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Individual Differences in the Association Between Subjective Stress and Heart Rate Are Related to Psychological and Physical Well-Being

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Abstract

The physiological response to stress is intertwined with, but distinct from, the subjective feeling of stress, although both systems must work in concert to enable adaptive responses. We investigated 1,065 participants from the Midlife in the United States 2 study who completed a self-report battery and a stress-induction procedure while physiological and self-report measures of stress were recorded. Individual differences in the association between heart rate and self-reported stress were analyzed in relation to measures that reflect psychological well-being (self-report measures of well-being, anxiety, depression), denial coping, and physical well-being (proinflammatory biomarkers interleukin-6 and C-reactive protein). The within-participants association between heart rate and self-reported stress was significantly related to higher psychological well-being, fewer depressive symptoms, lower trait anxiety, less use of denial coping, and lower levels of proinflammatory biomarkers. Our results highlight the importance of studying individual differences in coherence between physiological measures and subjective mental states in relation to well-being.

Keywords

subjective stress, physiology, well-being, coherence, open materials

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In response to stress, both physiological responses and experiential feeling states occur. Under acute stress, we may experience sweaty palms, a racing pulse, and shallow breathing but also a subjective mental state, represented as perceived stress. The functional significance of the association between these two response systems has received scant attention, although an older literature exists on the maladaptive consequences of decoupling between the physiological and experiential streams, particularly when physiology is responding in the direction of increased stress while experiential reports contain little or no representation of the stressful signature expressed in the physiology (Weinberger, Schwartz, & Davidson, 1979).

Many theories propose that emotional responses involve coordinated interactions across subjective experience, physiology, and behavior, in service of adaptive functioning (Darwin, 1872/2009; Ekman, 1992; Lang, 1988; Lazarus, 1991; Levenson, 1994; Plutchik, 1980). Yet the empirical evidence largely suggests a lack of coherence across response systems (for a review of studies of the relationship between subjective stress and biological markers in the context of the Trier Social Stress Test, see Campbell & Ehlert, 2012). Although it has been demonstrated that there is significant variability in this coherence across individuals (Sze, Gyurak, Yuan, & Levenson, 2010), few studies have examined these individual differences. In the studies that have, coherence has been shown to be related to differences in externalizing and

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internalizing problems (Hastings et al., 2009), attachment style (Ditzen et al., 2008), phase of menstrual cycle (Olson, 2006), gender (Avero & Calvo, 1999), and training in meditation and dance (Sze et al., 2010). No studies have examined whether coherence is related broadly to adaptive functioning. In the present study, the degree to which individuals' self-reports of their subjective stress experience is associated with their heart rate across phases of a stress-induction paradigm is a key measure of interest. We refer to this within-participants measure as *stress–heart rate coherence*. We examined the relationship between stress–heart rate coherence and multiple measures of psychological and physical well-being, as well as denial coping for construct validity.

Why Stress?

Stress reliably activates the sympathetic nervous system and thus modifies measurable physiological indicators, and we would expect variation in the degree of subjective stress that individuals experience. In response to perceived stressors, the brain initiates a physiological response preparing the animal to fight or flee the cause of stress. Through cascades of neurotransmitters, including norepinephrine and corticotropin-releasing hormone, stress activates the sympathetic nervous system, and its effects course through the body, accelerating heart and lung action, increasing blood pressure through constriction of blood vessels, and constricting pupils, while also increasing arousal and alertness, promoting vigilance, and focusing attention through more direct actions on the central nervous system (Chrousos, 2009).

Functional Significance

The extent to which self-reported experience mirrors physiology may have important functional significance. Weak stress–heart rate coherence reveals a disconnection between the state of the body and the mental state. Weak stress–heart rate coherence may be evident in individuals who lack awareness of their own mental states, have limited ability to appropriately label their mental states (as in alexithymia), or have a tendency to deny or suppress their feelings. In particular, denial coping, which is a tendency to cope with stress by denying the reality of a stressor or avoiding beliefs that the stressor exists (Carver, Scheier, & Weintraub, 1989), is likely to be tied to low stress–heart rate coherence.

Additionally, identification of mental states that correspond strongly with concomitant physiology may decrease the degree to which these states bias the perception of other unrelated stimuli. For example, Lapate, Rokers, Li, and Davidson (2014) demonstrated that physiological arousal in response to a previously presented fearful stimulus biased the evaluation of novel neutral faces only when that fearful stimulus was presented outside of awareness. When subjects are aware that they have seen a fearful stimulus, they may be better able to accurately ascribe their physiological arousal to its source and thus evaluate subsequently presented neutral faces with less bias. Even when subjects are aware of stimuli, they may not be aware of how stimuli influence their own mind and physiology. For example, Grupe et al. (2018) demonstrated that affective coloring of neutral stimuli by preceding emotional stimuli depends on individual differences in affective style. Awareness of ties between physiology and subjective experience may reduce affective bias to provide a more accurate depiction of reality and thus inform more adaptive responses to it.

Over time, patterns of effectively coping with stress and preventing its spillover to subsequent events should benefit psychological and physical well-being. Although the acute stress response is theorized to have evolved as an adaptive response, when it is ineffectively regulated and sustained, there can be negative consequences on behavior and physical health (Miller, Cohen, & Ritchey, 2002). Chronic stress has been linked to depression and anxiety (Chiba et al., 2012). These disorders are often associated with negative biases (Gotlib & Joormann, 2010), which may be a result of a disconnection between subjective experience and physiology. Chronic stress also affects the immune system by impairing effective termination of inflammatory responses. Levels of the proinflammatory cytokines interleukin-6 (IL-6) and C-reactive protein (CRP) are commonly found to be elevated in the context of chronic stress and are believed to be markers of chronic systemic inflammation. Stress–heart rate coherence has the potential to benefit psychological and physical well-being by contributing to a more accurate perception of the environment and more successful coping. Over time, these processes may buffer against negative consequences of chronic stress by facilitating efficient recovery from stress responses and preventing the initiation of additional stress responses to unrelated subsequent events that may otherwise be interpreted negatively through affective coloring.

