

Linking Daily Stress Processes and Laboratory-Based Heart Rate Variability in a National Sample of Midlife and Older Adults

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ABSTRACT

Objective: This study evaluates the associations between people's trait-like patterns of stress in daily life (stressor frequency, perceived stressor severity, affective reactivity to stressors, and negative affect) and laboratory-assessed heart rate variability (HRV).

Methods: Data were collected from 909 participants aged 35 to 85 years in the Midlife in the United States Study. Participants reported negative affect and minor stressful events during telephone interviews on 8 consecutive evenings. On a separate occasion, HRV was measured from electrocardiograph recordings taken at rest during a laboratory-based psychophysiology protocol. Regression models were used to evaluate the associations between daily stress processes and three log-transformed HRV indices: standard deviation of R-R intervals (SDRR), root mean square of successive differences (RMSSD), and high-frequency power (high-frequency HRV [HF-HRV]). Analyses were adjusted for demographics, body mass index, comorbid conditions, medications, physical activity, and smoking.

Results: Stressor frequency was unrelated to HRV (r values ranging from -0.04 to -0.01 , p values $>.20$). However, people with greater perceived stressor severity had lower resting SDRR (fully adjusted B [standard error {SE}] = -0.05 [0.02]), RMSSD (-0.08 [0.03]), and HF-HRV (-0.16 [0.07]). Individuals with more pronounced affective reactivity to stressors also had lower levels of all three HRV indices (SDRR: B [SE] = -0.28 [0.14]; RMSSD: -0.44 [0.19]; HF-HRV: -0.96 [0.37]). Furthermore, aggregated daily negative affect was linked to reduced RMSSD (B [SE] = -0.16 [0.08]) and HF-HRV (-0.35 [0.15]).

Conclusions: In a national sample, individual differences in daily negative affect and responses to daily stressors were more strongly related to cardiovascular autonomic regulation than the frequency of such stressors.

Key words: stress, negative affect, heart rate variability, vagal regulation, daily diary, national study.

INTRODUCTION

Negative emotional states—such as stress, depression, and hostility—increase the risk of cardiovascular disease in healthy populations and predict secondary cardiac events and mortality in patients with existing cardiovascular disease (1–4). Despite abundant evidence linking emotional states to cardiovascular disease, the mechanisms remain poorly understood. Dysregulation of the autonomic nervous system has been proposed as a key pathway, due to its role in physiological arousal, emotion regulation (5), and its associations with other processes involved in the pathophysiology of cardiovascular disease (e.g.,

inflammation, platelet activation, and endothelial dysfunction (4,6,7)).

Heart rate variability (HRV), the variation in intervals between consecutive heart beats, is a noninvasive measure of cardiac autonomic regulation that reflects the capacity to respond to physical and environmental challenges. Low HRV, assessed with 24-hour ambulatory monitoring, poses an increased risk for mortality after myocardial infarction

ECG = electrocardiographic, **HF-HRV** = high-frequency heart rate variability, **HRV** = heart rate variability, **MIDUS** = Midlife in the United States Study, **RMSSD** = root mean square of successive differences, **SDRR** = standard deviation of R-R intervals

SDC Supplemental Content

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(8,9) and in patients with heart failure (10). Reduced time and frequency domain indices of HRV also predict future cardiac events (11) and mortality (12) in community-based samples. A number of studies have linked negative emotional states to reduced HRV (see Refs. (13–15) for reviews). For example, depression and trait negative affect are inversely associated with HRV obtained either in the laboratory (16,17) or by ambulatory monitoring (18,19), although these effects may differ based on medication use (20) and specific depressive symptoms (21). Furthermore, laboratory-based mental stress tasks elicit vagal withdrawal and increased sympathetic predominance (22,23). Given longstanding interest in the correspondence between stress responses in the laboratory versus in real life (24,25), research on stress and HRV may benefit from assessments of naturally occurring stressful experiences across differing contexts.

Existing studies on real-life stress and cardiac autonomic control have produced inconsistent results, possibly due to heterogeneous assessments of stress processes and the use of smaller, select samples (e.g., college students and cardiac patients). Studies that measured participants' general level of stress (i.e., asking participants to recall how stressed they felt over the past week or month) showed that individuals with greater perceptions of chronic and recent stress tended to have diminished vagally mediated high-frequency HRV in comparison to less stressed individuals (26,27), but not all studies reported this finding (28). Less research has been devoted to examining the potential associations of HRV with stress processes in daily life using naturalistic methods (e.g., daily diaries and ecological momentary assessments). Several studies on naturalistic daily stressful experiences suggest that increases in negative affect (29,30) or worry (31,32) are linked to concurrent ambulatory reductions in HRV. However, other studies have shown no associations of daily negative affect or stress with HRV (32,33) or have only found interactive effects that were moderated by other personality or mental health characteristics (34,35).

