INTEGRATING AFFECTIVE AND COGNITIVE CORRELATES OF HEART RATE VARIABILITY: A STRUCTURAL EQUATION MODELING APPROACH

by

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ABSTRACT OF THE THESIS

Integrating Affective and Cognitive Correlates of Heart Rate Variability:

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High frequency heart rate variability (HRV) is a measure of neurocardiac communication that is thought to reflect predominantly parasympathetic cardiac regulation. Low HRV has been associated empirically with clinical and subclinical levels of anxiety and depression and, more recently, high levels of HRV have been associated with measures of executive functioning. These findings have informed theories proposing that HRV may provide an autonomic index of a broad, self-regulatory capacity underlying aspects of emotion regulation and cognitive control. This study sought to operationalize and test this proposition using a structural equation modeling approach by examining the relationships of HRV to negative affect (NA) and executive functioning (EF) in a large sample of U.S. adults spanning six decades of age (30s–80s). HRV was modeled as a predictor of an NA factor (self-reported trait anxiety and depression symptoms) and an EF factor (performance on three neuropsychological tests tapping facets of executive abilities). Alternative models also were tested to determine the utility of HRV for predicting EF, with and without statistical control of demographic and health-related covariates. In the initial structural, model HRV

showed a significant relationship to EF and a nonsignificant relationship to NA. When covariates were included in the model, HRV's associations with both constructs were nonsignificant. Age emerged as the only significant predictor of NA and EF in the final model, showing inverse relationships to both. Findings may reflect population and methodological differences between the present thesis and prior research, but they also suggest potential refinements to the interpretations of earlier findings and theoretical claims regarding HRV.

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I am extremely grateful to my advisor and thesis committee chair, Richard Contrada, whose help conceptualizing this study, finding an invaluable source of data in the Midlife Development in the U.S. survey (MIDUS), and working with me through the inevitable transformations and iterations made the project possible. I also owe enormous thanks to the members of my committee, Edward Selby and Marsha Bates, whose collegial guidance and generous feedback regarding the study of vagal control in clinical psychology and the use of structural equation modeling have allowed me to stretch and grow as a researcher. Finally, I would like to express my appreciation for the vast efforts of the many interdisciplinary researchers and staff at the University of Wisconsin—Madison's Institute on Aging and the University of Michigan—Ann Arbor's Institute for Social Research who work to carry out the MIDUS study and make its data publically available.

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Introduction

A variety of cognitive and affective measures have been linked to psychophysiological markers that reflect the biological bases, concomitants, or consequences of health-related psychological processes. Many of these markers are thought to reflect mechanisms underlying associations between psychosocial factors, including mental health problems, and physical disease (Contrada, 2011). In the study of cardiovascular health, one such psychophysiological marker is heart rate variability (HRV), which is measured using electrocardiographic data to quantify small variations over time in the intervals between successive heart beats. There is evidence to suggest that these beat-to-beat variations reflect parasympathetic cardiac regulation, among other influences, providing a measure of the deceleratory influence of the vagus nerve on heart rate (Levy, 1990). Consequently, HRV often is interpreted as a measure of vagal functioning or vagal tone (Berntson et al., 1997). Parasympathetic regulation is thought to exert tonic inhibitory effects on sympathetic activity, which is associated with stress and negative emotion and has been shown to mediate psychosocial influences on cardiovascular disease (Brooks, McCabe, & Schneiderman, 2011; Glick, Braunwald, & Lewis, 1965). As such, low HRV has been studied as possible marker of risk for cardiovascular disease (CVD), adverse cardiac events, and all cause mortality (Dekker et al., 1997, 2000; Tsuji et al., 1994, 1996).

Closely related to its significance for cardiovascular health, HRV also has been studied as a measure reflecting the bidirectional communication between the brain and the cardiovascular system. In particular, it is a possible reflection of brain mechanisms underlying various aspects of psychological self-regulation (Bates & Buckman, 2013; Benarroch, 1997; Berntson, Cacioppo & Grossman, 2007). Self-regulatory processes are defined broadly as individuals' means of purposefully controlling their predisposed affective,

cognitive, or behavioral responses to a situation, including resisting or changing those responses and initiating goal-directed efforts (Bandura, 1989; Heatherton, 2011; Shah, 2005).

In this context, HRV has been linked to both affective and cognitive facets of self-regulation. The relationship of low HRV to poor emotional regulation is noteworthy because, like HRV, negative emotional states and conditions have been implicated as possible risk factors for CVD and other physical health problems (Rozanzki, Blumenthal, & Kaplan, 1999; Suls & Bunde, 2005; Gianaros & Sheu, 2009). In addition, since the 1990s, a related literature has documented HRV's inverse relationships to symptoms of anxiety and depression in physically healthy individuals (Friedman, 2007; Rottenberg, 2007). Largely in parallel to this work, a smaller but growing literature on HRV's cognitive correlates has shown an association between HRV and some neuropsychological measures of executive functioning (Thayer et al., 2009). Executive functioning is a cognitive construct within the domain of self-regulation (Heatherton, 2011; Hoffman, Schmeichel, & Baddely, 2012) defined as cognitive control processes that support goal maintenance and the flexible implementation of task rules (e.g., Miller & Cohen, 2001; Miyake et al., 2000).

The status of HRV as a marker for neurocardiac communication, along with the broad range of psychological measures to which it has been linked, have prompted theoretical interest in integrating knowledge about autonomic cardiac regulation with findings delineating HRV's affective and cognitive correlates. These theories tend to hold in common the notion that HRV may reflect a broad, self-regulatory capacity that underlies aspects of emotion regulation and cognitive control, allowing individuals to respond in a flexible, adaptive way to a complex, changing environment (Appelhans & Leuken, 2006; Beauchaine, 2001; Porges, 2011, Thayer & Lane, 2009). One purpose of the present thesis was to test the proposition that HRV might serve as a predictor of individual differences in

these emotional and cognitive self-regulatory abilities by modeling its relationships to negative affect and executive functioning in a large sample of U.S. adults.

HRV and Negative Affect

Research on physically healthy individuals has documented associations between low HRV and elevated symptoms of anxiety and depression (Friedman, 2007; Rottenberg, 2007). Research into HRV's relationship to anxiety generally has focused on clinical and mixed samples. Among the anxiety disorders, the strongest relationships to low HRV have been found in panic disorder (PD) and post-traumatic stress disorder (PTSD), while studies of generalized anxiety disorder and phobias have been fewer and shown less consistent results (Friedman, 2007). Low HRV also has been associated with biased attention toward threat (Miskovic & Schmidt, 2010) and a delay in disengaging attention from threat (Cocia, Uscătescu, & Rusu, 2012), which are found in individuals with anxiety disorders (Clark, 1999).

As interest has grown in understanding the generalizability of these findings to nonclinical populations, a number of studies of nonclinical and mixed samples have examined HRV's relationship to emotion-modulated startle. Emotion-modulated startle is an implicit measure in the sense that it does not involve self-report. Instead, emotional states are inferred from changes in the amplitude of the eye blink response to a sudden stimulus such as an unexpected burst of loud white noise. This manifestation of the startle reflex is altered by pre-exposure to emotional stimuli (Grillon & Baas, 2003). This work has linked low resting HRV to exaggerated startle response under threat of shock (Melzig et al., 2009) as well as to increased startle magnitude following neutral stimuli (Ruiz-Padial, 2003). Based on these findings, the authors of these studies suggest that low HRV generally may be associated with elevated levels of anticipatory anxiety, which impedes adaptive emotional

regulation. This interpretation suggests that the emotion-modulated startle findings may point to a broader association between low HRV and higher trait anxiety (TA), a construct typically measured explicitly by self report and thought to be relatively stable across potentially threatening situations (Spielberger, 1983, 1989). Thus far, a few studies have found an inverse relationship between TA and low HRV in medical patient groups (e.g., Kogan, Allena, & Weihs, 2012), and the small number of studies to examine this association in physically healthy individuals have reported inconsistent findings (Bleil et al., 2008; Dishman et al., 2000; Fuller, 1992; Virtanen et al., 2003; Watkins et al., 1998).

Low HRV also has been associated with depressive symptoms, though much of this research has been conducted in patients with CVD, raising the possibility that CVD might confound or moderate this HRV–depression association (Kemp et al., 2010). Studies of HRV and depression in physically healthy participants are relatively recent and, compared to the HRV–anxiety literature, their findings have been more mixed and the associations more modest (Kemp et al., 2010; Rottenberg, 2007). Some major causes of inconsistencies in the HRV–depression literature include potentially confounding effects of (1) some antidepressant medications' effects on HRV, (2) unmeasured cardiovascular factors linked to both HRV and depression, and (3) potential effects on HRV due to unmeasured comorbid anxiety—each of which may cause or contribute to lower HRV in this population (Rottenberg, 2007).

