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Lower catecholamine activity is associated with greater levels of anger in adults☆

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ABSTRACT

Previous research has revealed a consistent association between heart rate at rest and during stress and behavioral problems, potentially implicating autonomic nervous system (ANS) functioning in the etiological development of antisocial behavior. A complementary line of research has focused on the potential independent and interactive role of the two subsystems that comprise the ANS, the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS), on behavioral problems. The current study aims to contribute to the existing literature by examining the influence of heart rate (HR) reactivity, high-frequency heart rate variability (HF-HRV) reactivity, and catecholamine activity on a comprehensive measure of anger in a large, nationally-representative sample of adults from the United States. Results from a series of structural equation models (SEMs) revealed that catecholamine activity was most consistently linked to anger, while associations involving HR and HF-HRV reactivity were nonsignificant. Additional analyses revealed that HF-HRV did not significantly moderate the association between catecholamine activity and anger. These findings highlight the importance of SNS activity in the development of more reactive forms of aggression such as anger.

1. Introduction

An impressive line of research has consistently demonstrated a significant and robust association between resting heart rate (HR) and various mental disorders including personality disorders (Lorber, 2004; Raine et al., 2000), internalizing problems such as depression (Stein et al., 2000), and lower overall levels of empathic response (Muñoz and Anastassiou-Hadjicharalambous, 2011). Perhaps the largest segment of the extant literature has been devoted to examining the potential association between resting HR and various externalizing behavior problems including delinquent and criminal behavior (Lorber, 2004; Ortiz and Raine, 2004; Portnoy and Farrington, 2015). Studies also find decreased heart rate in response to stressors in individuals with high levels of antisocial behavior (Ortiz and Raine, 2004; Popma et al., 2006; van Goozen et al., 2000). Taken together, findings flowing from this line of research have provided evidence indicating a significant association between lower HR at rest and during stress and greater levels of externalizing behavior problems. The results of a recent systematic review examining 114 studies and 115 independent effect sizes revealed a summary effect size of $d = -0.20$, indicating lower resting HR was associated with greater levels of antisocial behavior (Portnoy and Farrington, 2015). Importantly, this association persisted even after controlling for a host of study characteristics and across multiple measures of antisocial behavior, indicating a robust association.

Previous studies have indicated that HR is an indirect indicator of autonomic nervous system (ANS) activity, indicating that the reduced activity of the ANS may result in an increased predisposition toward externalizing behavior problems. While a significant number of studies have reported results that support this general hypothesis (Beauchaine, 2001; El-Sheikh et al., 2009), this particular line of research has continued to develop, identifying more details of the association between ANS activity and behavioral problems. For example, recent studies have examined the potential role of ANS activity in the development of more specific forms of behavioral problems (Hubbard et al., 2002; Pitts, 1997; Raine et al., 2014). The results of this line of research are decidedly mixed with some studies indicating that lower resting HR was significantly associated with both proactive and reactive forms of aggression (Pitts, 1997), while other studies have reported more

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consistent associations between resting HR and proactive forms of aggression (Hubbard et al., 2002; Raine et al., 2014). These findings suggest that heart rate may be differentially associated with reactive forms of aggression compared to proactive forms. Directly in line with this possibility, anger represents one particular facet of reactive aggression that may be differentially associated with resting HR, as previous studies have indicated that individuals with higher overall levels of anger are more likely to experience behavior problems including delinquent and criminal behavior (Carmichael and Piquero, 2004; Mazerolle et al., 2000).

Sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) activity have also been independently implicated in the development of behavioral problems (Beauchaine, 2001; de Vries-Bouw et al., 2012; El-Sheikh et al., 2009; Ortiz and Raine, 2004). Studies have identified significant associations between behavioral problems and a host of indirect indicators of SNS activity including lower electrodermal response (EDR) to stress tasks (Gao et al., 2010), lower pre-ejection period (PEP) reactivity (Hinnant et al., 2016), attenuated salivary alpha-amylase responsivity (Glenn et al., 2015), and lower overall levels of plasma or urinary catecholamine activity (McCaffery et al., 2000). Similarly, studies have implicated PNS activity as a significant risk factor for the development of behavioral problems (Grossman and Taylor, 2007; Beauchaine, 2001, 2015; El-Sheikh et al., 2009; Porges, 2007). For example, cardiac vagal control (typically operationalized by high frequency-heart rate variability [HF-HRV] and previously recognized as an indirect indicator of PNS activity) has been found to be significantly associated with behavioral problems (Beauchaine, 2015; Berntson et al., 2007; Porges, 2007). High baseline cardiac vagal control is thought to index appropriate emotional regulation (Beauchaine, 2001), and reduced cardiac vagal control at rest in response to emotional stimuli has been found to predict externalizing behavior problems in selected samples (Beauchaine, 2015; Porges, 2007).

Based on the differential responsibilities in the regulation of homeostatic reactions to stress-inducing stimuli of each subsystem, and in line with the concept of autonomic determinism (Berntson et al., 1991), previous studies have also examined whether the manner in which the PNS and SNS respond to one another also contributes to behavioral problems. More specifically, due to the reciprocal contributions of the PNS and SNS, previous studies have postulated that as activity in indicators of one subsystem increases, activity in indicators of the other should decrease (Berntson et al., 1994). However, other studies have found that concurrently low activity in both the PNS and SNS is associated with behavior problems in juveniles (Beauchaine et al., 2007; de Vries-Bouw et al., 2012; El-Sheikh et al., 2009). While previous studies have found preliminary evidence for both conditions (i.e., asymmetrical activity or concurrently low activity), the resulting limited literature remains decidedly mixed, leaving the potential moderating effects of one subsystem on the other unclear.