Whether the repeated stressful demands of day-to-day life are related to impairments in autonomic regulation is unclear. The objective of the current investigation was to link individual differences in daily stress processes with laboratory-based resting HRV in a national sample of 909 midlife and older adults. We used daily diary methods to capture four aspects of daily stress as they unfolded, namely, *stressor frequency*, *perceived stressor severity*, *affective reactivity*, and *daily negative affect*. These daily stress measures were conceptualized as an individual's trait-like patterns of exposure, perceptions, and reactivity to minor stressful events in everyday life. Emotional reactivity to daily stressors is stable during middle adulthood (yet can vary based on psychosocial contexts; 36) and increases the risks of subsequent psychological distress, chronic conditions, and mortality (37–40). On the other hand, the

relationship between stressor exposure (i.e., frequency of daily stressors) and health is less clear-cut because individuals with higher socioeconomic status tend to encounter more daily stressors (41). We therefore hypothesized that stressor frequency will be unrelated to laboratory-based measures of resting HRV, whereas subjective aspects of daily stress will be associated with diminished HRV. Specifically, people with more pronounced reactions to daily stressors, greater stressor severity, and elevated negative affect were expected to have lower levels of resting HRV, compared with people who were better able to handle the challenges of everyday life.

METHODS

Participants

This study uses cross-sectional data from the second wave of the Midlife in the United States Study (MIDUS II), a national survey of psychological and social factors accounting for age-related variations in health and well-being. MIDUS II consisted of 4963 noninstitutionalized, English-speaking respondents aged 35 to 85 years; an additional 592 African Americans from Milwaukee were recruited to increase the diversity of the study.

Data for the current study were drawn from two separate MIDUS sub-projects spanning from 2004 to 2009: the National Study of Daily Experiences and the Biomarker Project. A representative subset of MIDUS II respondents ($n = 2022$) participated in the National Study of Daily Experiences, a daily diary study consisting of short telephone interviews about daily experiences for 8 consecutive evenings (42). Of these, 928 respondents had HRV data obtained from a laboratory-based psychophysiology protocol in the Biomarker Project. Nineteen participants were excluded due to missing covariate data on household income. The current analyses were conducted on a final sample of 909 participants, including 128 African Americans from the Milwaukee cohort. Compared with the other 1113 daily diary participants who did not have HRV or covariate data, the final sample of 909 participants did not significantly differ in sex, number of daily interviews completed, daily negative affect, perceived stressor severity, or affective reactivity to stressors. However, participants in the current analyses were younger than the rest of the daily diary sample (mean age of 55 years versus 57 years, respectively, at entry in MIDUS II; $p < .001$) and experienced stressors more frequently (stressors on 42% of days versus 37% of days, respectively; $p < .001$). The order and timing of data collection varied among participants, with 569 participants (62.6%) completing the Biomarker Project first and 340 (37.4%) completing the daily diaries first. Assessments for the Biomarker Project and the daily diary study were separated by a median of 6 months (mean [standard deviation {SD}] = 14.5[10.8] months). Procedures were approved by institutional review boards at participating institutions, and all participants provided informed consent.

Procedure and Measures

Daily Diary Assessments

This study examined four predictor variables—stressor frequency, stressor severity, affective reactivity, and daily negative affect—that were obtained from telephone interviews for 8 consecutive evenings. Daily stressors were assessed using the Daily Inventory of Stressful Events, which consists of stem questions asking whether seven types of daily stressors occurred in the past 24 hours: argument, avoided an argument, stressful event at work or school, stressful event at home, discrimination, network stressor (i.e., stressful event that happened to a close friend or relative), and any other stressful event (43). Days on which at least one stressor occurred were

coded as “stressor days.” Most stressor days (74%) contained only one stressor. Thus, *stressor frequency* was defined as the percent of stressor days, based on the number of daily interviews completed (e.g., a person who experienced stressors on 4 of 8 days had a stressor frequency of 50%). Results were comparable to those reported when the total number of stressors, divided by number of days, was entered as a predictor. Participants also rated how stressful each event was, using a 4-point scale: 0 = *not at all*, 1 = *not very*, 2 = *somewhat*, 3 = *very*. *Perceived stressor severity* was calculated by averaging the ratings for all stressors within the day, and then aggregating scores across the 8 interview days. Stressor severity ratings were not available for 64 participants who did not experience any stressors during the study.