One recent study of the relationship of HRV to depression with and without comorbid anxiety sought to address these sources of variation by comparing physically healthy controls to physically healthy, unmedicated patients diagnosed with either depression alone or depression with comorbid anxiety (Kemp et al., 2012). In this case, the comparison of controls to patients with depression alone showed a moderate effect, while the

comparison of controls to the group with comorbid depression and anxiety yielded a large effect. Overall, results showed lower HRV in both patient groups, supporting the independent relationship of low HRV to depression and supporting a possible contributory role for comorbid anxiety. To our knowledge, only one study has examined HRV's relationship to symptoms of both anxiety and depression in a large community sample. Bleil et al. (2008) reported that in young and middle-aged adults (N=653) symptoms of depression and anxiety, but not anger, each independently predicted HRV and contributed to the higher-order latent variable of negative affect, which also predicted HRV.

Overall, research on the relationship of HRV to anxiety and depression provides some support for independent associations, while generally presenting a clearer relationship to anxiety than depression. This appears due, in part, to the medical and medication-related confounds in much of the depression-focused work, and the paucity of well-controlled studies examining comorbid anxiety and depression. At the same time, this research includes even fewer studies seeking to clarify the generalizability of the clinical and laboratory-based findings to large nonclinical samples. One way in which the present study aimed to address this question of generalizability was by testing the relationships of HRV to both anxiety symptoms and depressive symptoms, conceptualized as indicators of the latent variable of negative affect, in a large, nonclinical sample with a broad age range.

HRV and Executive Functioning

Executive functions (EF) are defined as cognitive control mechanisms for maintaining task goals and flexibly implementing task rules (e.g., Miller & Cohen, 2001; Miyake et al., 2000). One prominent theoretical framework for characterizing individual differences in EF describes a three-factor model comprising (1) monitoring and updating information in working memory, (2) task shifting, and (3) inhibition of prepotent responses

(Miyake et al., 2000). Factor analyses have shown these three component executive processes to be intercorrelated yet functionally separable (Miyake et al., 2000). Given the broad cognitive reach of these processes, the proper means of delimiting the executive functioning construct has been a topic of debate, as has its status as a coherent construct in cognitive research (e.g., Engle, 2002; Parkin, 1998). Nonetheless, there is a working consensus in the clinical and neuropsychological literatures that the abilities typically described as EF can show distinctive patterns of dysfunction and are critical for psychological and behavioral self-regulation in response to changing environmental demands (Chan et al., 2008; Heatherton, 2011; Jurado & Rosselli, 2007).

Several quasi-experimental studies have yielded associations between resting HRV and performance on two tasks with executive demands on working memory (Thayer et al., 2009). One such study, carried out with young, male members of the Royal Norwegian Navy, compared cognitive performance in high- and low-HRV participants grouped using a median split in resting HRV (Hansen, Johnsen, & Thayer, 2003). Compared to the low-HRV group, high-HRV participants showed superior accuracy on a two-back working memory measure. In this computerized task, participants are presented with a series of stimuli (e.g., letters) one at a time and asked to indicate when the current stimulus matches the one shown two steps earlier in the series. They also showed faster responding, with a trend toward better accuracy, on the components of a continuous performance test (CPT) interpreted as tapping executive functioning. Other studies of the HRV–EF relationship have used the same tasks and similar Norwegian Navy samples. They have reported improved executive performance under stress among low-HRV but not high-HRV participants, which was attributed to low-HRV individuals' higher anxiety; and coincident increases in HRV and improvements in executive task components after physical fitness training (Hansen et al.,

2004; Hansen, Johnson, & Thayer, 2009). A similarly designed study of the effects of physical fitness training on older, sedentary adults likewise found increased HRV and improved performance on the Wisconsin Card Sorting Test only in the group assigned to an exercise regimen (Albinet et al., 2010).

Overall, these studies provide some initial support for a correlation between higher resting HRV and better performance on several tasks tapping facets of EF. However, this work also resembles the HRV-negative affect research, in that it raises similar types of questions about the generalizability of findings. First, the extent to which these findings reflect associations in the general population is unclear, given their focus on small (N = 24-65), predominantly male samples drawn from the extremes of the nonclinical population in terms of physical fitness. Second, these studies also present a relatively narrow conceptualization of executive functioning, based either on a single task (WCST; Albinet et al., 2010) or on subdividing executive from nonexecutive components of the same two tasks (two-back and CPT; Hansen, Johnson, & Thayer, 2003; Hansen et al., 2004; Hansen, Johnson, & Thayer, 2009). Third, among the studies that measured HRV as a predictor of individual EF differences rather than change in HRV as an intervention outcome, HRV was dichotomized rather than treated as a continuous variable. As a result, it not clear whether the findings describe a continuous EF-HRV relationship or one characteristic of their extreme values.

The present thesis aimed to address several of the questions raised by these studies about HRV as a potential predictor of EF in the nonclinical adult population. This study aimed to shed new light on the strength and nature of the HRV-EF relationship by (1) using a larger, more diverse sample than much of the prior research in this area; (2) treating HRV

as a continuous variable, rather than dichotomizing it; and (3) conceptualizing EF as a latent variable measured by a set of tasks selected to measure its major theorized components.

Aims and Hypotheses

This study examined the relationships of both negative affect and executive functioning to resting HRV in a large, nonclinical adult sample spanning six decades of age (30s-80s). As noted, the relationships of anxiety and depression to HRV have rarely been examined together despite their high comorbidity (Gorman, 1998; Kessler et al., 1996), and only one study has examined both relationships to HRV in a large, nonclinical adult sample (Bleil et al., 2008), albeit one with a narrower age range (30-54 years). In part, the proposed analysis sought to provide a conceptual replication of that study by testing a model in which symptoms of anxiety and symptoms of depression were hypothesized to underlie a common, HRV-related negative affect factor.

In addition, this study aimed to extend previous findings by including in the model the relationship of HRV to an executive functioning factor. This aspect of the study was intended to serve two major purposes. First, as discussed above, it addressed several questions about the generalizability of previously reported EF-HRV associations by using a large, diverse sample, a more comprehensive conceptualization of EF, and a continuous measurement of HRV. Second, by incorporating the major proposed affective and cognitive correlates of HRV in a single model, it represents the first effort to operationalize and test current theories proposing HRV as a potential "index" reflecting individual differences in affective and cognitive self-regulatory processes (Porges, 2011, Thayer & Lane, 2009). Based on prior theoretical and empirical work, the hypotheses were: (1) HRV was expected to show an inverse relationship to NA and a direct relationship to EF; (2) NA and EF were

expected to show an inverse association; (3) age was predicted to relate inversely to EF; no prediction was made about its association with NA.

A structural equation modeling (SEM) approach was adopted for these analyses because this method is well-suited to examining relationships between measured and latent variables in large samples. It allows for the construction of latent factors (NA and EF) based on shared variance among observable indicators, in addition to path analyses to test the extent to which the variability in a given measure can explain the variability in other constructs in the model. Several limitations of SEM are important in the context of the present study: It cannot determine causality; it poses challenges for examining nonlinear relationships, which may characterize autonomic functioning; and it shares with other methods the potential to yield misleading conclusions if influential variables are omitted (Berntson et al., 1994; Tomarkin & Waller, 2005). Nonetheless, its ability to provide a global test of model fit for a set of linear relationships makes it appropriate for testing theory-driven predictions about such relationships between a physiological variable, HRV, and the psychological constructs of NA and EF.

Methods

Participants

Data were drawn from the second wave of the Midlife in the United States (MIDUS) study (MIDUS II; 2002-2006), which collected biomedical, psychosocial, cognitive, and psychophysiological data from a large, diverse sample of U.S. adults (N = 4,975) aged 33 to 84 years. MIDUS II included 9-year follow-ups of all four of the subsamples that comprised its first wave, MIDUS I: (1) a national random digit dialing (RDD) sample, (2) oversamples from 5 U.S. cities, (3) siblings of participants from the RDD sample, and (4) a national RDD sample of twin pairs. In addition, MIDUS II added an African-American subsample from

Milwaukee, Wisconsin. To be eligible to participate, individuals had to be non-institutionalized English-speakers living in the continental United States and aged 25 to 74 when they took part in MIDUS I.

Data for the current analyses were drawn from two study components initiated in MIDUS II: the Biomarker Project (n = 1,255), which collected data on psychophysiological, biomedical, and psychosocial parameters; and the Cognitive Project (n = 4,512), which collected data on cognitive functioning. The eligible sample for the currents study before exclusion criteria were applied included the subset of MIDUS II participants who took part in both of these new studies and had valid resting HRV data (n = 1,056).

