mean RSA, β = -.44, n.s.; at +2 SD, β = -.78, p < .06.

2.3.2.3 EC and CR

In contrast to the analyses for EF, none of the equations for EC and CR were significant; *F* statistics ranged from 1.33 to 1.70, with *p* values ranging from .121 to .246. However, the significant three-way interaction effect (F4-P4/F4-P8 frontoparietal alpha-power coherence *RSA*age) that we found for EF was of similar effect size and direction and approaching significance for EC (p < .08). Also, EC and CR were correlated .40, suggesting a potential general self-perceived cognitive regulation indicator. Thus, in a post-hoc analysis, we averaged the EC and CR scores (standardized) and estimated the equation for Aim 2 again using the composite EC-CR score as the dependent variable. The equation for F4-P4/F4-P8 was significant: F (7, 79) = 2.20, p = .043, R^2 = .16, and the three-way interaction term was marginally significant, β = -.25, p = .057. Analysis of simple slopes

showed a pattern that had some similarity to the pattern for EF. Among older women only, F4-P4/F4-P8 frontoparietal alpha-power coherence was a significant negative statistical predictor of EC-CR at higher levels of RSA (+1 SD, β = -.60, p < .02; +2 SD, β = -.83, p < .04). Also, among older women only, RSA was a significant positive statistical predictor of EC-CR at lower levels of F4-P4/F4-P8 frontoparietal alpha-power coherence (-1 SD, β = .44, p < .001; +2 SD, β = .73, p < .003).

We ran post-hoc simple slopes analyses for Aims 1 and 2 during *task only* to clarify whether the effects were driven by general RSA levels, or specifically the levels during cognitive tasks. All effects were in the same direction but were smaller than the resting/task averages results. For Aim 1 F4 composite predicting RSA moderated by age the effect sizes were +2SD β = .28, +1SD β = .11, -1SD β = -.25, -2SD β = -.42 and for Aim 2 Older moms F4 composite predicting EF moderated by RSA effect sizes were +2SD β = -.64, +1SD β = -.33, -1SD β = .29, -2SD β = .60.

2.4 DISCUSSION

The broad goal of the current study was to examine a potential developmental shift from optimization to compensation across adulthood (Baltes, 1997; Freund and Baltes, 1998) in mothers' system of cognitive regulation involving various physiological, behavioral, and survey-based indicators. These are key physiological aspects of well-regulated, nonreactive maternal parenting behavior that is instrumental to warm, sensitive, and responsive caregiving (e.g., Crandall et al., 2015; Deater-Deckard, & Bell, 2017). We had two primary aims. For Aim 1, we tested potential developmental changes in how cardiac

and cerebral physiological indicators of cognitive regulation covaried with each other as part of an integrated system. Results indicated an age difference in how frontoparietal alpha-power coherence and RSA covaried, though only for the F4-P4/F4-P8 composite.

2.4.1 Aim 1 Age and RSA predicting FPc & Age and FPc predicting RSA

Specifically, the results from Aim 1 indicate there was a significant negative association that dissipated with age. For younger mothers, having higher RSA predicted lower frontoparietal alpha-power coherence with the same pattern when frontoparietal alpha-power coherence predicted RSA—an association that did not hold for older mothers in the sample. RSA and frontoparietal alpha-power coherence both are considered as important indicators of the efficiency and effectiveness of regulatory capacity (for RSA, Balzarotti et al., 2012; Geisler et al., 2013; Wang et al., 2013; for frontoparietal coherence, Bell, 2001; Klimesch et al. 1999; Moore et al., 2008). A preliminary interpretation of the cross-sectional age difference in the current study is that it reflects the specialization between frontoparietal alpha-power coherence and RSA which was previously seen in adolescence and early adulthood samples. This may indicate that optimization is denoted by specialized activity of those two systems followed by a developmental shift toward less negative coupling. This is a pattern that may reflect a shift away from optimization toward a compensatory process involving greater flexibility in utilization of frontoparietal alpha-power coherence and RSA. This may represent the kind of compensatory process proposed by SOC theory (Baltes, 1997). A competing interpretation is that the age difference we detected may not reflect a shift from an optimized system toward a compensating process. They could instead reflect age-based degradation in the system. A fuller understanding of

whether the age-difference pattern just described reflects a shift toward compensation requires examination of whether and how frontoparietal alpha-power coherence and RSA work together to account for variation in multiple diverse indicators of maternal cognitive regulation.

4.4.2 Aim 2 Age moderating RSA and FPc interactions predicting EF

For Aim 2, we tested for a potential age difference in the additive and interactive associations of RSA and frontoparietal alpha-power coherence with maternal EF, EC, and CR. We examined maternal age as a statistical moderator, and the equations examined variance in EF, EC, and CR based on physiological statistical predictors. As with the analyses for the first aim, statistically significant equations were found only for the F4 composite. Furthermore, for the Aim 2 analyses, the equation was significant only for EF, and results indicated a significant three-way interaction effect between age, RSA, and frontoparietal alpha-power coherence. To interpret the statistical interaction, we performed post-hoc analyses. In post-hoc analyses, we used age as the first moderator by dividing the sample at the median. We then found significant results when frontoparietal alpha-power coherence was used as the second moderator for RSA predicting EF, *and* when RSA was used as a moderator for frontoparietal alpha-power coherence predicting EF (we also found a similar pattern of this effect for a composite of self-reported EC and CR). Results for Aim 2 indicate that younger women may be using fewer physiological resources while attending to and utilizing relevant information during cognitively demanding tasks. This could suggest that there is more automation in psychophysiological mechanisms in the central and peripheral nervous systems in young adulthood compared to older adulthood

(e.g., Cabeza et al., 2018; Hamilton & Allard., 2021). This may mean that their cognitive regulation is not contingent on the physiological components of self-regulation capacity in the way that it is for older mothers.

Among older mothers only, EF performance was associated with both RSA and frontoparietal alpha-power coherence in an interaction effect that may be indicative of compensatory processes. Lower RSA was offset by higher frontoparietal alpha-power coherence to predict higher EF, and higher RSA was offset by lower frontoparietal alpha-power coherence to predict higher EF. This may indicate that among older mothers, frontoparietal alpha-power coherence is able to compensate for poorer autonomic regulation of the heart, and better autonomic regulation of the heart is able to compensate for higher frontoparietal alpha-power coherence (representing less automatic, more effortful processing). Furthermore, this interactive pattern among older mothers also may be present for self-reported indicators of cognitive regulation (including effortful control and cognitive reappraisal), but our findings in this regard are tentative and require replication before interpreting further.

What are the implications of these results regarding harsh reactive versus well-regulated caregiving? In prior analyses with the same sample (Deater-Deckard & Bell, 2017), we reported that the well-established link in the literature between higher maternal EF and nonreactive supportive parenting may reflect the roles of both cognitive and cardiovascular activity—although those prior results were ambiguous with respect to the precise patterns of regulatory aspects of cognitive and cardiac functioning. The current results suggest that

age-based changes in maternal cardiac and cerebral regulation during effortful cognitive processing reflect developmental aging effects, and perhaps compensatory effects within the body, for promoting better cognitive regulation. Better cognitive regulation increases self-regulatory resources for mothers—perhaps especially among older mothers—to enact caregiving behaviors that decrease the frequency and strength of impulsive reactive responses to the acute and chronic stressors that arise when caring for young children (Crandall et al., 2015).

2.4.3 Limitations

The current study is, to our knowledge, the first of its kind and it also has several strengths (e.g., multi-method, wide age range of mothers, examination of multiple indicators of cognitive self-regulation capacity). These strengths aside, the study had several limitations that should be considered. First, the cross-sectional correlational study design limits the interpretation of developmental change and potential causality in the detected statistical associations between frontoparietal alpha-power coherence, RSA, and cognitive regulation measures.

In addition, we did not control for parity or child age (although this did not vary widely in the current sample); addressing these variables may be beneficial for future studies. Furthermore, although the study sample was broadly representative of the region where the families lived, it was not representative of the broader region of the country or the entire country. Given the overall lack of racial and ethnic diversity in the sample, we were not adequately powered to test for potential group differences in our study aims. Relatedly, the

current research also would be well complemented by future studies that consider age-based changes in non-maternal women, and paternal and non-paternal men.

Finally, our inferences regarding age-based differences in mothers' cognitive regulation implicates ovarian aging and other hormonal changes across adulthood, but we did not have measures of self-reported ovarian aging indicators or of hormones. Although some of the older individuals in the sample may be perimenopausal, hormone levels are quite stable within the age range of the current study, with only slight declines on average prior to the onset of menopause (usually between 40 and 44 years) (e.g., Burger et al., 2007 and Butler & Santoro, 2011).

Future research could address some of these limitations as well as test for replication and extension of our findings. For example, we will be testing for replication in a second larger cross-sectional study of mothers. In addition to our next study, it will be essential to elucidate within-person changes longitudinally, in the interacting effects of frontoparietal functioning and vagal regulation as related to cognitive regulation among mothers. Future studies could also include direct or indirect measures of ovarian age and menopausal status, to elucidate whether and how those aspects of physiological development may covary with or even explain the age differences we found.

With these caveats and future directions considered, there are several key conclusions to be drawn from the current study. We found that there are age-related differences in the interactions between RSA and frontoparietal alpha-power coherence (Figure 2.5.2), and

that those differences pertain to cognitive regulation in older mothers (Figure 2.5.3). This may reflect developmental shifts toward compensation for aging- and learning-related changes in cognitive regulation among mothers as they raise their children. These age differences probably reflect a gradual yet constantly developing whole-body system of physiological and cognitive self-regulation that is essential for maintaining responsive caregiving in the face of the many challenges of parenting. Also, the separate components of this whole-body system likely do not covary with each other with age, and likely interact with each other in their effects on cognitive regulation. Thus, researchers should not be deterred if they at first observe nonsignificant zero-order correlations or lack of significant additive statistical predictive effects. Finally, the evidence for developmental changes in this system of regulation will very likely depend on the specific methods and measures used for operationalizing key indicators captured by physiology, behavior, and self-perceptions.

2.5 TABLES AND FIGURES

Table 2.5.1

Descriptive Statistics and Bivariate Pearson Correlations

	EF	CR	EC	RSA	FPc^{F3}	FPc^{F4}	Age
EF	---						
CR	.00	---					
EC	.04	$.40^{**}$	---				
RSA	-.02	$.18^{+}$	.07	---			
FPc^{F3}	-.03	-.15	$-.24^{*}$	$-.19^{+}$	---		
FPc^{F4}	-.11	-.10	-.14	.01	$.50^{***}$	---	
Age (yrs)	$.23^{*}$	.11	.12	$-.26^{*}$	-.10	$-.30^{**}$	---
mean	.07	5.01	4.52	3.01	0.14	0.14	32.35
SD	1.03	0.99	0.75	0.63	0.03	0.03	5.86

Two-tailed p-values: + p < .10, * < .05, ** < .01, *** < .001.

Note: EF (executive function), CR (cognitive reappraisal), EC (effortful control), RSA (respiratory sinus arrhythmia), FPc-F3 (frontoparietal coherence for F3-P3/F3-P7 sites), FPc-F4 (frontoparietal coherence for F4-P4/F4-P8 sites).