The current study's guiding hypothesis was that greater stress–heart rate coherence would be associated with greater psychological and physical well-being. Psychological well-being was indexed by higher scores on a standardized scale of well-being as well as fewer depressive symptoms and lower trait anxiety. Physical well-being was indexed by lower levels of proinflammatory biomarkers IL-6 and CRP in blood plasma. We also examined the relationship between stress–heart

Table 1. Sample Characteristics

Variable	Value
Sample size (*N*)	1,065
Female	*n* = 610 (57.2%)
Age in years (at stress induction)	*M* = 56.4 (*SD* = 11.21), range = 35–86
Months between survey and stress induction	*M* = 25.89 (*SD* = 14.19), range = 0–62
Race	
Asian	*n* = 3 (0.2%)
Black	*n* = 193 (18.1%)
Native American or Alaska Native Aleutian Islander/Eskimo	*n* = 14 (1.3%)
White	*n* = 825 (77.5%)
Other	*n* = 27 (2.5%)
Don't know, refused to report, and missing	*n* = 3
MIDUS subsample	
Main	*n* = 521 (48.9%)
Sibling	*n* = 6 (0.5%)
Twin	*n* = 337 (31.9%)
City oversample	*n* = 19 (1.8%)
Milwaukee	*n* = 182 (17.2%)
Twins	
Twin pairs	*N* = 118 pairs (2 pairs from one family)
Monozygotic	*n* = 64 pairs
Dizygotic same sex	*n* = 28 pairs
Dizygotic different sex	*n* = 23 pairs
Unable to determine zygosity	*n* = 1 pair
Twin singletons	*N* = 101 (cotwin not in subsample for this analysis)
Nontwin siblings	
Three siblings	*n* = 3 (same family)
Two siblings	*n* = 8 (four families)

Note: MIDUS = Midlife in the United States.

rate coherence and denial coping to expand the nomological network of stress–heart rate coherence because we believed that denial coping would lead to a disconnection between physiology and subjective reports, to the degree that subjective reports deny the existence of a mental state.

Method

Participants

Data were collected from 2004 to 2009 as part of the second wave of the Midlife in the United States (MIDUS) study, a national longitudinal study of health and well-being (www.midus.wisc.edu). Participants completed surveys (*N* = 4,963), and a subsample participated in a biomarker project that included a stress-induction session (*n* = 1,255). The sample size for the current study was predetermined by existing MIDUS data and included all participants with sufficient data on the measures of interest. Participants without five complete and valid data points for self-reported stress were excluded from the analyses. The final total sample for the present study was 1,065, which is adequate to detect even small effects. Demographic statistics for the sample are provided in Table 1.

Briefly, participants were between the ages of 35 and 86 years (*M* = 56 years, *SD* = 11) at the time of the stress-induction substudy, and 57.2% were female (*n* = 610). Overall, the sample was predominantly White (77.5%), and a significant percentage (18.1%) was African American. The sample included 118 twin pairs and 11 nontwin siblings (one family with 3 siblings, and four families with 2 siblings). Because siblings present a source of nonindependence in the data, we adjusted for family membership in our models. Participants completed the biomarker substudy between 0 and 62 months (*M* = 25.9 months, *SD* = 14.19) following the survey study. Part II in Supplemental Results (see the Supplemental Material available online) includes a description of analyses investigating the impact of this lag between the two studies on the results. Lag did not significantly moderate results nor did adjusting for lag impact the significance of any findings.

Procedure

Participants completed a standardized laboratory-based experimental stress-induction paradigm designed to measure cardiovascular reactivity and recovery from stress (Crowley et al., 2011; Love, Seeman, Weinstein, & Ryff, 2010; Shcheslavskaya et al., 2010; detailed documentation of the study protocol is publicly available at http://www.midus.wisc.edu/midus2/project4/). The data were collected at the University of California, Los Angeles; Georgetown University; and the University of Wisconsin and processed at the Columbia University Medical Center in the laboratory of Richard Sloan. Figure 1 depicts the distribution of heart rate and self-reported stress levels across the course of the stress-induction paradigm.

The stress-induction paradigm involved a resting baseline (11 min); two cognitive-psychological stressor tasks (6 min each; counterbalanced across participants); a seated, resting period after each task (recovery period; 6 min each); and an orthostatic challenge, which involved moving from a seated to a standing position and remaining standing (6 min). The orthostatic phase of the task was not included in the analyses because changes in heart rate during this phase are confounded with physical movement. Thus, we examined five phases of interest: baseline, first stressor task, first recovery, second stressor task, and second recovery.

Participants' heart rate was measured using electrocardiograph electrodes placed on the left and right shoulders and in the left lower quadrant. Heart rate was measured continuously over every phase of the task. Heart rate was calculated as an average of all valid interbeat intervals and converted from interbeat-interval units (milliseconds) to beats-per-minute units. The average of a 5-min epoch was analyzed for each of the five phases of the task. Each epoch was scored for quality, and only epochs containing a full 5 min of good signal quality, without any designated invalid intervals of data that had to be omitted, were included in the analysis. We chose to examine the average heart rate for each phase of the task because the precise timing of each subjective report was not recorded on the physiological time series, and subjective reports did not necessarily occur during the peak physiological response. We focused on heart rate as our indicator of physiological arousal because it is accessible to conscious awareness, unlike heart rate variability and blood pressure, and is not liable to voluntary control, unlike respiration. However, we acknowledge that increases in heart rate do not purely reflect increases in sympathetic activation but also reflect parasympathetic withdrawal.