Daily negative affect was assessed using scales developed for the MIDUS study (44,45). Participants reported the frequency of negative emotions using a 5-point scale: 0 = *none of the time*, 1 = *a little of the time*, 2 = *some of the time*, 3 = *most of the time*, 4 = *all of the time*. The scale consisted of 14 items: restless or fidgety, nervous, worthless, so sad nothing could cheer you up, everything was an effort, hopeless, lonely, afraid, jittery, irritable, ashamed, upset, angry, and frustrated. Daily negative affect was calculated by averaging the respective items, and then aggregating scores across all interview days. Cronbach α ranged from .84 to .88 each day.

Affective reactivity was defined as the within-person change in negative affect on days when stressors occurred, compared with one's typical negative affect on nonstressor days. Following procedures established in other daily stress studies (38,46), affective reactivity scores were computed for each participant using a two-level multilevel model in which the occurrence of a daily stressor (yes/no) was entered as a predictor of negative affect on day d for person i :

Level 1 (day-level): Negative Affect $_{di} = a_{0i} + a_{1i}(\text{Stressor Day}_{di}) + e_{di}$

Level 2 (person-level): $a_{0i} = \beta_{00} + U_{0i}$

$a_{1i} = \beta_{10} + U_{1i}$

At Level 1, a_{0i} is the intercept representing negative affect on nonstressor days, a_{1i} is the slope representing person i 's change in negative affect on stressor days, and e_{di} is the residual representing day-to-day variability in negative affect for person i . At Level 2, β_{00} and β_{10} represent the sample average levels of negative affect and affective reactivity, respectively, and u_{0i} and u_{1i} are the variances reflecting person i 's deviations from the sample average levels of negative affect and affective reactivity. To calculate each person's affective reactivity slope, his or her deviation was outputted from the multilevel model and added to the sample fixed effect for affective reactivity. These reactivity slopes were subsequently entered as predictors of HRV in linear regression models for the primary analyses (37,39,40). For example, a person with an affective reactivity slope of 0.17 (the sample mean) had an increase of 0.17 in negative affect on stressor days, relative to nonstressor days. The calculation of affective reactivity requires that a person have both stressor and nonstressor days. Thus, affective reactivity could not be computed for 102 participants (11% of sample) because 63 did not experience any stressors and 39 had stressors every day.

Psychophysiology Protocol

The Biomarker Project required an overnight stay at one of three general clinical research centers (University of California, Los Angeles; Georgetown University; and the University of Wisconsin-Madison), where participants completed a detailed assessment of physical health and a laboratory-based psychophysiology protocol (47). Details on the psychophysiology protocol are published elsewhere (48,49). Briefly, the protocol took place in the morning after a light breakfast with no caffeinated beverages. Electrocardiographic (ECG) electrodes were placed on the left and right shoulders

and in the left lower quadrant. The protocol consisted of an 11-minute seated baseline period, followed by mental stress tasks and corresponding recovery periods. For the current analyses, we used HRV data from the resting baseline period.

Analog ECG signals were digitized at 500 Hz by a 16-bit National Instruments A/D Board and passed to a microcomputer for collection. The ECG waveform was submitted to an R-wave detection routine implemented by proprietary event detection software, resulting in an R-R interval time series. Errors in marking of R-waves were corrected interactively following established procedures (50).

The SD of the R-R interval (SDRR), root mean squared of successive differences (RMSSD), and spectral power in the high-frequency bands (0.15–0.50 Hz; high-frequency HRV[HF-HRV]) were computed from 5-minute epochs using an interval method for computing Fourier transforms (51). Before computing Fourier transforms, the mean of the R-R interval series was subtracted from each value in the series, the series was filtered using a Hanning window (52), and the power (in ms^2) over the high-frequency bands was summed. Estimates of spectral power were adjusted to account for attenuation produced by the filter (52). HRV was computed as the mean of the values from the two baseline 5-minute epochs. HRV variables were natural log-transformed to normalize the distributions.

