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Depressive Symptomatology and Respiratory Sinus Arrhythmia in a Non-Clinical Sample of Middle-Aged African Americans

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Abstract

Decreased heart rate variability and depression are both independent risk factors for cardiac mortality in clinical and non-clinical samples. The purpose of the current study is to examine the hypothesis that severity of depressive symptomatology is inversely associated with respiratory sinus arrhythmia (RSA) in a non-clinical sample of African Americans. The sample included 77 African Americans with a mean age of 48.4 (SD=11.7). Participants completed the Beck Depression Inventory-II (BDI-II) and a five-minute resting baseline measurement of RSA was collected. The BDI-II total score was positively associated with RSA ($\beta = .334$, $p = .008$). Given the unexpected direction of the association, we separated the BDI-II into cognitive and somatic affective subscales to identify which construct was driving the relationship. The somatic affective, was related to RSA ($\beta = .328$, $p = .010$), but not the cognitive subscale. Given this unexpected positive result, future research should further examine the nature of the relationship between depressive symptomatology and RSA in African Americans, as the relationship may vary based on levels of depressive symptomatology.

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Disclosure Statement

The authors declare that there are no conflicts of interest.

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Keywords

Heart Rate Variability; Depression; Depressive Symptomatology; Respiratory Sinus Arrhythmia; Somatic Affective; Cognitive

Introduction

Coronary heart disease (CHD) is the single leading cause of death in the United States for both men and women (Heron, et al., 2009) and all racial/ethnic groups (Heron, 2007). Psychological risk factors, such as depressive symptoms, are associated with increased risk for CHD (Cohen, Panguluri, & Whooley, 2010). The establishment of depressive symptoms as a risk factor for CHD stems from their consistent association with cardiovascular morbidity and mortality (Wulsin & Singal, 2003; Carney, Freedland & Vieth, 2000). Approximately one in five patients meet the criteria for major depressive disorder at the time of diagnoses with CHD or after a myocardial infarction (MI), while another one in five meet criteria for subclinical depression (Carney, et al., 2000). Studies also report that depression accelerates the progression of CHD (Carney, et al., 2000; Pizzi et al. 2008) and slows recovery from MI (Carney et al, 2001). Interestingly, researchers have suggested that autonomic dysfunction, as evidenced by poorer cardiac vagal tone, might be a key factor mitigating the relationship between depression and cardiac morbidity and mortality (Bosch et al. 2009, Kemp, et al. 2010; Rottenberg et al. 2007). In addition, research indicates that vagal control is the best measure for predicting near fatal to fatal cardiac arrhythmia (Huikuri et al., 2009; Kemp et al., 2010). Specifically, vagal control is inversely associated with increased likelihood of cardiac arrhythmia occurrence.

Heart rate variability (HRV) refers to temporal variations between successive heartbeats. It is partially mediated by the complex interplay between the parasympathetic and sympathetic branches of the autonomic nervous system (European Task Force, 1996; Allen et al, 2006; Porges, 2007). High vagal tone, (indicated by increased HRV) is essential for the maintenance of the heart's dynamic flexibility, as well as an indicator of cardiovascular health. Respiratory sinus arrhythmia (RSA) is an index of vagal activity and is inversely associated with poor cardiac health (Rottenberg, et al., 2002; Thayer & Lane, 2007). In addition to measuring vagal tone, RSA is used to assess overall vagal control (Chambers & Allen, 2002), which research has suggested to be clinically important to mitigating depressive symptomatology (Rottenberg, et al., 2002).

Research that has investigated the relationship between depression and autonomic activity has primarily examined clinical samples that have suffered a major cardiac event or patients with clinical depression (Lett, et al. 2004; Siepmann et al., 2008; Salomon et al., 2009; Licht et al., 2008). In the case of a cardiovascular event, such an event may leave the heart in a static, deleterious condition. In this case, the heart would not be healthy enough to deal with stressors, including those psychological stressors. In a clinically depressed sample, psychological stress could precipitate increases in anxiety (Dobson, 1985; Suls and Bunde, 2005). Increases in anxiety are associated with autonomic nervous system dysregulation (Friedman and Thayer, 1998). In addition, HRV has been linked to depression in those with

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cardiovascular disease (Vaccarino et al., 2008), with an association between low HRV and depression being reported after MI (De Jonge, Mangano, & Whooley, 2007) and other cardiovascular events (Carney, et al., 2006). Overall it appears that individuals, who are clinically depressed, have poorer HRV than their healthy counterparts (Van der Kooy et al. 2008).

