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Resting sympathetic arousal moderates the association between parasympathetic reactivity and working memory performance in adults reporting high levels of life stress

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Abstract

The neurovisceral integration model stipulates that autonomic function plays a critical role in the regulation of higher-order cognitive processes, yet most work to date has examined parasympathetic function in isolation from sympathetic function. Furthermore, the majority of work has been conducted on normative samples, which typically demonstrate parasympathetic withdrawal to increase arousal needed to complete cognitive tasks. Little is known about how autonomic regulation supports cognitive function in populations exposed to high levels of stress, which is critical given that chronic stress exposure alters autonomic function. To address this, we sought to characterize how parasympathetic (high-frequency heart rate variability, HF-HRV) and sympathetic (preejection period, PEP) measures of cardiac function contribute to individual differences in working memory (WM) capacity in a sample of high-risk women. HF-HRV and PEP were measured at rest and during a visual change detection measure of WM. Multilevel modeling was used to examine within-person fluctuations in WM performance throughout the task concurrently with HF-HRV and PEP, as well as between-person differences as a function of resting HF-HRV and PEP levels. Results indicate that resting PEP moderated the association between HF-HRV reactivity and WM capacity. Increases in WM capacity across the task were associated with increases in parasympathetic activity, but only among individuals with longer resting PEP (lower sympathetic arousal). Follow-up analyses showed that shorter resting PEP was associated with greater cumulative risk exposure. These results support the autonomic space framework, in that the relationship between behavior and parasympathetic function appears dependent on resting sympathetic activation.

Keywords

heart rate variability; impedance cardiography; stress; working memory

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Full dataset is available online on Open Science Framework at <https://osf.io/6kjqb/>.

1 | INTRODUCTION

Working memory (WM) is established as a critical component of executive function, and is implicated in a broad range of skills from basic cognitive processes, such as attentional control and fluid intelligence measures (Conway & Engle, 2001; Cowan et al., 2005; Engle & Kane, 2003; Fukuda, Vogel, Mayr, & Awh, 2010; Giuliano, Karns, Neville, & Hillyard, 2014; Kane, Bleckley, Fukuda, & Vogel, 2009, 2011; Unsworth, Brewer, & Spillers, 2009; Unsworth & Engle, 2007) to more complex interpersonal skills such as parenting (Crandall, Deater-Deckard, & Riley, 2015; Johnston, Mash, Miller, & Ninowski, 2012).

Psychophysiological research on the correlates of WM performance has produced a confluence of evidence that greater parasympathetic control of cardiac arousal is associated with better WM performance (Thayer, Hansen, Saus-Rose, & Johnsen, 2009). According to the neurovisceral integration model, higher resting high-frequency heart rate variability (HF-HRV), a marker of parasympathetic influence over cardiac function, reflects greater activation of the prefrontal cortex suggesting a greater capacity for cognitive engagement (Thayer et al., 2009). This model additionally specifies that prefrontal activation is associated with inhibitory suppression of sympathetic cardiac input at rest (Thayer & Lane, 2009), although the sympathetic component of the model has rarely been examined. In addition, the neurovisceral integration model speculates that this integrated network of prefrontal cortex and autonomic cardiac control is implicated in the regulation of affect and is reactive to conditions of chronic stress that may lead to changes in allostatic load (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). However, associations between WM and autonomic psychophysiology are predominantly examined in normative samples, most often with university students. Thus, an evaluation of how both sympathetic and parasympathetic cardiac processes are associated with WM performance in individuals exposed to chronic stress would provide an opportunity to expand the literature and provide a more comprehensive framework for how neurovisceral integration functions in different populations.

1.1 | Parasympathetic regulation and cognitive function

The neurovisceral integration theory (Thayer & Lane, 2000, 2009) posits that HF-HRV is strongly associated with attentional control functions of the brain due to functional and anatomic linkages between prefrontal and cingulate cortices and brainstem structures that regulate parasympathetic influences on heart rate. According to this framework, an overlapping set of brain structures underlying the control of cognitive behaviors, such as WM, are involved in the inhibition of arousal via activation of the parasympathetic system (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). For WM function, these networks most heavily emphasize the role of the dorsolateral prefrontal cortex (Barbey, Koenigs, & Grafman, 2013), while regulation of parasympathetic activity involves networks emphasizing the medial prefrontal cortex and insula (Thayer et al., 2012). As such, tonic, or resting, HF-HRV is conceptualized to be a traitlike predictor of individual differences in cognitive control (Beauchaine & Thayer, 2015). A large body of research supports this conceptualization, with higher resting levels of HF-HRV linked to better performance on a range of tasks measuring executive function (Jennings, Allen, Gianaros, Thayer, & Manuck,

2015; Kimhy et al., 2013), selective attention (Park, Vasey, Van Bavel, & Thayer, 2013), and emotion regulation (Applehans & Luecken, 2006; Thayer & Lane, 2009).

In general, most studies demonstrate reductions in HF-HRV from baseline to experimental task, commonly referred to as HF-HRV withdrawal, although whether and how such reductions are correlated with task performance is less consistent. Polyvagal theory (Porges, 2001, 2007) proposes that task-related reductions in HF-HRV (i.e., parasympathetic withdrawal) reflect a regulated approach to mobilizing energy resources needed to meet the metabolic demands of active cognitive and behavioral engagement. Consistent with this theory, many studies have found associations between HF-HRV withdrawal and performance on cognitive tasks (e.g., Lenneman & Backs, 2009; Overbeek, van Boxtel, & Westerink, 2014). However, other studies have reported opposing patterns of association, in which greater augmentation of HF-HRV is associated with better attentional control (e.g., Park, Vasey, Van Bevel, & Thayer, 2014). If withdrawal of HF-HRV reflects an adaptive increase in arousal to meet cognitive demands, it is not inherently clear how increases in HF-HRV (i.e., reduction in arousal) support better cognitive function.