Participants were informed at the beginning of the session that, periodically, they would be asked for a verbal stress rating on a scale from 1 (*not stressed at all*) to 10 (*extremely stressed*). The experimenter prompted each participant to verbally report his or her level of stress approximately 20 to 30 s before the end of each phase of the task. Thus, a total of six self-reports of stress were collected during the session, near the end of each phase: baseline, during each stressor task, during the recovery period following each stressor task, and after the orthostatic challenge. The first five self-reports of stress were used, excluding the orthostatic time point.

Psychological stressors

Stroop color-word task. Participants completed a modified Stroop color-word task (Stroop, 1935). One of four color-name words was presented in a font color that was either congruent or incongruent with the word itself. The colored color-name stimulus appeared on screen, and participants pressed one of four keys on a keypad corresponding to the color of the letters in the word, not the color name. The rate of stimuli was modified according to participant performance to roughly standardize the degree of stressfulness. This standardization was set so that participants achieved an overall accuracy of 67%.

Morgan and Turner Hewitt (MATH) task. The MATH task (Turner et al., 1986; Turner, Sims, Carroll, Morgan, & Hewitt, 1987) is a mental arithmetic task designed for use as a psychological stressor in laboratory studies of cardiovascular reactivity. Participants were required to solve problems of mental addition or subtraction of two numbers. Problem difficulty could vary across five levels, ranging from problems of 1-digit ± 1-digit numbers (Level 1) to 3-digit ± 3-digit numbers (Level 5). The task always began at Level 3; difficulty was adjusted on each trial by accuracy on the previous trial.

Psychological well-being

Psychological Well-Being (PWB) scale. Participants completed the 42-item version of Ryff's (1989) PWB scale as part of the survey project in MIDUS. The scale consists of six subscales with 7 items each: autonomy, environmental mastery, personal growth, positive relations with others, purpose in life, and self-acceptance. Participants indicate on a 7-point Likert-type scale how true each statement is of themselves; higher scores indicate greater well-being. In the survey sample of 4,019 participants (precise sample sizes vary because of missing data for different scales), of which the current sample is a subset, Cronbach's alpha for subscales were .40 for autonomy, .54 for environmental mastery, .54 for personal growth, .63 for positive relations with others, .29 for purpose in life, and .66 for self-acceptance. PWB scores were divided by 10 for better representation of estimates and standard errors.