However, there are studies that have reported no association between depression and HRV (Watkins et al. 1999; Carney et al., 2000; Sayar et al. 2002). Additionally, in the follow up study to the Rottenberg, et al. (2002), Rottenberg et al. (2007) found no relationship between RSA and depression severity. An even smaller body of research presents a positive association between RSA, a measure of HRV, and depressive symptomatology in depressed persons (Rottenberg, et al., 2002). Additionally, the influence of medication usage in each sample could cause variation in findings (Rottenberg, et al., 2002). Another factor contributing to the mixed results in previous research is the ethnic differences in both depression symptomatology (Riolo et al., 2005) and HRV (Wang et al., 2005; Sloan et al. 2008). Taking the inconsistent and scant findings together, the explanation of the relationship between depressive symptomatology and parasympathetic activity remains unclear.

Given the disproportionate rate to which African Americans are at risk for CHD (Douglas, et al., 2003), and the limited amount of research regarding depressive symptomatology and HRV in African Americans, it is critical to explore the complex interplay between depressive symptoms and HRV in this group. Therefore the purpose of our study was to examine the relationship between HRV and depressive symptomatology in a community-based sample of middle-aged African Americans. Given the presented literature, we hypothesize that RSA will be inversely related to BDI-II scores. Specifically, we expect that higher RSA levels will be associated with lower BDI-II total scores.

Method

Participants

The current study was part of a larger cross-sectional study entitled *Stress and Psychoneuroimmunological Factors in Renal Health and Disease*. It explored the associations among neuropsychological, psychosocial and biological factors, with a focus on renal health in 214 community-based African Americans. Participants were recruited from the Washington, DC metropolitan area at local health fairs and advertisements at Howard University Hospital. Individuals with diagnoses of psychological disorders or a history of cardiovascular events were excluded from the study. Written informed consent was obtained from all participants, and all data were collected as approved by the Howard University Institutional Review Board. Upon completion of the study, participants received monetary compensation. A subsample of 77 African Americans was used for the current study due to missing data in the ambulatory monitoring system data collection (43%) and approximately 20% having incomplete Beck Depression Inventory items. This subsample included everyone with complete biological, cardiac, and psychosocial data. The mean age of the sample is 48.4 (SD = 11.7), with a mean years of education of 13.9 (SD = 2.5). More demographic information is provided in Table 1.

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Procedure

Data collection occurred between the hours of 9 a.m. and 4 p.m. at the General Clinical Research Center located at Howard University Hospital. After the participant provided consent, a nurse entered the room to take blood pressure and other vital information. After assessment by the nurse, a trained research assistant attached the six-lead ambulatory monitoring system to the chest and back of the participant. Participants were then seated upright in a hospital room chair and instructed to relax for ten minutes. After ten minutes, each participant completed a demographic questionnaire, which included medical history, psychological trauma and medication usage. The participant then completed a battery of neuropsychological and psychosocial measures, which included the Beck Depression Inventory II.

Depressive Symptoms

Depressive symptoms were measured by the Beck Depression Inventory-II (BDI-II), which is a self-report questionnaire addressing both cognitive and somatic affective factors of depression (Beck, Steer & Brown, 1996). This scale includes 21 items, with a response scale ranging from 0–3. The responses to each item are summed into a total score. For research purposes, the BDI-II can be split into two subscales: cognitive and somatic affective (Arnau et al., 2001). The cognitive subscale items represent the mental aspects of depression, such as sadness, self-accusations and self-dislike. The somatic affective subscale is characterized by items, which represent more physical symptoms of depression, such as changes in appetite, lack of sleep, and body aches and pains. The current study uses both subscales in the analyses because previous research has demonstrated a link between somatic depressive symptoms and lower HRV (De Jonge, Mangano, & Whooley, 2007). In addition, research has also determined that African Americans more likely to present with somatic complaints (Brown, Schulberg, & Madonia, 1996; Kirmayer, 2001). Therefore, the analysis of the scales separately acknowledges the cultural differences in depressive symptom presentation (Tylee & Gandhi, 2005) and is consistent with prior research on depressive symptoms and HRV. The overall scale in the current sample yielded a Cronbach's alpha coefficient of $\alpha = .928$. The Cronbach's alpha for the subscales were $\alpha = .885$ and $\alpha = .877$, for the somatic affective and cognitive, respectively.