The relative value of withdrawal versus augmentation may be dependent on the nature of the task or the nature of the sample (Park & Thayer, 2014). For instance, several studies have reported that, when experimental task demands specifically involve regulation of negative affect, better emotion regulation is typically associated with increases in HF-HRV (see Thayer & Lane, 2009), suggesting that the nature of the task itself (cognitive or affective) may moderate the adaptive direction of physiological change. Although researchers may consider the affective nature of a given laboratory task to be quite low (e.g., basic WM tasks), whether the task is emotionally challenging is, in fact, in the eye of the beholder and may therefore differ across individuals and across samples. One study of preschool-aged children found that children from typical families showed a significant association between HRV withdrawal and better inhibitory control, consistent with findings from many studies in adults; however, children from families with a history of maltreatment showed the opposite association, with better inhibitory control associated with greater augmentation of HF-HRV (Skowron, Cipriano-Essel, Gatzke-Kopp, Teti, & Ammerman, 2014).

The opposing associations between HF-HRV and better cognitive control observed between the maltreated and non-maltreated groups raise an interesting question about whether the implication of task-related changes in HF-HRV is dependent on the individual's basal state of arousal. Following the basic premise of optimal arousal (Yerkes & Dodson, 1908), individuals in a relatively calm resting state may benefit from an increase in arousal needed to meet the cognitive demands placed on them (i.e., these individuals are to the left of the optimum peak of the inverted U). Conversely, individuals exposed to significant stress may be in a chronic state of hypervigilance that may interfere with cognitive performance unless they are able to regulate emotionally in order to allocate resources to perform the cognitive task (i.e., these individuals are to the right of the optimum peak of the inverted U). Thus, individuals with high levels of anxiety or hypervigilance may need an increase in HF-HRV to support better cognitive performance.

1.2 | Sympathetic regulation and cognitive function

It is important to note that both the sympathetic and parasympathetic systems contribute to overall cardiac arousal in ways that are nondependent. Thus, the influence of the sympathetic system cannot be estimated or assumed from quantifying the influence of the parasympathetic system (Berntson, Cacioppo, & Quigley, 1993). Because each autonomic branch is likely to have different psychophysiological implications, it is important to isolate their relative contributions. Measures of HF-HRV can be relatively easily extracted from a basic electrocardiograph signal by quantifying the variation in the interbeat interval across the respiration cycle (e.g., respiratory sinus arrhythmia). However, isolating the contribution of the sympathetic nervous system cannot be accomplished from the electrocardiograph alone. Some studies have examined other measures of the sympathetic system, such as blood pressure (Capuana et al., 2014) or skin conductance (El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008), but the most directly parallel measure of the sympathetic contribution to cardiac function is in the calculation of the cardiac preejection period (PEP; Sherwood et al., 1990). PEP is the amount of time between the depolarization of the sinoatrial node and the ejection of blood into the aorta, and reflects the unique influence of the sympathetic system (Sherwood, Allen, Obrist, & Langer, 1986). Increases in sympathetic activation shorten the preejection time, which contributes to a more rapid completion of the cardiac cycle and thus an increase in heart rate. Because calculating PEP requires additional equipment and electrodes, many studies have not been able to account for this additional source of variance in an individual's physiological arousal.

In addition to the anatomical influence of the prefrontal cortex on parasympathetically mediated cardiac function, the neurovisceral integration model also emphasizes the role of the prefrontal cortex in the tonic inhibition of sympathetic innervation to the heart (Amat, Paul, Zarza, Watkins, & Maier, 2006; Thayer, 2006). Thus, individuals with better prefrontal function are likely to have lower resting state sympathetic arousal, or longer PEP times. Because the sympathetic system is associated with robust increases in arousal, it is ideally reserved for meeting demands associated with threat (i.e., fight or flight). By inhibiting the sympathetic system during resting conditions, the prefrontal cortex prioritizes the parasympathetic system in regulating arousal needs through moment-to-moment changes in reactivity that can provide fewer systemic, and better regulated, increases in arousal to meet nonthreatening demands (Thayer & Lane, 2009). Consistent with this perspective, research has demonstrated that individuals with better executive function are less physiologically reactive to acute stress, suggesting that strong trait-level prefrontal regulatory control buffers individuals' state-level physiological reactivity (Hendrawan, Yamakawa, Kimura, Murakami, & Ohira, 2012).

Despite the predictions of the neurovisceral model, associations between state-level sympathetic reactivity and cognitive performance have been inconsistent. In studies in which arousal was experimentally manipulated through acute administration of stimulating substances (e.g., caffeine, cortisol), greater induced arousal was found to impede cognitive performance (Shields, Bonner, & Moons, 2015; Watters, Martin, & Schreier, 1997). However, studies assessing naturally occurring correlations between concurrent sympathetic arousal and cognitive performance frequently failed to detect an association (Benikos,

Johnstone, & Roodenrys, 2013; Capuana et al., 2014). Given that these studies focused on peripheral measures of sympathetic activation such as skin conductance and blood pressure, it remains unclear whether a direct measure of sympathetic influence of cardiac activity through PEP provides a better index of cortical-visceral integration.