Additional methodological limitations exist within the current literature examining the association between indirect indicators of the SNS and PNS and behavioral problems. First, few studies focused on behavioral problems have simultaneously examined HF-HRV and catecholamine activity, with the majority of the existing literature focused on indicators of overall ANS activity, such as HR (Lorber, 2004; Ortiz and Raine, 2004; Portnoy and Farrington, 2015). Second, the majority of previous studies examining ANS activity and behavioral problems have focused almost exclusively on earlier stages of the life course such as childhood and adolescence (e.g., Crowell et al., 2006; de Vries-Bouw et al., 2012; El-Sheikh et al., 2009), with few studies examining the association during adulthood (but see Chiang et al., 2001; Hansen et al., 2007; Lobbestael et al., 2009). Third, many of the existing studies that do examine indicators of PNS and SNS activity in the development of externalizing behavior problems seem to be underpowered and rely on relatively small convenience samples (for an overview of studies examining externalizing behavior problems among children and adolescents, see van Goozen et al., 2007). Finally, many existing studies rely on bivariate correlations or multivariate statistical models with a limited number of covariates (for a comprehensive summary, see Portnoy and Farrington, 2015), effectively increasing the likelihood of detecting a spurious association.

In light of the status of the existing literature examining the associations involving HR, HF-HRV, catecholamine activity, and behavioral problems, the current study aims to accomplish two related objectives. First, the current study aims to examine the potential associations between HR reactivity, HF-HRV reactivity, catecholamine activity and anger in a large, nationally-representative sample of adults from the Survey of Midlife Development in the United States (MIDUS). This particular aspect of the current study is aimed at more directly examining the external validity of previous studies, while also increasing overall levels of statistical power in an effort to detect potentially smaller effects. Second, the current study also examines the potential interaction between HF-HRV and catecholamine activity in predicting anger. Previous simulation studies have revealed that within smaller samples ($N < 120$) even moderate to large moderating effects can be difficult to detect (Stone-Romero and Anderson, 1994). Since the current study employs a sufficiently powered sample (ranging between 682 and 742, depending on the estimated model), the likelihood of detecting a small, or even moderately sized, moderating effect increases significantly.

2. Methods

2.1. Data

The current study analyzes data from the MIDUS, a prospective, two-wave study consisting of a nationally representative sample of adults and funded by the National Institute on Aging (Brim et al., 1996). The first wave was carried out between 1995 and 1996 and included over 7000 adults from the United States, ranging in age from 25 to 74 years old ($M = 46.38$, SD = 13.00). Participants were selected using random-digit dialing sampling procedures and were asked to participate in a 30-minute telephone interview and complete two self-administered questionnaires (Brim et al., 2004). The final sample was comprised of slightly more females (51.69%) than males (48.31%) and was predominantly Caucasian (90.67%). The second wave of data collection was carried out between 2004 and 2006, when respondents were between 32 and 84 years old ($M = 55.43$, SD = 12.45). A total of 4963 respondents who participated in the first wave of the study were contacted a second time (70 percent retention rate; Love et al., 2010; Radler and Ryff, 2010). Participants were asked to complete a brief telephone interview and two self-administered questionnaires with an overall response rate of approximately 81%.

During the second wave of data collection, a subsample of participants was also asked to participate in the Biomarker Project, an extensive physical and mental health assessment carried out over two days (Love et al., 2010). Inclusion criteria for the Biomarker Project included the completion of the Wave 2 telephone interview and self-administered questionnaires, as well as existing health information indicating that the participant would be able to travel to one of three General Clinical Research Centers: University of California, Los Angeles (UCLA), University of Wisconsin, or Georgetown University. The resulting sample ($n = 1255$) did not significantly differ from the larger, nationally representative sample across age ($M_{Full} = 55.50$; $M_{Biomarker} = 55.26$; $t = 0.57$, $p = 0.57$) and sex (full sample = 47.06% males; biomarker sample = 45.26% males; $\chi^2 = 1.08$, $p = 0.30$), but was comprised of significantly more Caucasian participants (full sample = 89.97% Caucasian; biomarker sample = 93.05% Caucasian; $\chi^2 = 9.27$, $p = 0.002$) and participants with significantly higher socioeconomic status ($M_{Full} = 41.71$; $M_{Biomarker} = 43.40$; $t = 2.84$, $p = 0.01$). Medical professionals collected information related to a wide variety of factors related to medical history, medication use, sleep quality, and psychosocial experiences over a 24-hour period.

Table 1
Descriptive statistics for study measures.