Covariates

Demographic covariates for age, sex, race (white versus nonwhite), and household income quintile were obtained by a telephone survey as part of the parent MIDUS Study. During the clinic visit for the Biomarker Project, medical comorbidity was assessed using a checklist of 20 physician-diagnosed chronic conditions (e.g., heart disease, high blood pressure, asthma, diabetes, depression); the total number of chronic conditions was included in the analyses as a continuous variable. Height and weight were measured in the clinic and used to calculate body mass index. Dummy-coded variables were included to control for the use of blood pressure, cholesterol-lowering, and corticosteroid medications. Physical activity was assessed with an item asking whether the participant engaged in physical activity for 20 minutes or more at least 3 times per week. Two dummy-coded variables were created to control for current smoking and past smoking, with never smoked as the reference group. In an alternative analysis, average minutes of vigorous physical activity each day ($n = 909$) and daily cigarettes smoked ($n = 895$) from the daily diary interviews were entered as covariates, instead of the physical activity and smoking status measures collected from the clinic visit. The time interval in months between the daily diary and psychophysiology protocol was calculated by subtracting the date of the HRV assessment from the date of the first daily diary interview; positive values refer to completion of the daily diary first.

Data Analysis

For descriptive purposes, we examined correlations between HRV (ln-transformed SDRR, RMSSD, and HF-HRV power) and all other variables. For the primary analyses, separate linear regression models were run to test each daily stress variable as a predictor of HRV indices. All covariates were entered in the first step, followed by the predictor in the second step. The covariates included the time interval between assessments, as well as factors that were related to HRV in prior research: demographics (age, sex, race, income), physical health (body mass index, number of comorbid conditions), medication use (cholesterol-lowering, blood pressure, and corticosteroid medications), and health behaviors (physical activity, smoking status). Continuous variables were centered at the sample mean, except that the time interval between assessments was centered at zero to indicate no lag. Interactions were tested among the daily stress variables, as well as between daily stress measures and

demographics. Analyses were conducted using SAS version 9.4 (SAS Institute Inc, Cary, NC).

RESULTS

Sample Characteristics

Participants completed an average (SD) of 7.43 (1.20) of 8 possible daily interviews, for a total of 6754 daily

interviews across the entire sample. Table 1 describes the sample of 909 participants. Participants were, on average, 57 years old at the time of the HRV assessment. Most were female (57%), and the median household income was \$59,500. The sample was primarily white (80.6%); 141 participants (15.5%) were African American/black, 12 (1.3%) were Native American or Alaskan Native, and 24 (2.6%)

TABLE 1. Sample Characteristics and Correlations With Heart Rate Variability ($n = 909$)

Variable	M (SD) or n (%)	Correlations With In-Transformed HRV		
		SDRR	RMSSD	HF-HRV
Demographics				
Age, y	57.39 (11.34)	-0.32***	-0.21***	-0.28***
Male	391 (43.01%)	0.06 [†]	-0.01	-0.06 [†]
White race (versus nonwhite)	733 (80.64%)	-0.07*	-0.19***	-0.22***
Household income, median (Q1–Q3)	\$59,500 (\$30,650–\$96,250)	0.06 [†]	-0.05	-0.03
Physical health covariates				
Body mass index, kg/m ²	29.63 (6.40)	-0.07*	-0.00	0.01
No. comorbid conditions	4.02 (2.91)	-0.22***	-0.10**	-0.12***
Medication use				
Cholesterol-lowering	252 (27.72%)	-0.18***	-0.12***	-0.15***
Corticosteroid	37 (4.07%)	-0.07*	-0.06 [†]	-0.05
Blood pressure	316 (34.76%)	-0.19***	-0.03	-0.05
Health behaviors from clinic visit				
Physical activity, 20+ min 3×/wk	702 (77.23%)	0.03	-0.02	0.007
Cigarette smoking status				
Never smoked	483 (53.14%)	0.04	-0.02	0.00
Former smoker	301 (33.11%)	-0.12***	-0.08*	-0.10**
Current smoker	125 (13.75%)	0.11***	0.14***	0.14***
Health behaviors from daily interviews				
Daily physical activity, min	41.32 (57.64)	0.05	0.02	0.01
Daily cigarettes smoked ($n = 895$)	1.74 (5.19)	0.06 [†]	0.09**	0.10**
Daily stress processes^a				
Daily negative affect (range, 0–4)	0.21 (0.27)	-0.06 [†]	-0.06 [†]	-0.07*
Stressor frequency (% stressor days)	42.48% (26.35%)	-0.01	-0.04	-0.04
Stressor severity ($n = 846$) ^b	1.74 (0.65)	-0.10**	-0.09*	-0.08*
Affective reactivity ($n = 807$) ^c	0.17 (0.12)	-0.08*	-0.07*	-0.08*
Heart rate variability (ln-transformed)				
SDRR, ms	3.46 (0.47)	—	0.83***	0.80***
RMSSD, ms	2.91 (0.63)	—	—	0.96***
High frequency (0.15–0.50 Hz), ms ²	4.89 (1.29)	—	—	—

M = mean; SD = standard deviation; SDRR = standard deviation of R-R intervals; RMSSD = root mean square of successive differences; HF-HRV = high-frequency heart rate variability.