1.3 | Implications of chronic stress

In addition to the state-specific effects of arousal, evidence indicates that temperamental traitlike predisposition toward higher or lower levels of arousal may be an important moderator of state effects. One study found that dogs selected for low basal arousal performed better on tasks requiring behavioral regulation when provided stimulating encouragement by humans, whereas dogs higher in basal arousal performed worse under these conditions (Bray, MacLean, & Hare, 2015). This pattern simply takes into account that individuals fall at different points along the inverted U at baseline, indicating that the optimal direction of change could be in opposite directions for different individuals. For instance, attention-deficit/hyperactivity disorder (ADHD) has been proposed to be a manifestation of chronic basal underarousal (Satterfield, Cantwell, & Satterfield, 1974). Consistent with this hypothesis, children diagnosed with ADHD perform better on memory tasks when tested with white noise in the background, whereas typically developing children show impaired performance under white noise conditions (Söderlund, Sikström, & Smart, 2007).

Given the importance of trait characteristics in understanding neurovisceral integration in response to state demands, it is necessary to expand our knowledge base beyond the typically developing and high functioning samples most frequently represented in the literature (see Gatzke-Kopp, 2016). Since prefrontal cortical function is essential in performing WM tasks, as well as exerting a regulatory influence over autonomic arousal, prolonged exposure to stress may be particularly disruptive to both cognitive and physiological function (Thayer & Lane, 2009). Indeed, chronic stress exposure is associated with arterial stiffening and basic changes in the perfusion of blood flow to the brain, particularly to the prefrontal cortex (McEwen & Gianaros, 2011). Individuals exposed to high levels of chronic stress are more likely to evidence lower WM (Karlmann et al., 2014) and greater levels of resting arousal as demonstrated by higher heart rate and lower HF-HRV (Propper & Holochwest, 2013). However, less is known about whether the effects of stress on physiological arousal directly mediate associations between stress exposure and cognitive performance, or if in fact the physiological processes that support WM capacity function differently among chronically stressed individuals.

Chronic exposure to stressful life experiences has been shown to affect autonomic nervous system (ANS) function across a number of studies, with greater stress exposure generally associated with higher sympathetic nervous system (SNS) activation (i.e., greater fight-or-flight arousal) and lower parasympathetic nervous system (PNS) activation (i.e., reduced calming of heart rate; Brosschot, 2010; Lucini, Di Fede, Parati, & Pagani, 2005; Pike et al., 1997; Propper & Holochwest, 2013). This pattern is consistent with the neurovisceral integration model, which proposes that increases in arousal through both autonomic branches result from the disruptive effect of chronic stress exposure on prefrontal function

(Arnsten & Goldman-Rakic, 1998; Thayer & Lane, 2009). If these individuals experience heightened arousal or hypervigilance at rest, WM performance is likely to be facilitated by an increase in HF-HRV reflecting a reduction in arousal and greater engagement of the prefrontal cortex. Thus, we hypothesize that greater arousal in the resting state will moderate the effects of HF-HRV reactivity across task performance.

1.4 | The present study

The present study investigates the association between both sympathetic and parasympathetic function in supporting WM performance in a sample of women exposed to high levels of stressful life events. Participants completed a visual measure of WM capacity that has been shown to robustly emphasize attentional control abilities (Vogel, McCollough, & Machizawa, 2005; Vogel, Woodman, & Luck, 2001), while measures of resting and task-reactivity levels of HF-HRV and PEP were continuously recorded. Specifically following the basic tenets of the neurovisceral integration model, we examined whether higher resting HF-HRV and longer resting PEP were both predictive of WM performance. We further sought to examine whether there was an association between autonomic reactivity during the task that was associated with performance, and whether performance was predicted by both autonomic branches or solely through HF-HRV reactivity. Next, we examined whether resting autonomic activity moderated associations between autonomic reactivity to the task and task performance, such as whether changes in arousal were only related to WM performance when taking into account an individual's arousal at rest. Finally, we examined whether profiles of ANS activity linked to WM performance would be associated with chronic stress exposure.

2 | METHOD

2.1 | Participants

Fifty-two mother-child dyads at risk for high exposure to life stressors were recruited through child welfare and Early Head Start, as well as through an ongoing study of mothers who had been involved with child welfare services as children. Prescreening was performed via phone call to ensure that all participants met the inclusion criteria: older than 18 years of age, fluent in English, having primary custody of a 3- to 5-year-old child at the time of enrolment, and no history of head trauma or neurological disorder. Usable physiological data were available for 42 of the mothers. This final sample was approximately 30 years old ($M = 30.42$, $SD = 6.54$), and included mothers who identified racially as Caucasian (69%), Hispanic American/Latino (14.3%), African American (7.15%), and Native American (9.5%). Parent education ranged from seventh grade or less to completion of a graduate degree, with completion of high school or GED certificate the most frequently reported (52.4% of mothers).

Income-to-needs ratios were calculated for each family on the basis of the 2015 poverty thresholds (U.S. Census Bureau; <http://aspe.hhs.gov/poverty-guidelines>). Household need was determined by size of the family unit while accounting for number of related children younger than 18 years of age. Participants self-reported monthly or annual household income. Household incomes ranged from yearly estimates of \$3,533 to \$70,088 ($M =$

\$25,870, $SD = \$13,682$). When considering household needs, 51.3% of participating families were living below the U. S. federal poverty guidelines at the time of their laboratory visit.

2.2 | Measures

2.2.1 | Cumulative risk—Mothers completed a comprehensive demographic interview to assess socioeconomic, physical, and psychosocial risk factors, from which a cumulative risk score was computed (e.g., Evans & Kim, 2007). Cumulative risk scores could range from 0 to 8, with scores based on three socioeconomic risk factors, two physical risk factors, and three psychosocial risk factors. Socioeconomic risk factors included (a) mother not having completed high school, (b) being a single parent, and (c) household income-to-needs ratio < 1.0 . Physical risk factors were assigned if participants' responses were in the top quartile for (a) household crowding (> 1 person per room), and (b) housing problems (e.g., presence of pests, structural damage, plumbing or electrical failures). Psychosocial risk factors were assigned if participants' responses were in the top quartile for (a) parent-child separation events (e.g., hospitalizations, lost custody, incarceration, or institutionalization), (b) family turmoil events (e.g., residential instability, being known to protective services, domestic disturbance, and adult substance abuse), and (c) exposure to violence inside or outside of the home. For each of these eight factors, risk was scored dichotomously as present (1) or absent (0).