	Mean/%	SD/N	Min–max
Heart rate (HR) measures			
Baseline HR (bpm)	72.73	10.84	45.20–109.75
HR after stress tasks (bpm)	76.53	11.39	46.25–114.95
HR reactivity	3.74	3.82	− 6.40-29.40
Heart rate variability (HRV) measures			
Baseline HRV	4.78	1.24	0.89–9.66
HRV after stress tasks	4.43	1.21	0.95–8.91
HRV reactivity	− 0.35	0.63	− 3.03-2.75
Catecholamine measures			
Dopamine (μg/g)	144.31	49.66	2.50–305.88
Epinephrine (μg/g)	24.10	7.96	3.50–41.86
Norepinephrine (μg/g)	1.77	0.80	0.09–3.91
Covariates			
Respiration rate	17.19	1.81	12.00–28.00
Socioeconomic status	43.40	14.15	13.85–80.53
Exercise 3 + times/week			0.00–1.00
Yes (%)	78.84	831	
No (%)	21.16	223	
Weekly fast food consumption	2.43	0.91	1.00–5.00
Currently use tobacco			0.00–1.00
Yes (%)	13.47	142	
No (%)	86.53	912	
Alcohol use in past month	2.53	1.58	1.00–6.00
Body mass index	29.18	6.01	14.99–60.39
Age (years)	55.26	11.78	34.00–84.00
Sex			0.00–1.00
Male (%)	45.26	477	
Female (%)	54.74	577	
Race			0.00–1.00
Caucasian (%)	93.05	978	
All other races (%)	6.95	73	

Note: Baseline HR and HRV measures are the average of the two baseline measures. HR and HRV after stress task measures were calculated as the mean of each measure following the verbal and mathematical stress tasks. HR and HRV reactivity were calculated as the difference between each baseline measure and the stress score for the same measure. Due to positive skew a natural-log transformation was applied to the HRV measures. Weekly fast food consumption reflects how often participants ate at a fast food restaurant or ordered takeout or delivery during a typical week, with responses ranging between 1 (*never*) to 5 (*seven or more times per week*). Alcohol use in the past month reflects the number of times each participant consumed alcohol in the past month with responses ranging between 1 (*never*) to 6 (*everyday*). Sample sizes reflect the total number of cases with valid (i.e., nonmissing) values on each measure.

Participants were subjected to a comprehensive physical exam and provided 12-hour urine samples, fasting blood draws, and saliva tissue specimens. The final analytic sample examined in the current study was limited to participants who completed the Biomarker Project (n = 1255), had complete information on all cardiovascular measures (n = 967), and complete information on all the included covariates (ranging between n = 682 and n = 742 depending on the model). Descriptive statistics, including means, standard deviations, and proportions, for the final analytic sample are presented in Table 1.

2.2. Measures

2.2.1. Anger

Anger was assessed using the Speilberger State-Trait Anger Expression Inventory (STAXI; Spielberger, 1996), a widely-used, psychometric scale tapping five subscales: Anger Expression-In (8 items); Anger Expression-Out (8 items); Anger Expression-Control (4 items); Angry Temperament (4 items); and Angry Reaction (4 items). For all STAXI items, respondents were asked to indicate how frequent they felt as described using the following answer categories: 1 = *almost never*; 2 = *sometimes*; 3 = *often*; 4 = *almost always*. In addition to the STAXI measures, the aggression scale of the Multidimensional Personality Questionnaire (MPQ) was also included in the current study. The aggression scale is contained within the Negative Emotionality dimension of the MPQ and is comprised of four items tapping general levels of aggression. (Patrick et al., 2002). Additional information regarding the employed data reduction strategies is provided in the Plan of Analysis section below.

2.2.2. Cardiac reactivity

On the second day of the two-day Biomarker Project protocol, participants engaged in an experimental protocol carried out by clinical nursing staff at one of the General Clinical Research Centers. The primary purpose of this protocol was to assess response to and recovery from commonly encountered physical and cognitive challenges (Love et al., 2010). The protocol consisted to two randomized cognitive challenges (one related to verbal skills, the other related to mathematical skills) in addition to a physical challenge in which participants were asked to stand from a sitting position as many times as possible over a six-minute period. Each challenge was followed by a six-minute recovery period. The clinical nursing staff collected information on several physiological indicators immediately following each stage of the protocol including blood pressure, respiration, and HRV. In addition, two baseline measures were also recorded prior to beginning the protocol. Fig. 1 summarizes the experimental protocol, including the measurement of cardiovascular measures, duration of recovery periods, and timing of stress tasks.

During the psychophysiological assessment conditions, electrocardiograph (ECG) electrodes were placed using a standard lead-II electrode configuration (placed on the left and right shoulders and in the lower left quadrant). Prior to attaching electrodes, skin was prepared using alcohol preps. Beat-to-beat waveforms were continuously monitored and digitized at 500 Hz[1] by a 16-bit National Instruments analog-to-digital board installed in a microcomputer (Ryff et al., 2010) and then processed by proprietary, user-written software (Gmark by Delano McFarlane) by implementing an algorithm for identifying R waves based on maximum voltage recordings. Additional visual inspection was also performed to correct for any software errors. HR during each examination period was calculated as the average of all valid RR intervals during the specified timeframe and converted to beats per minute units. HF-HRV (0.15–0.50 Hz) was calculated based on 300 second epochs using an interval method for computing Fourier transformations similar to that described by DeBoer et al. (1984). This method is described in more detail in Ryff et al. (2010) and has been used in prior studies of HF-HRV with this sample (Cooper et al., 2015; Donoho et al., 2015). Due to overall levels of positive skew, a natural-log transformation was applied to the resulting HF-HRV measures. Baseline HR and HF-HRV was calculated as the mean of two baseline measures collected prior to the implementation of the employed stress tasks. Consistent with previous studies analyzing the MIDUS sample (Čukić and Bates, 2015; Lin et al., 2014), the HR and HF-HRV stress measures were calculated as the mean of each participant's measure across the mathematical and verbal challenge tasks. HR and HRV reactivity were then calculated by subtracting the average baseline measures from the stress measures.