* $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$, [†] $p \leq .10$.

^a Correlations between daily stress processes and HRV were partialled for age, income, and race, due to strong confounding relationships of demographics with stress and HRV.

^b Participants rated how stressful each event was, using a 0 (not at all) to 3 (very) scale. Seven percent of the sample ($n = 63$) did not experience a stressor during the 8 days of interviews.

^c Affective reactivity was defined as the change in negative affect on a stressor day, compared with a nonstressor day. Affective reactivity was not computed for 102 participants (63 did not have a stressor; 39 had stressors every day).

were of another race. Participants had an average of four medical conditions, and the mean body mass index of 29.6 kg/m² was close to the standard cutoff of 30 kg/m² for obesity. More than half of participants had never smoked cigarettes regularly, and 77% engaged in 20 minutes of physical activity at least 3 times per week. On average, participants reported having at least one stressful experience on 42% of interview days (range, 0%–100%), and these experiences were self-rated “somewhat” stressful. Daily negative affect was low and increased by a mean of 0.17 on stressor days.

Correlations

The daily stress variables were significantly and positively correlated with each other and ranged in magnitude from $r=0.16$ to $r=0.91$ (p values $<.001$; see Table S1, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A269>). For example, compared with participants with lower affective reactivity, those who experienced greater affective reactivity to stressors were also more likely to have higher daily negative affect ($r = 0.91$), to encounter stressors more frequently ($r = 0.21$), and to perceive their stressors as more severe ($r = 0.34$).

As shown in Table 1, natural log-transformed SDRR, RMSSD, and HF-HRV power were strongly, positively associated with one another (p values $<.001$). In correlations that partialled out the effects of demographics (age, race, and income), people who had greater stressor severity and affective reactivity tended to have lower levels of all three indices of HRV, whereas the frequency of stressors was not related to HRV. Higher daily negative affect, aggregated across interview days, was significantly correlated with lower HF-HRV power and marginally correlated with lower SDRR and lower RMSSD. Bivariate correlations between covariates and HRV showed that older age, white race, higher comorbidity, use of cholesterol-lowering medications, and former smoking were associated with lower HRV. Current smokers had higher HRV, although these correlations were largely explained by age and race in multivariate analyses. There were no associations between self-reported physical activity and HRV. Health behaviors assessed by daily diary showed the same pattern, such that the average number of daily cigarettes was correlated with higher RMSSD and HF-HRV, and the average minutes of daily physical activity was unrelated to HRV.

Regression Models of Daily Stress Constructs as Predictors of HRV

Table 2 and Figure 1 show the results of separate regression models for each daily stress predictor, adjusted for demographics, body mass index, comorbid conditions, medication use, health behaviors, and the time interval between assessments. Participants who experienced more negative affect on a daily basis tended to have lower RMSSD and

lower HF-HRV power, both indices of cardiac vagal modulation; daily negative affect was not associated with SDRR, a more global HRV index influenced by both the vagal and sympathetic systems. The frequency of daily stressors—defined as the percent of days in which a stressor occurred—was not related to any index of HRV. However, subjective responses to stressors were consequential for HRV. Participants' ratings of stressor severity and their affective reactivity to stressors (i.e., increases in negative affect on stressor days, compared with nonstressor days) were associated with lower levels of all three laboratory-based HRV indices. The daily stress variables did not interact with one another, nor did they interact with age, sex, race, or income to predict any measure of HRV.

DISCUSSION

Negative emotional states and psychological distress are known to be inversely related to HRV, but evidence delineating the roles of naturalistic daily stress and affect in HRV remains unclear. The purpose of this study was to evaluate the associations between people's patterns of stress responses in daily life and laboratory measures of HRV (SDRR, RMSSD, and HF-HRV power) in a national sample of midlife and older adults. We found that stressor exposure (i.e., frequency of daily stressors) was not associated with HRV. In contrast, individuals who perceived their stressors to be more severe or who experienced greater increases in negative affect when faced with stressors tended to have lower levels of all three HRV indices, compared with those with less pronounced stressor severity or affective reactivity. People who had higher aggregated daily negative affect were also relatively more likely to have reduced RMSSD and HF-HRV power. These results suggest that exposure to daily stressors may be less important for cardiac autonomic control than how people perceive and respond to those stressors, as well as their overall daily experiences of negative affect.