2.2.2 | Working memory—WM capacity was assessed with a visual change detection task (Luck & Vogel, 1997; Vogel et al., 2001) implemented in E-Prime 2.0 presentation software (Psychology Software Tools, Pittsburgh, PA). Each trial began with a 150-ms sample array of 2, 3, or 6 isoluminant colored squares (black, blue, brown, cyan, green, orange, purple, red, white, or yellow) randomly distributed around a $14'' \times 10''$ video display. The color of the squares was sampled randomly with replacement, such that multiple squares could be presented in the sample color on a given trial. Participants were instructed to remember the location of each colored square during a retention interval of 1,000 ms of a blank display. In the test array, one of the squares from the sample appeared in either the same or a different color and with an additional red circle around it. Participants were instructed to respond "same" or "change" regarding the color of the square in the test array that was cued by the red circle. Responses were registered via a response pad placed on a desk in front of the participants, who used their left and right index fingers for same and change responses, respectively. To emphasize accuracy over speed, the test array remained on the display until the participant responded. The full task was split into six blocks of 60 trials each, totaling 360 trials with 120 trials at each set size (2, 3, and 6), evenly divided between same and change trials, and with each trial type represented equally in each block. Individual estimates of WM capacity (k) were calculated for each participant through the use of a standard formula (e.g., Cowan, 2001) in which $k = \text{set size} \times (\text{hit rate} - \text{alarm rate})$, averaged across all three set sizes. As a manipulation check, WM capacity estimates for set sizes 2, 3, and 6 were assessed at the task level using a repeated measures analysis of variance (ANOVA) with age as a covariate. Overall, WM capacity estimates (k) showed the expected set-size effect, with k values increasing with the number of items to be remembered, $F(2, 80) = 9.06$, $p = .004$. Pairwise comparisons revealed that Set Size 2 ($M =$

1.82, $SD = .17$), Set Size 3 ($M = 2.59$, $SD = .28$), and Set Size 6 ($M = 3.61$, $SD = .87$) were all significantly different from each other (all $ps < .001$). There was no main effect of age or Age $\times$ Set Size interaction (both $ps > .81$).

2.3 | Autonomic physiology recording and analysis

A montage of 11 electrodes was used for the measurement of HF-HRV and PEP. Electrocardiogram (ECG) was obtained from three disposable pregelled electrodes placed in a modified Lead II configuration on the distal right clavicle, lower left rib, and lower right abdomen. Cardiovascular impedance (Z0) was recorded from eight electrodes placed in a tetrapolar configuration on the left and right lateral neck and torso, from a vertical maximum of the jawline down to the diaphragm. Data were acquired wirelessly via Biopac Nomadix BN-RSPEC and BN-NICO transmitters (Biopac Systems Inc., Goleta, CA) sending ECG and impedance signals respectively to a Biopac MP-150 acquisition unit placed in the room with the participant. A respiration signal was derived from the raw impedance cardiogram for the inspection of respiration values.¹ High-frequency HRV values were derived from natural log-transformed values of the spectral power in the high frequency range (.12–.40 Hz) of the ECG signal. PEP was calculated from the first-order derivative of the cardiovascular impedance signal (dZ/dt), as the length of time from the Q point of the ECG waveform to the B point of the dZ/dt waveform (Berntson, Lozano, Chen, & Cacioppo, 2004).

Autonomic data were processed separately for a 5-min baseline period and for the six blocks of the WM task (approximately 5 min per block). Data processing was performed using Mindware HRV and IMP software (Gahanna, OH). First, ECG signals were inspected by trained research assistants to ensure the correct identification of individual R peaks. Edited ECG files were then used for the processing of PEP values, whereby visual inspection was used to verify that both the Q and B points were present and correctly placed in 30-s averages of ECG and dZ/dt waveforms. HF-HRV and PEP values were exported in 30-s epochs, then averaged across epochs to yield a single baseline value and a separate value for each of the six 5-min blocks of the WM task. Physiological reactivity values during the WM task were calculated for both HF-HRV and PEP as difference scores from baseline values (task minus baseline), such that positive HF-HRV reactivity scores indicated greater PNS activation and negative scores indicated PNS withdrawal relative to baseline levels. Because longer PEP intervals reflect less SNS activation, positive PEP change scores reflect SNS withdrawal during the task, and negative PEP change scores reflect SNS activation during the task, relative to baseline.

2.4 | Procedure

Before visiting the laboratory for testing, adult participants completed eligibility screening during which demographic information was obtained. Upon arrival at the laboratory,

¹We found no evidence of respiration rate influencing the results presented herein. Respiration rate did not significantly differ between long and short PEP subjects at baseline, $t(38) = -.84$, $p = .407$, or in response to the task, $t(38) = -.95$, $p = .347$. When including respiration rate at baseline and respiration changes to the task as covariates, neither the association between HF-HRV and WM capacity observed in the long PEP group, $r(17) = .66$, $p = .002$, nor the lack of relationship in the short PEP group, $r(15) = -.15$, $p = .580$, were changed. Furthermore, WM capacity was not associated with respiration rate at baseline, $r(38) = .01$, $p = .931$, or in response to the task, $r(38) = -.26$, $p = .100$.

electrodes were applied to the mother's and the child's torso for psychophysiological recordings. Then, mother and child sat on a couch and watched a neutral 5-min video clip, followed by a 5-min interaction task with child sitting on the mother's lap. Results from these tasks are discussed elsewhere (Clark, Skowron, Giuliano, & Fisher, 2016). After the interaction task, mothers were escorted into a separate room for the WM task, while the child continued with behavioral tasks. Baseline physiology data were collected for 5 min while mothers sat in a chair facing a computer screen in the same position that they maintained throughout the WM task. Mothers then completed six blocks of the visual change detection task. Participation throughout the task was self-paced, with short breaks given between blocks as needed by the participant. Once the last block of the WM task was completed, mothers were reunited with their child for collection of a variety of biomarkers not discussed here.