Several exclusion criteria were applied to limit major confounding influences on HRV, executive functioning, and negative affect. First, eligible participants who did not deny a history of stroke were excluded (i.e., those who affirmed having had a stroke [n=20] or for whom data was missing [n = 59]). Stroke has been associated with low HRV, significant decrements in neurocognitive functioning, and a increased risk of depression (Dütsch et al., 2007; Robinson, 2006). Individuals taking antidepressant medications (ADMs; n = 157) and those taking antihypertensive medications (n = 367) also were excluded, as these medications are widely prescribed and have been shown to affect cardiac autonomic functioning (Lampert et al., 2003; Licht et al., 2008, 2009; Rottenberg, 2007; Toivonen, 1993). Table 1 presents the demographic and clinical characteristics of the present sample (N=533).

Procedure

Data for the MIDUS II Biomarker Project were collected from July 2004 to June 2006. All MIDUS II participants who completed the MIDUS I follow-up phone interview and self-administered questionnaire (Project 1) were eligible to take part, except those drawn from the city oversamples. Biomarker Project participants traveled to one of 3 regional

research centers (Georgetown University, UCLA, or University of Wisconsin—Madison) for an overnight stay (Ryff, Seeman, & Weinstein, 2010). The protocol at all sites included collection of medical history and completion of psychosocial self-report questionnaires (day 1), laboratory-based collection of psychophsyiological data (day 2), and physical exams (both days). At all sites, the psychophysiological recording session was conducted in the morning of the second day, following a light breakfast with no caffeinated beverages (Ryff, Seeman, & Weinstein, 2010).

HRV data were collected with electrocardiograph (ECG) electrodes placed on each shoulder and in the left lower quadrant. Because HRV is sensitive to respiration rate, respiration bands on the chest and abdomen were used to measure respiration. The participant was seated at a computer that was used later in the session to present the cognitive stress tasks. Participants received instructions for two stress tasks (mental arithmetic and a the Stroop color–word task) and practiced keyboard-based responding; these tasks were administered later in the session, after the baseline resting HRV recording. After this brief practice interval, recording instruments were calibrated (up to 10 min), signal quality was checked (up to 10 min), and instruments were recalibrated as needed (up to 4.67 min; Kimhy et al., 2013). Following the instruction to breathe normally, the two 5-min baseline ECG and respiration recordings were obtained.

As described elsewhere (Shcheslavskaya et al., 2010), a National Instruments A/D board was used to digitize the analog ECG signals at 500 Hz and to pass them to a microcomputer. Proprietary event detection software was used to submit the ECG waveform to an R-wave detection routine, which yielded an RR interval series. Errors in R-wave marking were corrected following established procedures (see Shcheslavskaya et al., 2010). The spectra of the RR interval series were calculated using an interval method for

computing Fourier transforms, in which the mean of the RR interval series was first subtracted from each series value (see DeBoer, Karemaker, & Strackee, 1984). Next, the series was then filtered using a Hanning window (Harris, 1978) and the variance (in $msec^2$), over the LF (0.04-0.15 Hz) and HF (0.15-0.50 Hz) bands was summed. Estimates of spectral power were adjusted to offset any attenuation caused by this filter (Harris, 1978).

All MIDUS II participants were eligible to take part in the Cognitive Project. Those who participated were administered the Brief Test of Adult Cognition by Telephone (BTACT) to collect data on six domains of cognitive functioning: episodic verbal memory, inductive reasoning, speed of processing, working memory span, verbal fluency, and task-switching (Lachman & Tun, 2008; Tun & Lachman, 2006). A detailed description of BTACT administration is available elsewhere (Tun & Lachman, 2006).

Measures

Resting Heart Rate Variability. High-frequency R-R interval variability (HF-HRV; bandwidth 0.15-0.40 Hz, $msec^2$) was used to measure resting HRV. Time- and frequency-domain measures of HRV are closely correlated (Berntson, Lozano, & Chen, 2005) and both are available in the MIDUS II data. The frequency domain measure was selected for these analyses because time-domain measures include some low-frequency contributions to R-R interval, which may conflate sympathetic and vagal influences (Berntson, Lozano, & Chen, 2005). The MIDUS II Biomarker Project collected baseline data on resting HRV in 2 epochs of 300 seconds each, prior to a series of stress responsivity assessments. Only the first recording epoch was used in these analyses, as all participants who lacked valid epoch 1 data also lacked valid data for epoch 2. Resting HRV has shown good test-reliability over intervals of three weeks (.81–.99; Bertsch et al., 2012) to several months (.76–.80; Sinnreich et al., 1998) in healthy adults.

Spielberger State Trait Anxiety Inventory—Trait version (STAI-T). This widely used, 20-item inventory uses a 4-point Likert scale to measure individual differences in the likelihood of experiencing anxiety symptoms in response to potentially stressful situations (Spielberger, 1983, 1989). The trait scale has shown high test-retest reliability and good convergent validity with other self-report anxiety measures (Spielberger, 1983). Its reliability in the current sample was adequate (Cronbach's alpha = .75).

Center for Epidemiologic Studies Depression Scale (CESD). The CESD is 20-item measure of depressive symptoms designed for large-scale surveys (Radloff, 1977; Roberts & Vernon, 1983). It has shown a stable factor structure across large clinical and nonclinical samples, high internal and adequate test-retest reliability, and good convergent and discriminant validity (Contrada et al., 2006; Radloff, 1977). Given the broad age span of MIDUS II participants and concerns about the accuracy of some common depression measures with elderly populations (Christensen et al., 1999), it is notable that the CESD has shown good psychometric properties in older populations (Herzog et al., 1990). It showed good reliability in the present sample (Cronbach's alpha =.88).

Digits Backward. The Digits Backward task, which measures the longest series of digits an individual can mentally resequence in reverse order, is a common measure of sustained attention and working memory (Tun & Lachman, 2006). This task demands sustained attention and active manipulation of information (Kaneko et al., 2011), making it a viable indicator of the executive capacity for continuous monitoring and updating information in working memory (Engle, 2002). Reliability data are unavailable for the present sample, to whom the cognitive battery was administered only once. Among a large, demographically representative sample of U.S. adults, it has shown good test-retest reliability (r = .83; Weschler, 1981).

Red/Green. The BTACT Red/Green task is a variant of the classic Go/No Go measure; this version of the task requires verbal responses only and can therefore be administered by telephone. Like the classic task, it is thought to draw on the executive processes of task switching and inhibition of prepotent responses (Kramer, Hahn, & Gopher, 1999; Tun & Lachman, 2006). In the BTACT protocol, this measure included a "normal" block of trials (participants said "stop" when the examiner said *red* and "go" when the examiner said *green*) a "reverse" block ("stop"—*green*; "go"—*red*), and a mixed block. In the mixed block, participants were cued with the words *normal* and *reverse* to switch between response types at unpredictable intervals. Earlier analyses of age and gender effects on these data indicated high accuracy (> 94%) across age groups and task conditions and reported that speed was not compromised for accuracy, even when controlling for age (Tun & Lachman, 2008). The present analyses used the average response latency of the switch and non-switch trials during the mixed block. For ease of interpretation in the context of the other cognitive measures, for which better performance corresponded to higher values, the latency values were subtracted from zero prior to analyses. Test-retest reliability over a 6-month interval was examined in a representative subset of Cognitive Project participants a and found to be adequate ($r = .77$) for the mixed-task condition (Tun & Lachman, 2008).

Category Fluency. The Category Fluency task measures the number of unique items from a semantic category (e.g., animals) an individual can generate in 1 minute. This task is conventionally interpreted as a measure of the executive processes of active self-monitoring and inhibition (Lezak, Howieson, & Loring, 2004; Tun & Lachman, 2006). This measure has shown adequate test-retest reliability ($r = .70$) in middle-aged and elderly samples (Harrison, Buxton, & Husain, 2000; Snow et al., 1988).

HRV Covariates

Demographic Factors. The demographic factors of age and sex, which have been associated with differences in resting HRV in previous research (e.g., Kou et al., 1999; Stein, Kleiger, & Rottman, 1997), were included in the analyses in order to account for their potential influence on HRV and for potential age- and sex-related differences in negative affect and executive functioning. Differences in resting HRV across racial groups also have been reported (e.g., Choi, 2006; Liao et al., 1995), but the relative lack of racial diversity in the sample (see Table 1) precluded an examination of race as a major factor in this study.

Cardiovascular Health. Given that low HRV has been associated with poor cardiovascular heath (Dekker et al., 1997, 2000; Tsuji et al., 1994, 1996), the relationships of several major CVD risk factors to HRV were also examined. These included hypertension, hypercholesterolemia, diabetes, smoking, low levels of physical activity, and BMI (NHLBI, 2012), each of which previously has been associated with low HRV (Thayer & Lane, 2007).