Several prior studies have examined associations of HRV with daily negative affect and stressors. For example, negative emotions reported throughout the day were associated with transient decreases in ambulatory ECG-assessed HRV in 33 healthy adults (30) and in 135 patients with coronary heart disease (29). Our study supports and extends these previous findings due to the wide range of stressors reported in daily life (spanning work, home, and interpersonal domains), a consistent pattern of results across multiple indices of HRV, and the larger, representative sample of American adults.

Yet, our results differed from some prior findings in the literature on psychological stress reactions and HRV. A study of 38 university students reported no main effects between laboratory measures of resting HRV and problematic daily outcomes (e.g., stressors specific to college students, negative affect, and rumination in response to stressors)

TABLE 2. Daily Stress Constructs as Predictors of Heart Rate Variability

Variables	<i>n</i>	<i>B</i> (SE) for In-Transformed Heart Rate Variability Outcomes		
		SDRR (ms)	RMSSD (ms)	HF-HRV (ms ²)
Step 1: Covariates (entered simultaneously)				
Intercept	909	3.534 (0.052)***	3.147 (0.072)***	5.403 (0.143)***
Age		−0.010 (0.002)***	−0.010 (0.002)***	−0.027 (0.004)***
Sex (reference: male)		0.081 (0.030)**	0.023 (0.042)	−0.060 (0.083)
White race (versus nonwhite)		−0.090 (0.040)*	−0.269 (0.055)***	−0.607 (0.110)***
Household income quintile		−0.001 (0.011)	−0.018 (0.015)	−0.029 (0.031)
Body mass index		−0.005 (0.002)*	−0.005 (0.003)	−0.008 (0.007)
No. comorbid conditions		−0.012 (0.006)*	−0.011 (0.008)	−0.020 (0.016)
Cholesterol medication		−0.082 (0.036)*	−0.089 (0.050)†	−0.185 (0.099)†
Corticosteroid medication		−0.158 (0.075)*	−0.233 (0.103)*	−0.450 (0.205)*
Blood pressure medication		−0.034 (0.037)	0.094 (0.050)†	0.180 (0.100)†
Physical activity		0.004 (0.036)	−0.001 (0.049)	0.0645 (0.098)
Smoking status				
Current smoker		0.064 (0.046)	0.148 (0.063)*	0.282 (0.125)*
Past smoker		−0.035 (0.033)	−0.026 (0.046)	−0.073 (0.091)
Never smoked		Reference	Reference	Reference
Time interval between assessments		0.001 (0.001)	0.001 (0.001)	0.003 (0.002)
Step 1 <i>R</i> ²	909	0.148	0.098	0.139
Step 1 <i>R</i> ²	846	0.151	0.097	0.134
Step 1 <i>R</i> ²	807	0.147	0.090	0.124
Step 2: Daily stress predictors (separate models for each predictor)				
Daily negative affect	909	−0.070 (0.057)	−0.157 (0.078)*	−0.354 (0.155)*
Step 2 <i>R</i> ²		0.149	0.102	0.144
Stressor frequency (% stressor days)	909	0.031 (0.058)	−0.054 (0.080)	−0.143 (0.159)
Step 2 <i>R</i> ²		0.146	0.098	0.140
Stressor severity	846	−0.051 (0.024)*	−0.080 (0.033)*	−0.164 (0.067)*
Step 2 <i>R</i> ²		0.155	0.103	0.140
Affective reactivity	807	−0.279 (0.136)*	−0.439 (0.187)*	−0.958 (0.371)**
Step 2 <i>R</i> ²		0.152	0.096	0.152

SE = standard error; SDRR = standard deviation of R-R intervals; RMSSD = root mean square of successive differences; HF-HRV = high-frequency heart rate variability.

*R*² differs based on the sample size. The sample sizes correspond to analyses for daily negative affect and stressor frequency (*n* = 909), stressor severity (*n* = 846), and affective reactivity (*n* = 807).

* *p* ≤ .05, ** *p* ≤ .01, *** *p* ≤ .001, † *p* ≤ .10.