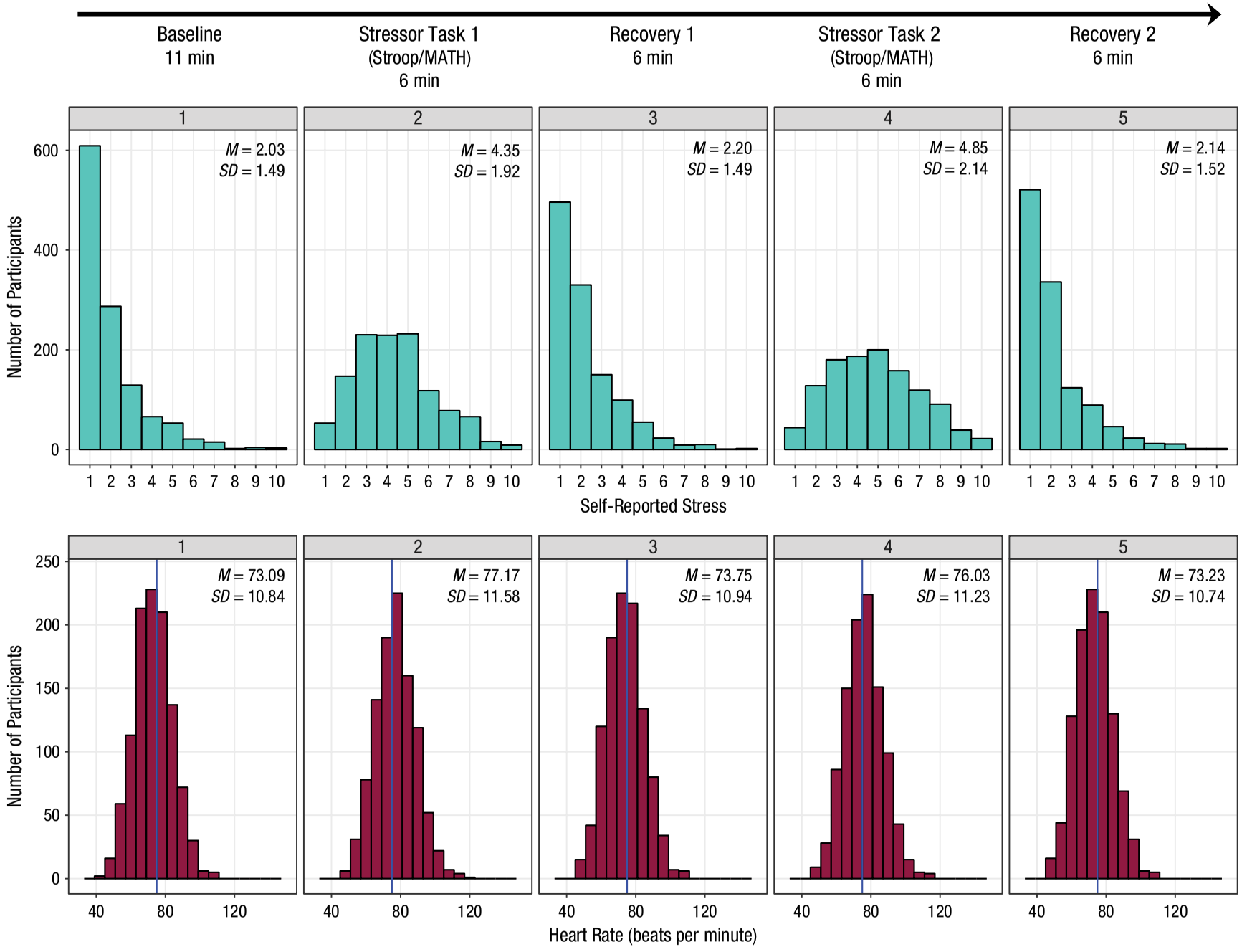

Fig. 1. *(continued on next page)*

Fig. 1. Distributions of self-reported stress levels (top row) and heart rate (bottom row) for each phase of the stress-induction paradigm. Self-reported stress and heart rate histograms, with means and standard deviations, are represented for each of the five phases of the stress-induction testing procedure. The blue vertical line in the heart rate histograms represents a constant of 75 to support comparison across phases of the task. Heart rate is the average across 5 min for each phase of the task. Self-reported stress is a verbal report on a simple scale from 1 (*not stressed at all*) to 10 (*extremely stressed*), reported once near the end of each phase of the task. Thus, there was one average heart rate measure and one self-reported stress measure per participant for each of the five phases of the task; their association composed the stress–heart rate coherence measure. Stroop = Stroop color-word task; MATH = Morgan and Turner Hewitt task.

Center for Epidemiological Studies Depression Inventory (CES-D). Participants completed the CES-D (Radloff, 1977) as part of the stress-induction substudy. The CES-D includes 20 items assessing depression symptoms over the past week, rated on a 4-point scale (0 = *rarely or none of the time*, 1 = *some or little of the time*, 2 = *moderately or much of the time*, 3 = *most or almost all the time*). Scores on the CES-D range from 0 to 60, with higher scores indicating more depressive symptoms. In the biomarker sample of 1,255 participants, of which the current sample is a subset, Cronbach's alpha was .89 for the CES-D. CES-D scores were divided by 10 for better representation of estimates and standard errors.

Spielberger Trait Anxiety Inventory (STAI). Participants completed the STAI (Spielberger, 1983, 1989) as part of the stress-induction substudy. The STAI includes 20 items designed to assess trait anxiety. Participants rate items such as, "I worry too much over something that really doesn't matter," on a 4-point Likert-type scale (1 = *almost never*, 4 = *almost always*). Cronbach's alpha was .91 for the STAI in the biomarker sample. STAI scores were divided by 10 for better representation of estimates and standard errors.

Coping strategies

Participants completed a subset of scales from the COPE Inventory (Carver et al., 1989) as part of the survey project of MIDUS 2. Only one of the subscales was theoretically relevant for our purposes: The denial subscale measures participants' tendency to cope with stress by denying the reality of a stressor or avoiding beliefs that the stressor exists (four items). Cronbach's alpha was .76 for the denial subscale in the survey sample. The other subscales administered were positive reinterpretation and growth (a tendency to identify positive aspects of stressors), active coping (a tendency to take action to deal with the stressor), planning (a tendency to think of plans to deal with the stressor), behavioral disengagement (a tendency to give up on goals that the stressor is interfering with), focus on and venting of emotion (a tendency to focus on distress and express those feelings), and using food to cope. These were not tested because they were not relevant to the hypothesis.

Physical well-being

Fasting blood draws were collected as part of the stress-induction substudy. We examined two inflammatory biomarkers: IL-6 was assayed in the MIDUS Biocore Laboratory (University of Wisconsin–Madison) using a Quantikine high-sensitivity enzyme-linked immunosorbent assay (ELISA) kit HS600B (R&D Systems, Minneapolis, MN). CRP was assayed at the Laboratory for Clinical Biochemistry Research (The University of Vermont) using a BN II nephelometer (Dade Behring, Deerfield, IL) and a particle-enhanced immunonephelometric assay. Distributions for IL-6 and CRP values were positively skewed and therefore log transformed, with bases 2 and 10, respectively.

Statistical analysis

Statistical analyses were conducted in RStudio (Version 1.1.453; RStudio Team, 2016), in the R programming environment (Version 3.5; R Core Team, 2008), and using the *lme4* package (Bates, Maechler, Bolker, & Walker, 2018; complete analysis scripts are publicly available at https://zenodo.org/record/3237927#.XPaWBBZKgWo). An R Markdown (https://rmarkdown.rstudio.com/) document including the output of the analysis code is included in the Supplemental Material. Our hypothesis is that the within-participants association between self-reported stress and heart rate is positively related to psychological and physical well-being and negatively related to denial coping at the between-participants level.

There are two statistical approaches to examine the relationship between a within-participants association and an individual-differences variable. First, one can derive for each participant an indicator of the strength of the within-participants association (e.g., compute a within-participants correlation coefficient between subjective stress and heart rate) and then correlate this indicator with the individual-differences variable. Second, one can estimate a linear mixed-effects model (LMEM) to examine whether the (statistical) effect of one of the Level 1 variables (e.g., subjective stress) on the other Level 1 variable (e.g., heart rate) is moderated by the individual-differences variable. If, for example, the effect of subjective stress on heart rate is stronger for participants high in psychological well-being, then

the within-participants association is positively related to psychological well-being. The second approach is preferable from a statistical standpoint (Hox, Moerbeek, & Van de Schoot, 2018) but somewhat less intuitive. We therefore report the LMEM approach in the main text but include the within-participants correlation-coefficient approach in Part I in Supplemental Results. Findings were consistent across the two approaches, except for those for CRP, which showed an effect in the same direction but was not significant in the correlation approach.

For the LMEM approach, we regressed heart rate on self-reported stress (centered around each participant's own mean), the well-being indicator under consideration (mean centered; e.g., PWB), and their interaction, adjusting for age, the interaction between self-reported stress and age, and nonindependence due to participants and families (Brauer & Curtin, 2018). Our model thus includes six fixed effects: self-reported stress (Level 1), the well-being indicator of interest (Level 2), their interaction, age (Level 2), the interaction of self-reported stress and age, and the intercept. The model includes a by-participant random intercept, a by-participant random slope for stress, and a by-family random intercept. The two by-participant random effects were allowed to correlate.