2.2.3. Catecholamines

During the Biomarker Project, overall levels of three catecholamines were recorded: dopamine; epinephrine; and norepinephrine. Beginning on the first evening of the two-day Biomarker Project protocol, 12-hour overnight urine samples were collected for each participant. Participants were provided specific collection instructions and were asked to inform trained nursing staff after each void. Collected samples were stored in a − 60° to − 80 °C freezer and shipped from each General Clinical Research Center to the Mayo Medical Laboratory for

[1] Importantly, 500 Hz is at the lower limit of reasonable resolution for HRV analysis. While this resolution aligns with described guidelines (Task Force of the European Society of Cardiology, 1996), lower sampling rages may result in jitter in the estimation of R-wave fiducial points and bias the resulting spectrum.

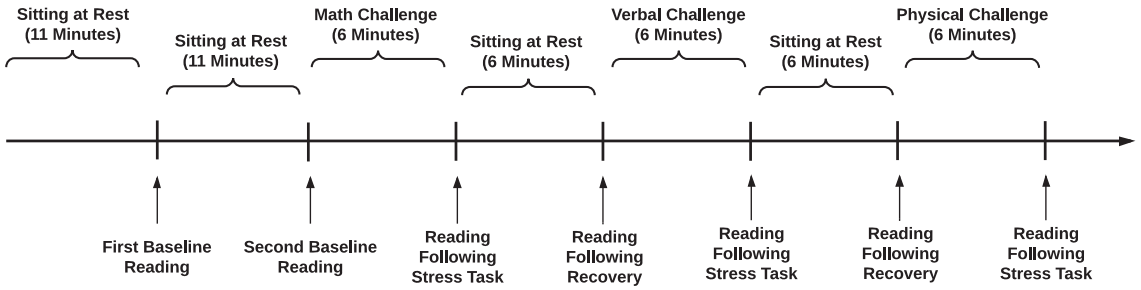

Fig. 1. Illustration of psychophysiological assessment conditions.

processing. Samples were processed using high-pressure liquid chromatography (HPLC) and adjusted for urine creatine levels to account for variation in hormone output based on body size (Ryff et al., 2010). The resulting catecholamines were measured in microgram/gram units (μg/g). Outliers were identified as participants with catecholamine scores more than three standard deviations from the grand mean and were removed from the final analysis.

2.2.4. Statistical covariates

A total of nine statistical covariates were also considered for the analysis. First, socioeconomic status (SES) was estimated using income, educational attainment, and occupation categories from the 1990 Census (Hauser and Warren, 1996) and coded such that higher scores indicate higher SES. Second, a dichotomous measure was used to identify participants that exercised for at least 20 min three or more times per week (0 = *no*, 1 = *yes*). Third, in an effort to assess dietary habits, participants were asked to indicate how often they ate at a fast food restaurant or order takeout or delivery during a typical week, with responses coded such that greater values indicated more frequent fast food consumption (1 = *never* to 5 = *seven or more times per week*). Fourth, participants were asked to indicate how often they consumed alcoholic beverages in the past month, and were coded such that greater values indicated more frequent use (1 = *never drinks* to 6 = *everyday*). Fifth, body mass index (BMI) was measured by dividing weight in kilograms by height measured in meters squared. Height and weight were measured by trained clinical staff during the Biomarker Project protocol. Three additional demographic covariates were also included in the statistical models: age (measured continuously in years); sex (0 = *female*; 1 = *male*); and race (0 = *Caucasian*: 1 = *all other races*). Finally, since respiration rates have previously been found to be predictive of HF-HRV (Grossman and Kollai, 1993; Grossman and Taylor, 2007), the resulting HF-HRV reactivity measures were regressed on respiration rate and the residuals were preserved and included in subsequent analyses. This statistical procedure has been recommended to reduce variance from the resulting HRV measures explained by the respiration rate measure (Grossman and Taylor, 2007).[2] Respiration was assessed during the experimental protocol using an Inductotrace respirometer (Ambulatory Monitoring Systems, Ardsley, NY) by placing stretch bands around the chest and abdomen and a Finometer beat-to-beat blood pressure monitor around the middle finger of the non-dominant hand. Signals from the stretch bands were collected by the A/D board at 20 Hz and analyzed by proprietary software on a minute-by-minute basis and reported as the average observed respiration rate (Ryff et al., 2010). Prior to beginning the experimental protocol, respiration was calibrated using an 800 cm^3 spirobag. With the exception of the respiration measure, all statistical covariates were included in the estimated multivariate SEMs. Based on the results of these models, and in the interest of parsimony, only those covariates that reached conventional levels of statistical significance ($p < 0.05$) were retained in the final models.