2.5 | Analytic procedure

Descriptive statistics and zero-order correlations were first conducted to identify the basic associations among all variables of interest. As a manipulation check of the WM task, capacity estimates for each set size of the WM task were averaged across all six blocks of the task and analyzed via a repeated measures ANOVA. To analyze within- and between-person variability in WM capacity, multilevel modeling was utilized to predict WM scores during each of six blocks of the task on the basis of HF-HRV and PEP reactivity during each of the same six blocks, while at the second level examining the effects of baseline HF-HRV and baseline PEP levels, as well as controlling for maternal age. A multilevel model was specified using Hierarchical Linear Modeling software (Raudenbush & Bryk, 2002), as shown below.

$$\text{Level 1: WM capacity}_{ij} = b_{0j} + b_{1j} * (\text{HRV reactivity}_{ij}) + b_{2j} * (\text{PEP reactivity}_{ij}) + r_{ij} \quad (1)$$

$$\text{Level 2: } b_{0j} = g_{00} + g_{01} * (\text{HRV baseline}_j) + g_{02} * (\text{PEP baseline}_j) + g_{03} * (\text{Age}_j) + u_{0j} \quad (2)$$

$$b_{1j} = g_{10} + g_{11} * (\text{HRV baseline}_j) + g_{12} * (\text{PEP baseline}_j) + g_{13} * (\text{Age}_j) \quad (3)$$

$$b_{2j} = g_{20} + g_{21} * (\text{HRV baseline}_j) + g_{22} * (\text{PEP baseline}_j) + g_{23} * (\text{Age}_j) \quad (4)$$

Follow-up analyses were then performed to explore significant interactions in the multilevel model. Participants were median split on baseline PEP values and compared on the association between HF-HRV reactivity and WM capacity, and also compared on scores for the cumulative risk index assessing exposure to socioeconomic, physical, and psychosocial stressors.

3 | RESULTS

3.1 | Descriptive statistics

Descriptive statistics and zero-order correlations between all variables of interest for all participants are presented in Table 1 and 2, respectively. Younger maternal age was significantly associated with higher HF-HRV (i.e., more PNS activation) at baseline ($r = -.43, p = .005$) and during the WM task ($r = -.43, p = .005$), but not associated with HF-HRV reactivity scores ($p = .99$). Younger maternal age was also significantly associated with longer PEP intervals (i.e., less SNS activation) at baseline ($r = .35, p = .024$) and during the WM task ($r = .31, p = .046$), but not associated with PEP reactivity scores ($p = .62$).

Overall WM capacity scores were not related to psycho-physiological scores at rest (HF-HRV, $p > .57$; PEP, $p > .15$) or reactivity (HF-HRV, $p > .36$; PEP, $p > .15$). Higher cumulative risk scores showed a trend toward relating to lower resting HF-HRV ($r = -.30, p = .058$) and were significantly related to less HF-HRV withdrawal during the WM task ($r = .33, p = .035$). There was no relationship between cumulative risk and resting PEP ($p > .32$) or PEP reactivity ($p > .28$).

3.2 | Working memory behavioral results

To characterize fluctuations in WM capacity across the task, WM capacity estimates were collapsed across set size within each block and assessed using a repeated measures ANOVA with six levels of a block factor and with age as a covariate. As shown in Figure 1, WM capacity estimates displayed a quadratic change across the task, (quadratic contrast, $F(1, 38) = 8.80, p = .005$) such that WM capacity increased from the first block to the second block, then exhibited a slight decline across the remainder of the task. There was a significant Age $\times$ Block interaction, $F(5, 190) = 2.52, p = .042$. However, follow-up comparisons of WM capacity between age groups split at the sample median revealed no significant difference between young and older participants in any of the six task blocks (all $ps > .32$).

3.3 | Modeling WM fluctuations and psychophysiology

Multilevel modeling was used to examine fluctuations in repeated measures of WM capacity across the six blocks of the task as a function of HF-HRV and PEP, controlling for maternal age. As shown in Table 3, this model is characterized by a significant cross-level interaction between HF-HRV reactivity from block to block at Level 1, and resting PEP at Level 2, in predicting block-to-block changes in WM capacity ($g_{12} = .02, t(202) = 2.44, p = .016$).

To interpret this cross-level interaction, we examined how the relationship between HF-HRV reactivity and WM capacity varied as a function of resting PEP values, based on a median split of resting PEP (i.e., shorter and longer resting PEP groups). Given that there was a significant difference in age between the shorter resting PEP group ($n = 21; M = 27.93, SD = 5.21$) and longer resting PEP group ($n = 21; M = 32.91, SD = 6.89$), $t(40) = -2.64, p = .012$, partial correlations were examined while controlling for age. This analysis revealed that, for participants with longer resting PEP (i.e., less SNS activation), increases in HF-HRV during the WM task were associated with higher WM capacity estimates overall, $r(18) = .54, p = .015$. However, there was no association between increases in HF-HRV and WM

capacity among participants with shorter PEP, $r(18) = -.12$, $p = .62$ (see Figure 2). At the individual block level, participants with longer resting PEP also showed more robust associations between block-to-block values of HF-HRV reactivity and WM capacity. As seen in Table 4, individuals with longer resting PEP showed a pattern of positive associations between HF-HRV reactivity in the first two blocks of the task and WM capacity across the majority of WM task blocks. Thus, the correlation between HF-HRV reactivity and WM capacity observed among participants with longer resting PEP appears to be carried by augmentations in HF-HRV activity during the beginning blocks of the WM task. Although the shorter resting PEP group showed no overall relationship between their HF-HRV reactivity and WM capacity, they did show a significant negative association between WM capacity in the last block of the task and HF-HRV reactivity across the last three WM task blocks.