Data Analytic Strategy

The general mode of analysis involved structural equation modeling (SEM), which incorporates both measured and latent variables and can model interrelationships between multiple variables simultaneously, allowing a test of the hypothesized models' respective fits to the MIDUS II data. The maximum likelihood (ML) method was used to analyze covariance matrices. This method uses all available data to compute parameters, standard errors, and test statistics that are unbiased when data is missing at random or completely at random and data are multivariate normal (Brown, 2006); it also has performed well relative to other available methods when data is nonnormal (Savelei & Bentler, 2005).

Model fit was evaluated using the normed chi square (χ^2/df), comparative fit index (CFI), root mean square error of approximation (RMSEA), and Non-normed Fit Index (NFI; Kline, 2005). According to standard criteria, values of (χ^2/df) < 5 were considered a

good fit (Bollen, 1989). Model fit was considered good at RMSEA values ≤ 0.05, reasonable at values 0.5 to 0.8, or poor at values > 0.10 (Browne & Cudeck, 1993). CFI and NFI values ≥ 0.95 each were considered a good fit (Hu & Bentler, 1999). The respective fits of nested models were compared using the chi-square difference test, in which a significant difference indicates that the additional estimated parameters in the more complex model provide a sufficiently improved fit to the data to justify less parsimony, compared to the simpler model (Hoyle, 2012). In addition, the models' Akaike information Criterion (AIC) and Browne-Cudeck Criterion (BCC) values were compared, with a decrease on these measures of at least 10 units indicating a significantly better fitting model (Burnham & Anderson, 2004).

Results

Preliminary Analyses

Preliminary analyses were performed in SPSS Statistics 20 (IBM Corporation, 2012) to check for skew, kurtosis, and univariate outliers with z-scores greater than ±4.0 (Kline, 2005). Because HRV and the responses on the CESD and STAI were not normally distributed, these variables were subjected to a logarithmic transformation (after adding 1 to each observed CESD score to eliminate values of zero) that improved normality. Two variables included univariate outliers with z-scores greater than ±4.0. For BMI, 2 such outliers (BMI = 57.40, z = 5.12; BMI = 57.28, z = 5.00) were "brought to the fence" and set to the highest non-outlier value (BMI = 50.15, z = 3.77). For Red/Green (RG) task response latency, 3 outliers (RG = -2.30 s; z = -6.06; RG = -2.01 s, z = -4.69; RG = -1.91 s; z = -4.20) were likewise "brought to the fence" and set to the lowest non-outlier value of -1.79 s (z = -3.65). The means, standard deviations, and skewness and kurtosis statistics for all variables are shown in Table 2, and simple bivariate correlations between variables are

shown in Table 3. Multicollinearity was evaluated using tolerance values (O'Brien, 2007) and found to be inconsequential.

Measurement Model

Modeling analyses were conducted with AMOS 21 software (Arbuckle, 2012). Following recommended practices (e.g., Kline, 2005), the measurement model was examined first, followed by the structural model. The measurement model included two correlated latent variables, NA and EF functioning. NA was modeled with two indicators: the Center for Epidemiological Studies Depression Scale (CESD) and the Spielberger Trait Anxiety Inventory (STAI). EF was modeled with three indicators: category fluency, backward digit span, and the Red/Green task. Results are shown in Figure 1. Based on accepted standards (e.g., Kline, 2005), the model showed good fit to the data ($\chi^2 = 2.723$, $df = 4$, $p = .605$, NFI = .994, CFI = 1.00, RMSEA < .001). All indicators had significant factor loadings ($p < .05$). NA and EF showed a relationship in the expected direction but that was vanishingly small in magnitude (-.01) and did not reach significance ($p = .092$).

In light of theories proposing that HRV may reflect individual differences in a broad self-regulatory capacity for adaptive responding that underlies both cognitive and affective self-regulation (Porges, 2011, Thayer & Lane, 2009), an additional measurement model also was evaluated. In this model, a higher-order construct potentially reflecting a broad dimension of flexible responding was added as a predictor of the negative affect and executive functioning constructs. This additional higher-order factor failed to improve model fit. Consequently, structural analyses proceeded based on the model that included the first-order latent variables (NA and EF) without the higher-order flexible responding construct.

Structural Model

The full model showed good fit to the data (χ^2 = 5.981, *df* = 7, *p* = .542, NFI = .998, CFI = 1.00, RMSEA < .001; Figure 2). The path from HRV to executive functioning was significant (β = .16, *B* = .014, *SE* = .006, *p* < .05). However, the path from HRV to negative affect was not significant (β = -.03, *B* = -.015, *SE* = .023, *p* >.05). The path from negative affect to executive functioning showed a trend toward significance (β = -.13, *B* = -.022, *SE* = .011, *p* = .05).

Alternative Model

An alternative model was also evaluated in which the subset of hypothesized covariates that showed significant bivariate correlations with HRV was incorporated . This analysis estimated these covariates' respective relationships to HRV, as well as their utility as predictors of negative affect and executive functioning. As shown in Table 4, among the hypothesized demographic- and CVD-related covariates, HRV showed significant bivariate correlations with age (*r* = - .31, *p* < .001), BMI (*r* = -.12 *p* < .01), and the presence of hyperglycemia or diabetes in the past 12 months (*r* = -.10, *p* < .05),. In the alternative model, these three covariates were hypothesized to be correlated with one another and with HRV (NHLBI, 2012; Thayer & Lane, 2007), and they were examined as predictors of negative affect and executive functioning.

The alternative model yielded a significant chi-square (χ^2 = 32.52, *df* = 16, *p* < .05), indicating inadequate fit (Figure 3). However, the chi-square test is known to be inflated by large sample size and the presence of higher correlations within a model (Kenny, 2014), so additional fit indices also were examined. These indicated a good fit to the data (NFI = .951, CFI = .973, RMSEA = .044). In this model, the path from negative affect to executive functioning was significant (β = -.22, *B* = -.03, *SE* =.010, *p* < .01). Age significantly

predicted both executive functioning (β = -.48, B = -.005, SE =.001, p < .001) and negative affect (β =-.20, B =-.014, SE =.004, p < .001. The path from HRV to executive functioning was no longer significant (β =-.005, B < .001, SE =.005, p >.05).

Model Comparison

In a final analysis, the alternative model including covariates was compared to a version of this model in which the covariates were allowed to intercorrelate, but their relationships with the key variables (HRV, negative affect, and executive functioning) were constrained to zero (Figure 4). Retaining the full set of paths in the model while constraining the covariates' relationships to the variables of interest allows for a direct comparison of two structurally identical, nested models: one in which the covariates' relationships to HRV are accounted for, and a second model, nested within the first, in which HRV is the sole predictor of NA and EF. The aim of this comparison was to determine the utility of HRV as an independent predictor of NA and EF relative to its predictive value after accounting for variance in these relationships that is attributable to covariates.

The constrained version of the model showed poor fit to the data across all indices (χ^2 = 174.855, df = 25, p < .001, NFI = .738, CFI = .759, RMSEA = .106; Figure 5). A chi-square difference test yielded a significant result (χ^2_{diff} = 142.334, df_{diff} = 9, p < .001), indicating that the model in which covariates' relationships to key variables were measured provided a significantly better fit to the data compared to the model in which these relationships were constrained to zero. Comparing these models' AIC and BCC values likewise showed that the model in which covariates' relationships to the variables of interest were estimated (AIC = 108.521, BCC = 109.977) was superior to the model in which these paths were constrained (AIC = 232.855, BCC = 233.966).

Discussion

In light of the aim to examine relationships between HFV, negative affect, and executive functioning, the most salient findings from this thesis are as follows: (1) Contrary to expectations, resting HRV showed a negligible relationship to negative affect and no relationship to executive functioning when the demographic- and health- related covariates were included in the model. (2) As expected, the negative affect and executive functioning constructs showed a significant inverse relationship to one another. (3) Among the variables included in the final model, age was the only significant predictor of NA and EF, showing the expected inverse relationship to EF and an inverse relationship to NA.

The finding that HRV did not predict NA (self-reported symptoms of anxiety and depression) or EF in this sample contrasts with several prior findings of such relationships. These disparities raise several possibilities. First, the current results can be interpreted in light of methodological differences between this thesis and the prior research, as well as some limitations of this study which could be addressed in follow-up analyses. Second, it suggests some potential limitations on or refinements to the interpretations of prior findings and to theories proposing HRV as an "index" measure reflecting a broad capacity for efficient emotional and cognitive self-regulation (e.g., Porges, 2011; Thayer et al., 2009).