Respiratory Sinus Arrhythmia

RSA was derived from the interbeat interval series collected by the six-lead ambulatory monitoring system (Groot, Geus, & Vries, 1998) while the participant sat in a quiet hospital room for ten minutes. The ambulatory monitoring system device provides a continuous measure of movement and segments were obtained from the periods where movement was minimal for a continuous five-minute period in the middle of the ten minute measurement as recommended by the European Task Force (1996). Researchers focused on the high frequency bandwidth, which reflects vagal activity (0.15–0.40 hz). The interbeat intervals were then transformed using the Fast Fourier transformation by the CMetx software (Allen, Chambers & Towers, 2007). Data were hand checked for artifacts. If artifacts were found in the data, they were removed prior to the imputation into the CMetX software. Utilizing the CMetX software and protocol, the natural log of RSA was computed. RSA is a frequency

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domain measure of HRV that is positively related to other candidate indices of cardiac parasympathetic activity, including successive differences in the interbeat interval, the percentage of interbeat intervals greater than 50 msec, and Tochi's Cardiac Vagal Index (Allen, Chambers & Towers, 2007). RSA has been shown to be associated with cardiovascular disease, diabetes and hypertension (Masi, Hawkley, Rickett & Cacioppo, 2007).

Covariates

Covariates were used to aid in the clarification of the relationship between depression and HRV. Demographic factors such as age, gender, and years of education were entered as covariates because of their statistical relationship to depression and HRV and because of the literature-based conceptual framework. Age and education were represented in years. Physiological factors such as body mass index (BMI) and blood pressure have been associated with depression and HRV and were also included as covariates. BMI was calculated using the formula weight (in pounds) by the square of height with quotient multiplied by 703. Lastly, selfreport of hypertension and medication usages were also included as covariates.

Statistical Analysis

All data analyses were performed using the Statistical Package for the Social Sciences 16 (SPSS, 2008). Given the negative skewness of BDI-II scores and BMI, a log and square root transformations were used. RSA values were also log transformed. Zero-order correlations were performed to determine the unadjusted associations among the variables. Hierarchical linear regression analyses were performed to explore associations between HRV and depressive symptomatology. Covariates were entered in the analysis prospectively to adjust for variance in either the depressive factors or HRV. The first block of the regression models included the demographic covariates, while the physiological covariates were included in the second block, followed by the depression variable.

Results

Table 1 contains characteristics of the study sample. BDI-II scores ranged from 0 to 40 (mean 7.51 ± 9.05). BMI scores ranged from 16 to 55 (mean 30.39 ± 8.36).

Table 2 presents BDI-II items in rank-order. Highest item percentage endorsements for the Somatic items were "Changes in Sleeping Pattern" (45.5%), "Loss of Energy" (44.2%), "Changes in Appetite" (40.3%), "Tiredness or Fatigue" (35.1%), and "Loss of Interest in Sex (33.8%). The highest item percentage endorsements for the Cognitive items were "Guilty Feelings" (35.1%), "Self-Criticism" (29.9%), "Past Failure" (24.7%), and "Punishment Feelings" (20.8%).

Table 3 presents zero-order correlations between log transformed RSA and demographic, physiological and depressive variables. RSA was inversely associated with Systolic Blood Pressure (SBP) ($r = -.211$, $p = .033$) and self-reported hypertension ($-.222$, $p = .026$). Positive associations were found between RSA and total BDI-II scores ($r = .278$, $p = .007$),

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the Cognitive subscale of the BDI-II (r = .195, p = .045) and the Somatic Affective subscale of the BDI-II (r = .248, p = .015).

Utilizing multiple regression analysis, demographic and other covariates were entered prior to the BDI-II total score to predict RSA. As seen in Table 4, BDI-II total score significantly predicted RSA levels (β = .334, p = .008).