Post hoc tests were performed to examine whether the longer and shorter baseline PEP groups differed on exposure to cumulative risk. Results of an ANOVA controlling for participant age as a covariate demonstrated that cumulative risk was significantly higher in the group with shorter baseline PEP ($M = 3.33$, $SD = 2.08$) than in the group with longer baseline PEP ($M = 2.57$, $SD = 1.43$) $F(1, 39) = 5.20$, $p = .028$. A similar ANOVA examining WM capacity while controlling for age demonstrated no significant difference in WM capacity between the shorter ($M = 2.75$, $SD = .35$) and longer PEP groups ($M = 2.71$, $SD = .41$) $F(1, 39) = 0.117$, $p = .734$.

4 | DISCUSSION

This study examined whether exposure to life stress affected associations between patterns of autonomic activity and cognitive control. Both the neurovisceral integration model and polyvagal theory propose associations between higher resting HF-HRV and better cognitive function, a finding frequently supported in the literature (e.g., Williams, Thayer, & Koenig, 2016). The majority of research, however, has examined typical samples, with less attention to participants exposed to high levels of stress thought to affect autonomic regulation. Our results indicate that, among individuals identified as at risk for exposure to a range of chronic stressors, neither resting HF-HRV nor HF-HRV reactivity were directly correlated with WM capacity. However, a significant interaction was observed such that associations of HF-HRV reactivity and WM performance were moderated by resting PEP (see Figure 3). Participants with longer resting PEP (i.e., lower SNS activity) who also evidenced an increase in HF-HRV from baseline to the WM task had higher WM scores on average. Within-person analyses also indicated that, among participants with longer resting PEP, increases in WM performance were associated with concurrent increases in HF-HRV reactivity dynamically across the task. Participants with shorter resting PEP (i.e., higher SNS activity) showed no associations between HF-HRV reactivity and WM capacity on average and few associations across individual blocks of the task. These results suggest that the role of autonomic function in support of cognitive capacity may need to be considered in light of the individuals' resting physiological arousal, which may be affected by life history.

The lack of association between resting HF-HRV and WM capacity may be a function of this sample, which differs from most samples used to assess psychophysiological correlates

of cognitive processes with regard to psychosocial risk. Higher resting HF-HRV reflects greater parasympathetic control over cardiac arousal, which is believed to support more flexible regulation of arousal and engagement of pre-frontal cortical activation (Thayer & Lane, 2000, 2009). In this sample, resting HF-HRV was not associated with WM capacity, but was associated with measures of stress exposure. Individuals who reported a greater confluence of stress exposure had lower resting HF-HRV, consistent with research demonstrating an effect of stress exposure on autonomic state (Cacioppo et al., 2000). It is possible that higher resting HF-HRV can promote better cognitive function, but is itself not sufficient for this purpose. Very little research examining HF-HRV and cognitive processes has conducted a comprehensive assessment of both parasympathetic and sympathetic function, despite both autonomic branches having implications for regulated arousal needed to engage in higher-order cognitive processing (Berntson et al., 1993). In this sample, resting PEP was positively correlated with resting HF-HRV such that individuals with lower resting sympathetic activation (longer PEP scores) had higher resting parasympathetic activation (higher HF-HRV scores). In contrast, reactivity scores for both PEP and HF-HRV were uncorrelated, indicating that the cooperative association in the resting state is not predictive of a cooperative association between the parasympathetic and sympathetic systems in how they respond to cognitive demands.

Reactivity in the sympathetic and parasympathetic systems did not independently predict WM capacity. However, interactions between resting state and reactivity were found. Specifically, resting PEP moderated the association between HF-HRV reactivity and WM capacity such that, among participants with longer resting PEP (less sympathetic arousal), better WM performance was associated with increases in HF-HRV. The association between increased HF-HRV and WM performance is consistent with the neurovisceral model, which proposes that HF-HRV reflects the autonomic output of increased prefrontal activation (Thayer & Lane, 2009). However, this pattern is specific to individuals with lower resting sympathetic arousal. It may be that a calmer physiological state at rest facilitates this pathway of cognitive engagement. This is consistent with other reports of heightened PNS activity being associated with better selective attention performance (Park et al., 2014), given that selective attention is highly correlated with individual differences in working memory (Cowan et al., 2005; Fukuda & Vogel, 2009; Giuliano et al., 2014). Although these results are somewhat conflicting with other reports that PNS activity can be suppressed during WM performance (Hansen, Johnsen, & Thayer, 2003; Overbeek et al., 2014), the contexts of previous WM measures were different in that they required continuous speeded performance, which is likely facilitated by higher arousal levels (i.e., PNS withdrawal). In contrast, the visual WM task used in the present study does not require speeded responses or motor demands and was designed to emphasize attentional control (Vogel et al., 2001), which is likely facilitated by regulated arousal (i.e., PNS augmentation). We suggest that PNS flexibility to contextual demands is relevant to cognitive performance, but likely differs based on task demands. Even though the context of the WM task used here could be considered more cognitively “cold” than “hot” (Zelazo, Qu, & Miller, 2005), it is notable that PNS activity is indeed associated with behavioral performance. This supports the notion that PNS activity is generally relevant to cognitive control and not unique to emotion regulation (Thayer & Lane, 2009). In the context of low resting SNS activity, the finding that

increased PNS reactivity predicts better WM performance is consistent with findings of reciprocal PNS dominance as an optimal physiological profile associated with cognitive regulation (Duschek, Muckenthaler, Werner, & del Paso, 2009; El-Sheikh et al., 2009).