HRV and Negative Affect

In the present analyses HRV showed a negligible relationship to NA, which contrasts with a number of prior findings associating low HRV with symptoms of anxiety and depression. One set of factors that distinguish this study from much of the prior research in this area, and which likely contributed to the discrepant findings, concerns population differences. The majority of previous findings have described associations between low HRV in clinical populations, whereas the current study examined an epidemiological, largely

nonclinical sample. (e.g., Friedman, 2007; Levin et al., 2007; Rottenberg, 2007, though see also Bleil, 2008). Therefore, the predominantly subclinical levels of negative affect in this epidemiological sample may help to explain its lack of association with HRV. Along similar lines, another factor that may have contributed to the lack of relationship between NA and HRV was the exclusion of potential participants who were taking antidepressant medications (ADMs), in order to control for the potential negative effects of antidepressants on HRV (Rottenberg, 2007). It is possible that this exclusion criterion may have prevented the detection of a real but modest NA-HRV relationship, if such a relationship were to be driven by persistent, clinical levels of depression and/or anxiety among participants taking ADMs (Kemp, 2012).

A second interpretation is that the relationship between low HRV and depression in the clinical literature may be overstated due insufficient controls for ADM use. In this case, excluding those participants taking ADMs from the present study may have eliminated a spurious NA-HRV relationship reflected in the literature, driven by the effects of ADMs on HRV. Consistent with this possibility, findings from the Netherlands Study of Depression and Anxiety, which examined very large samples (N > 2000) comprised of individuals with current psychological disorders, those with remitted disorders, and healthy controls, showed that the effects on HRV of major depression (Licht et al., 2008) and of three anxiety disorders (panic disorder, social phobia, and generalized anxiety disorder; Licht et al., 2009) were due to autonomic effects of ADMs rather than clinical symptoms per se. Although the current biomarker and cognitive data is cross-sectional, future waves of MIDUS data collection will allow longitudinal analyses that could help to disentangle the HRV-NA relationship from effects of ADM use.

Given that anxiety generally has shown stronger, more reliable associations with low HRV than depression has (Kemp et al., 2010; Rottenberg, 2007) and that this line of research has focused less consistently on clinical samples, an additional difference between the analyses reported here and prior studies reporting HRV–anxiety relationships also comes to the fore. This issue pertains to the ways anxiety has been defined and operationalized. Several prior investigations have linked low resting HRV with greater emotion-modulated startle magnitude and potentiation, in order to infer a relationship between low resting HRV and high anticipatory anxiety under laboratory induction of mild arousal or "threat" (e.g., Melzig et al., 2009; Ruiz-Padial, 2003). By contrast, self-reported trait anxiety (TA), which was examined in this study, is a construct generally measured by explicit self-report, and which pertains to individual differences in the level of subjectively experienced anxiety elicited by threatening situations (Spielberger, 1983, 1989). Findings of a lack of relationship between TA and fear-potentiated startle in nonclinical samples (Cook et al., 1992; Grillon et al., 1993) suggest that caution is warranted in inferring greater anticipatory anxiety in those with low HRV based on the reported association of low HRV with greater startle magnitude, or at least in equating this anxiety construct with that associated with TA. To explain the discrepancy between TA and startle measures, the latter have been interpreted more conservatively as a function of the fear elicited by the immediate stimulus, rather than as an indicator of generally higher anxiety levels (Grillon & Baas, 2003).

Interestingly, this narrower interpretation of the startle measure may shed some light on the more reliable findings of low HRV in panic disorder (PD) and posttraumatic stress disorder (PTSD) compared to other anxiety disorders (Friedman, 2007). These two disorders are characterized by experiences of acute physiological hyperarousal associated with fear of imminent threat (Blechert et al., 2007; Tuescher et al., 2011). In physiological terms, this

hyperaroused state may more closely resemble the induced fear in the startle paradigm than it does the persistent, pervasive distress seen in some other anxiety disorders—which have more often shown blunted startle response and little relationship to HRV(Friedman, 2007; McTeague & Lang, 2012). Taken together, these variations in the HRV findings across different anxiety disorders and anxiety-related measures suggest that resting HRV may be more associated with implicit, physiological measurements of acute, reactive fear than with explicit, trait-level anxiety.

HRV and Executive Functioning

Executive functioning showed a significant relationship to HRV before covariates (age, BMI, diabetes/hyperglycemia) were included in the model, but this relationship was reduced to zero when covariates were incorporated. This null finding after controlling for covariates contrasts with associations between EF and HRV found in smaller, more homogenous samples in which such controls were not required (reviewed in Thayer et al., 2009). However, one recent investigation of HRV and executive functioning using MIDUS II data likewise found a significant relationship between a multifaceted EF factor and resting HF-HRV that was no longer significant after adding demographic covariates (age, sex, education) to the model. Those findings and the present results raise questions about how to interpret a significant EF-HRV association that appears to be "accounted for" by age. Age was the most powerful predictor of both HRV and EF in the present model, whereas sex showed no significant relationship to HRV (see Table 4). Potential pitfalls of controlling for education in analyses of EF are discussed below, in the context of the present study's limitations.

On one hand, the significant relationship between EF and HRV in the structural model before the addition of covariates (Figure 2) may have been spurious (Type I error), in

the sense that it can be statistically attributed to both variables' significant age-related decline. Given the myriad of psychological and physiological changes with age, these two variables' respective associations with age need not reflect any meaningful common influence or shared mechanism of change. Yet, on the other hand, the finding that HRV and EF each showed stronger relationships to age than to one another does not preclude the possibility that their initial significant association reflected a meaningful relationship in which both variables also show significant age-related change. Age essentially provides a summary measure of a multitude of physiological and psychological changes over time, without reference to mechanisms. Therefore, controlling for age, particularly in a sample with a broad age range, runs the risk of "washing out" subtler associations between age-affected processes (Type II error; Consonni, Bertazzi, & Zocchetti, 1997).

To take a purely speculative example, if age-related decline in both HRV and EF were due in part to one or more common underlying mechanisms, such as age-related decline in the functional integrity of prefrontal cortical structures, which have been associated with both processes (Thayer et al., 2012), a real HRV–EF relationship theoretically could be reduced to nonsignificance by introducing age as a covariate. Thus, under the current conditions, the extent to which controlling for age simply reallocates to the age variable a moderate but real HRV–EF association subserved by mechanisms that, themselves, undergo age-related change, remains unclear. While the wide age range of the present sample is a strength of the study, it also increases the likelihood that the strong relationship of age to both HRV and EF, particularly among the oldest participants, could overshadow a more nuanced HRV–EF relationship. The planned tests of these models in subsamples with narrower age ranges will help shed light on this issue.

In addition to these questions about the interpretation of age as a covariate, several key difference between these two compatible analyses of MIDUS data and the prior reports of an association between HRV and executive abilities (Thayer et al., 2009) may help to explain the discrepant results. First, the prior studies have generally collected cognitive and HRV data at the same time points (Hansen, Johnson, & Thayer, 2003; Hansen et al., 2004; Hansen, Johnson, & Thayer, 2009), whereas in MIDUS II collected these measures separately, sometimes with several years intervening. While resting HRV is considered reasonably stable over time (and more stable than HRV reactivity to stress; Bertsch et al., 2012; Kleiger et al., 1991; Sinnreich et al., 1998), it remains possible that the interval between data collection points allowed confounding influences to reduce a relationship between these measures that would have been evident had they been collected at closer time points.

A second methodological difference that may have contributed to the disparity in findings is that some prior studies (e.g., Hansen, Johnson, & Thayer, 2003; 2009) have used median splits based on their respective HRV measures to define "high" versus "low" HRV groups for comparison on cognitive tasks. In these cases, dichotomizing the HRV variable may have sharpened HRV-related group differences that might have been less evident in analyses that incorporated both cognitive performance and HRV as continuous measures, such as the present study and that of Kimhy et al. (2013).

Perhaps most importantly, the positive and negative findings are based on different approaches to defining and measuring EF. The present study and Kimhy et al.'s (2013) recent MIDUS study each modeled executive functioning as a latent variable measured with cognitive tests thought to tap different facets of executive functioning (Miyake, 2000; Tun & Lachman, 2006). In contrast, the few prior studies in which HRV was used as a predictor of EF have examined a narrower set of individual differences in cognitive performance. These

studies examined performance on subtasks of a continuous performance task (CPT) and a two-back working memory task (WMT) by conducting separate analyses of response times on specific components of the measures and the number of true and false positive responses on particular task components (Hansen, Johnson, & Thayer, 2003; Hansen et al., 2004; Hansen, Johnson, & Thayer, 2009). Based on their assessments of these subtasks' cognitive demands, the authors effectively split moment-to-moment task performance into nonexecutive and executive components, highlighting HRV- related differences in the latter. Interestingly, one of these studies also included a "threat" condition, in which the threat of shock was never carried out; in this condition only the low HRV group's performance improved. This was interpreted as indicative of their greater anxiety under threat (Hansen, Johnson, & Thayer, 2009), although the converse seems equally plausible.