Table 2.5.2

Aim 1 Regression Results for Age as Moderator of Association between Frontoparietal Coherence (for F4-P4/F4-P8) and RSA

Age as Moderator of FPc predicting RSA

F (3, 86) = 4.515, p = .005, R^2 = .136

	B	(se)	β	p
Age	-.300	(.101)	-.315	.004
FPc	-.051	(.098)	-.055	.606
Age*FPc	.190	(.088)	.219	.033

Age as Moderator of RSA predicting FPc

F (3, 86) = 4.905, p = .003, R^2 = .146

	B	(se)	β	p
Age	-.339	(.109)	-.326	.003
RSA	-.028	(.117)	-.026	.811
Age*RSA	.190	(.101)	.194	.062

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Table 2.5.3

Aim 2 Regression Results for Age as Moderator of Association between Frontoparietal Coherence (for F4-P4/F4-P8) and RSA predicting Executive Function

Age, FPc, and RSA predicting EF

F (7, 89) = 3.094, p = .006, R^2 = .141

	B	(se)	β	p
Age	.193	(.113)	.190	.092
FPc	-.094	(.109)	-.097	.388
RSA	-.115	(.130)	-.109	.337
Age*FPc	.268	(.105)	.290	.012
Age*RSA	-.013	(.121)	-.013	.916
FPc*RSA	.023	(.119)	.024	.849
Age*FPc*RSA	-.211	(.086)	-.303	.016

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

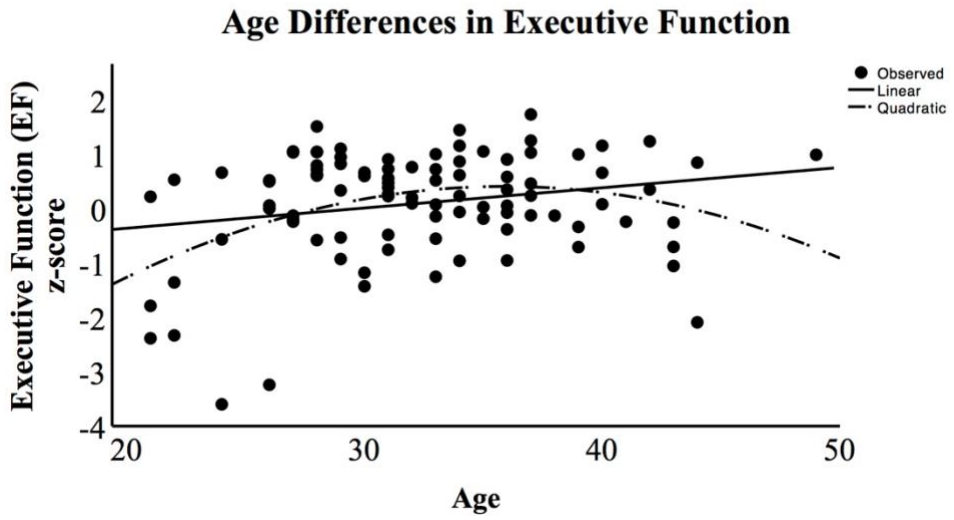


Figure 2.5.1

Age (x-axis) association with Executive Function (EF) composite z-score (y-axis).

This scatterplot includes the linear and quadratic functions representing the association between maternal age and EF task performance, indicating a negative parabolic function with the apex of performance occurring in the mid-30s with a decrease that continues through the older half of the sample.

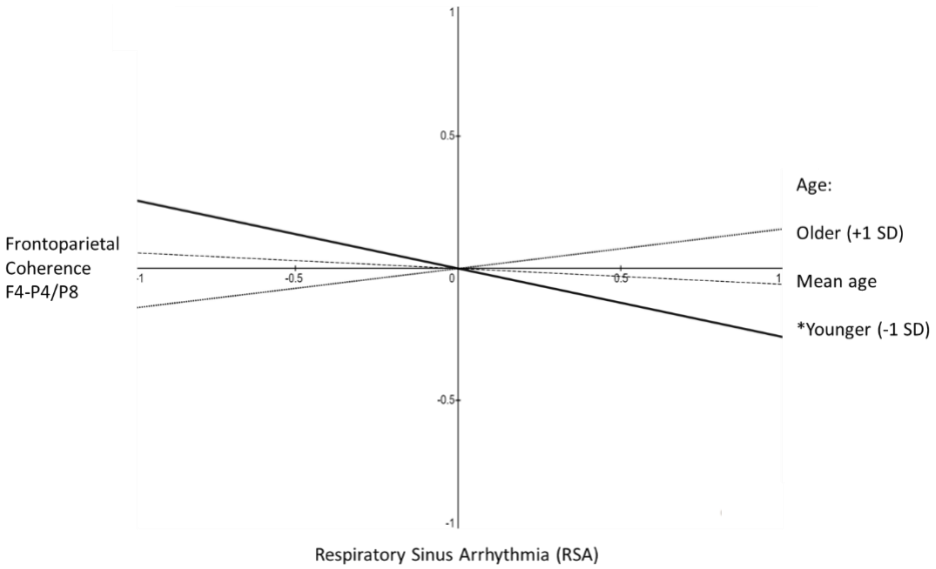


Figure 2.5.2

Aim 1 simple slopes.

Shown are the simple slopes reflecting the statistical prediction of frontoparietal coherence (F4-P4/F4-P8) from RSA as a function of maternal age. Slopes are shown at one standard deviation below mean age (solid line, * $p < .05$), at mean age (dashed line, nonsignificant slope), and one standard deviation above mean age (dotted line, nonsignificant slope).

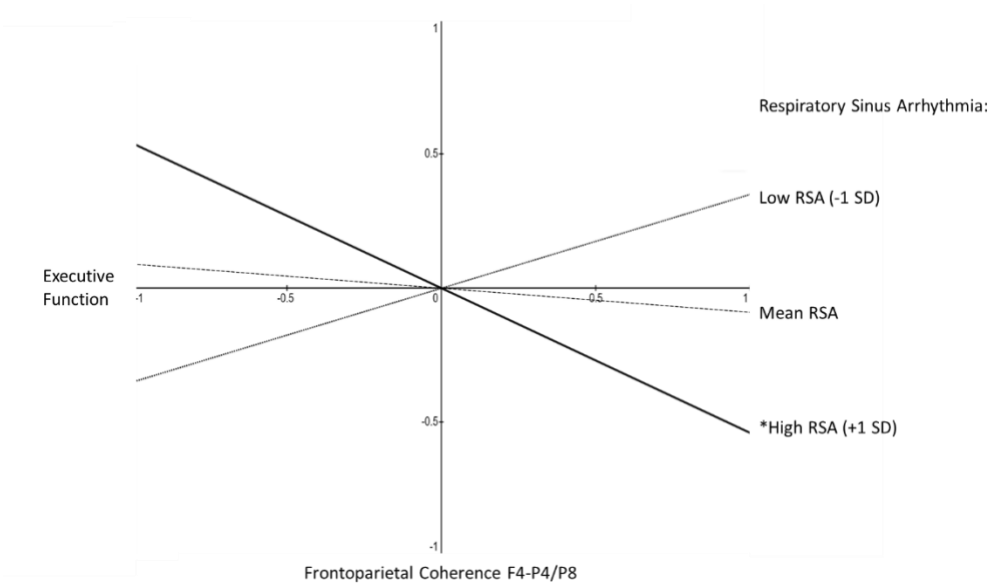


Figure 2.5.3

Aim 2 simple slopes, for older half of sample

Shown are the simple slopes for the older half of the sample, reflecting the statistical prediction of executive function from frontoparietal coherence (F4-P4/F4-P8) as a function of respiratory sinus arrhythmia (RSA). Slopes are shown at one standard deviation above mean RSA (solid line, * $p < .05$), at mean RSA (dashed line, nonsignificant slope), and one standard deviation below mean RSA (dotted line, nonsignificant slope). In contrast, none of the simple slopes

III. CHAPTER 3: STUDY 2 – REPLICATION OF STUDY 1, VARIABLE-CENTERED ANALYSIS

3.1 INTRODUCTION

As discussed in the background section in Chapter 1, identifying potential age differences in the associations between respiratory sinus arrythmia (RSA) and frontoparietal alpha-power coherence (FPc) in women, and in their statistical interaction in predicting cognitive regulation, is important to our understanding of the developing integrated system of cognitive regulation as described in the Neurovisceral Integration (NVI) Model (Thayer & Lane, 2000). The current study replicated the methods from the previous study presented in Chapter 2 with a demographically similar but larger sample of women. To delineate the specific details of the replication implemented of the prior study, we **2a)** examined whether there were distinguishable age-based patterns in the correlations between RSA and FPc. We then **2b)** determined if there were similar patterns of age differences (age as a statistical moderator) in the interaction between RSA and FPc in predicting cognitive regulation. We acknowledge at the outset that replication attempts can be equivocal and can present new results that reflect potentially useful and meaningful variability across studies.

It is fortunate that we have the data and resources to perform a direct replication of Study 1, given the ongoing debate regarding the replication crisis in the sciences. One clear potential benefit to this replication is that it *could* determine the statistical reproducibility of the findings from Study 1 (Malich & Munafõ, 2022). Simon (2014) pointed out that replication can offer validity (or reliability) *for* or *against* previous findings. Unfortunately,

it is easier to accept and publish results that appear to support the reliability of the previously published original findings. However, since 2015 when the Open Science Collaboration report – which scrutinized the idea of replicability in psychology – was published, it has been widely accepted that the sciences have faced significant challenges with replicability. Wiggins and Christopherson (2019) suggested that there has been agreement in the field that any "true phenomena" would be replicated. But Brandt et al. (2014) previously cautioned that true, direct replication is nearly impossible, because it is difficult to ensure all circumstances are exactly the same. For example, two studies may have a different technician administering the tests or might require distinct participant recruitment methods.

Why try to directly replicate a study if there are so many concerns about doing so? Mülberger (2022) positioned the concept of replication within a historical framework. Historically, replication was a big proponent of what was accepted as knowledge. Mülberger goes on to suggest that replication has a larger purpose beyond confirming prior findings; it also is important as a tool for scrutinizing methods, techniques, and theories. In addition, replication can sometimes even be about approaching a question or hypothesis from a different angle with the goal of generating a different result. Morawski (2019) notes that there are two camps that have grown from discussion about replication. One group assumes there is enough stability in systems possessing a "true phenomenon" that direct replication should be possible; Wiggins and Christopherson (2019) call this universalism. The other group assumes that complexities are so prevalent that this state of affairs needs to just always be assumed. Simonsohn (2015) suggests that we use caution before

committing to the stance (i.e., conclusion) that replication or non-replication determines the reliability of the original findings (and therefore the veracity of the conclusions), and the stability of the systems being studied.

This debate requires scientists to consider whether there is sufficient stability (e.g., across time, contexts, or populations) in the systems of interest that are being studied, to merit replication testing. If the systems being investigated are not sufficiently stable, replicating a study design yet finding different results across the original and replication study still can be useful because it generates new ideas, questions and hypotheses that also can be contributed to the ongoing literature on those systems.

Thus, with all of the above cautions above in mind, in the current Study 2 we used a direct replication approach. As we did with Study 1, we separated the current study into two aims: (Aim 1) we tested age effects on the association between RSA and FPc, and (Aim 2) we tested age-based moderation of the additive and interactive effects of RSA and FPc predicting cognitive regulation, assessed as EF (See end of 2.1 Introduction for details). Note that effortful control and cognitive reappraisal (EC and CR) were not part of the replication analysis because the Study 1 results indicated they had no age-based additive or interactive effects on predicting cognitive regulation. Based on the results from Study 1 (Aim 1) results we anticipated seeing a significant moderation effect of age on the association between RSA and FPc^{F4}. Based on the EF results from Study 1 (Aim 2), we anticipated a negatively signed three-way interaction between age, RSA, and FPc^{F4}

predicting EF. Specifically, we expected to see a negative association between RSA and FPc^{F4} with EF only among the younger women (see end of 2.1 Introduction for details).