In contrast, individuals with higher resting sympathetic arousal (i.e., shorter PEP) showed no association between WM performance and HF-HRV reactivity. Importantly, we did not observe a relationship between PNS activity and WM performance in the subset of participants with higher SNS levels at rest, who also reported higher cumulative risk exposure. This finding is consistent with prior work with patients with chronic psychosocial stress exposure that have reported higher SNS activity at rest (Cacioppo et al., 2000; Luciniet al., 2005). Given that PNS activity was not linked to WM performance in this group, it is possible that experiences of chronic stress linked to heightened SNS activity may limit individuals' ability to flexibly engage the PNS to meet moment-to-moment demands in arousal relevant to WM performance. The interrelationship between chronic stress, WM performance, and cardiac activity is possibly due to changes in blood perfusion and arterial hardening, which have been shown to particularly impact prefrontal areas (McEwen & Gianaros, 2011). In particular, stressors have been shown to impact blood flow to dorsolateral prefrontal cortex and the insula (Wang et al., 2005), regions that are highly implicated in WM performance and cardiac regulation, respectively.

These findings have key implications for research on neurovisceral integration theory, which emphasizes the role of both ANS branches for behavior, yet often measures only PNS engagement (i.e., Hansen et al., 2003; Overbeek et al., 2014; Park et al., 2013, 2014). It is an open empirical question whether the often documented relationship between parasympathetic activity and behavior, typically assessed in lower-risk samples, is particular to the cardiac autonomic space wherein sympathetic activity is low at rest (Berntson et al., 1993). Given the trend observed here between higher resting parasympathetic activation and lower resting sympathetic activation, measuring only HF-HRV may inherently capture some variance associated with sympathetic tone. In low-risk samples, this association may be stronger and statistically significant, thus the implications of resting sympathetic tone for such samples might be minimal. Our results indicate, however, that resting sympathetic activation is associated with stress exposure and important for understanding how autonomic reactivity relates to cognitive performance. As observed here, it may be true that, for individuals whose autonomic space is marked by high SNS activity, engagement of the PNS may be less optimal for modifying behavior than it is for individuals with lower SNS activity. Future research seeking to understand links between ANS regulation and cognitive performance should measure both SNS and PNS branches to investigate possible interactions linked to cognitive performance, similar to those reported here.

Despite chronic stress differences between the short and long resting PEP groups, we did not observe between-group differences in WM capacity. Thus, although individuals with longer PEP displayed higher WM performance concurrent with higher HF-HRV reactivity, their WM performance as a group was not significantly different from that of higher-risk individuals with shorter PEP, for whom WM performance was not associated with HF-HRV reactivity. Given that our sample as a whole was at relatively higher risk, this result might reflect the biological costs of high-risk individuals achieving similar levels of performance

as their lower-risk counterparts. Brody and colleagues (2013) have reported that high-risk African American youths who show low levels of maladaptive behaviors display greater levels of allostatic load later in life. Within this framework, higher-risk moms might achieve cognitive performance similar to that of their lower-risk peers via a more costly pathway of heightened resting SNS activation, which might jeopardize long-term cardiovascular health, immune function, and other health-related outcomes. As suggested by other researchers, such behavioral resilience from chronic life stress might be only skin deep and associated with suboptimal health-related outcomes (Brody et al., 2013). More research is needed to elucidate the extent and nature of these possible hidden costs of maintaining adequate levels of cognitive functioning.

Although the sample reported on here is overrepresentative of mothers at high risk for stress exposure, it is similar to populations of individuals typically recruited for studies of parenting at risk (Neville et al., 2013; Skowron, Cipriano-Essel, Benjamin, Pincus, & Van Ryzin, 2013). In such populations, the regulation of autonomic physiology is a promising biomarker underlying positive parenting behaviors and cognitive control (Crouch et al., 2015; Skowron et al., 2013). Clarifying the role of PNS and SNS function in predicting WM performance in at-risk mothers may have important implications for understanding how PNS and SNS function underlie effective parenting behaviors that are targeted by a variety of interventions (Shonkoff & Fisher, 2013). Our results suggest that, within a generally risky sample, heightened SNS arousal is more likely to be observed in mothers with higher chronic stress exposure, which should be taken into consideration as a potential moderator of the proposed PNS-mediated regulation of behavior (Thayer et al., 2009). Emotion regulation techniques that typically increase PNS activity (e.g., paced breathing; Fisher, Ellis, & Chamberlain, 1999) may have differential benefits for parents, depending upon chronic SNS arousal levels. It may be useful for interventions to assess SNS function in addition to PNS function, to determine if heightened resting SNS activation can be targeted by interventions or if such heightened activation in high-risk parents may limit their ability to benefit from a regulation-focused intervention.