2.3. Plan of analysis

The plan of analysis was carried out in a series of interconnected steps performed using the statistical software program M*plus* 7.4 (Muthén and Muthén, 2012). First, a series of measurement models were estimated to examine the underlying factor structure of the externalizing behavior and catecholamine indicators. The second step of the analysis involved the estimation of a series of structural equation models (SEMs) to examine the associations between the cardiac reactivity measures, catecholamines, and anger. The multivariate models examined the association between each ANS activity measure separately and then collectively. Finally, in an effort to more closely examine the dynamical aspects of PNS and SNS activity, a final model was estimated that included a latent interaction term between the catecholamine factor and HF-HRV reactivity. A significant interaction term would indicate that the association between HF-HRV reactivity (or catecholamine activity) and the examined externalizing behavior factor is moderated by catecholamine activity (or HF-HRV reactivity). All of the estimated SEMs included all statistical covariates and missing data were handled using full information maximum likelihood (FIML) estimation. Finally, to aid in replication efforts, a variance-covariance matrix from the SEM involving all ANS measures is presented as an appendix (Table S1).

3. Results

The first step of the analysis was to estimate a series of measurement models. First, a second-order confirmatory factor analysis (CFA) revealed that the STAXI indicators and the negative emotionality aggression indicators loaded on a single, higher-order latent factor of anger (comparative fit index [CFI] = 0.92; Tucker-Lewis Index [TLI] = 0.92; root mean square error of approximation [RMSEA] = 0.06). Second, an additional model that allowed the catecholamine activity indicators to load on a single latent factor was estimated. Due to a limited number of indicators, the resulting model was just-identified and yielded perfect (yet trivial) model fit indices. However, the resulting standardized factor loadings ranged between 0.57 (epinephrine) and 0.73 (norepinephrine), and the results of a measurement model that included the catecholamine activity factor and the second-order anger factor provided an acceptable fit to the data (CFI = 0.93; TLI = 0.92; RMSEA = 0.05).

The next stage of the analysis involved the estimation of a series of multivariate SEMs aimed at examining the potential associations between HR reactivity, HF-HRV reactivity, catecholamine activity, and anger. The cardiac reactivity and catecholamine activity measures were entered into the SEM in a stepwise fashion, with the results presented in

[2] In this way, respiration rate is not included in the multivariate SEMs as a traditional covariate since such an approach would distinguish the variance in the examined anger measure explained by HF-HRV reactivity from the variance explained by the respiration rate. The approach employed in the current study instead adjusts the HF-HRV reactivity measure prior to estimating the association.

Table 2
Multivariate structural equation models estimating the association between the ANS activity measures and anger.

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Beta	SE	Beta	SE	Beta	SE	Beta	SE	Beta	SE
ANS activity measures										
HR reactivity	− 0.04	0.04					− 0.02	0.04		
HRV reactivity			0.06	0.04			0.05	0.04		
Catecholamines					− 0.14**	0.05	− 0.14**	0.05		
HRV reactivity × catecholamines									0.02	0.04
Covariates										
Socioeconomic status	− 0.11*	0.04	− 0.12**	0.04	− 0.12**	0.04	− 0.12**	0.04	− 0.11*	0.05
Exercise	ns		ns		ns		ns		ns	
Weekly fast food consumption	ns		ns		ns		ns		ns	
Alcohol use	0.12**	0.04	0.11**	0.04	0.11**	0.04	0.11**	0.04	0.10*	0.04
Body mass index	0.11*	0.05	0.10*	0.04	0.10*	0.04	0.10*	0.04	0.12*	0.05
Age	− 0.24**	0.04	− 0.24**	0.04	− 0.24**	0.04	− 0.24**	0.04	− 0.22**	0.04
Sex	ns		ns		ns		ns		ns	
Race	ns		ns		ns		ns		ns	
Model fit statistics										
χ^2 (df)	1473.88 (509)**		1576.16 (542)**		1880.05 (684)**		1720.78 (681)**		–	
CFI	0.91		0.92		0.91		0.92		–	
TLI	0.91		0.91		0.91		0.92		–	
RMSEA	0.05		0.05		0.05		0.05		–	
N	686		741		741		741		684	

Abbreviations: ANS = autonomic nervous system; HR = heart rate; HRV = heart rate variability; ns = nonsignificant.
Note: Standardized parameter estimates presented. Models 1–4 were estimated using a weighted least squares estimator (WLSMV) and Model 5 was estimated using a maximum likelihood estimator (MLR). All models include robust standard errors. Variance in HRV reactivity explained by respiration rate was residualized prior to the estimation of Models 2 and 5. Covariates that emerged as nonsignificant were omitted from the final models (flagged as "ns" above). Model fit statistics are not available for Model 5 due to the inclusion of a latent interaction term.
$p < 0.10$.
* $p < 0.05$.
** $p < 0.01$.