This model was represented in R as follows:

```
lmer(heartRate ~ stressClusterMeanCentered *
wellbeingCentered + StressClusterMeanCentered *
ageCentered (1 + stressClusterMeanCentered | subject) +
(1 | family), data = dfLong).
```

Our focus was on the interaction effect in this model, which represents the degree to which within-participants associations between self-reported stress and heart rate were related to the well-being indicator (PWB, depression, anxiety, IL-6, and CRP) or denial coping. Age was included as a covariate because of the broad age range of the sample, extending from early to late adulthood and because older participants had lower stress–heart rate coherence, $b = -0.008$, $F(1, 843.0) = 7.754$, $p = .005$. Gender was not associated with stress–heart rate coherence, $b = 0.051$, $F(1, 850.0) = 0.560$, $p = .455$, and so was not included as a covariate in the analyses. We fitted a separate model for each of the five well-being indicators of interest and denial coping (six total tests). The Anova() function in the *car* package (Version 3.0.0; Fox & Weisberg, 2011) provided estimates of *F*, error *df* (via Kenward-Roger approximation), and *p*. Multiple comparisons of the six different tests were corrected using the Holm-Bonferroni method.

Results

Stress–heart rate coherence and well-being

Stress–heart rate coherence was examined in relation to multiple markers of psychological and physical well-being. Table 2 and Figure 2 summarize these results. The statistical effect of stress on heart rate was found to be moderated by PWB, $b = 0.050$, $F(1, 822.8) = 26.70$, $p < .0001$; participants with higher stress–heart rate coherence also reported higher psychological well-being. The opposite was true for depressive symptoms, $b = -0.249$, $F(1, 783.7) = 36.77$, $p < .0001$, and trait anxiety, $b = -0.211$, $F(1, 769.4) = 32.49$, $p < .0001$; individuals with higher stress–heart rate coherence reported fewer depressive symptoms and had lower trait anxiety. Part III in Supplemental Results describes exploratory analyses investigating PWB subscales. For physical well-being, the statistical effect of stress on heart rate was found to be significantly moderated by IL-6 and CRP; participants with higher stress–heart rate coherence also

Table 2. Relationships Between Stress–Heart Rate Coherence and Well-Being Indicators and Between Stress–Heart Rate Coherence and Denial Coping, Adjusted for Age

Variable	*M* (*SD*)	*b*	*SE*	*F*	Error *df*	*p*	Adjusted *p*[a]
Psychological well-being							
Psychological well-being (divided by 10)	23.28 (3.52)	0.050	0.010	26.70	822.8	< .0001	< .0001
Depression (divided by 10)	0.86 (0.81)	–0.249	0.041	36.77	783.7	< .0001	< .0001
Anxiety (divided by 10)	3.42 (0.90)	–0.211	0.037	32.49	769.4	< .0001	< .0001
Physical well-being							
Interleukin-6 (log2)	2.96 (2.89)	–0.145	0.031	22.20	762.3	< .0001	< .0001
C-reactive protein (log10)	2.85 (4.26)	–0.175	0.065	7.16	827.2	.008	.008
Denial coping	6.09 (2.22)	–0.069	0.015	20.69	853.3	< .0001	< .0001

[a]The *p*s in this column were Holm-Bonferroni adjusted.