3.2 METHODS

3.2.1 Participants

Participants were all self-identifying women (n=137) with children who participated in a longitudinal, developmental, childhood study. The mothers were assessed when the children were between the ages 6 and 9 years. The sample size for the current study was smaller than the sample size of child-focused analyses in previously published papers from this same longitudinal study sample, because not all the children's mothers completed their own assessment. The mean age was 37.23 (*SD* = 6.02). Half of the participants in the child study were recruited from a college town in Virginia and the other half from a small city in North Carolina. The total sample included individuals that identified as White (83%), Black or African American (14%), multi-racial (1%), Asian (1%), and Native Hawaiian or other Pacific Islander (1%). Education level consisted of 2% individuals that had not completed high school, 31% high school or equivalent graduates, 48% were college graduates, and 19% had advanced or professional degrees.

3.2.2 Procedures

A signed consent was obtained for the mothers separately from the children at the time of the mothers' data collection. Participants also submitted a series of questionnaires related to emotional control, home environment, and demographics. An honorarium was given to each participant.

IBM SPSS Statistics (Version 26, 2019) was used for all data analyses: descriptive statistics, bivariate correlations, and multiple regressions. Variables were standardized to center them, for use in regression analyses. For Aim 1, we used multiple regression models to test if there was an interaction between age and RSA predicting FPc, and conversely if the interaction between age and FPc predicted RSA. For Aim 2, we used multiple regression models to test for three-way interactions between age, RSA, and FPc predicting EF and EC.

3.2.3 Measures

3.2.3.1 Executive Function (EF)

Four variables were utilized to assess the three main domains of executive function: working memory/updating, set shifting/attentional control, and inhibitory control (Miyake & Friedman, 2012). These variables have been used by other researchers as a measure of cognitive processes necessary for efficient and appropriate thought and behavior during a cognitive challenge. A composite score representing an executive function general factor was calculated by averaging the z-scores of the four tasks: Tower of Hanoi, Backward Digit Span Task, Wisconsin Card Sort, and Stroop Color-word Task.

3.2.3.1.1 *Tower of Hanoi*

This general problem-solving task measures working memory, planning, and response inhibition. Participants were presented with a computerized version of the classic three peg task with a set of three or five disks of varying sizes. At the outset of the task, the disks were stacked from largest to smallest on one peg. Then, moving one disk at a time, the goal

was to end with all the disks stacked similarly on one of the other pegs. The caveat was that a larger disk was never allowed to rest atop a smaller disk. The task was scored based on seconds to completion with a 60 second maximum (Davis & Keller, 1998; Zook et. al, 2005).

3.2.3.1.2 *Backward Digit Span Task*

This task primarily measures working memory/updating. Participants were asked to verbally repeat an experimenter's verbalized series of randomized numbers (0-9) in reverse order with the number of digits in the series increasing with each trial. When a mistake was made, participants were given a second chance before either moving on to the next sequence of digits if they passed or concluding the task if they failed a second time. The score was the highest number of digits repeated correctly (e.g., Blankenship et al., 2019; Garon et al., 2008).

3.2.3.1.3 *Wisconsin Card Sorting Test*

This computerized task primarily measures updating and set shifting. Participants were shown four cards with symbols differing in quantity, color, and shape. They then were shown a stack of 128 cards. Moving through the stack, the participants were tasked with matching each card to one of the four original cards based on quantity, color, or size. The "matching rule" determining if a card was a match were periodically changed without warning. Participants received feedback (correct/incorrect) for each trial. Participants had to deduce which different characteristic should be used when the matching rule changed. The score for this task was the total number of errors (Heaton & PAR Staff, 2003).

3.2.3.1.4 *Stroop Color-word Task*

This computerized task primarily measures response inhibition in service of resolving conflicting information (Stroop, 1935). This task involves suppressing a prepotent response to generate a correct response using a particular key stroke to identify the ink color of a color word representing a different color (e.g., the word "red" written in blue ink, and the correct response is "blue"). Participants were presented with congruent and incongruent word/ink stimuli in 20-trial blocks that varied in difficulty. The score was the number of correct responses in the most difficult 20-trial block of mixed congruent/incongruent stimuli.

3.2.3.2 Respiratory Sinus Arrythmia (RSA)

RSA was extracted from heart rate collected through electrocardiography (ECG) as the high-frequency heart rate variability of the inter-beat-interval (IBI) (Stern, Ray, & Guigley, 2001). The IBI was determined by the R peaks with a four-pass detection. Data were manually inspected and cleaned to remove any potential errors in detection. James Long Bioamp (Caroga Lake, NY) was used to amplify the signal with a bandpass from 0.1 to 100Hz and Snapshot-Snapstream analyzation software (HEM Data Corp.; Southfield, MI) was used to digitize at 512 samples per second. The RSA was collected during the four EF tasks which were then averaged together along with a two-minute resting condition for an overall average RSA score.

3.2.3.3 Frontoparietal Alpha-power Coherence (FPc)

FPc was collected using electroencephalography (EEG). We used the International 10-20 System facilitated by Electro-Cap (Eaton, OH) with 16 electrodes per hemisphere. The James Long Bioamp (Caroga Lake, NY) was used to amplify the signal between 1 to 100 Hz pass with impediment below 10K ohms. Analysis System software (James Long Company; Caroga Lake, NY) was used for analysis of the data. Data impacted by eye and gross motor events were removed and then Hamming windows (1 second) with a 50% overlap and discrete Fourier processing methods were utilized to convert and transform the data. The alpha-power band was considered 8-13 Hz expressed as mean square microvolts. FPc was calculated as the squared correlation for frontal and parietal sites F3, F4, P3, P4, P7, and P8 using an algorithm by Saltzberg et al. (1986, Equation 9). Then those two sites per hemisphere were averaged to arrive at our two measures: FPc^{F3} and FPc^{F4}. As with RSA, FPc was collected during a two-minute resting condition and the four EF tasks and was averaged for overall FPc scores (FPc^{F3}, FPc^{F4}).

3.3 RESULTS

Descriptive statistics and bivariate correlations can be found in Table 3.5.1. There was a normal distribution for age, RSA, FPc^{F3}, FPc^{F4}, and EC (skewness from -.30 to .37) with a modest or moderate kurtosis (-.22 to .17). Means and standard deviations for all frontoparietal sites had expected and similar variability. The distribution for EF was slightly skewed and kurtotic (skewedness =1.02, kurtosis = 3.54) due mainly to two outliers. Results did not change with those outliers excluded, so we report results with those cases retained.

Regarding bivariate correlations (also shown in Table 3.5.1), age showed negative associations with EF and RSA. RSA was positively correlated with FPc^{F4}. There was also a positive correlation between the two EEG composite measures (i.e., FPc^{F3} with FPc^{F4}). There was a positive correlation between FPc^{F3} and EC.

We also estimated potential nonlinear associations between age and our study variables given the evidence of potential nonlinear developmental change patterns in prior research. The only significant nonlinear association was between age and RSA (see Figure 3.5.1). With older age, the negative association between age and RSA decelerated (i.e., "flattened out"), $F(2, 133) = 3.939$, $p = .022$, $R^2 = .06$. All other nonlinear associations with age were nonsignificant: EF, $F(2,134) = 2.15$, $p = .12$, $R^2 = .03$; EC, $F(2,132) = .806$, $p = .256$, $R^2 = .010$; FPc composites, FPc^{F3}, $F(2, 134) = .511$, $p = .601$, $R^2 = .01$, and FPc^{F4}, $F(2, 134) = 1.067$, $p = .347$, $R^2 = .02$.

3.3.1 Aim 1 Age and RSA predicting FPc & Age and FPc predicting RSA

Based on our goal for Aim 1, we estimated two-way interaction effects with age for each of the frontoparietal hemispheres or RSA. We did this twice: once testing age interacting with RSA predicting frontoparietal coherence, and again testing age interacting with frontoparietal coherence predicting RSA.

3.3.1.1 Age, FPc^{F3} and RSA

Results for this analysis are shown in Table 3.5.2. The multiple regression equation for age and FPc^{3F3} predicting RSA was significant, $F(3,132) = 2.85$, $p = 0.04$, $R^2 = 0.06$. Age was

a main effect in negatively predicting RSA, *standardized* β= -0.23, p = 0.01, t = -2.72. There were no other significant effects.

3.3.1.2 Age, FPc^{F4} and RSA

Results for this analysis are shown in Table 3.5.3. The equation for age and FPc^{F4} predicting RSA was significant, $F(3,132) = 4.11$, $p = 0.01$, $R^2 = 0.09$. Age and FPc^{F4} were each main effects predicting RSA: for age, β = -.22 p = 0.01, t = -2.63; for FPc^{F4}, β = 0.17, p = 0.05, t = 2.02. There were no other significant effects.

The equation for age and RSA interacting in predicting FPc^{F4} was also significant, $F(3,132) = 1.88$, $p = 0.14$ $R^2 = 0.04$. RSA had a main effect, $\beta = 0.18$, $p = 0.04$, $t = 2.06$. There were no other significant effects.

3.3.2 Aim 2 Age moderating RSA and FPc interactions predicting EF

The second aim was to test for additive and interactive effects of age, RSA, and frontoparietal alpha-power coherence in predicting EF.

3.3.2.1 EF, Age, RSA, and FPc^{F3}

There were no significant effects for this equation.

3.3.2.2 EF, Age, RSA, and FPc^{F4}

There were no significant effects for this equation.

3.4 DISCUSSION

The goal of this study was to replicate the methodology from the previous Study 1 in Chapter 2, that assessed whether there are developmental shifts (operationalized as cross-sectional age differences) in adult women in the associations between behavioral and physiological measures and in their statistical prediction of cognitive regulation. As with the previous study, there were two aims. For Aim 1, we calculated the association between RSA and FPc as a function of age. There were no significant interactions. For Aim 2, we tested the additive and interactive statistical effects of age, RSA, and FPc on EF. There were also no significant interactions for this model.

In turning to interpretation of replication of results, it is important to note that virtually none of the findings from Study 1 were replicated in the current Study 2. However, this is not uncommon, and we fully accepted at the outset that non-replication was a valid expectation—an important attitude to adopt when performing replication studies. Strickland and Cruz (2021) noted that replication failure is more likely when scientists look to confirm their own prior work. Strickland's suggestion is to not approach a replication with the expectation based on the original research. Instead, researchers should be prepared to explain *why* results might not have replicated. To this end, after describing the current study results compared to Study 1, we will then offer some potential insights about non-replication.

3.4.1 Aim 1 Age and RSA predicting FPc & Age and FPc predicting RSA

3.4.1.1 FPc^{F3}, RSA, and Age

The only finding that replicated for this Aim and statistical model was age predicting RSA. In both studies, there was a negative association between age and RSA such that older women had lower RSA values. This age effect on cardiac regulation was anticipated given previous literature (e.g., Almeida-Santo et al., 2016; Simoyi, 2020). Developmental changes in cardiac regulation have been interpreted as an indication of the gradual decline in the regulatory capacity of the cardiovascular system due to the effects of aging throughout the central and peripheral nervous system (Kaye & Esler, 2008).