(35). Also, a study of 117 healthy young adults found that momentary positive affect predicted ambulatory HRV, but negative affect did not (53). Our findings likely differed from these previous studies due to differences in the nature of the stressors, samples, and methodology, such as the covariates and negative emotions assessed. Alternatively, because negative affect and stressor exposure tend to decrease with age (54), it is possible that higher levels of negative affect or pronounced reactivity to stressors are more detrimental to health in mid-to-late adulthood than in adolescence or early adulthood.

The current study highlights the importance of examining reactions to naturalistic stress—including affective, cognitive, and behavioral responses—to understand their implications for cardiac autonomic control and, more generally, for physical and mental well-being. Cognitive representations of stress (e.g., rumination, worry, and anticipatory stress) may amplify and prolong physiological activation to stressors. For example, a previous study found that momentary assessments of worry episodes and stressors were concurrently associated with higher ambulatory heart rate and lower ambulatory RMSSD (31). The cardiac effects

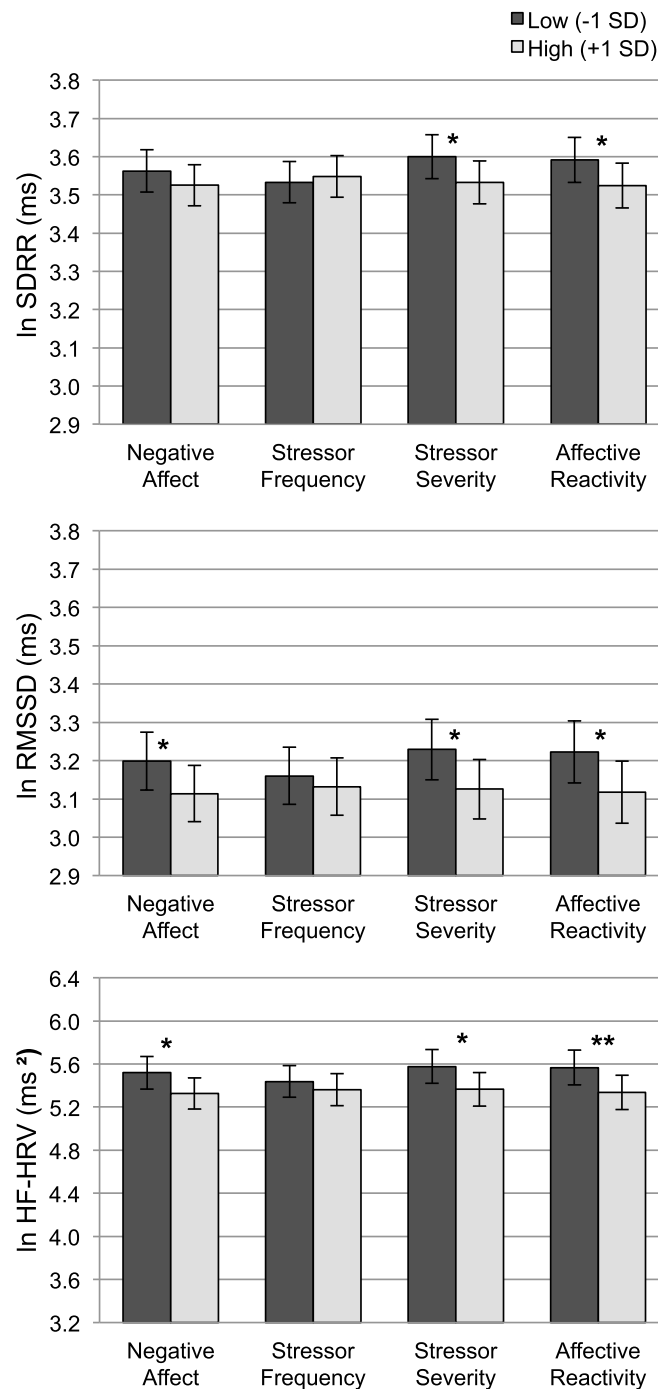


FIGURE 1. Associations between daily stress variables and natural log-transformed HRV outcomes, controlling for all covariates. Daily stress variables were depicted at -1 and $+1$ SD from the means, with standard error bars. HRV = heart rate variability; SD = standard deviation. SDRR = standard deviation of R-R intervals; RMSSD = root mean square of successive differences; HF-HRV = high-frequency heart rate variability. * $p \leq .05$, ** $p \leq .01$.