This study aimed to extend Hansen and colleagues' findings by testing whether resting HRV, as a continuous variable, could predict performance on a multifaceted EF construct in a large, diverse sample. The lack of a significant HRV-EF relationship under these conditions contrasts with Hansen et al.'s work, yet it parallels findings by Kimhy and colleagues (2013). They also used a multifaceted EF construct, which was comprised of the three indicators used in the current study and two additional tests tapping processing speed and fluid intelligence/reasoning. They also found no relationship between EF and either resting or post-challenge HRV. Their exploratory post hoc analyses of individual tests' relationships to HRV at rest and during recovery from cognitive stressor revealed one significant relationship: Faster vagal recovery from stress was significantly associated with faster response on the mixed-trial Red/Green task (Kimhy et al., 2013). This task's demands for rapid task-switching and inhibition of rote, prepotent responses bares some notable resemblance to the demands of the CPT used by Hansen and colleagues, suggesting that

these measures requiring rapid switching between rote tasks under time pressure may show a relationship to HRV, though the extent to which this relationship is due to "executive" task demands or other features of task performance is unclear. Overall, these findings suggest that the relationships between HRV and cognitive performance may be best understood by focusing analyses and interpretations on specific, implicated abilities rather than generalized relationships to "executive" tasks at large. Furthermore, Kimhy's (2013) results parallel the startle findings in the suggestion that HRV's relations to these cognitive abilities reflect individual differences in a particular state of physiological arousal—in this case, induced by reactivity to cognitive challenge—rather than differences in specific types of cognitive abilities per se.

Effects of Age

Age and HRV. The introduction of age as a covariate drove many of the significant relationships in the analyses. Among all study variables, age showed the strongest relationship with HRV. This finding is consistent with the substantial support in the literature for an inverse relationship between age and HRV (e.g., O'Brien, O'Hare, & Corrall, 1986; Kuo, 1999; Sinnreich et al., 1998). In addition, although race was not included in the analyses due to the large proportion of white participants, the racial make-up of the sample may have accentuated the age-HRV relationship. Earlier research has shown that on average the magnitude of the age-HRV association is stronger in whites than in African-Americans, due to lower HRV earlier in adulthood among African-Americans (Choi, 2006).

Age and Negative Affect. Age was also the only significant predictor of EF and NA in the study, exempting the significant relationship between NA and EF themselves. Though research into the effects of age on adults' anxiety and depression symptoms has yielded mixed results, those studies that have controlled for covarying risk factors (e.g., sex,

education, marital status, socioeconomic status) generally have shown a clearer pattern of decreased anxiety and depression risk with increasing age (Jorm, 2000). In addition, several characteristics of the sample may have strengthened the inverse relationship between age and NA in the present study.

First, there are several reasons why older participants in the current sample may have had lower NA than older adults in the U.S. population at large. Among older individuals, ill health is one of the most powerful predictors of anxiety and depression (Jorm, 2000; Wade & Cairney, 2000). A recent comparison of longitudinal retention of participants from MIDUS I to MIDUS II cohorts found health status to be a key modifier of older individuals' continued participation. That is, older participants in MIDUS I who were in poor physical health were among the least likely individuals to be retained in MIDUS II (44%), whereas the likelihood of retention was unusually high among older first-wave participants in excellent health (83%; Radler & Ryff, 2010). Along similar lines, participation in the MIDUS II Biomarker Study involved an overnight stay at a psychophysiology laboratory in order to standardize data collection conditions. This requirement would have prevented participation by otherwise-eligible MIDUS II participants whose physical health was too poor to accommodate it.

The present study's exclusion criteria also may have had differential effects on levels of NA across the age range represented in the sample. As noted, depression is associated with poor physical health among older people (Jorm, 2000). Excluding individuals who had suffered a stroke and those taking antihypertensives from the sample is likely to have excluded a larger proportion of older participants than young ones, due the greater prevalence of these conditions with advanced age (Davies, Chung, & Juarez, 2011). Furthermore, among the participants excluded by this criterion, the prevalence of comorbid

conditions such as high cholesterol, diabetes, and heart disease was likely higher among the older adults, based on these conditions' comorbidity patterns across the lifespan (Davies, Chung, & Juarez, 2011). Thus, the exclusion of individuals taking antihypertensives may have disproportionately removed older individuals from the sample whose ill health—hypertension and its common comorbid conditions—put them at increased risk of anxiety and depression. As a point of contrast, an especially strong predictor of depression among younger adults is social stress (Wade & Cairney, 2000), which is unlikely to have interacted with any exclusion criteria apart from ADM use.

One caveat to the current finding of an inverse relationship between age and NA is that this pattern may be specific to the portion of the lifespan represented in MIDUS II data. Some large-scale studies examining anxiety and depression symptoms cross-sectionally across the whole adult lifespan, rather than from midlife to old-age, have reported a quadratic relationship in which, on average, negative affect is relatively low during early adulthood, peaks at midlife, and declines in old age (Blanchflower & Oswald, 2008). In this case, the present finding of a linear, inverse relationship between age and NA may capture only the down-slope of a more complex curvilinear relationship.

Age and Executive Functioning. In the present study, age also showed an inverse relationship with executive functioning. This finding is consistent with prior research on the differential decline of various cognitive abilities with age (e.g., Bryan & Luszcz, 2000; van Hooren et al., 2007; reviewed in Luszcz, 2011). It also accords with neurobiological evidence that some frontal lobe structures that subserve executive abilities may show relatively early and/or more pronounced structural and functional changes compared to other cortical regions in normal aging (Raz et al., 1997, 2005; Spreng, Wojtowicz, & Grady, 2010; West, 1996, 2000).

Against the background of generally broad support for age-related decline in executive functioning, the individual studies within this body of research continue to reckon with differences in the ways that executive functioning is conceptualized and operationalized, which have compromised generalizability and contributed to some discrepant findings (Luszcz, 2011). As a result, one question that has arisen in this area pertains to the role of processing speed in many tasks used to measure executive functioning. Some theorists have proposed that "global" age-related declines in processing speed, rather than more "local" declines in capacities for specific types of processing (i.e., executive functioning), might better explain patterns of cognitive aging (Salthouse, 1996). This alternative theory suggests that caution is warranted in interpreting the present finding specifically as an age-EF relationship, given that two of the three EF measures (Category Fluency and Red/Green) were timed tasks dependent on rapid performance.

This potential confounding of the effects of slowed processing and executive decline with age, however, may be inherent to these cognitive constructs themselves, which have been shown to make interrelated and/or partially overlapping contributions to age-related decline (e.g., Albinet et al., 2012; Borella, Ghisletta, & de Ribaupierre, 2011; Matthews, 2011;), suggesting that the competing *frontal/executive* and *processing speed* theories of cognitive aging are not mutually exclusive (Albinet et al., 2012; Schretlen et al., 2000). In this context, one advantage of the structural equation modeling approach to operationalizing EF is that it allows EF to be measured in terms of the shared variance among several types of tasks thought to tap different kinds of executive processes (Miyake et al., 2000). By partialling out the tasks' shared variance, this method helps to account for the inherent "task impurity" in measures of higher-order processes, which, by definition, rely on the efficient use of other

capacities such as attentional control and speed of processing (Albinet et al., 2012; Miyake et al., 2000).

In addition, the present study's exclusion criteria may have had differential effects on EF that interacted with the effects of age. That is, excluding individuals who were taking antihypertensive medications may have attenuated the age-EF relationship somewhat by removing from the sample a subgroup of older individuals who were at greater risk than age-matched study participants for executive deficits. Older individuals with hypertension have shown significantly increased risk of executive dysfunction relative to normotensive individuals, even when they have been carefully screened for common comorbidities (Vicario et al., 2005) and when hypertension is medically controlled (Raz, Roderigue, & Acker, 2003). Furthermore, as discussed in the context of the age-NA relationship, the individuals excluded for use of antihypertensives were, by virtue of the diagnosis of hypertension, also at increased risk for certain comorbidities including vascular risk factors (e.g., diabetes, high cholesterol; Davies, Chung, & Juarez, 2011) and cardiac diseases (e.g., atrial fibrillation, coronary heart disease, congestive heart failure; Stamler, Stamler, & Neaton, 1993). These conditions would, if present, further increase the risk of executive impairments among those who were excluded from the sample (Pugh et al., 2003; Roberts et al., 2013).

