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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, though both systems must work in concert to enable adaptive responses. We investigated 1,065 participants from the MIDUS 2 study (www.midus.wisc.edu) 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 (pro-inflammatory biomarkers interleukin-6 and C-reactive protein). Within-subject association between heart rate and self-reported stress was significantly associated with higher psychological well-being, fewer depressive symptoms, lower trait anxiety, less use of denial coping, and lower levels of pro-inflammatory biomarkers. 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

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

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; Plutchik, 1980; Lang, 1988; Lazarus, 1991; Ekman, 1992; Levenson, 1994). Yet the empirical evidence largely suggests a lack of coherence across response systems (e.g., Campbell & Ehlert, 2012 review studies of the relationship between subjective stress and biological markers in the context of the Trier Social Stress Test (TSST)). Although it has been demonstrated that there is significant variability in this coherence across individuals (Sze et al., 2010), few studies have examined these individual differences. The few studies that have, show coherence to be related to differences in externalizing and 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-report 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-subject measure as ‘stress-heart rate coherence.’ We examine 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 measureable 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, 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 disconnect between the state of the body and the mental state. Weak stress-heart rate coherence may be evident in individuals who lack awareness into 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 et al. (2014) demonstrated that physiological arousal 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 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 so as 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. While 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 disconnect between subjective experience and physiology. Chronic stress also affects the immune system by impairing effective termination of inflammatory responses. Levels of the pro-inflammatory 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 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 pro-inflammatory biomarkers interleukin-6 and CRP in blood plasma. We also examined the relationship with denial coping to expand the nomological network of stress-heart rate coherence, as we believe denial coping would lead to a disconnect between physiology and subjective reports, to the degree that subjective reports deny the existence of a mental state.

Methods

Participants

Data were collected from 2004-2009 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 pre-determined 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 sample was $N = 1,065$, which is adequate to detect even small effects. A detailed description of the

sample is provided in Table 1. Briefly, participants were aged 35 to 86 years ($M = 56$, $SD = 11$) at the time of the stress-induction sub-study, and 57.2% were female ($N = 610$). Overall, the sample was predominantly White (77.5%), and a significant proportion (18.1%) were African American. The sample included $N = 118$ twin pairs, and $N = 11$ non-twin siblings (one family with three siblings, and four families with two siblings). As siblings present a source of non-independence in the data, we adjusted for family membership in our models. Participants completed the biomarker sub-study between 0 and 62 months ($M = 25.9$ months, $SD = 14.19$) following the survey study. Supplemental Results, part II includes a description of analyses investigating the impact of this lag between the two studies on results. Lag did not significantly moderate results nor did adjusting for lag impact the significance of any findings.

Table 1. Sample characteristics

N	1,065
Gender	$N = 610$ Female (57.2%)
Age in years (at stress-induction)	$M = 56.4$ ($SD = 11.21$) Range: 35-86
Months between survey & stress-induction	$M = 25.89$ ($SD = 14.19$) Range: 0 – 62
Race	Asian: $N = 3$ (.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: $N = 1$; Refused to report: $N = 1$; Missing: $N = 1$
MIDUS Subsample	Main: $N = 521$ (48.9%) Sibling: $N = 6$ (.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 1 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: $N = 101$ (co-twin not in subsample for this analysis)
Non-twin siblings	Three siblings: $N = 3$ (same family) Two siblings: $N = 8$ (4 families)

Procedure

Participants completed a standardized laboratory-based experimental stress-induction paradigm designed to measure cardiovascular reactivity and recovery from stress (Love, Seeman, Weinstein, & Ryff, 2010; Crowley et al. 2011; 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 (CUMC) in the laboratory of Dr. 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 minutes), two cognitive/psychological stressor tasks (6 minutes each; counterbalanced across subjects), a seated, resting period after each task (recovery period; 6 minutes each), as well as in response to orthostatic challenge: moving from a seated to a standing position and remaining standing (6 minutes). The orthostatic phase of the task is not included in analyses, as changes in heart rate during this phase are confounded with physical movement. Thus, we examined 5 phases of interest: baseline, stressor task 1, recovery 1, stressor task 2, recovery 2.