of stressors were not significant after accounting for worry, suggesting that cognitive stress responses may have prolonged physiological effects beyond the actual occurrence of a stressor (32,55). Similarly, our study demonstrated that people with greater affective reactivity to daily stressors,

rather than exposure to stressors per se, tended to have lower levels of all three indices of resting HRV in the laboratory. Accumulating evidence suggests that affective reactivity to stressors in daily life is concurrently associated with poor sleep (56) and inflammation (57), as well

as future risk of affective disorders (37), health conditions (40), and mortality (39). Our findings add to this literature by proposing autonomic dysregulation as a possible pathway that links daily stress processes to long-term health outcomes. Although we did not collect data on behavioral coping responses to daily stressors (such as actively addressing the stressful situation or seeking emotional support), the role of coping strategies in cardiac autonomic control is a potentially fruitful area for future research.

Several intriguing findings emerged regarding race, life-style factors, and HRV. Consistent with other studies, white participants in our sample had lower HRV than African American/black participants (for recent review, see Ref. (58)). Given the greater burden of cardiovascular disease-related mortality among African Americans (59), these paradoxical results support the notion that racial groups may have differential patterns of risk factors (58) and that other aspects of autonomic regulation besides resting HRV (such as HRV reactivity to stress tasks (60)) should also be considered when investigating health disparities. In addition, our analyses showed that current smokers had higher HRV. Because younger and black participants were relatively more likely to smoke and to have higher HRV, the effect of smoking was mostly (but not entirely) explained by demographic factors. Previous studies have shown that cigarette smoking is associated with reduced HRV, but much of this research is based on small samples of healthy young adults (61,62). In line with our results, however, an analysis of more than 6800 adults in the Multi-Ethnic Study of Atherosclerosis showed that current smokers had marginally higher HRV than did nonsmokers (17). Also unexpected was the lack of association between physical activity and HRV, yet an epidemiological study of middle-aged adults reported a link between vigorous physical activity and higher HRV in men only, but found no effects in women or for moderate physical activity (63). Additional work using population-based samples and more refined assessments of life-style factors are needed to understand how daily health behaviors relate to HRV.

Limitations and Future Directions

Study limitations should be considered when interpreting these findings. First, the cross-sectional, observational design of the study does not allow us to draw conclusions about causality or the direction of association. Perceptions of and reactions to stressful events in daily life may pile up over time to influence autonomic function, yet it is also possible that HRV is a marker of emotion regulatory ability (5).

Second, our measure of basal HRV was obtained in a controlled laboratory setting separately from the daily diary interviews. Although this is a valid and accurate assessment of resting HRV, we do not have data on physiological responses during the stressful moments. Because the interval

between the daily diary and HRV assessments (spanning a median of 6 months and mean of more than 14 months), the daily stress measures were conceptualized as trait-like characteristics. We were unable to examine within-person covariation in daily stress processes with HRV. Ecological momentary assessments of affect and stress processes, coupled with ambulatory ECG monitoring, may be ideal for capturing stress responses as they unfold in real time, provided that confounding factors (e.g., posture, speaking, and substance use) are controlled.

Third, because affect was measured at the end of each day, it was unclear whether affective reactivity truly reflected concurrent emotional responses to stressors, or if it represented prolonged emotional activation or slower emotional recovery. Our end-of-day assessment of stressors may have lacked sensitivity for capturing very minor, transient stressors. In addition, it is possible that 8 days was long enough to assess an individual's typical stress responses and negative affect, but too short for capturing one's typical exposure to stressors. Future studies should assess affect and stress throughout the day to disentangle the time-course of stress processes and to provide a more nuanced portrayal of the anticipatory, reactivity, and recovery phases of stress responses. Despite these limitations, this study has notable strengths that include a nationally representative sample of US adults, multiple indicators of HRV and of daily stress, and careful adjustment for demographic, physical, and behavioral confounding factors.

CONCLUSIONS

In summary, this study indicates that adults who exhibited greater subjective stress in daily life—specifically, negative affect, perceived stressor severity, and affective reactivity to stressors—had lower levels of resting laboratory-based HRV, compared with those who were better equipped to handle day-to-day challenges. By contrast, the frequency of daily stressors was unrelated to HRV. These findings provide support for autonomic dysregulation as a potential pathway whereby the perceived demands of everyday life pile up to influence morbidity and mortality. Although hassles and disruptions are common and often unavoidable, how a person responds to these seemingly minor stressors is important for cardiovascular health.

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