3.4.1.2 FPc^{F4}, RSA, and Age

In the replication, Study 2, age and FPc^{F4} were *main effects* predicting RSA (negative and positive effects, respectively), whereas in Study 1, they showed a significant positive statistical *interaction* with each other. In Study 1, the FPc^{F4} model showed a significant *negative* effect predicting RSA, but the sign of the effect shifted to *positive* in Study 2 (see 2.3.1 Aim 1). Thus, it was apparent that none of the effects in this model replicated across the two studies.

3.4.2 Aim 2 Age moderating RSA and FPc interactions predicting EF

There were no significant effects in the analyses for Aim 2 involving FPc^{F4}, so those are not discussed further here. For analyses involving FPc^{F3} , analyses for Aim 2 showed a significant negative two-way interaction effect between RSA and FPc^{F3} in the prediction

of EF. This contrasts with the same analysis in Study 1 which showed a negative three-way interaction between RSA, FPc^{F3} and age in the prediction of EF.

It may be noteworthy that in the current study, age did not show a significant main effect in the prediction of EF. We were not completely surprised by this given previous literature suggesting that EF capacity peaks in one's 30-40s and begins to only gradually decrease across the 40s and beyond (e.g., Ferguson et al., 2012; Hartshorne and Germine, 2015; Strittmatter et al, 2020). However, the sign of the statistical effect of age predicting EF was negative (albeit very small and nonsignificant) in the current study. This negative sign for the effect is what we would anticipate with a sample spanning the age range in the current study.

3.4.3 Studies 1 and 2 Estimated Effects Quantitative and Visual Comparison

As a final step of the replication analysis, we conducted a comprehensive quantitative and visual comparison of all of the estimated effects across current Study 2 and prior Study 1. Results are show in in Tables 3.5.4 and 3.5.5. In both tables, the sign and standardized effect size of each estimated parameter is presented, alongside a column indicating if the sign of the effect was the same, and another column showing the calculated absolute difference between effect sizes, across Studies 1 and 2.

Turning first to the comparison of results for Aim 1 (Table 3.5.4), of the twelve main and interactive effects that were estimated, half had the same sign (+/-) in Study 2 compared to Study 1 and the averaged absolute difference in effect sizes was 0.22. For Aim 2 (Table

3.5.5), of the fourteen main and interactive effects that were estimated, only four showed the same sign (+/-) and the averaged absolute difference in effect sizes was 0.22. Perhaps these results indicate that the signs and effect sizes in both studies were largely due to chance. However, because the average age was older in current Study 2 compared to Study 1, we believe possible age moderating effects related to cognitive regulation warrants further exploration.

3.4.4 Limitations

Before turning to limitations specific to the current study, it is important to emphasize that our attempt to replicate the results from Study 1 was conducted within a broader ongoing conversation in our fields about replication in psychology and indeed all sciences. In 2017, Tackett et. al declared, "It's time to broaden the replicability conversation." Their review went on to say that some fields in psychology are more susceptible to problems with replication. They say this can be due to a variety of reasons, including but not limited to the level of peer-scientist scrutiny, a field's overconfidence in the interpretation of particular results, detection bias (i.e., detecting results in one sample but not another due to a difference in sample sizes), or data analysis approach (Tackett et al., 2017). It has also been suggested that subfields and specific theories face specific challenges when it comes to replication, because the specific theories and accompanying methods that are used means there are a limited number of researchers and resources focused on any particular topic using that specific theory and methodology (Sweller, 2023). The current work may be representative of this problem. Both studies were guided by the very specific NVI model theoretical framework (Thayer & Lane, 2000). Thus, replication may be susceptible to

issues within that framework that have not yet been identified. But fortunately for future work at least, the NVI model has a growing literature supporting its ideas and hypotheses. The looming limitations that this theory faces are whether the specific developmental, behavioral, and physiological systems we and others are investigating emerge as predicted significant associations, and whether such findings can be replicated and also fit within the expected results based on the NVI model.

The current study is, to our knowledge, the only one of its kind. Still, there are some limitations worth noting that perhaps contributed to non-replication. First, as Simons (2014) cautions, scientists typically must assume that sample-specific results are representative of a broader population. However, it is often necessary to be aware of specific characteristics of the sample population that might influence results. Thus, it is necessary for us to scrutinize potential characteristics that might account for our results. One such variable is that fact that the previous Study 1 included a sample of women that were familiar with the research process because their children had been part of an ongoing longitudinal study. Perhaps their familiarity with the process influenced their willingness to participate in the maternal assessment and affected the resulting data. Also, prior Study 1 was carried out mostly in a different geographic location. This too might influence participant self-selection and aspects of the data that might contribute to non-replication.

Second, the effect sizes in Study 1 were modest to moderate in size. Effect size has been cited as a common issue with replication studies, with the odds of replicating a result

increasing as a function of the standardized effect size that is being tested for replication (e.g., Flora, 2020; Ward & Kemp, 2019).

Third, as pointed out in the discussion section (see 3.4.2 Aim 2), there was a noteworthy difference in the age distribution across Study 1 and the current replication Study 2. The current Study 2 dataset had fewer 20–29-year-old women, with the entire age distribution shifting upward (by about six years at the sample mean) compared to Study 1. This difference could have implications pertaining to the lack of replication, given that there were significant results in the first study found only for the youngest women (who were in their 20s). This is especially of interest given that some of the models show age effects. We might be seeing part of the age-related plateau in effects described in Study 1. For example, Aim 1 for Model FPc^{F4} shows an interaction between age and FPc^{f4} predicting RSA for Study 1, but for Study 2 there is only an age main effect.

Lastly, the calendar year was different for the two studies. Calendar secular time differences are a confound given potential cohort effects the contribute to different study results due to ongoing changes in local and global culture and societally meaningful events.

3.5.4 Future Directions

In closing, we offer several possible future directions. First, a measurement design that allows for a resting condition prior to and after each task condition would allow for an analysis of resting-to-task physiological change far more precisely. In both Study 2 and prior Study 1, there was only one brief resting state period at the very beginning of the

assessment. This point is potentially consequential, because adding more resting state periods would allow for a finer-grained distinction between so called “state and trait” sources of variance that could be clarifying. This was not possible with only one two-minute resting state condition at the beginning of the assessment. We would be able to say something more confidently about the association between the physiological data predicting behavioral task performance if we could repeatedly compare a resting state to a task state.

Second, future research would do well to operationalize FPc using EEG along with fMRI or fNIRS (both of which have far more precise spatial resolution compared to EEG). Although EEG is the superior measure for temporal resolution, it is a far less sensitive measure of individual differences in brain region connectivity (Warbrick, 2022).

Lastly, as is often the case with developmental cross-sectional studies, a longitudinal design would benefit this line of inquiry. Rather than having only a one-time snapshot of each individual’s current functioning, a longitudinal study would offer insight into changes in each individual over time. Longitudinal data are essential for making strong inferences not only about the stability (or instability) over time of between-person differences, but also about within-person changes that may be better markers of real developmental shifts within an individual. By comparison, cross-sectional age differences may tell us relatively little about actual developmental processes in the systems of interest that we are studying.

On this final point, the current study's dataset is cross-sectional with regard to women's ages. However, it still might be feasible to examine short term within-person changes during a single assessment session. To that end, we used the current Study 2 dataset to extend the analyses by employing a person-centered measurement approach. These analyses and results are reported as Study 3 in the following Chapter 4.

3.5 TABLES AND FIGURES

Table 3.5.1

Descriptive Statistics and Bivariate Pearson Correlations

	EF	EC	RSA	FPc^{F3}	FPc^{F4}	Age
EF	---					
EC	.03	---				
RSA	-.04	.10	---			
FPc^{F3}	.04	$.06^{*}$	.04	---		
FPc^{F4}	.11	.08	$.18^{*}$	$.62^{**}$	---	
Age (yrs)	$-.13^{*}$	-.10	$-.24^{**}$	-.05	-.11	---
mean	.00	4.60	2.84	0.13	0.12	37.23
SD	.59	0.70	0.57	0.03	0.03	6.02

Two-tailed p-values: + $p < .10$, * < .05, ** < .01, *** < .001.

Note: EF (executive function), CR (cognitive reappraisal), EC (effortful control), RSA (respiratory sinus arrhythmia), FPc^{F3} (frontoparietal coherence for F3-P3/F3-P7 sites), FPc^{F4} (frontoparietal coherence for F4-P4/F4-P8 sites).

Table 3.5.2

Aim 1 Regression Results for Age as Moderator of Association between FPc^{F4} and RSA

Age as Moderator of FPc^{F4} predicting RSA				
F (3, 132) = 4.11, p = .008, R^2 = .090				
	B	(se)	β	p
Age	-.220	(.084)	-.220	.010**
FPc ^{F4}	.173	(.086)	.170	.046*
Age*FPc ^{F4}	-.014	(.085)	-.014	.867

Age as Moderator of RSA predicting FPc^{F4}				
F (3, 132) = 1.88, p = .136, R^2 = .040				
	B	(se)	β	p
Age	-.056	(.086)	-.057	.518
RSA	.181	(.088)	.184	.041*
Age*RSA	.014	(.072)	.017	.842

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Table 3.5.3

Aim 2 Regression Results for Age as Moderator of Association between FPc^{F4} and RSA predicting Executive Function

Age, FPc^{F4}, and RSA predicting EF

$F(7, 128) = .979$, $p = .450$, $R^2 = .050$

	B	(se)	β	p
Age	-.153	(.090)	-.154	.091
FPc^{F4}	.106	(.098)	.105	.281
RSA	-.104	(.092)	-.104	.261
Age*FPc^{F4}	-.019	(.099)	-.019	.846
Age*RSA	-.006	(.075)	-.007	.938
FPc^{F4}*RSA	-.113	(.093)	-.121	.227
Age*FPc^{F4}*RSA	.058	(.076)	.074	.447

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Figure 3.5.1
Age (x-axis) and Executive Function (standardized composite) (y-axis) curvilinear relationship. This scatterplot includes the linear (solid line) and quadratic (dotted line) functions representing the association between maternal age and EF task performance, indicating a decline in EF with an increase in age.

Table 3.5.4

Effect size comparison between Aim 1 for Study 1 and Study 2

	Study 1		Study 2			
	sign	*β*	sign	*β*	same sign	absolute difference
Model FPc^{F3}						
Predicting RSA						
Age	negative	.31*	negative	.23*	yes	.08
FPc^{F3}	negative	.21*	positive	.02	no	.23
$Age*FPc^{F3}$	negative	.07	positive	.07	no	.14
Predicting FPc^{F3}						
Age	negative	.07	negative	.04	yes	.03
RSA	negative	.25*	positive	.04	no	.29
Age*RSA	negative	.14*	positive	.07	no	.21
Model FPc^{F4}						
Predicting RSA						
Age	negative	.32**	negative	.22**	yes	.10
FPc^{F4}	negative	.06	positive	.17*	no	.23
$Age*FPc^{F4}$	positive	.22*	positive	.87	yes	.65
Predicting FPc^{F4}						
Age	negative	.33**	negative	.06	yes	.27
RSA	negative	.03	positive	.18	no	.21
Age*RSA	positive	.19	positive	.02	yes	.17

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Two-tailed p-values: + $p < .10$, * < .05, ** < .01, *** < .001.