Participants' heart rate was measured using electrocardiograph (ECG) 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 inter-beat (RR) intervals and converted from RR interval units (milliseconds) to beats per minute units. The average of a 5-minute epoch was analyzed for each of the five phases of the task. Each epoch was scored for quality, and only epochs containing full 5-minutes of good signal quality, without any designated invalid intervals of data that had to be omitted from analysis, were included in analysis. We chose to examine the average heart rate for each phase of the task as 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 instructed at the beginning of the session that periodically they would be asked for a verbal stress rating on a scale of 1-10, 1 representing not stressed at all, and 10 representing extremely stressed. The experimenter prompted the participant to verbally report their level of stress approximately 20-30 seconds 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, 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.

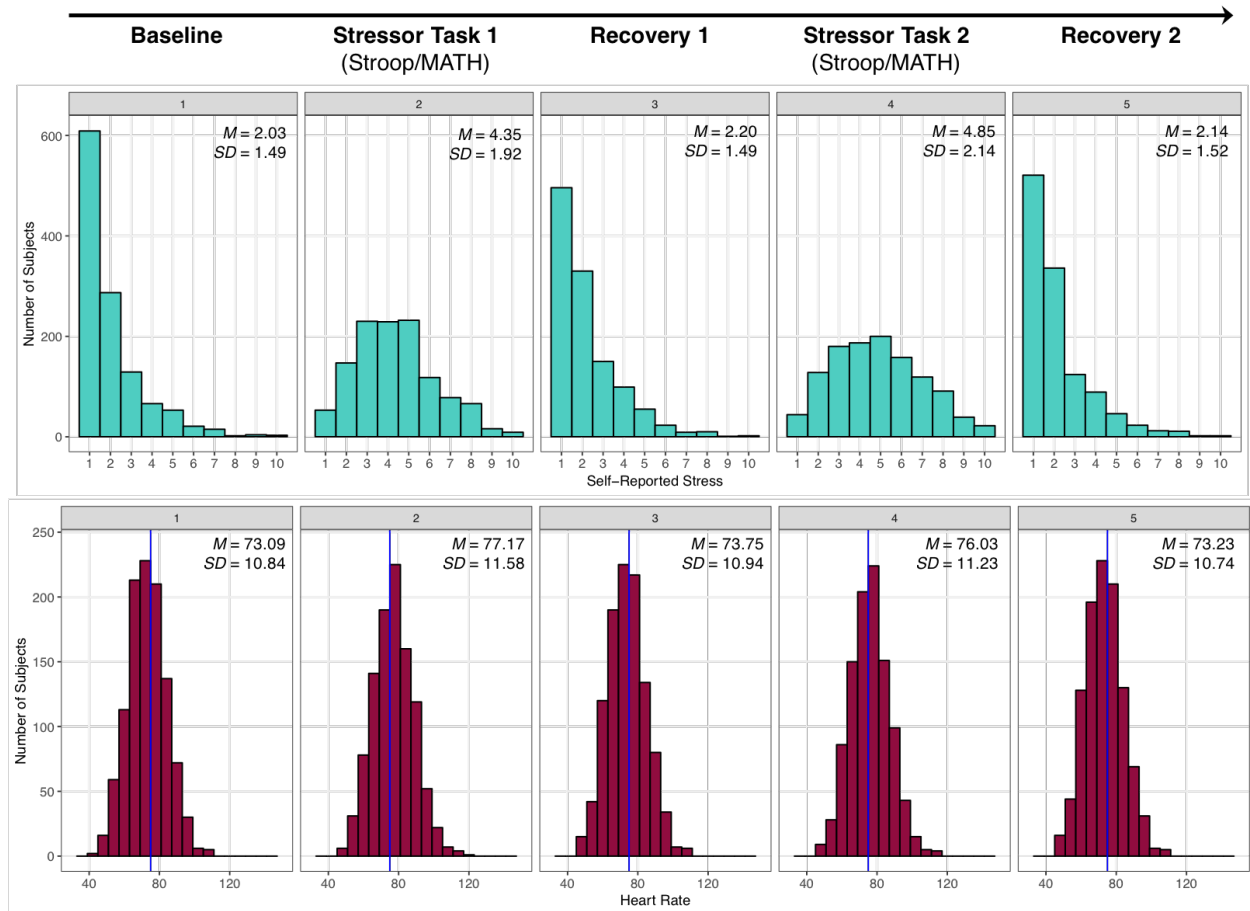


Figure 1. Distributions of self-reported stress levels and heart rate for each phase of the stress-induction paradigm. Turquoise arrows indicate approximate timing of stress self-reports in the procedure, which is near the end of each phase. Self-reported stress (turquoise) and heart rate (burgundy) histograms, with means and standard deviations for each of the 5 phases of the stress-induction testing procedure are represented. The blue vertical line in heart rate histograms represents a constant of 75 to support comparison across phases of the task. Heart rate is the average across 5 minutes for each phase of the task. Self-reported stress is a verbal report on a simple scale of 1-10 (1 representing not stressed at all, and 10 representing extremely stressed), reported once during 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 comprised the stress-heart rate coherence measure.

Psychological stressors

Stroop (Stroop, 1935). Participants completed a modified Stroop color-word task. One of four color-name words was presented in a font color that was either congruent or incongruent with the name. 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; Turner et al., 1986; Turner, Sims, Carroll, Morgan, & Hewitt, 1987). The MATH task 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 \pm 1-digit numbers (level 1) to 3-digit \pm 3-digit numbers (level 5). The task always began at level 3, then difficulty was adjusted at each trial by accuracy on the previous trial.

Psychological well-being