After separating the BDI-II into the cognitive and somatic affective subscales, each was put into the multiple regression analysis to determine which subscale better explained the relationship between BDI-II total score and RSA. The order of entry of demographic and covariate variables remained the same but the BDI-II total score was replaced with each subscale. The somatic affective subscale significantly predicted RSA levels (β = .328, p = .010), found in Table 5. However, as seen in Table 6, the cognitive subscale was not a significant predictor of RSA levels (β = .191, p = .116). When both subscales were entered into the same regression analyses alongside the covariates, as reported in Table 7, the subscales were not significantly associated RSA levels.

Discussion

Results from this study illustrated a positive relationship between the total BDI-II score and RSA, contrary to our hypotheses. The direction of the association was not expected; therefore, additional analyses were conducted to elucidate this finding. Our analyses identified a significant association between the somatic affective subscale of the BDI-II and RSA. However, there was no significant association with the cognitive subscale, suggesting that the somatic affective subscale is driving the relationship between the total BDI-II score and RSA. The associations were present when analyses adjusted for demographic and medical risk factors, including age, BMI, blood pressure, self-reported medication usage and self-reported diagnosis of hypertension. It is important to note that participants in this study were not clinically depressed. The middle-aged sample presented in the current study examines the depressive symptoms that non-clinically depressed individuals carry with them on a daily basis.

Previous research reports an inverse relationship between HRV and depression (De Jonge, Mangano, & Whooley, 2007; Glassman et al., 2007; Vaccarina et al., 2008). Specifically, depressed groups have lower HRV levels in comparison to controls (Thayer et al., 1998; Vaccarina et al., 2008; Van der Kooy et al. 2008; Licht et al., 2008). Further, De Jonge et al (2007) also reported a negative association between HRV and somatic depressive symptomatology in a large sample of patients with stable cardiovascular disease. However, the current study is the first to identify a positive association between RSA, a measure of HRV, and depressive symptomatology in a middle-aged, non-clinical sample of African Americans. In line with this finding, Thayer et al (1998) also reported a positive association between depression and heart period variability in a subsample of women. These results are similar to findings reported in a sample of preadolescents by Bosch et al (2009). Bosch and colleagues found a positive relationship between HRV and the cognitive subscale of the Youth Self-Report Affective Problems Scale in a sample of Dutch adolescents. Further evidence for this claim is found when examining the cognitive subscale created by Bosch et

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al. (2009), which included questions similar to the items on the somatic affective subscale used in the current study. However, Bosch et al., (2009) also reported an inverse association between HRV and the somatic subscale. These inconsistencies among the studies are a clear indication of the heterogeneity in sample types, depressive symptomatology experienced by them, and potential differences in autonomic activity.

Previous research presents the differential effects of the cognitive and somatic affective subscales of depression measures (De Jonge, Mangano, & Whooley, 2007; Bosch et al., 2009). The somatic affective subscale contains items such as sadness, loss of pleasure, self-criticalness, irritability and changes in appetite. These items fall under the psychological domain of emotion. This is quite interesting, as previous research has also shown a positive relationship between emotionality, specifically sadness and HRV (Miller & Wood, 1997; Rottenberg, et al., 2002; Koelsch et al., 2007), however, these associations were found in depressed and/or clinical samples.

In a study exploring the association between RSA and depression severity, higher RSA levels were not associated with depression symptomatology (Rottenberg, et al., 2002). However, there was a relationship presented between RSA and sadness and suicide items of the BDI-II. Rottenberg, Wilhelm, Gross and Gotlib (2002) reported a smaller sample size and utilized participants who were being treated with medication for their depression in comparison to the current study. Given the adjustment for medication usage and exclusion criteria for psychological disorders, findings in the current study support those of Rottenberg, Wilhelm, Gross and Gotlib (2002) and add to the limited literature regarding RSA and depressive symptomatology.

African Americans are more likely to suffer from dysthymia, as compared to non- Hispanic Whites, who are more likely to have Major Depressive Disorder (Riolo et al., 2005). Given the prevalence of depression overall, it seems imperative to adapt the assessment and research of this construct to different samples types. Refining measurements of constructs for each ethnicity may yield more reliable and robust results. The lack of relationships in some studies versus others could be due to sample composition. Given the predominately non- Hispanic White samples presented in previous research, results may differ from a predominately African American or Hispanic sample. These dysthymic symptoms may provide insight not only into the individual, but could also lend understanding to the social and physiological function of the participants. Moreover, the current sample reported relatively low levels of depression on the BDI-II, which limits generalizability to moderately depressed and clinical samples. However, this is not the first study to examine the consequences of depressive symptoms in non-depressed samples (For example see Dunkel, Kendel, Lehmkuhl, Hetzer, & Regitz-Zagrosek, 2011; Kinder, Kamarck, Baum, & Orchard, 2002).