A major limitation of the present study is the relatively small sample size and limited statistical power. This may have hindered the ability to detect significant associations that might emerge in a larger data set. A close inspection of Table 3 reveals the possibility that baseline HF-HRV might interact with PEP reactivity in a similar manner as the significant interaction reported between baseline PEP and HF-HRV reactivity, yet the larger degree of variability in the interaction between baseline HF-HRV and PEP reactivity may preclude the detection of statistical significance without a large sample size. Thus, the results reported here should not be taken as strong evidence against the role of baseline HF-HRV in cognitive function. Additionally, Table 2 and Table 4 highlight a number of nonsignificant correlations that show trends in the same direction as other significant correlations ($p < .10$) or the same direction as expected by previous research. Given the large number of correlations examined in the present analyses and relatively small effect sizes reported, these nonsignificant associations should be noted with caution. We hope, however, that future research with larger sample sizes will benefit from the identification of such nonsignificant trends when developing and testing hypotheses.

In sum, these results provide a nuanced support to models of neurovisceral integration (Thayer & Lane, 2000, 2009) within the autonomic space framework (Berntson et al., 1993). As predicted by neurovisceral models, engagement of the parasympathetic system measured by high-frequency heart-rate variability was associated with enhanced WM performance in mothers; however, this was observed only in individuals with lower resting SNS activity as measured by PEP during a neutral baseline period. In a higher-risk group of mothers with greater resting sympathetic activity, parasympathetic function was not associated with WM performance. Given that stressful life experiences have been associated with greater sympathetic and reduced parasympathetic influence (Propper & Holochwest, 2013), these results suggest that studies of autonomic-behavioral associations would benefit from including participants from a wider range of life experiences, as results drawn from lower-risk university samples might be overrepresentative of traitlike autonomic activity that is higher in parasympathetic and lower in sympathetic contributions.

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FIGURE 1.
WM capacity averaged across all participants for each block of the WM task

FIGURE 2.

Scatter plots of the relationship between HF-HRV reactivity and WM performance for longer and shorter resting PEP groups

FIGURE 3.

Model of the relationship between HF-HRV reactivity, resting PEP, and WM performance

TABLE 1

Descriptive statistics

	<i>M</i>	<i>SD</i>	Range
Participant age	30.42	6.54	29.9–48.1
Baseline HF-HRV	6.31	0.98	3.71–8.70
Task HF-HRV	6.47	0.98	4.42–9.31
HF-HRV reactivity	0.16	0.56	–1.45–1.69
Baseline PEP	116.25	10.81	87.50–139.20
Task PEP	116.95	11.17	87.32–139.00
PEP reactivity	0.60	2.94	–5.58–8.52
WM capacity (all set sizes)	2.73	0.38	1.82–3.42
Cumulative risk	2.95	1.81	0.00–7.00

TABLE 2

Zero-order correlations among all variables of interest

	1	2	3	4	5	6	7	8	9
1. Maternal age	–								
2. Baseline HF-HRV	–.43 ^{**}	–							
3. Task HF-HRV	–.43 ^{**}	.84 ^{**}	–						
4. HF-HRV reactivity	.002	–.29 [^]	.28 [^]	–					
5. Baseline PEP	.35 [*]	–.23	–.25	–.03	–				
6. Task PEP	.31 [*]	–.26 [^]	–.24	.04	.96 ^{**}	–			
7. PEP reactivity	–.08	–.18	–.07	.20	.01	.28 [^]	–		
8. WM capacity	–.01	.09	–.01	.14	–.22	–.21	–.01	–	
9. Cumulative risk	.24	–.30 ^{***}	.11	.33 [*]	–.16	–.10	.17	.17	–

Note. Dashes indicate no data available.

^{*} $p < .05$.
^{**} $p < .01$.
[^] $p < .10$.

TABLE 3

Multilevel models predicting WM capacity fluctuations across the task

	Coefficient	SE
Intercept, π_0		
Intercept, β_{00}	2.77 ***	0.057 ***
Age, β_{01}	-0.001	0.010
Baseline HF-HRV, β_{02}	-0.101	0.072
Baseline PEP, β_{03}	0.009	0.006
HF-HRV reactivity, π_1		
Intercept, β_{10}	0.027	0.072
Age $\times$ HRV Reactivity, β_{11}	0.020	0.013
Baseline HRV $\times$ HRV Reactivity, β_{12}	0.082	0.062
Baseline PEP $\times$ HRV Reactivity, β_{13}	0.020 *	0.008 *
PEP Reactivity, π_2		
Intercept, β_{20}	0.008	0.013
Age $\times$ PEP Reactivity, β_{21}	0.004	0.002
Baseline HRV $\times$ PEP Reactivity, β_{22}	0.017	0.013
Baseline PEP $\times$ PEP Reactivity, β_{23}	0.001	0.001

*
 $p < .05$.**
 $p < .01$.***
 $p < .001$.

TABLE 4
Correlations between fluctuations in WM capacity and HF-HRV reactivity for longer and shorter resting PEP groups

	HF-HRV reactivity					
	Block 1	Block 2	Block 3	Block 4	Block 5	Block 6
Longer resting PEP ($n = 21$)						
WM Block 1	.170	.180	.092	.060	.147	.188
WM Block 2	.455*	.427 [^]	.385 [^]	.525*	.453*	.092
WM Block 3	.448*	.575**	.243	.279	.217	.269
WM Block 4	.488*	.324	.321	.259	.197	.003
WM Block 5	.510*	.371 [^]	.434*	.304	.378	.535*
WM Block 6	.202	.152	.330	.293	.298	.249
Shorter resting PEP ($n = 21$)						
WM Block 1	.391***	.210	.334	.302	.236	.310
WM Block 2	.095	.076	-.016	-.036	-.130	-.009
WM Block 3	-.329	-.099	-.065	-.157	-.263	-.148
WM Block 4	.188	.094	.005	-.005	-.011	.175
WM Block 5	-.142	-.159	-.044	-.151	-.217	-.165
WM Block 6	-.288	-.315	-.372	-.542*	-.556*	-.405 [^]

* $p < .05$.** $p < .01$.*** $p < .001$.[^] $p < .10$.