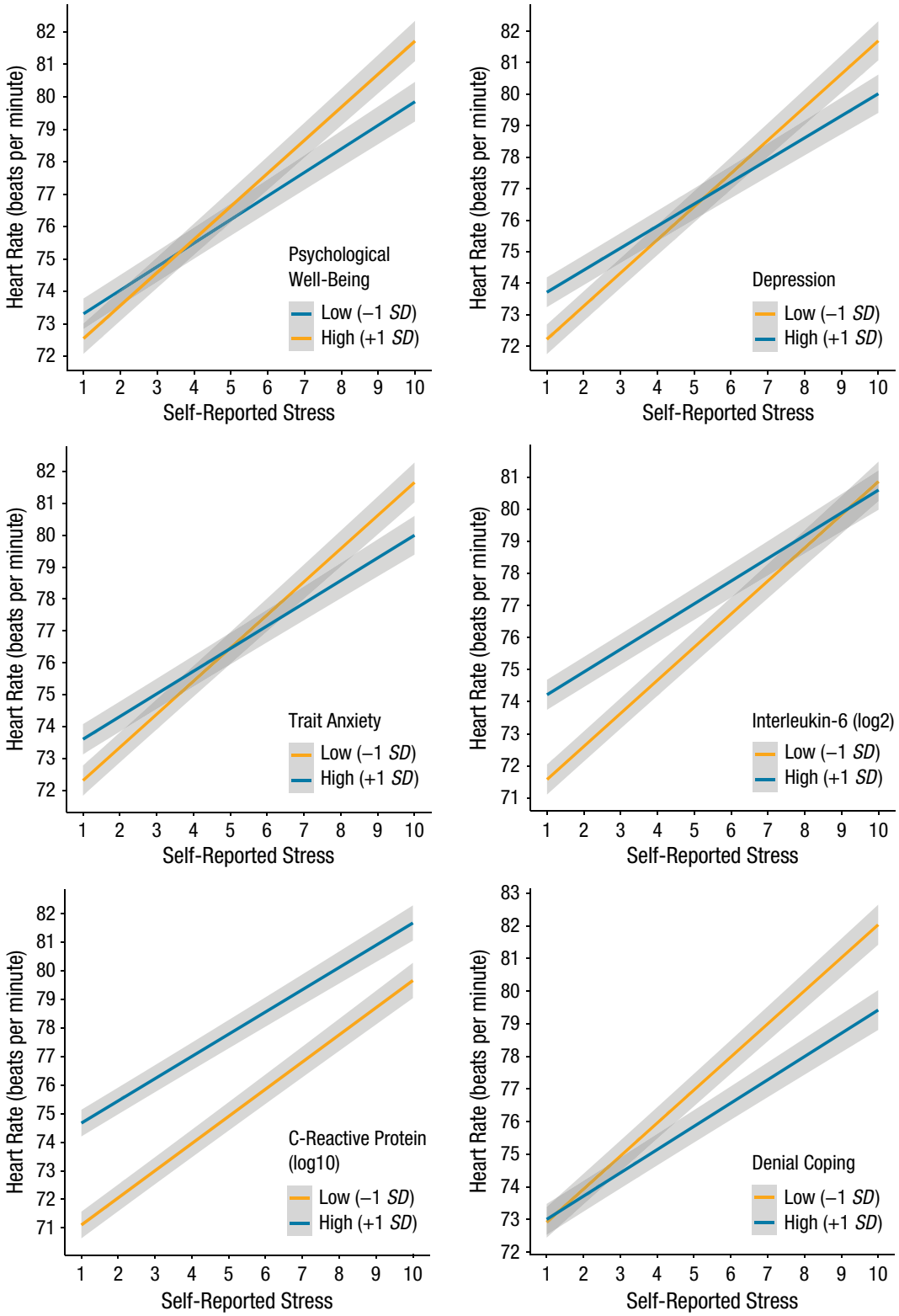

Fig. 2. Association between stress and heart rate for high (1 *SD* above the mean) and low (1 *SD* below the mean) levels of each well-being indicator and of denial coping. Gray shading represents 95% confidence intervals.

Table 3. Relationship Between Reactivity and Recovery Measures and Stress–Heart Rate Coherence

Measure	*M* (*SD*)	*b*	*SE*	*F*	Error *df*	*p*
Reactivity						
Subjective stress	2.6 (1.75)	−0.062	0.019	10.35	714.5	.001
Heart rate	3.42 (3.81)	0.196	0.005	1,318.70	752.5	< .0001
Recovery						
Subjective stress	−2.46 (1.66)	0.046	0.020	5.25	711.3	.022
Heart rate	−3.06 (3.33)	−0.216	0.006	1,306.21	672.1	< .0001

had lower IL-6, $b = -0.145$, $F(1, 762.3) = 22.20$, $p < .0001$, and lower CRP, $b = -0.175$, $F(1, 827.2) = 7.16$, $p = .008$.

Denial coping

We also investigated whether stress–heart rate coherence was associated with use of denial as a coping strategy. The statistical effect of stress on heart rate was found to be moderated by denial; higher stress–heart rate coherence was associated with less tendency toward the use of denial as a coping strategy, $b = -0.069$, $F(1, 853.3) = 20.69$, $p < .0001$.

Reactivity and recovery

Stress reactivity and recovery from stress are distinct theoretical constructs that may share overlapping variance with stress–heart rate coherence and may be associated with well-being. Thus, exploratory analyses investigated whether the associations between stress–heart rate coherence and well-being markers (and between stress–heart rate coherence and denial coping) may be due to shared variance with reactivity and recovery indices.

We computed heart rate reactivity for each participant by taking the difference in average heart rate from baseline to each stressor task and then averaging that participant's scores across the two stressor tasks. We computed subjective stress reactivity in the same way. This resulted in two measures: heart rate reactivity and subjective stress reactivity. We also computed recovery measures for heart rate and subjective stress by taking the difference in average heart rate or subjective stress from each recovery period to the previous stressor task and then averaging across the two recovery periods. This resulted in two more measures: heart rate recovery and subjective stress recovery.

Briefly, we fitted the same interaction LMEM, replacing the well-being indicator for each reactivity and recovery measure (four separate models). The statistical effect of stress on heart rate was found to be moderated by each reactivity and recovery measure. Table 3 details these results. We also fitted our original LMEM but included the reactivity and recovery measures as covariates. In models adjusting for the two reactivity and two recovery measures, stress–heart rate coherence was still significantly associated with the well-being markers and denial coping. Table 4 details these results.

Reactivity and recovery measures generally were not associated with the well-being indicators, with a few exceptions. We fitted an LMEM, regressing each well-being indicator on the two reactivity and two recovery measures, adjusting for age and nonindependence due to families (for complete details on the model, see R Markdown in the Supplemental Material). Heart rate recovery was significantly associated with PWB, $b = -1.239$, $F(1, 1009.1) = 6.15$, $p = .013$, as well as trait anxiety, $b = 0.284$, $F(1, 974.5) = 5.04$, $p = .025$; greater

Table 4. Relationship Between Stress–Heart Rate Coherence and Well-Being Indicators and Between Stress–Heart Rate Coherence and Denial Coping, Adjusted for Stress and Heart Rate Reactivity and Recovery

Variable	*b*	*SE*	*F*	Error *df*	*p*
Psychological well-being					
Psychological well-being (divided by 10)	0.051	0.010	26.94	814.3	< .0001
Depression (divided by 10)	−0.250	0.041	36.52	775.5	< .0001
Anxiety (divided by 10)	−0.210	0.037	31.88	761.2	< .0001
Physical well-being					
Interleukin-6 (log2)	−0.150	0.031	23.14	754.1	< .0001
C-reactive protein (log10)	−0.183	0.066	7.68	815.5	.006
Denial coping	−0.070	0.015	20.92	844.9	< .0001

decreases in heart rate from stressor to recovery periods were associated with higher psychological well-being and lower trait anxiety. Heart rate reactivity was significantly associated with CRP, $b = -0.017$, $F(1, 985.5) = 7.12$, $p = .008$; greater increases in heart rate from baseline to stressor periods were associated with lower CRP. All other results were not significant (for the full model results, see "Reactivity and Recovery" in the R Markdown file).