Table 2.[3] The columns labeled Model 1 display the results of an SEM estimating the association between HR reactivity and the second-order anger factor net of the included covariates. The results revealed a negative, but nonsignificant association ($\beta = -0.04$, $p = 0.36$). The second set of columns present the results of the SEM examining the association between HF-HRV reactivity and anger. While the resulting coefficient was in the expected direction, the results indicated a nonsignificant association ($\beta = 0.06$, $p = 0.14$). The results of the SEM examining the association between catecholamine activity and anger are presented in the third column (Model 3). The results revealed a significant and negative association ($\beta = -0.14$, $p = 0.002$), indicating that individuals with lower catecholamine activity at rest displayed greater overall levels of anger. The fourth set of columns presents the results of a SEM in which the HR reactivity, HF-HRV reactivity, and catecholamine activity measures were all included. The results were similar to those from the preceding models and revealed that catecholamine activity was significantly associated with anger ($\beta = -0.14$, $p = 0.002$), while associations involving HR reactivity ($\beta = -0.02$, $p = 0.70$) and HF-HRV reactivity ($\beta = 0.05$, $p = 0.21$) were nonsignificant. The fifth set of columns presents results from an SEM examining the potential dynamical aspects of HF-HRV and catecholamine activity with the inclusion of a latent interaction term between both measures. The results revealed a nonsignificant association between the latent interaction term and externalizing behavior ($\beta = 0.02$, $p = 0.65$), indicating that the previously reported associations between catecholamine activity and anger is not significantly moderated by HF-HRV reactivity.

[3] Additional sensitivity analyses (results not presented but available upon request) examined the associations between the ANS activity measures (HR reactivity, HF-HRV reactivity, and catecholamines) and each of the examined anger subfactors were estimated. The overall pattern of results was similar to those presented for the higher-order factor of anger.

3.1. Sensitivity analyses

In addition to the primary analyses, a series of sensitivity analyses were also performed to examine the robustness of the findings, with the results presented in Table 3. Model 1 displays the results of an SEM in which the HR and HF-HRV reactivity measures were calculated using estimates following the verbal challenge task (as well as the baseline measures). The results from this model directly aligned with the results from the primary analysis and revealed nonsignificant associations between HR reactivity and anger ($\beta = -0.02$, $p = 0.65$) as well as HF-HRV reactivity and anger ($\beta = 0.03$, $p = 0.56$). Similar to the primary analysis, the association between catecholamine activity and anger was negative and significant ($\beta = -0.14$, $p = 0.002$). Model 2 was similar, but included HR and HF-HRV measures that were calculated using estimates following the math challenge task. Once again, the results revealed nonsignificant associations involving HR ($\beta = -0.004$, $p = 0.92$) and HF-HRV reactivity ($\beta = 0.04$, $p = 0.30$), but a significant association between catecholamine activity and anger ($\beta = -0.13$, $p = 0.004$). In an effort to remain consistent with the law of initial values, Model 3 estimates associations involving HR and HF-HRV reactivity by including the stress *and* baseline HR and HF-HRV measures (along with all other covariates) in the estimated model. The results revealed a nonsignificant association between HR reactivity and anger ($\beta = -0.07$, $p = 0.57$) as well as a nonsignificant association between HF-HRV reactivity and anger ($\beta = 0.08$, $p = 0.40$). The association between catecholamines and anger remained significant ($\beta = -0.10$, $p = 0.04$). Model 4 displays the results of a similar model in which HR and HF-HRV were measured as latent factors using CFA and making use of the baseline, stress task, and recovery indicators, with the resulting model providing an adequate fit to the data (CFI = 0.93; TLI = 0.93; RMSEA = 0.04). Once again, the overall results were similar to those presented in the primary analysis and revealed nonsignificant associations involving HR ($\beta = -0.01$, $p = 0.85$) and HF-HRV reactivity ($\beta = -0.03$, $p = 0.54$), and the association involving catecholamine activity was negative and significant ($\beta = -0.14$, $p = 0.003$).

Table 3
Sensitivity analyses examining the association between ANS activity measures and anger.

	Model 1		Model 2		Model 3		Model 4	
	Beta	SE	Beta	SE	Beta	SE	Beta	SE
ANS activity measures								
HR reactivity	− 0.02	0.04	− 0.004	0.04	− 0.07	0.12	− 0.01	0.05
HR baseline					0.04	0.13		
HRV reactivity	0.03	0.04	0.04	0.04	0.05	0.09	− 0.03	0.05
HRV baseline					− 0.10	0.09		
Catecholamines	− 0.14**	0.05	− 0.13**	0.05	− 0.10*	0.05	− 0.14**	0.05
Covariates								
Socioeconomic status	− 0.12**	0.04	− 0.12**	0.04	− 0.10*	0.04	− 0.12**	0.04
Exercise	ns		ns		ns		ns	
Weekly fast food consumption	ns		ns		ns		ns	
Alcohol use	0.11**	0.04	0.11**	0.04	0.11**	0.04	0.11**	0.04
Body mass index	0.10*	0.04	0.11*	0.04	0.11**	0.05	0.10*	0.04
Age	− 0.24**	0.04	− 0.23**	0.04	− 0.25**	0.04	− 0.24**	0.04
Sex	ns		ns		ns		ns	
Race	ns		ns		ns		ns	
Model fit statistics								
χ^2 (df)	1709.70 (681)**		1754.78 (681)**		1639.03 (743)**		1917.78 (920)**	
CFI	0.93		0.92		0.92		0.93	
TLI	0.92		0.91		0.92		0.93	
RMSEA	0.05		0.05		0.04		0.04	
N	741		741		741		741	

Abbreviations: ANS = autonomic nervous system; HR = heart rate; HRV = heart rate variability; ns = nonsignificant.
Note: Standardized parameter estimates presented. All models were estimated using a weighted least squares estimator with robust standard errors (WLSMV). Covariates that emerged as nonsignificant were omitted from the final models (flagged as "ns" above). Variance in all HRV measures explained by respiration rate was residualized prior to the estimation of all models. Model 1 includes HR and HRV reactivity measures calculated using estimates following the verbal challenge task. Model 2 includes HR and HRV reactivity measures calculated using estimates following the math challenge task. Model 3 estimates HR and HRV reactivity by including the measures following the two stress tasks and baseline HR and HRV measures as covariates. Model 4 estimates HR and HRV as latent factors.
$p < 0.10$.
* $p < 0.05$.
** $p < 0.01$.