The positive association presented in this study may be indicative of the presence of protective mechanisms in these participants. The ability to regulate emotions and control ones behavior is one of the fundamental pieces of coping (Barbarin, 1993). Instead of ruminating over stressful experiences, African Americans may be prone to employ various coping strategies, which may have been taught to them as children (Brown et al., 1986). As

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individuals age, they become more adaptive. This is due to the increase in experiences and the learning of dynamic coping strategies. Along these lines, Porges (2007) asserts that a high resting HRV indicates that the person perceives their environment to be a safe one and that this perception allows them to express their emotions. This notion of emotive expression, along with finding sanctuary in your environment may lead to an increase in HRV.

There are a few limitations of the study that should be considered. We examined the influence of both BDI-II subscales in the same regression model. Given the moderate association between the two subscales, results from this exploratory analysis may be influenced by multicollinearity. Thus, the outcome of the regression may be less precise in determining the exact influence of each subscale. Data were collected using a cross-sectional study design yielding small sample sizes, which limits the ability to generalize these findings to different samples. The same can also be said regarding our sample type, as all participants were African American urban community dwellers, with relatively high levels of obesity and hypertension. Other health-related behaviors that were not collected, such as sleeping habits, smoking, use of caffeine, or the taking of medications that may influence autonomic activity would be informative covariates for future research. Lastly, participants were administered protocol at various times during the day and this may have affected the cardiac impedance data.

Overall, the current results show a positive association between HRV and depressive symptomatology, with the somatic affective subscale driving the relationship in a middle-aged sample. This could be indicative of the difference in depressive style in African Americans, or that here may be an ethnically specific protective mechanism at work. Future research should focus on an ethnically specific research question and look to understand the biobehavioral profiles within a given group. Employing social support and personality measures as modulating mechanisms may elucidate the nature of the relationship.

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Highlights

1. Baseline respiratory sinus arrhythmia positively associated with BDI-II total score
2. Examined bot Cognitive and Somatic Affective subscales with RSA
3. Community-based sample
4. Non-Clinical sample of Middle-Aged African Americans

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Table 1

Subject Characteristics

N=77	M ±SD/n (%)
Age (yrs)	48.40 ± 11.74
Ed (yrs)	13.9 ± 2.45
Men	50 (64.9%)
Women	27 (35.1%)
SBP (mm hg)	134.86 ± 17.34
DBP (mm hg)	80.58 ± 12.61
Hypertension	23 (30%)
Use of Medications	37 (48%)
BMI (kg/m^2)	30.39 ±8.36
BDI-II	7.51 ± 9.05
Somatic-Affective	5.22 ± 5.97
Cognitive	2.29 ± 3.61
RSA (ms^2)	5.69 ± 1.53

Ed=years of education, SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure, BMI=Body Mass Index, BDI-II=Beck Depression Inventory-II, Somatic-Affective = Somatic Affective Subscale of BDI, Cognitive = Cognitive Affective Subscale of BDI, RSA=Respiratory Sinus Arrhythmia

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Table 2

Items of the Somatic Affective and Cognitive Subscales of the BDI-II

	Mean (SD)	% Item Endorsement
Somatic Items		
Changes in Sleeping Pattern	.66 (.88)	45.5%
Loss of Energy	.51 (.66)	44.2%
Changes in Appetite	.47 (.62)	40.3%
Tiredness or Fatigue	.48 (.77)	35.1%
Loss of interest in Sex	.45 (.75)	33.8%
Loss of Pleasure	.39 (.69)	29.9%
Concentration Difficulty	.39 (.69)	28.6%
Loss of Interest	.35 (.68)	26.0%
Indecisiveness	.34 (.72)	23.4%
Crying	.36 (.82)	20.8%
Agitation	.38 (.80)	22.1%
Sadness	.19 (.39)	19.5%
Irritability	.25 (.59)	18.2%
Cognitive Items		
Guilty Feelings	.40 (.61)	35.1%
Self-Criticism	.43 (.73)	29.9%
Past Failure	.32 (.61)	24.7%
Punishment Feelings	.30 (.67)	20.8%
Self-Dislike	.30 (.76)	15.6%
Worthlessness	.21 (.55)	14.3%
Pessimism	.18 (.50)	14.3%
Suicidal Thoughts or Wishes	.14 (.38)	13.0%