Variability in stress–heart rate coherence

We also examined variability in stress–heart rate coherence. We estimated an LMEM predicting heart rate from self-reported stress, taking into account that both variables were repeated measures (five data points per variable and per participant across the course of the stress-induction paradigm). We used the "coef()" method in the R package *lmer* to extract each participant's empirical best linear unbiased predictor (EBLUP). We emphasize that EBLUPs were extracted only to plot their distribution and were not used in any models. Participants whose self-reported stress was positively and strongly associated with their heart rate will have larger EBLUPs, and participants whose self-reported stress was not strongly associated with their heart rate will have EBLUPs closer to 0. Participants whose self-reported stress increased while their heart rate decreased, or whose self-reported stress decreased while their heart rate increased, will have EBLUPs less than 0. Figure 3 depicts associations between heart rate and self-reported stress for each individual in the sample to graphically display the variability in stress–heart rate coherence.

Discussion

We examined the functional significance of coherence between physiology and the subjective experience of stress within individuals and found it to be tied to multiple markers of well-being. Although coherence across subjective experience and physiology has often been theorized as important to adaptive functioning (e.g., Darwin, 1872/2009; Ekman, 1992; Lang, 1988; Lazarus, 1991; Levenson, 1994; Plutchik, 1980), it has rarely been demonstrated, with little consideration of whether coherence across response systems may be present primarily in high-functioning individuals.

This work constitutes an initial nomological network (Cronbach & Meehl, 1955) supporting stress–heart rate coherence as a measure perhaps tied to awareness and acceptance of mental states by demonstrating predicted interrelationships between stress–heart rate coherence and indices of denial coping and well-being. Specifically, we revealed positive associations between stress–heart rate coherence and psychological well-being and inverse associations between stress–heart rate coherence and factors commonly associated with reduced well-being, including anxiety, depression, and proinflammatory markers. Furthermore, stress–heart rate coherence was shown to be inversely associated with denial coping, suggesting that for at least some individuals, low stress–heart rate coherence may be due to the attempt to deny one's own feelings and the reality of stressors. Additional work is necessary to further specify this nomological network.

Because our study was cross-sectional and observational (i.e., lacked any experimental manipulation of stress–heart rate coherence or well-being), the directionality of the observed associations cannot be determined. For example, high stress–heart rate coherence is likely to support effective emotion regulation by affording signals on which effortful emotion regulation can operate. However, it is possible that individuals skilled at regulating their emotions may be more willing to attend to, confront, and accept their feelings because they are confident in their ability to successfully manage them. Similarly, individuals with high psychological and physical well-being may have more resources available to confront and correctly identify their own stress responses. The cross-sectional nature of this study also precludes evidence to support stress–heart rate coherence as a more stable trait measure. Future studies measuring stress–heart rate coherence at multiple points in time and across different contexts will help to establish the stability of stress–heart rate coherence within individuals and what states, such as fatigue, might impact it.

Additional studies are also needed to evaluate the causal status of stress–heart rate coherence as a contributor to well-being. For example, it would be fruitful to examine changes in stress–heart rate coherence over the course of interventions thought to improve metacognitive awareness, such as cognitive behavioral therapy or mindfulness. It is also important to examine whether increases in stress–heart rate coherence track with or precede improvements in symptomatology over the course of treatment. Investigating stress–heart rate coherence in relation to other measures of awareness of mental states would suggest convergent validity. For example, individuals scoring high on measures of emotional intelligence or cognitive insight would be expected to have high stress–heart rate coherence.

An important caveat of this study is that changes in heart rate are not purely due to stress. Individuals who reported low levels of stress but demonstrated elevated heart rate may have been experiencing a mental state other than stress that elevated their heart rate, such as