Table 3.5.5

Effect size comparison between Aim 2 for Study 1 and Study 2

	Study 1		Study 2			
	sign	*β*	sign	*β*	same sign	absolute difference
Model FPc^{F3}						
Age	positive	.33**	negative	.13	no	.46
FPc^{F3}	positive	.16	positive	.03	yes	.13
RSA	positive	.02	negative	.06	no	.08
$Age*FPc^{F3}$	positive	.18	negative	.13	no	.31
Age*RSA	positive	.04	negative	.01	no	.05
$RSA*FPc^{F3}$	positive	.18	negative	.20*	no	.38
$Age*RSA*FPc^{F3}$	positive	.30*	positive	.01	yes	.29
Model FPc^{F4}						
Age	positive	.19	negative	.15	no	.34
FPc^{F4}	negative	.10	positive	.11	no	.21
RSA	negative	.11	negative	.10	yes	.01
$Age*FPc^{F4}$	positive	.29*	negative	.02	no	.31
Age*RSA	negative	.01	negative	.01	yes	.00
$RSA*FPc^{F4}$	positive	.02	negative	.12	no	.14
$Age*RSA*FPc^{F4}$	negative	.30*	positive	.07	no	.37

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Two-tailed p-values: + $p < .10$, * < .05, ** < .01, *** < .001.

IV. CHAPTER 4: STUDY 3 – EXTENSION, PERSON-CENTERED ANALYSIS

4.1 PREAMBLE

This study is presented as a separate chapter from Chapter 3 (as was originally proposed). However, in the event that the results of the current Chapter 4 are disseminated, it will be combined with Chapter 3 into a single integrated publication. As a Study 2 extension, the current chapter is concise by design. It shares much of the same background and rationale and utilizes the same dataset as Study 2 (see 3.2 Methods). The innovation in current Study 3 is its distinct operationalization and analysis of the connection between cardiac and brain indicators of cognitive regulation. We use the Q-methodology, described in detail below, to transform the cardiac and neural physiological measures into one integrated cognitive regulation variable. The Q-methodology transformation facilitates a conceptual shift from a variable-centered to person-centered focus to analysis and interpretation of findings.

Given the similarities between the current Study 3 and both Study 2 and Study 1, the reader should refer to Chapters 2 and 3 for the in-depth background literature and data collection methods information. Specifically, the Introduction sections of Chapter 2 and Chapter 3 detail the rationale and guiding theories for the entire dissertation, including the current study (see 3.1 and 4.1 Introduction). For details about the participants and data collection, refer to Study 2 Methods (see 3.2 Methods). In this brief Introduction that follows, the overarching goals of the previous two studies and key points and theories guiding the dissertation are summarized.

4.2 INTRODUCTION

Utilizing a developmental framework, the current study examines potential patterns in the associations between indicators of neural (i.e., EEG), cardiac (i.e., EKG), and behavioral (i.e., task performance, self-report survey) indicators of cognitive self-regulation in adult women. The Neurovisceral Integration (NVI) model informed our decision to use these variables (e.g., Thayer et al., 2009). These variables are known indicators of cognitive regulation, and previous research has found associations between some of these measures at single points of development as well as across developmental periods (see 1.1 Introduction).

The commonly used *variable-centered* analysis approach, like that utilized in Study 1 and 2, assumes that there is only one "class" (group) of individuals, for whom the identified developmental process is operating in the same way. That is, the variables of interest vary and covary with each other in the same way for all individuals, because they presumably represent one population. In contrast, a *person-centered* approach does not assume a single population as one class (group) for whom the variables of interest vary and covary in the same way. Instead, it assumes the patterns of covariation between variables *within* each individual vary within those individuals, and between individuals in potentially very distinct ways.

Thus, person-centered, within-person methods examine whether and how within-person variation and covariation between the variables of interest are distinctly associated with other variables (e.g., Howard & Hoffman, 2018; Woo et al., 2024). Furthermore, there is

another fundamental difference between variable- and person-centered approaches that influences how one conceptualizes variation, covariation, and a "system" of constructs working together. For our research in current Study 3, the person-centered approach we use considers the covariation between the physiological cognitive regulation indicators from EEG and EKG *within each person*. Conceptually, this shifts the focus from the between-person correlations between each pair of indicators, toward within-person correlations among all of the indicators simultaneously with a more *holistic* point of view (Watts & Stenner, 2005; Watts & Stenner, 2007).

Although person-centered approaches that quantify within-person variation and covariation of variables gradually are becoming more common as a method for investigation (e.g., latent profile or class analysis, latent growth mixture modeling, and Q-Methodology—a form of an intraclass correlation), they still are vastly underutilized. This is despite the emphasis on both variable- and person-centered analysis as key features of Developmental Systems Theory (Moore, 2016) to devise ways to optimize healthy development. To this end, the person-centered within-person analysis approach has been utilized to a limited degree, to more comprehensively address questions related to health and development than variable-centered approaches can do on their own (e.g., Göktas, 2022; Ludlow, et al. 2021; Valenta et al., 1997). Still, variable-centered approaches continue to predominate in all fields that use quantitative methods.

As noted above, there are a variety of person-centered analysis methods. We will be using the Q-methodology in the current study. This method was originally developed for

qualitative (often narrative) research where subjects rank ordered their own open statements or attitudes about a topic. The within-person variation came from the "statement" ranking operationalized as the Q-sort Score (Kampen & Tamás, 2013), which is a quantitative measure of a "connected series" (Watts & Stenner, 2005). In their review, Watts and Stenner say that – based on Moscovici's interpretation of the function of the Q-methodology it was useful in developing "…constructions and representations of a social kind…" (Moscovici, 1981). But the Q-methodology application has evolved and expanded, from its original use in case studies, to be even just as suitable for calculating a within-person score that then can be applied to far more complex arrays of quantitative data (Watts & Stenner, 2005). It has done this by evolving from just the use of ranked statements (via the Q-sort) to now being used to calculate a "Q-correlation" which assesses nominally distinct (but dimensionally distributed) variables at repeated distinct conditions and occasions (e.g., trial to trial, or task to task, or monthly, or annual, etc.). One can then compute an overall Q-correlation from those different conditions and time points to derive a Q-Score (described more in depth below) which then can be thought of as a new variable that captures distinct patterns of covarying indicators within each individual in a sample of individuals.

In the current study, we are utilizing the Q-methodology to develop a construction or representation of the *physiological* kind instead of the *social* kind (Moscovici, 1981). Cattell (1952) describes the Q-methodology as a "derivation of abstract factors." Our abstract factor (represented by the Q-score) is essentially an intraclass correlation between two conceptually and functionally distinct physiological cognitive regulation variable sets.

Specifically, we are exploring whether there are individual differences in the patterns of covariation between RSA and FPc physiological indicators of cognitive regulation, by computing a Q-correlation across short periods of time in different resting state and task conditions. The resulting computed Q-correlation can then be analyzed as a unique Q-score for each individual (Damino, 2016; Duncan Millar et al., 2022; van Montfort, 2018, Watts & Stenner, 2005; Watts & Stenner, 2007; Yin et al., 2021). This Q-score allows us to describe within-person covariation between variables across all conditions (i.e., resting and three cognitive tasks performances), describe the Q-score distribution in a sample of individuals, and then examine whether that distribution of Q-scores is associated or interacts with other variables of interest (such as age) in the statistical prediction of standard measures of cognitive regulation capacity, including executive function (EF; see prior chapters for more detailed description of EF and its role as a key aspect of self-regulation).

The traditional and widely used "R-correlation" method represents the standardized covariation between two variables based on the between-person variation only. In contrast, the Q-correlation generates a correlation for each individual. By definition, and just like an R-correlation, a Q-correlation (and its resulting Q-score) can range from -1.00 to +1.00 (Duncan Millar et al., 2022). A positive Q-score within the context of our research would mean that the RSA measures during each condition shows a high level of covariation with the FPc measures during those same conditions. A negative Q-score means that higher levels on RSA indicators is related to lower levels of FPc measures during those same conditions. A Q-score near 0 indicates no covarying pattern between the RSA and FPc

indicators within the individual. Thus, the Q-correlation and Q-score represent the *magnitude* and *direction* of covariation based on the similarity/dissimilarity of score *profiles* across two variable arrays, within each person (e.g., Aiken, 1988; Block, 1961; Cattell, 1952; Stephenson, 1953; Watts & Stenner, 2007). The Q-correlation analysis results in a Q-score which can be described in its own right and used for subsequent traditional variable-centered analysis. In the current study, the two arrays of variables are the physiological variables from ECG (i.e., RSA) and EEG (i.e., FPc), aligned in the same order according to assessment condition (i.e., resting state, task 1, task 2, etc.). The Q-score is a new variable representing the within-person score *profile* similarity (i.e., covariation) for FPc and RSA. We then used this Q-score in more traditional, variable-centered multiple regression analyses to test for additive and interactive effects for predicting EF. We did not include EC and CR in the replication. There were not any additive or interactive effect for either of these variables in Study 1.

The fundamental premise of the current study is that each person is a unique system (i.e., RSA, FPc, and cognitive regulation measures covary in a distinct way for each individual) —without assuming there is only one class (i.e., group or population) for whom the variables covary in the same way. This is not to say the traditional R-correlation methodology is flawed or has no value; indeed, we have utilized that approach in Studies 1 and 2 and do so in some of the analyses in current Study 3. Rather, R-correlation methodology has been widely utilized because it does have many strengths and is the only way to estimate covariation of variables between individuals. Cattell (1952) highlights that, following standard R-correlation analyses with Q-correlation analysis, one can begin to

look within the single sample or population for indications of non-homogeny, i.e., heterogeneity, in how key indicators covary with each other. As we do in the current study, R- and Q-correlation methodology can be used together, for a fuller understanding of both within- and between-person patterns of covariation among constructs of interest (Watt & Stenner, 2012).

To this end, in the current Study 3, we first calculated two Q-scores using one RSA array and one FPc array; this was done twice, once for RSA and FPc^{F3}, and again for RSA and FPc^{F4}. We then used each Q-score to take a person-centered approach addressing two aims: (Aim 1) tested associations between age and the Q-scores, and (Aim 2) tested additive and interactive effects of age and the Q-scores predicting executive function (EF). We expected to see individual difference in the association between age and Q-scores (Aim 1), and additive and interactive effects of age and Q-scores on EF (Aim 2) based on the Study 1 results that pointed toward developmental difference in cognitive regulation and the NVI and SOC models. However, based on the lack of replication in Study 2, it is difficult to be confident with this prediction. Despite the inconsistent results between Studies 1 and 2, based on the analytical and conceptual shift as explained previously, we do expect to see person-centered age-based differences in the additive and interactive effect of age and Q-score predicting EF.

4.3 METHODS

4.3.1 Participants

The participants in this study are the same sample as those in Study 2. To summarize, there were 137 individuals who self-identified as women (age, *M* = 37.23 yrs, *SD* = 6.02 yrs). For more details about the participants, refer to the Study 2 Methods (see 3.2 Methods).

4.3.2 Procedures and Measure

4.3.2.1 Executive Function

Four cognitive tasks were administered using a computer. The variables of interest were standardized and averaged to calculate a general executive function composite z-score variable (Tower of Hanoi, Wisconsin Card Sort Test, Stroop Color-word Task, and Backward Digit Span Task. Together these tasks test for working memory/updating, set shifting/attentional control, and inhibitory control (Miyake & Friedman, 2012). (See 3.2.3.1 EF for details).