* An item is “endorsed” if a person has a score other than 0; SD= standard deviation

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Table 3

Zero-Order Correlations

	Age	Sex	Ed	SBP	DBP	Hyper	Meds	BMI	BDI-II	Cog	Som	RSA
Sex	−.294**											
Ed	−.057	−.237*										
SBP	.191*	.154	−.155									
DBP	.103	.165	−.116	.735**								
Hyper	.372**	−.115	−.026	.391**	.119							
Meds	.408**	−.274**	.053	.138	−.049	.565**						
BMI	.016	−.177	−.096	.294**	.107	.330**	.330**					
BDI-II	−.192*	−.191*	−.211*	−.059	.027	−.099	−.099	.156				
Cog	−.208*	−.087	−.094	−.079	.066	−.079	−.079	.066	.766**			
Som	−.074	−.225*	−.254	−.048	.034	−.054	−.054	.191*	.956**	.634**		
RSA	−.223	−.026	−.026	−.211*	−.176	−.222*	−.222*	−.189	.278*	.195*	.248*	

* =p<.05,

** =p<.01,

Ed=years of education, SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure ,Hyper=Hypertension, Meds=currently on any medication, BMI=Body Mass Index, BDI-II=Beck Depression Inventory-II, Som = Somatic Affective Subscale of BDI, Cog = Cognitive Affective Subscale of BDI, RSA=Respiratory Sinus Arrhythmia

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Table 4

RSA regressed on BDI-II

		Beta	t	R^2
Model 1	Age	−.161	−2.021	.064
	Gender	−.079	−.644	
	Education	.075	.638	
Model 2	SBP	.024	.122	.128
	DBP	−.139	−.794	
	BMI	−.142	−1.118	
	Hypertension	−.045	−.288	
	Medication	−.123	.404	
Model 3	BDI-II	.334**	2.713	.215

* =p<.05,

** =p<.01,

SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure, BMI=Body Mass Index, BDI-II=Beck Depression Inventory-II

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Table 5

RSA regressed on Somatic Affective Subscale

		Beta	t	R^2
Model 1	Age	−.161	−2.021	.064
	Gender	−.079	−.644	
	Education	.075	.638	
Model 2	SBP	.024	.122	.128
	DBP	−.139	−.794	
	BMI	−.142	−1.118	
	Hypertension	−.045	−.288	
	Medication	−.123	.404	
Model 3	Somatic Affective	.328**	2.657	.211

* =p<.05,

** =p<.01,

SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure, Hyper=Hypertension, BMI=Body Mass Index, Somatic Affective= Somatic Affective Subscale of BDI-II, RSA=Respiratory Sinus Arrhythmia

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Table 6

RSA regressed on Cognitive Subscale

		Beta	t	R^2
Model 1	Age	−.161	−2.021	.064
	Gender	−.079	−.644	
	Education	.075	.638	
Model 2	SBP	.024	.122	.128
	DBP	−.139	−.794	
	BMI	−.142	−1.118	
	Hypertension	−.045	−.288	
	Medication	−.123	.404	
Model 3	Cognitive	.191	−.902	.160

* =p<.05,

** =p<.01,

SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure ,Hyper=Hypertension, BMI=Body Mass Index, Cognitive = Cognitive Affective Subscale of BDI-II, RSA=Respiratory Sinus Arrhythmia

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Table 7

RSA regressed on Somatic Affective Subscale

		Beta	t	R^2
Model 1	Age	−.161	−2.021	.064
	Gender	.079	−.644	
	Education	.075	.638	
Model 2	SBP	.024	.122	.128
	DBP	−.139	−.794	
	BMI	−.142	−1.118	
	Hypertension	−.045	−.288	
	Medication	−.123	.404	
Model 3	Somatic Affective	.288	1.521	.214
	Cognitive	.042	.237	

* =p<.05,

** =p<.01,

SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure, Hyper=Hypertension, BMI=Body Mass Index, Somatic Affective= Somatic Affective Subscale of BDI-II, RSA=Respiratory Sinus Arrhythmia