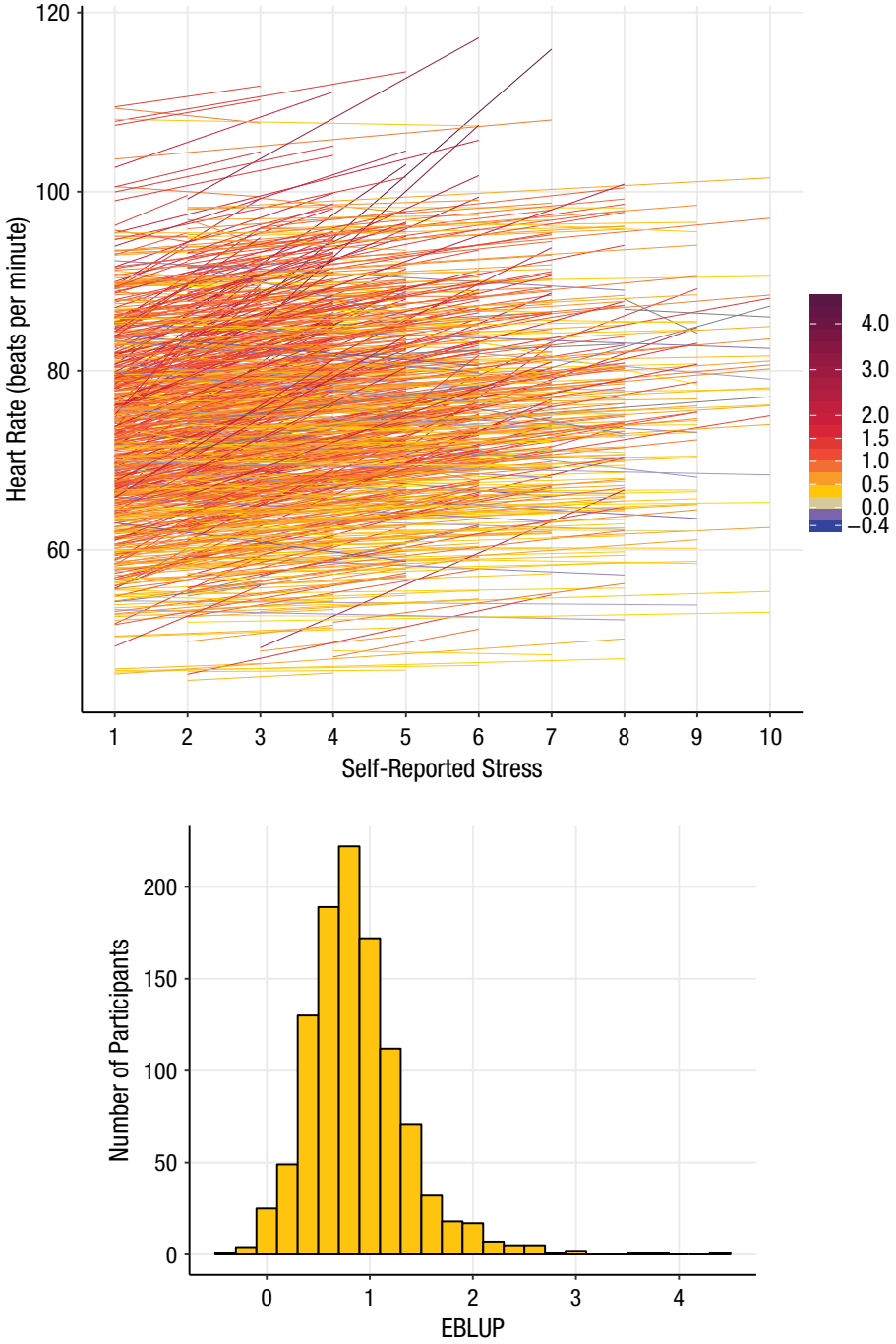

Fig. 3. Stress–heart rate coherence across the sample. The top panel shows slopes for the association between self-reported stress and heart rate. Each line represents a separate participant, and colors indicate the strength of the association. The bottom panel shows the distribution of empirical best linear unbiased predictors (EBLUPs) for within-participants associations between stress and heart rate.

greater arousal or engagement with the task. If queried about such experiences, they may have reported levels of arousal that tracked strongly with their heart rate and thus demonstrated strong associations between physiology and a subjective experience that was not stress. However, sitting at a computer to complete psychological stressor tasks helps to eliminate most physical-activity explanations for changes in heart rate, although future studies should also measure smaller movements (e.g., attaching an accelerometer to the

chair) for more complete examination of this potential confound.

It is important to assess whether the relationship between well-being and subjective experience-physiology coherence in the context of stress generalizes to other emotions, which have less clear physiological indicators. Likewise, assessing the coherence of subjective experience with physiological variables other than heart rate (e.g., corrugator and zygomatic facial electromyography, skin conductance response, pupil dilation) to compute an aggregate measure of physiological arousal would also benefit future research. An aggregate measure of multiple indices may better reflect physiological arousal across diverse individuals who respond to stress through changes in different physiological systems.

The current study benefited from extensive and detailed assessments from a large and sociodemographically diverse sample. However, the study was not designed specifically to investigate stress–heart rate coherence, which may require more precise measurements. For example, our finding that age was associated with lower stress–heart rate coherence may suggest that a more precise scale for measuring subjective stress is needed. Older individuals may have a reduced range of cardiac reactivity but perhaps perceive more precise changes in mental stress garnered from the breadth of experience across their lives that may not have been captured by the current 10-point subjective stress scale. A higher density of self-reports may also allow for a more reliable measurement within each participant. However, including additional self-reports must be balanced with the aim to induce stress or emotion. Asking participants to repeatedly report subjective experience can elicit increased awareness or even change the emotional response (Kassam & Mendes, 2013) and, thus, influence the measurement of stress–heart rate coherence. However, Mauss, Levenson, McCarter, Wilhelm, and Gross (2005) demonstrated comparable emotion-relevant experiential and physiological responses between participants who completed continuous ratings of their emotion while watching emotion-eliciting films and participants who provided no ratings.

Conclusion

Within-participants stress–heart rate coherence across stress induction and recovery shows promise as a novel characteristic that may contribute not only to psychological but also to physical well-being. The current work is part of an emerging program of inquiry on stress–heart rate coherence as a measure of awareness and acceptance of mental states that is linked to adaptive functioning. Broadly, this work underscores the importance of considering the coherence between measures as an index that can offer information beyond what either measure provides in isolation. The findings raise the possibility that awareness of the coupling between mental states and physiology is adaptive and may represent a key ingredient for psychological and physical well-being.

Action Editor

Ian H. Gotlib served as action editor for this article.

Author Contributions

S. L. Sommerfeldt developed the current study concept. S. L. Sommerfeldt analyzed the data with help from M. Brauer, S. M. Schaefer, and R. J. Davidson. C. D. Ryff led the Midlife in the United States study design. S. L. Sommerfeldt drafted the manuscript, and all other authors provided critical revisions. All the authors approved the final manuscript for submission.

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Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/0956797619849555

Open Practices

All data are publicly available and can be accessed at http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/29282 and http://www.icpsr.umich.edu/icpsrweb/ICPSR/studies/04652. Data-analysis code is available at https://zenodo.org/record/3237927#.XPaWBBZKgWo. The design and analysis plans for this study were not preregistered. The complete Open Practices Disclosure for this article can be found at http://journals.sagepub.com/doi/suppl/10.1177/0956797619849555. This article has received the badge for Open Materials. More information about the Open Practices badges can be found at http://www.psychologicalscience.org/publications/badges.

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