4.3.2.2 Respiratory sinus arrythmia

Electrocardiogram was used to collect heart rate data. Respiratory sinus arrythmia was calculated as the high-frequency band of heart rate variability. (See 3.2.3.2 Respiratory Sinus Arrythmia for details).

4.3.2.3 Frontoparietal alpha-power coherence

Electroencephalography was used to collect electrical brain activity. Frontoparietal alpha-power coherence was considered the coherence in alpha waves (8 to 12Hz) between F3-

P3, F3-P7 F4-P4, and F4-P8 sites. The two site sets per hemisphere were then averaged to derive composite scores: FPc^{F3} and FPc^{F4} (See 3.2.3.3 Frontoparietal Alpha-power Coherence for details).

4.3.3 Data Analysis

Data analyses were performed in *IBM SPSS Statistics* (Version 26, 2019): descriptive statistics, bivariate correlations, and multiple regressions. All variables were standardized to center them, for use in regression analyses to estimate statistical interaction effects.

The Q-correlation, referred to in the analyses as the Q-score, was calculated in *Excel*. Using the correlation function in Excel, the Q-score was calculated using two arrays of variables within each person. The first array consisted of the RSA variables for each condition (i.e., resting state, Tower of Hanoi, Wisconsin Card Sort Task, and Stroop Task). The second array consisted of the FPc variables for the same conditions in the same order (i.e., resting state, Tower of Hanoi, Wisconsin Card Sort Task, and Stroop Task). The possible range for the Q-scores was from -1.00 to 1.00.

There were two Q-score variables calculated for each individual: RSA-FPc^{F3} Q-score and RSA-FPc^{F4} Q-score.

4.4 RESULTS

Descriptive statistics and bivariate correlations were calculated and can be found in Table 4.8.1. A summary of this information is first offered, followed by more detailed

consideration of these preliminary results. In sum, only two correlations were significant. There was a moderate positive correlation between RSA-FPc^{F3} and RSA-FPc^{F4} Q-scores, and a modest negative correlation between age and EF.

The Q-scores for FPc^{F3} and FPc^{F4} were not normally distributed, but instead had wide distributions approaching a uniform distribution (see Figure 4.8.1). Thus, there was ample variation in Q-scores between participants.

4.4.1 Aim 1 Correlation between Age and RSA-FPc Q-scores

There were no significant correlations between age and the FPc^{F3} Q-score or the FPc^{F4} Q-score. To examine potential curvilinear associations between age and each Q-score, quadratic functions also were estimated and were not significant (see Figures 4.8.2 and 4.8.3).

4.4.2 Aim 2 Age and RSA-FPc Q-score interaction predicting EP

Though the Q-scores were not normally distributed, we proceeded with a traditional parametric general linear model analysis method. Normality is an assumption for using a standard linear regression model, but such analyses are highly robust to violations of normality if there is ample variability. Also, two of the most vital assumptions were met: linearity ($\delta = 0$, $SD = 1$) and independence of the errors (Ernst & Albers, 2017). It should also be noted that the distribution is uniform distribution, there is no transformation that will convert to a normal distribution.

The first equation involved estimating the additive and interactive predictive effects of age and the FPc^{F3} Q-score in the statistical prediction of EF. None was significant (see Table 4.8.2).

The second equation involved estimating the additive and interactive predictive effects of age and the FPc^{F4} Q-score in the statistical prediction of EF. Again, none was significant (see Table 4.8.2).

4.4.3 Post-Hoc Analysis Consideration of Quadratic Interaction Effects

To be as thorough as possible, a final additional analysis was conducted including age-squared and Q-score-squared as additive and interactive effects into an extended regression equation—the event that the linear effects reported above were not capturing potential age differences. However, as with the linear-effects only analyses, none of the effects was significant (see Tables 4.8.3 and 4.8.4).

4.5 DISCUSSION

There were two aims in this study: (Aim 1) we analyzed the associations between age and the Q-scores, and (Aim 2) we estimated the addictive and interactive effects of age and the Q-scores in predicting EF. These aims used a person-centered approach by using Q-correlation methodology (a special case of intra-class correlation) to first calculate two Q-scores for each individual (RSA-FPc^{F3} and RSA-FPc^{F4} Q-scores), then analyze those as new variables. For both Aims 1 and 2, there were essentially no significant results.

It is worth noting that there was a significant positive correlation between the two Q-scores with each other (i.e., Q-score for RSA-FPc^{F3} with Q-score for RSA-FPc^{F4}). However, the set of variable arrays for both Q-scores used the exact same RSA array when calculating the FPc^{F3} and FPc^{F4} Q-correlations; there was no alternative to this, since unlike FPc, RSA is not hemispheric. This use of identical information for RSA might be accounting for some or all of the correlation between the two Q-scores. In addition, the two hemisphere FPc scores in Studies 1 and 2 also were moderately correlated, so that also could be contributing to this positive correlation between the two Q-scores in the current study.

The Q-score distributions for RSA-FPc^{F3} and RSA-FPc^{F4} were not normal but were widely distributed. They can best be described as continuously uniform, given they were not distinctly modal or multimodal. Despite the lack of normality, we proceeded with traditional parametric general linear model analysis methods, because they are highly robust to variable distributions that are not normal (Ernst & Albers, 2017). It should also be noted that due to the uniform distribution of the Q-correlation, it is not feasible to transform the data. Within the uniform distribution, the average Q-score for both FPc^{F3} and FPc^{F4} were near 0, which suggests there was about an equal number of women showing a negative and positive association between RSA and FPc indicator arrays. We can say then that there is no prevailing or normative pattern of positive (or negative) correlation between RSA and FPc when using within-person calculation method.

The conventional interpretation of a uniform distribution, like that found for the Q-score distribution in the current study, is that there is an equal probability for each outcome to

occur. So, what exactly is this distribution telling us about the Q-scores in the current study? One possibility is that there is not a meaningful covariation between the two physiological variable arrays in their predicting variance in women's cognitive regulation. In other words, it could just be random noise. One counterpoint to this interpretation is that the two Q-scores (for FPc^{F3} and FPc^{F4}) were both at least moderately positively correlated with each other, suggesting within-person reliability in the Q-score measurement approach. A second counterpoint is that a third (or more) unmeasured construct is accounting for underlying patterns of covariation, and the current study model is not able to detect this. A third possibility is that there are indeed additive and interactive effects of the physiological and behavioral cognitive regulation indicators included in our research. It is likely that because this is a complex system, we are missing essential features of the system in our approach to detection or operationalization of the key underlying mechanisms.

As established in the introduction, the unique contribution of the Q-correlation and derived Q-score is that it allows for inclusion of potentially important variation in the multiple measures that then can be used to assesses within-person variability by intentionally *not* averaging across those measures for each person (Damino, 2016; Duncan Millar et al., 2022; van Montfort, 2018, Watts & Stenner, 2005; Watts & Stenner, 2007; Yin et al., 2021).The Q-score variables that we computed in the current study captured the covariation of the RSA and FPc physiological data *within* each individual. But there are limitations with this analytical approach. We discuss these and other possible limitations in the next section.

4.6 LIMITATIONS

Given that the current study used the same dataset as Study 2, some of the same limitations apply (see 3.4.4 Limitations). First, replication itself has limitations. Tackett et al. (2017) suggest a couple of potential issues in particular. They consider the possibility of detection bias or surveillance bias, which is defined as detecting certain results due to increased testing or screening. While our sample populations we both tested only once, some were familiar with the laboratory setting. Our datasets of women were used in two difference prior studies and the level of prior exposure to the data collection setting of the laboratory varied between them (given that the women in Study 2 had been visiting the lab for years with their child who was part of an ongoing longitudinal study, but that was not the case for the women in Study 1). Given that we were collecting physiological and behavioral data that could have been subjected to influence from *stressors* within the environment, it is good to note this difference between the two datasets. Tackett et al. also caution that sometimes there is overconfidence when we find a significant result; paying closer attention to statistical power and effect sizes can serve to diminish that overconfidence. Relatedly, there are other investigators concerned with small effect sizes and how they hinder the replicability of a statistical finding in a prior study or studies (e.g., Flora, 2020; Ward & Kemp, 2019).

Second, another limitation discussed in Study 2 relates to the "nicheness" of many fields of research (Sweller, 2023). When there is a tight focus on only a particular method and way to operationalize each construct, there is often not a lot of literature that can corroborate methods and theories. We consider our research within a few well-established

frameworks including SOC and NVI models, but the attempt to find developmental links between physiological and behavioral measures using a very specific and rarely used person-centered approach could be considered contributing to only one niche in science.

Third, it also is important to consider unique and unknown characteristic of the sample being investigated (Simons, 2014). While there are specific features of our samples that we know might have impeded replication – for example, each prior study had distinct age distributions and were collected in different calendar years – it is highly plausible that there are additional features unknown to us that are distinctly different between the two datasets; these distinctions might have influenced the results of our attempts at replication.

There are additional limitations specific to the Q-correlation method. First, the sparseness of the number of variables used to calculate the Q-scores could raise concern. Studies in the field of psychology that utilize the Q-methodology often have used many more variables in the arrays than we did in the current study (e.g., van Tuijl et al. 2005; Wood & Furr, 2016). One way to address this possible issue would be to add a series of resting conditions to precede and follow each task condition, in a future study. In Study 2 and current Study 3, there was only one resting state condition at the very beginning of the assessment session. This multiple resting state design was suggested in the Future Directions section of Study 2 (see 3.5 Future Directions) as a means for distinguishing between state and trait variation between people. However, this suggested study design modification would also carry the added benefit of allowing for a more robust estimation of meaningful within-person covariation in Q-correlation estimates by providing more

indicators in the variable arrays. This concern aside, the Q-correlation is essentially a special case of intra-class correlation and as such does not require a specific number of variables in the arrays. For instance, intra-class correlations are usually calculated with just two variables.

A second limitation specific to the Q-methodology is that it is vastly underutilized. Though it was developed in conjunction with some of the more common correlational analytical methodologies in the early history of quantitative measurement methods (Turner, 1955), it is rarely used in most fields of research, including psychology and neuroscience. The result of this is a lack of literature using this method for within-person measurement and analysis. Thus, there is not a literature to read and reflect on for a better understanding of null and significant results using Q-scores. Nevertheless, Q-correlation methodology has a sound rationale even though it is rarely used. Turner (1955) eloquently stated in his review of W. Stephenson's book, *The study of Behavior: Q-Technique and Its Methodology*, that this method is essential for anyone exploring "…analysis of self, with individual role conceptions, and with other phases of microsociology…". In all future explorations of individual differences in women's adult development and health, we argue that including more within-person person-centered measurement and analysis methods will benefit our understanding while also contributing to much-needed development of the Q-correlation literature generally.

4.7 FUTURE DIRECTIONS

All three Future Direction suggested in Chapter 3 also are applicable here in this person-centered analysis extension. To reiterate, we concluded previously that adding more resting conditions would lend to a better and more robust calculation for the Q-correlation as it would also allow for analysis of state-to-state changes between resting periods and task periods.

We also highlighted the possibility of considering other head and heart physiology measures as operationalizations for cognitive regulation. We have specific rationale for using RSA and EEG, but it could be argued that there is just as much rationale for using HRV (heart rate variability) and fMRI (functional magnetic resonance imaging) or NIRS (near-infrared spectroscopy).