4. Discussion

The current study contributes to an existing literature focused on exploring associations involving cardiac reactivity, catecholamine activity and behavioral problems. Despite the large number of studies comprising this literature (which includes three separate meta-analyses and over 100 individual studies) and the robustness of the resulting associations ($d = -0.20$; Portnoy and Farrington, 2015), the existing literature suffers from a number of methodological limitations including low levels of statistical power, limited generalizability of findings, and the examination of a narrow range of behavioral problems. In addition, few studies have considered the potential moderating effects of both HF-HRV reactivity and catecholamine activity in the development of behavioral problems (but see de Vries-Bouw et al., 2012). In this way, the current study is the first to examine the independent and interactive influences of HR reactivity, HF-HRV reactivity, and catecholamine activity on a broad measure of anger in a large, nationally-representative sample of adults from the United States. The results revealed three key findings, all of which will be discussed in more detail below.

First, the multivariate models examining the association between the cardiac reactivity measures and anger revealed small and nonsignificant associations in all estimated models. While this pattern of findings was somewhat unexpected, a substantive number of previous studies have also reported nonsignificant associations involving either HR (Crowell et al., 2006; de Wied et al., 2012) or HRV measures (Beauchaine, 2001; Beauchaine et al., 2008; de Vries-Bouw et al., 2012) and various behavioral problems. In addition, the measure of anger used in the current study taps more reactive (as opposed to proactive) forms of aggression. For example, items included in the STAXI tap the frequency in which participants respond to a particular situation with anger (e.g., I strike out at whatever infuriates me). The results of a developing line of research have demonstrated a more consistent link between lower overall cardiac reactivity and proactive forms of aggression (for an overview, see Raine et al., 2014). Complementary findings have also demonstrated a relatively consistent link between lower levels of HRV and factors more directly linked to proactive forms of aggression such as emotional regulation (Appelhans and Luecken, 2006) and self-regulatory strength (Segerstrom and Nes, 2007). While the findings of the current study align with previous studies examining reactive forms of aggression, due to data limitations of the MIDUS, we were unable to examine proactive forms of aggression. Based on these observations and the limited number of previous studies that have distinguished between proactive and reactive forms of aggression, future research would benefit from examining associations involving additional forms of aggressive behavior.

In addition, a further specification of the underlying psychological processes that potentially link SNS activity and reactive forms of aggression may provide a better understanding of inconsistencies in the examined associations. The SNS branch of the ANS is activated during times of stress, resulting in the secretion of catecholamines and additional hormones that ultimately increase heart rate and oxygen flow (El-Sheikh et al., 2009). At the same time, polyvagal theory (Porges, 2007) asserts that, to aid in the physiological responses that accompany SNS activation, PNS activity is inhibited. This process is reversed once the stressor or challenge diminishes, resulting in normative homeostatic functioning. In this way, reactive forms of aggression, particularly in response to an external threat or stressor, would be expected to be more directly tied to increases in SNS activity. Alternatively, PNS activity has been found to be more directly linked to maintaining homeostasis and contributing various phenotypes related to future outlook and planning including attentional focus (Porges, 2007) and self-regulation (Segerstrom and Nes, 2007). In this way, an association between PNS activity and proactive forms of aggressive behavior would be more likely, as this particular form of behavior would be more likely to require future planning and foresight.

Second, compared to the cardiac reactivity measures, catecholamine activity was most consistently linked to anger. Importantly, this finding directly aligns with previous studies reporting associations between other indirect indicators of lower SNS activity and various behavioral problems (de Vries-Bouw et al., 2012; Lorber, 2004; Scarpa and Raine, 1997), but such studies rarely consider indirect indicators of PNS activity, making it difficult to distinguish the specific role of SNS activity in the resulting associations. In this way, the models estimated in the current study address this limitation by distinguishing resting catecholamine activity from cardiac reactivity. While the resulting pattern of findings still requires replication and should be interpreted with caution, such findings provide evidence suggesting that catecholamine activity, relative to cardiac reactivity, may be more closely linked to anger.

While only speculation, additional environmental sources of influence may also contribute to this pattern of results. Previous studies have revealed that individuals with greater exposure to early life trauma (Dodge et al., 1990) and chronic victimization (Sullivan et al., 2007) experience behavioral problems at a greater rate compared to their counterparts. Chronic and sustained levels of stress not only result in increased SNS activity, but can also result in simultaneous increases in hypothalamic-pituitary-adrenal (HPA) axis activity (Gordis et al., 2006) and overall increases in cortisol levels. Previous studies have found that greater levels of cortisol may nave neurotoxic effects on brain regions directly implicated in executive functions and emotional regulation, such as the prefrontal cortex, anterior cingulate cortex, and hippocampus (De Bellis et al., 1999; Carrion et al., 2001; Carrion et al., 2009). Deficits stemming from sustained exposure to increased levels of cortisol may further exacerbate underlying psychophysiological influences on externalizing behavior problems including SNS activity. In this way, future research would benefit from a more comprehensive examination of the potential role of HPA activity, as well as neuropsychological deficits, in the association between ANS activity and behavior problems.