Limitations

A number of limitations of the present study should be kept in mind. First, the use of cross-sectional rather than longitudinal data precludes any causal or developmental inferences regarding the observed associations (Kraemer et al., 2000). This is a common issue for large epidemiological studies in which follow-up may not be practical. However, future waves of MIDUS will continue to gather cognitive and psychophysiological data using

the protocols initiated in MIDUS II. These data will allow for future analyses of longitudinal change in the variables of interest, including the extent to which low resting HRV is a predictor versus a symptom of the health-related covariates to which it was related. Second, as noted earlier, the low representation of racial and ethnic minorities among MIDUS II participants for whom cognitive and biomarker data were available limits the generalizability of the current findings, particularly given the differences in resting HRV and in its patterns of age-related change between U.S. racial groups (Choi, 2006; Liao et al., 1995). Third, although the medication-based exclusion criteria were selected to avoid confounding effects on HRV, they may have enhanced the age-NA relationship and/or attenuated the age-EF relationship somewhat. Both of these possibilities reflect the increased risk of poorer cardiovascular health among the excluded individuals, which is associated with increased risk of depression and anxiety as well as with a vascular risk profile thought to confer executive impairments.

Fourth, as in any large, cross-sectional study including a broad age range, cohort effects on the relationships of key variables to age cannot be ruled out. Along these lines, meta-analyses comparing the levels of anxiety and neuroticism, a trait-like construct closely related to NA, across birth cohorts found that younger cohorts report significantly higher levels of both traits than older cohorts did at comparable ages (Twenge, 2000). This effect complicates the inference of developmental change from the inverse age-NA relationship—an interpretation that will be testable when longitudinal data on the MIDUS II Biomarker Project measures become available.

Similar cohort effects are possible on EF, in relation to the unexamined variable of educational attainment. That is, older participants, whose HRV has declined with age (Stein, Kleiger, & Rottman, 1997), also may tend to be less educated, given the increasing

proportion of U.S. adults attending college and earning advanced degrees in recent decades (U.S. Bureau of the Census, 1977, 1987; U.S. Department of Education, 1996).Given that educational attainment has shown associations with measures of executive skills (e.g., Tun & Lachman, 2008; van Hooren et al., 2007), education level may interact with age as a confounding influence on EF. However, controlling EF for education level from the outset also could be problematic, because the causal connection between education and many aspects of cognitive functioning is theoretically bidirectional (Cesi, 1991), so that controlling initially for education level might artificially reduce real EF variance.

Conclusion

Under the conditions of the current analyses, resting high frequency HRV showed no predictive value in relation to either NA or EF. As described above, population and methodological differences may help to explain the discrepancy between these null findings and those of prior studies linking HRV to related aspects of emotional and cognitive self-regulation. However, it is also notable that individual findings in this area often have been interpreted broadly, as being consistent with theories such as the Polyvagal Theory (Porges, 2011) and the Neurovisceral Integration Model (Thayer et al., 2009). These theories' claims for HRV as a potential "physiological metaphor for the regulation of emotional states" or indicator of "[ability] to produce context appropriate responses, including appropriate recovery after [a] stressor has ended" (Thayer et al., 2012) strongly suggest that it might provide a meaningful, easily accessible, implicit metric for assessing individual differences in these aspects of psychosocial functioning. To the contrary, in the current study the utility of a readily accessible demographic factor, age, significantly outstripped that of HRV for predicting NA and EF, respectively. Nonetheless, this finding does not preclude meaningful, psychologically relevant interpretations of individual differences in HRV. Instead, it suggests

that refinements in the theoretical understanding of HRV's significance could be informed by (1) more conservative interpretations of the constructs with which HRV has been associated in individual studies, and (2) more large-scale tests of those interpretations in demographically diverse samples that allow controls for medication effects on autonomic functioning. Rather than remaining a potential correlate of self-regulation or adaptive responding at large, HRV could become a more clearly interpretable variable in the study of neurocardiac communication, stress responsivity, and the psychophysiological relations between personality, psychological disorders, and medical health.

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Tables

Table 1. Demographic and clinical characteristics of the sample

		N (%)	Mean (SD)
DEMOGRAPHICS			
Age		533 (100)	54.9 (10.7)
Sex	Male	247 (46.3)	--
	Female	286 (53.7)	--
Racial Origin	White	493 (92.5)	
	Black / African-American	11 (2.1)	--
	Native American or Alaska Native / Aleutian Islander / Eskimo	8 (1.5)	--
	Asian	3 (0.6)	--
	Other	17 (3.2)	
	Refused	1 (0.2)	
Highest Level of Education Completed	No school / some grade school	1 (0.2)	
	Some high school (no diploma/no GED)	12 (2.3)	--
	Graduated from high school or received GED	109 (20.5)	--
	1-2 Years of college, no degree	78 (14.6)	--
	3 or More years of college, no degree	25 (4.7)	--
	Graduated from 2-year college, vocational school, or associate degree	37 (6.9)	--
	Graduated from a 4- or 5-year college, or bachelor degree	138 (25.9)	--
	Some graduate school	22 (4.1)	--
	Master's degree	84 (15.8)	--
	PhD, EdD, MD, LLB, JD, or other professional degree	24 (4.5)	--
CLINICAL CHARACTERTISTICS			
Health-Related Covariates			
	Body Mass Index (BMI)	533 (100)	28.18 (5.7)
	Have ever had heart disease	17 (3.2)	--
	Have had high BP / hypertension ever (12 mo)	29 (5.4)	--
	Have had diabetes / high blood sugar ever (12 mo)	22 (4.1)	--
	Blood LDL cholesterol (mg/dL)	532 (99.8)	110.2 (34.0)
	Ever smoked cigarettes regularly	203 (38.1)	--
	Getting regular exercise at least 20 min. 3x/wk	493 (82.4)	--
Additional Health-Related Characteristics			
	Currently smoke cigarettes regularly	59 (11.1)	--
	Have ever had depression	74 (13.9)	--
	Have ever had cholesterol problems	162 (30.4)	
	Taking corticosteroid medications	52 (9.8)	--
	Taking cholesterol medications	85 (15.9)	--

Table 2. Mean, standard deviation, skewness, and kurtosis statistics for HRV and measures of negative affect and executive functioning.

	Mean	SD	Skewness	Kurtosis
B1 natural log of HF-HRV	4.89	1.25	.074	.380
Category Fluency	20.44	5.82	.372	.446
Digits Backward	5.18	1.42	.116	-.074
Red/Green	-1.03	.201	-1.20	1.72
log CESD + 1	1.76	.877	-.211	-.524
log STAI	3.46	.246	.437	-.383
Non-normally distributed variables before log transformation				
HF-HRV	309.40	815.37	13.14	227.04
STAI	32.74	8.55	.988	.666
CESD	7.23	7.12	1.84	4.25

Table 3. Simple bivariate correlations between HRV and measures of negative affect and executive functioning.

		log HF-HRV	log STAI	log CESD + 1	Category Fluency	Digits Backward
Log STAI	Pearson r	-.031				
	Sig.	.482				
Log CESD + 1	Pearson r	.021	**.727****			
	Sig.	.635	.000			
Category Fluency	Pearson r	.064	**-.105***	-.067		
	Sig.	.141	.015	.125		
Digits Backward	Pearson r	.053	-.041	-.042	**.143****	
	Sig.	.226	.347	.331	.001	
Red/Green	Pearson r	.108*	-.033	-.046	**.281****	**.159****
	Sig.	.013	.447	.292	.000	.000

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Table 4. Simple bivariate correlations between HRV and hypothesized covariates.

		LN HF-HRV			LN HF-HRV
DEMOGRAPHIC COVARIATES					
Age	Pearson r	**-.310****	BMI	Pearson r	**-.123****
	Sig. (2-tailed)	.000		Sig.	.004
Gender	Pearson r	.047			
	Sig.	.279			
HEALTH-RELATED COVARIATES					
Diabetes/high blood	Pearson r	**.103***	High BP/hypertension	Pearson r	.035
sugar ever (12 mo)	Sig. (2-tailed)	.018	ever (12 mo)	Sig.	.423
Ever had heart disease	Pearson r	.054	Blood LDL	Pearson r	-.040
	Sig.	.216	Cholesterol (mg/dL)	Sig.	.356
Reg exercise at least	Pearson r	-.045	Ever smoked	Pearson r	-.070
20 mins 3 times/wk	Sig.	.299	cigarettes regularly	Sig.	.182

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Figures

Figure 1. Measurement model. (N=533) $\chi^2 = 2.723$, $df = 4$, $p = .605$, NFI = .994, CFI = 1.00, RMSEA < .001.