Lastly, as is true for most developmental studies, a longitudinal design would offer a better framework for examining within person individual differences. We have merely taken a snapshot of one short period of time for each individual during a single laboratory visit session. We employed specific methods to be able to infer developmental processes (specifically, by examining a sample with a wide age range), but it would be very advantageous to see the stability or changes within individuals over longer periods of time. (3.5.4 Future Directions).

There are also possible future directions related specifically to the Q-methodology. It has been recommended that a normative profile be developed for models using the Q-

methodology. This allows for identifying extreme difference between individuals. With the normative profile, it may be possible to detect subpopulations based on the profiles that are distinguishable from the others. In addition, it would be advantageous to increase the number of resting conditions in each physiological array so there are resting conditions matching each task condition. Lastly, we propose determining an appropriate number of participants based on a Q-methodology specific power analyses.

4.8 TABLES AND FIGURES

Table 4.8.1

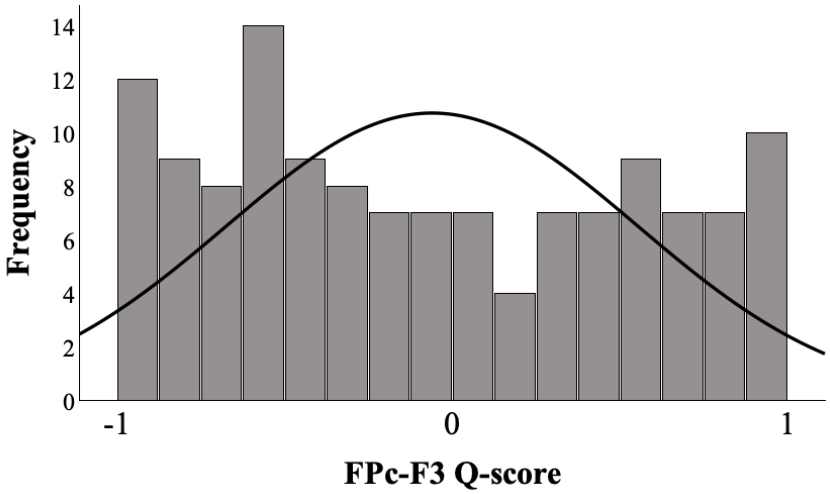
Descriptive Statistics and Bivariate Pearson Correlations

	EF	Q-FPc^{F3}	Q-FPc^{F4}	Age
EF	---			
Q-score FPc^{F3}	.09	---		
Q-score FPc^{F4}	.07	.36**	---	
Age (yrs)	-.13*	-.05	.13	---
mean	.00	-.06	-.05	37.23
SD	1.0	0.61	0.57	6.02

Two-tailed p-values: + $p < .10$, * < .05, ** < .01, *** < .001.

Note: EF (executive function), CR (cognitive reappraisal), EC (effortful control), RSA (respiratory sinus arrhythmia), FPc^{F3} (frontoparietal coherence for F3-P3/F3-P7 sites), FPc^{F4} (frontoparietal coherence for F4-P4/F4-P8 sites).

a)



b)

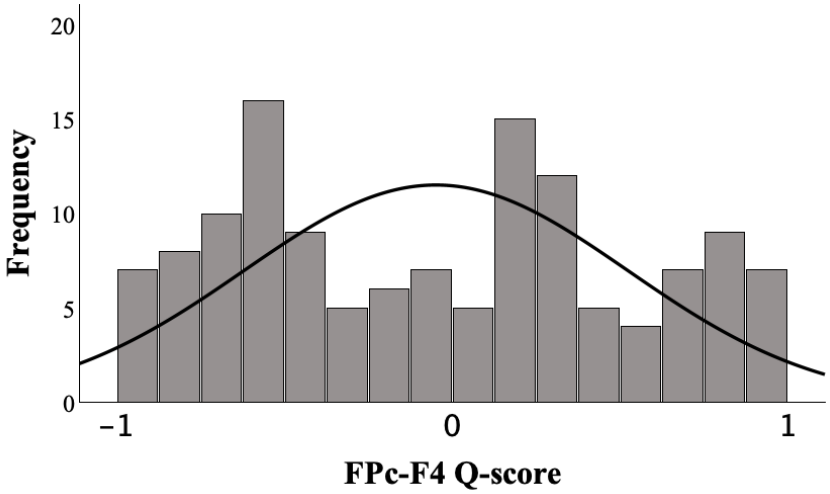


Figure 4.8.1
Histogram for Q-score Distribution a) FPc^{F3} Q-score and b) FPc^{F4} Q-score

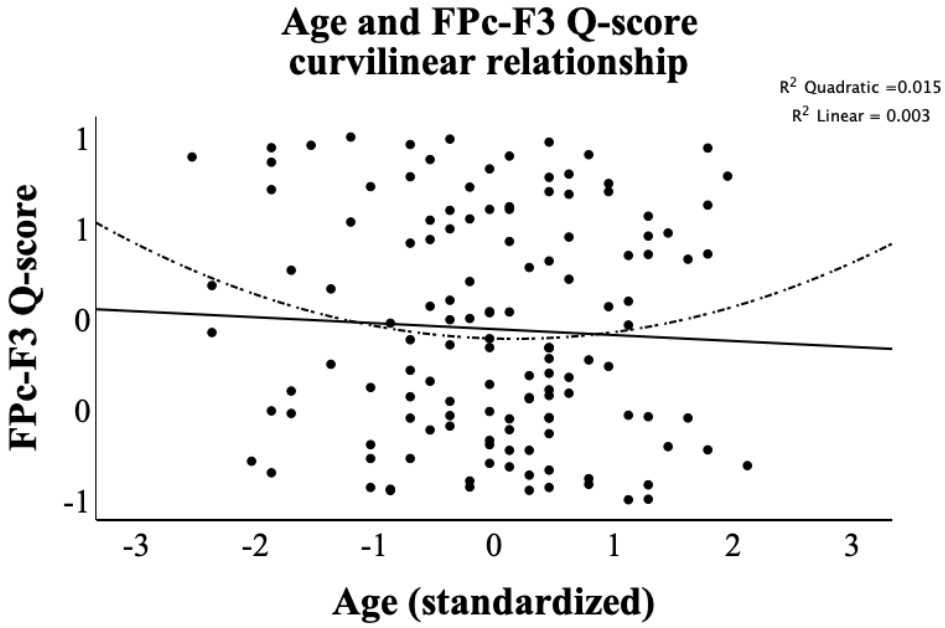


Figure 4.8.2
Linear and curvilinear graph. Correlation between Age and Q-score for RSA and Frontoparietal Coherence FPc^{F3} arrays (FPc-F3 Q-score).

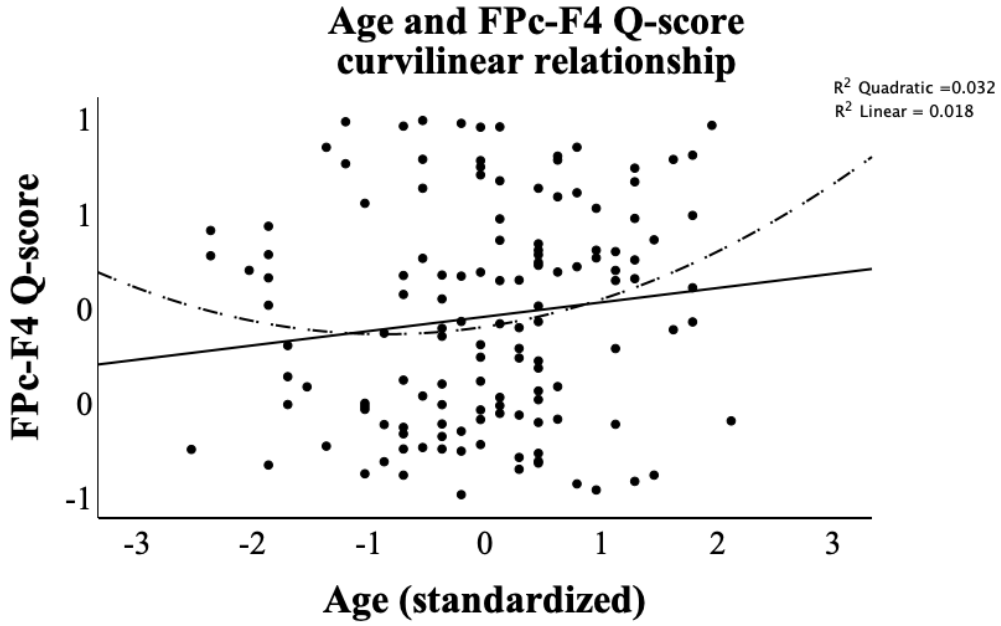


Figure 4.8.3
Linear and curvilinear graph. Correlation between Age and Q-score for RSA and Frontoparietal Coherence FPc^{F4} arrays (FPc-F4 Q-score).

Table 4.8.2

Multiple regression between Age and Q-score arrays (resting and task RSA and Frontoparietal Coherence FPc^{F3} and FPc^{F4} predicting EF.

	Age x RSA-FPc^{F3} Q-score $F(3, 128) = 1.18$, $p = .32$, $R^2 = .03$				Age x RSA-FPc^{F4} Q-score $F(3, 128) = 1.87$, $p = .14$, $R^2 = .04$			
	B	(*se*)	*β*	*p*	*B*	(*se*)	*β*	*p*
Age	-.133	(.089)	-.132	.138	-.158	(.089)	-.156	.078
Q-score	.089	(.088)	.088	.316	.087	(.088)	.086	.324
Age x Q-score	.051	(.083)	.054	.543	.141	(.091)	.135	.124

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Table 4.8.3

Multiple regression between Age^2 and Q-score arrays (resting and task RSA and Frontoparietal Coherence, FPc^{F3} and FPc^{F4}) predicting EF.

	Age^2 x RSA-FPc ^{F3} Q-score $F(5, 126) = 1.10$, $p = .37$, $R^2 = .20$				Age^2 x RSA-FPc ^{F4} Q-score $F(5, 126) = 1.99$, $p = .08$, $R^2 = .27$			
	B	*(se)*	*β*	*p*	*B*	*(se)*	*β*	*p*
Age	-.100	(.091)	-.099	.271	-.103	(.091)	-.101	.581
Q-score	.063	(.187)	.038	.738	.334	(.191)	.189	.084
Age^2	.085	(.069)	.122	.219	.070	(.068)	.092	.303
Age x Q-score	.111	(.138)	.072	.803	.205	(.159)	.112	.200
Age^2 x Q-score	.056	(.106)	.522	.602	-.215	(.122)	-.192	.079

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

Table 4.8.4

Multiple regression between Age and Q-score arrays[2] (resting and task RSA and Frontoparietal Coherence, FPc[F3] and FPc[F4]) predicting EF.

	Age x RSA-FPc ^{F3} Q-score^2				Age x RSA-FPc ^{F4} Q-score^2			
	$F(5, 126) = 1.00$, $p = .42$, $R^2 = .20$				$F(5, 126) = 1.36$, $p = .24$, $R^2 = .23$			
	B	*(se)*	*β*	*p*	*B*	*(se)*	*β*	*p*
Age	-.015	(.149)	-.015	.919	-.067	(.130)	-.066	.607
Q-score	.124	(.147)	.075	.400	.156	(.155)	.088	.317
Q-score^2	.214	(.292)	.065	.466	.228	(.302)	.066	.451
Age x Q-score	.110	(.138)	.071	.797	.237	(.160)	.129	.140
Age x Q-score^2	-.265	(.285)	-.139	.354	-.257	(.305)	-.108	.401

Note: FPc = frontoparietal coherence; RSA = respiratory sinus arrhythmia

V. CHAPTER 5: INTEGRATION

5.1 SUMMARY

With the large body of developmental research focusing on cognitive regulation, much is known about the function, formation, interruption, and degradation in self-regulation over the lifespan on average (e.g., Reuter-Lorenz & Lustig, 2005). Far less is known about individual differences in development of the components of the complex cognitive regulation system, such as those set forth in the Neurovisceral Integration (NVI) model (Thayer & Lane, 2000).