Third, based on the results of previous studies suggesting moderating effects of one ANS subsystem on associations involving the other (Beauchaine et al., 2007; Berntson et al., 1994; de Vries-Bouw et al., 2012; El-Sheikh et al., 2009), additional multivariate SEMs were estimated to examine the potential interaction between HF-HRV reactivity and catecholamine activity. The results of these models failed to identify a significant interaction, indicating that the identified associations between catecholamine activity and anger were not significantly moderated by HF-HRV reactivity. Importantly, previous studies examining the potential moderating effects of one subsystem on the other have produced mixed results, with some studies finding support for the asymmetrical activity in both systems (Salomon et al., 2000), others for concurrently low activity levels in both systems (Beauchaine et al., 2007; El-Sheikh et al., 2009), and yet others finding no support for either model (de Vries-Bouw et al., 2012). While the findings from the current study more directly implicate the role of catecholamine activity on anger, these findings require replication in additional samples. In addition, future research would also benefit from a closer examination of the relationship between SNS and PNS activity within the context of more diverse sets of behavioral problems (e.g., criminal activity), personality disorders such as antisocial personality disorder, and other traits that have been found to be closely linked to criminal behavior like psychopathy (Marsh et al., 2008).

While the current study addresses several methodological limitations of previous studies examining ANS functioning and externalizing behavior problems, it is not without its own limitations. First, and as mentioned above, HR and HF-HRV reactivity were operationalized as stress specific measures, while catecholamine activity was assessed at a different time point and not directly linked to the employed experimental protocol, resulting in a trait-based measure of catecholamine activity. In this way, HR and HF-HRV reactivity are measured in response to stress, while catecholamine activity reflects more general functioning of the SNS. As discussed above, these differences in operationalization may represent another reason for the lack of an association between cardiac reactivity and anger. Future research would benefit from the use of a sample that allows for the simultaneous measurement of cardiac reactivity and SNS activity during the same experimental protocol. Second, while the examined anger measure was comprised of a diverse set of behavioral indicators, these measures were focused on reactive forms of aggressive behavior since indicators of proactive aggression were not collected by the MIDUS research team. Future research would benefit from more directly distinguishing between additional forms of aggressive behavior. Third, while the employed cardiac reactivity and catecholamine activity measures tap underlying ANS activity, they do not reflect overall functioning of the ANS or its subsystems. While additional physiological indicators of ANS functioning (e.g., baroreflex-sensitivity of heart rate control) have been previously identified, such measures were not available in the MIDUS. In addition, the majority of previous research examining the potential role of the ANS, PNS, or SNS in the development of behavioral problems has relied on measures tapping activity rather than functioning. With that said, future research would benefit from examining a wider range of indicators tapping various aspects of ANS functioning and activity. Fourth, and related, while overall catecholamine activity is related to SNS activity, catecholamines are released as a result of SNS activation and are, therefore, not directly tied to SNS activity. While other studies have used similar measures to tap SNS activity (de Vries-Bouw et al., 2012; McCaffery et al., 2000; Thoma et al., 2012), the distinction is worth noting and future research would benefit from examining how additional measures of SNS activity (e.g., salivary alpha-amylase) may moderate the findings reported in the current study.

In addition, the MIDUS includes a nationally representative sample of adults in the U.S. While this sample enjoys many strengths and provides insight into an important subset of the U.S. population, previous research has indicated that overall levels of antisocial behavior tend to peak during adolescence and decrease precipitously from early adulthood onward (Moffitt, 1993). In this way, overall variability in the employed anger measure is likely truncated in the examined sample, effectively increasing the likelihood of Type II error. Finally, the current study relied on a cross-sectional design, making it impossible to detect causal effects. The employed study design was focused on contemporaneous associations involving cardiac reactivity, catecholamine activity, and anger, as opposed to a longitudinal association that would allow for the examination of stability and change in such measures over time. Future research analyzing longitudinal data would provide greater insight into the role of cardiac reactivity and catecholamine activity on anger, while also providing the opportunity to more closely explore the underlying mechanisms driving such associations.

Despite these limitations, the findings of the current study contribute to a developing literature demonstrating the underlying complexity of the association between the ANS and behavior problems. While a large number of studies have demonstrated this association, much still remains unknown, including the potential roles of the PNS and SNS. The current study attempted to better specify the independent and interactive roles of indirect indicators of both subsystems on anger within a sample of U.S. adults. While the findings from the current study more directly implicate the role of catecholamine activity on the etiological development of anger, the findings also effectively demonstrate the importance of simultaneously examining the roles of both SNS and PNS activity in future studies. Each subsystem appears to be differently implicated in the development of specific forms of behavior problems, indicating that the etiological pathways connecting neurological functioning and physiological responses may vary across different forms of behavioral problems. Gaining a better understanding of the underlying processes and identifying differences would not only contribute to understanding the underlying physiological processes that contribute to behavioral variation, but may also provide additional insight into more effective and efficient forms of behavioral intervention.

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