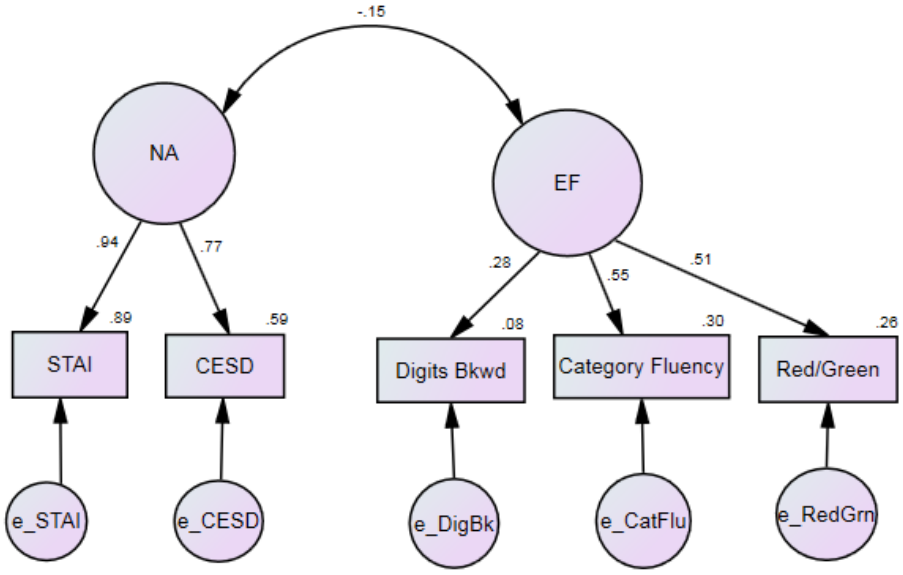

Figure 2. Structural model. (N=533) $\chi^2 = 5.981$, df = 7, $p = .542$, NFI = .998, CFI = 1.00, RMSEA < .001. $^{++}$ $p = .05$; * $p < .05$.

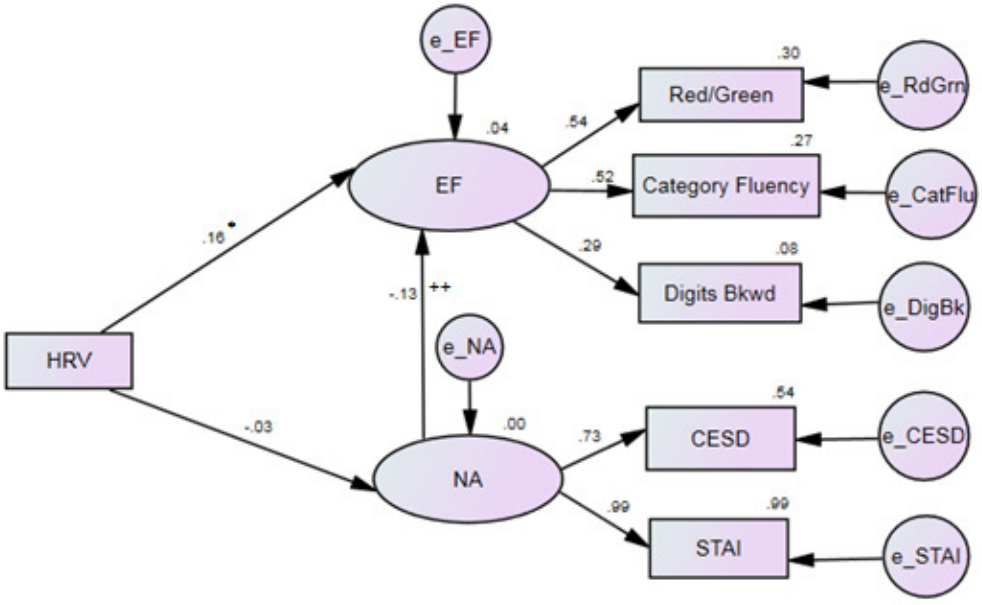

Figure 3. Full alternative model including covariates. (N=533) χ^2 = 32.52 (p < .05); NFI = .951, CFI = .973, RMSEA = .044. * p < .05; ** p < .01; *** p < .001.

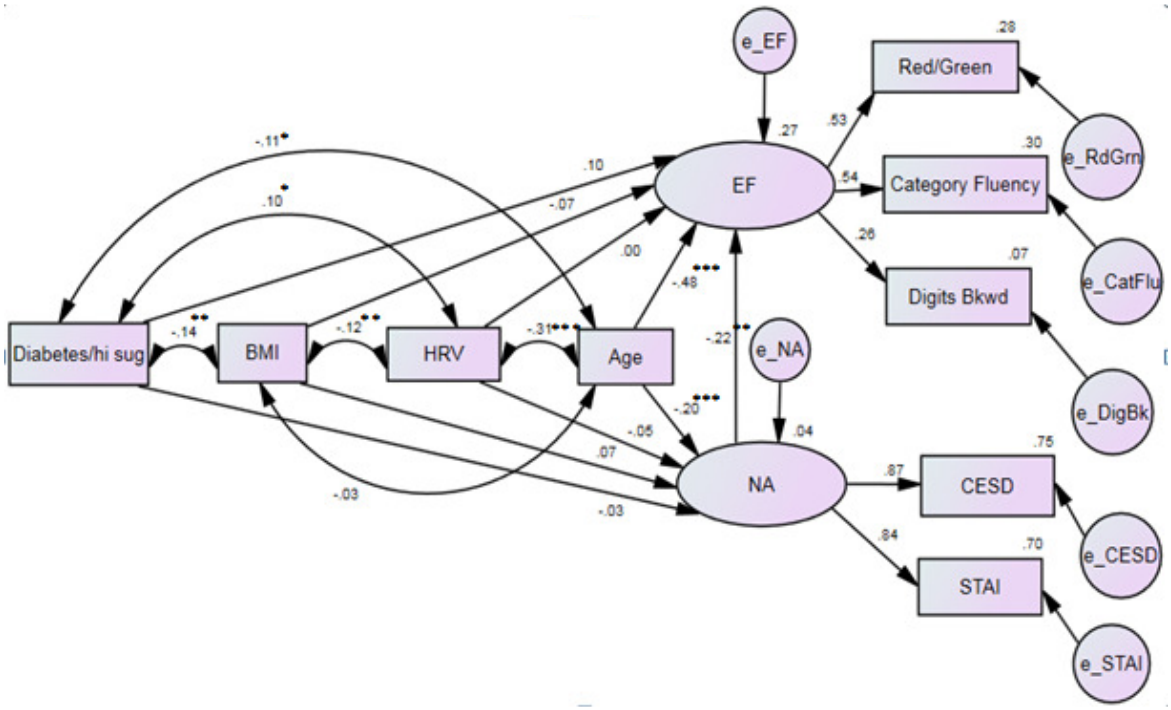

Figure 4. Alternative nested model with covariates allowed to intercorrelate and their relationships to HRV, NA, and EF set to zero.

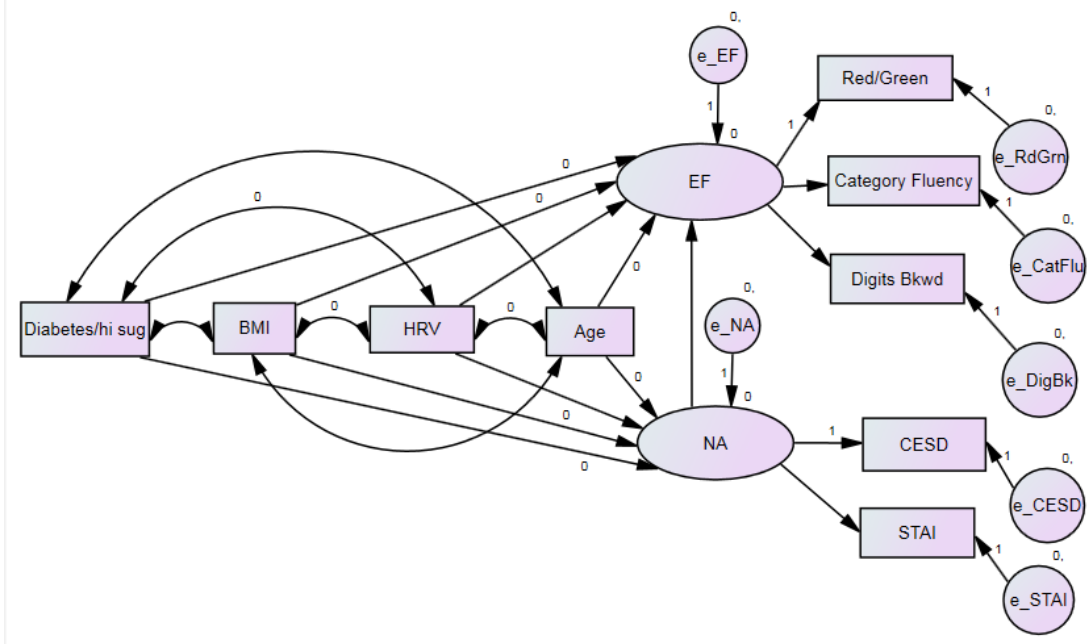

Figure 5. Alternative nested model. (N=533) χ^2 = 174.855, df = 25, p < .001, NFI = .738, CFI = .759, RMSEA = .106. $^{++}$ p = .05; * p <.05.