As cognitive regulation indicators that have been implicated in the NVI model, executive function (EF), respiratory sinus arrythmia (RSA) and frontoparietal coherence (FPc) show possible developmental patterns that can be discussed within the Selection, Optimization, and Compensation (SOC) model (Baltes, 1997). RSA and EF show increasing efficiency through childhood and adolescence, then a plateau during early or mid-adulthood, followed by increasing are-related decline. Less is known known about FPc in adulthood, but we see increasing efficiency in childhood and increased decline with age. There are clear covariations and interactions between RSA and EF or FPc and EF. For this dissertation, we hypothesized that age is a moderator of additive and interactive relationships between *all three* of these cognitive regulation indicators (see 1.1 Introduction for details).

The three studies together indicated no replicated additive or interactive patterns between age and the physiological and behavioral cognitive regulation indicators that we

investigated. Nevertheless, the results have inspired much thought and consideration that can inform future research. There were some age-based moderation effects in Study 1; however, Study 2 essentially did not replicate those findings. In Study 3, the intra-individual person-centered approach also did not show definitive age-based differences in results. Before we conclude that age and these physiological and behavioral indicators are not part of a developmental process, we consider below some of the limitations and possible ways to continue exploring the development of these interrelated systems of regulation across adulthood among women.

In conclusion, we have assumed some risks in attempting a replication and in utilizing the Q-methodology. Despite the inability to replicate and the null results of the person-centered approach, we still consider these variables as integrated regulators of cognitive regulation based on the rationale and previous findings (see 1.1 Introduction). We propose a complex system and when we add the lens of individuals as independent systems, there is yet another layer of complexity. The nature of a complex developmental systems is, well – hard to tease apart. We will discussion some of the limitation of this dissertation followed by some suggestions for future dictions in the next sections.

5.2 LIMITATIONS

First, Study 2 might not have replicated the results from Study 1 because the second study's sample was lacking younger individuals compared to the sample in Study 1. Study 1's sample had a wider age range, with many more women in their 20s. The results for Aim 1 in Study 1 indicated that it was only among the younger women that we found RSA and

FPc^{F4} to be correlated. This might mean that most of the developmental changes occur over the 20s and into the 30s (see 2.4 Discussion). Study 2's sample, by comparison, might be capturing a developmental plateau because it included older women.

Second, cross-sectional data inherently represents a snapshot in time, so we could be only detecting each woman's functioning on one occasion. Developmental hypotheses typically assume that change is occurring, so ascertaining individual differences in physiology and behavior is more difficult when using cross-sectional study designs.

Third, our samples probably included some sampling biases. For Study 2, there may be some self-selection bias not only because participants were recruited as volunteers through clinics and advertisements, but also because they had children already participating in an ongoing longitudinal study (Heckman, 1979). Survivor bias is also an issue originating from the most rudimentary subject recruitment period, whereby selective attrition occurs that goes unrecognized (Vansteelandt et al., 2018). We did not consider how many and why individuals dropped out of the original data collection. Volunteer bias is historically an issue for various reasons such as willingness, personality traits, and interests. This may be especially true in women's physiological research due to physical activity requirements during participation that might be perceived as potentially "painful" as might be the case with ECG and EEG data collection. In addition, women are less likely to participate in cardiovascular research (Nuzzo, 2021).

Fourth, studies involving only women or only men often raise questions surrounding the operationalization and measurement of "biological" sex and gender. These concerns are typically rooted in the complexity of sex and gender determination, identification, and expression, which includes genes, hormones, and gender-based socialization. Our original datasets did not include separate sex and gender variables, so we were not able to scrutinize these concepts (e.g., Schiappa, 2022;).

Fifth, RSA and FPc could have limitations as cognitive regulation indicators. RSA is thought to be associated primarily with autonomic sympathetic activity in regulation and might not detect cardiac reactivity to environmental inputs or stress which is an important part of the regulatory process (e.g., Allen et al., 2007). In addition, our FPc measure was derived from EEG which has relatively poor spatial resolution and as a result may not capture frontoparietal connectivity as directly as it would seem (e.g., Awada et al., 1997; Darva et al., 2004). Furthermore, we averaged the RSA and FPc resting and task conditions to get at the most reliable individual difference measures, but resting state-to-task state reactivity is a more commonly used approach in physiological research, and our approach may be overlooking some important sources of variation that are minimized when averaged across long periods of time during an assessment session.

Sixth, the Q-correlation methodology presents a few unique challenges that may well be limitations. The Q-methodology has been underutilized in quantitative research (Watts & Stenner, 2005; Watts & Stenner, 2007). While this method is appropriate for our Study 3 (Watts & Stenner, 2007), it has not been widely used in a developmental framework. With

sparse literature, it is difficult to investigate potential constraints and problems with Q-correlation methodology generally in the field and within the context of our specific line of research.

In addition, Wood and Furr (2016) proposed that one way to augment the Q-correlation approach is to build a *normative profile* of Q-correlation "scores" that is derived from the commonalities in the correlations within a large population of individuals. An extreme Q-score is difficult to detect without comparison individual profiles to *normativeness*. This allows for analysis of difference. Wood and Furr also suggest that sometimes one or more elements could be extreme for a subpopulation of condition and subtracting out the normative values, it is possible to identifying distinct profiles. Without this profile of Q-correlation scores, it is difficult to determine how and if an individual is different from what is typical in the population, and we only have an estimation of similarities based on a relatively small set of measured indicators per person.

Eighth, beyond the Q-methodology statistical power issue, the question of statistical power for our variable-centered analyses in Studies 1 and 2 also arises. For example, within the cardiac regulation literature, there is inconsistency in the correlation between RSA and behavioral indicators of cognitive regulation—but most effect sizes are modest in size (Balzarotti et al., 2017; Borges et al., 2020). Even within individual studies, we find that there are often null results reported in conjunction with statistically significant findings (e.g., Capuana et al., 2014; Wang et al., 2013). And upon closer inspection, significant results are often small or moderate at their largest. But we often take small effects sizes

and null results as they stand, in order to not inflate Type 1 error. Low statistical power makes it much more difficult to know whether a small or moderate effect size is a "true" effect, and getting to that conclusion requires numerous studies with very large sample sizes. The samples for Study 1 and 2 were adequately powered to detect the range of effect sizes found in the relevant literatures, but it still may be the case that we were underpowered to detect significant interaction effects in the variable-centered analyses that we conducted.

5.3 FUTURE DIRECTIONS

Despite the study limitations, we argue that there is some evidence supporting a developmental process across adulthood that pertains to these cognitive regulation indicators, given that Study 1 appeared to follow the expected pattern. Specifically, there was an association between RSA and FPc only in the younger women (see 2.4 Results). Thus, we propose possible future research exploring this topic perhaps using other analytic and data collection and design methods.

First, a longitudinal approach would be a major conceptual shift in exploring our research. Longitudinal data are essential for calculating intra-individual change patterns allow for measuring the actual change across time for one individual. The current design infers this change based on individuals at potentially difference states in development. Analyzing actual difference between one time point and another helps to elucidate if individuals are traversing a stage that incudes developmental change. We proposed that we might be seeing a shift from an optimized physiological and behavioral system of cognition where

there is more automation of the system to one where compensation for decline in RSA and executive function is moderated by FPc. A longitudinal approach could better capture individual decline and compensation. Some individuals may not experience decline in physiological and behavioral indicators of cognition until well past the ages included in this study. A longitudinal study could utilize the change variables rather than considering age as the variable inferring developmental *change*.

In addition, more observations per individual would also increase statistical power given that repeated longitudinal measurement increases power for variable-centered analysis. Analyzing a variable of *change* is a way to boost power. The latent change model (Kievit et al., 2018) or more specifically the multiple indicator univariate latent change score model could inform the developmental aspect of our research. This model takes a longitudinal approach and considers a latent factor. The latent change score derived from the difference from one time point to the next then informs within-person change. With multiple time points rather than just two, we could describe within-person change that might be due to developmental processes and stages. Then we can look at developmental patterns within the sample population.

Longitudinal studies present some significant limitations worth noting. First and foremost, there is a greater demand for time and monetary commitment on the part of the participants and researchers. This design also introduces potential sampling bias challenges as individuals might not volunteer due to the long-term commitment, and there may be a high dropout rate. Longitudinal studies also are subjected to intrinsic age effects that might

confound, but not relate directly to the research. In addition, exposure to the laboratory setting and research process (test-retest) can also interfere with longitudinal results. Addressing these issues is often a balancing act to determine an appropriate length of time between data collection. Increasing the time span could lead greater age effects and more dropout, but decreasing the span between samples might conflate the test-retest where individuals remember clearly and are more comfortable with the laboratory setting if it is recent.

Next, using a design that includes more resting conditions between each task condition is also an added element for future consideration. This will allow for analysis of cognitive and physiological demand reactivity and regulation over short periods of time, which may be the more important piece of information to consider when testing ideas from the Neurovisceral Integration model.

Third, utilizing a random coefficient model in the variable-centered analysis work could be good method for studying stable individual differences (Ployhart et al., 2002). This method is similar to (conceptually) but distinct from (computationally) the Q-correlation method.

Lastly, it is good to acknowledge the constraints and limitations are a basic level: each physiological and behavioral measure. It might be advantageous to utilize different operationalizations of cognitive regulation in the future. We chose to utilize RSA, but there are arguments for using HVR which is a less specific measure of cardiac regulation (e.g., ;

ChuDuc et al., 2013; Ernst, 2017; Kaye & Esler, 2008; Xhyeri el al., 2012; Yasuma, 2004). We can also rationalize using fMRI or fNIRS over or in tandem with EEG due to variations in temporal and spatial fine tuning (e.g., Zhan et al., 2011).

5.4 POSSIBLE APPLICATIONS

When thinking about the broad implications and application of this, we first acknowledge the obvious as suggested in the introduction of this chapter. Understanding patterns of development in women can be beneficial to families and by extension, society. Predicting cognitive decline and compensation through these physiological and behavioral indicators could be used to develop improved screening tools for clinical practice. The questions are – can we use these markers or indicators of cognition and physiology to predict decline and compensation that is happening in the real world? And can changes in the interactions between physiological cognitive regulation indicators predict changes in behavioral indicators of cognitive function?

Quickly moving beyond those questions, we caution against considering any indicator or compilation of indicator(s) of cognitive regulation as a *gold standard.* While a standard in the form of a short check list is a good thing (for instance, as is commonly used by clinicians for many types of disease or disorder states or conditions), it is far too early to build one for this complex system of cognitive regulation. We need to consider how to develop a screening measurement method and progress monitoring (i.e., developmental) measurement method that is actually meaningful and feasible. In the current studies, we used the "state of the art" technology for measuring physiology and cognitive functioning.

These technologies are not always or even seldom available for screening and progress monitoring purposes in clinical settings. But continued research related to the developmental patterns in physiological and behavioral indicators of cognitive regulation could eventually help in an endeavor to develop screening method.

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