Psychological Well-Being (PWB; Ryff, 1989). Participants completed the 42-item version of Carol Ryff's PWB Scale as part of the survey project in MIDUS. The scale consists of 6 subscales with 7 items each: autonomy, environmental mastery, personal growth, positive relations with others, purpose in life, and self-acceptance. Participants indicated on a 7-point Likert scale how true each statement is of themselves; higher scores indicate greater well-being. In the survey sample of $N = 4,019$ (precise sample sizes vary due to missing data for different scales), of which the current sample is a subset, Cronbach's alpha for subscales were: autonomy

($\alpha = .40$), environmental mastery ($\alpha = .54$), personal growth ($\alpha = .54$), positive relations with others ($\alpha = .63$), purpose in life ($\alpha = .29$), and self-acceptance ($\alpha = .66$).

Center for Epidemiological Studies Depression Inventory (CES-D; Radloff, 1977). Participants completed the CES-D as part of the stress-induction sub-study. 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 high scores indicating more depressive symptoms. In the biomarker sample of $N = 1,255$, of which the current sample is a subset, Cronbach's alpha was .89 for the CES-D.

Spielberger Trait Anxiety Inventory (STAI, Spielberger, 1983; 1989). Participants completed the STAI as part of the stress-induction sub-study. 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 scale: 1 representing almost never, 4 representing almost always. Cronbach's alpha was .91 for the STAI in the biomarker sample.

Coping strategies

Participants completed a subset of scales from the *COPE Inventory* (Carver, Scheier, & Weintraub, 1989) as part of the survey project of MIDUS II. Only one of the subscales was theoretically relevant for our purposes: The denial subscale measures respondents' tendency to cope with stress by denying the reality of a stressor or avoiding beliefs that the stressor exists (4 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 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 sub-study. We examined two inflammatory biomarkers: IL-6 was assayed in the MIDUS Biocore Laboratory (University of Wisconsin, Madison, WI) using the Quantikine® High-sensitivity ELISA kit #HS600B (R & D Systems, Minneapolis, MN). CRP was assayed at the Laboratory for Clinical Biochemistry Research (University of Vermont, Burlington, VT) using the BNII nephelometer from Dade Behring utilizing a particle enhanced immunonephelometric assay. Distributions for IL-6 and CRP values were positively skewed and therefore log-transformed for statistical analyses.

Statistical Analysis

Statistical analyses were conducted in RStudio version 1.1.453, R version 3.5, and using the lme4 package (<https://cran.r-project.org/web/packages/lme4/lme4.pdf>; complete analysis scripts are attached to this submission and will be made publicly available at https://github.com/sashasomms/coherence_behavioral/ after this work is accepted). An R Markdown (<https://rmarkdown.rstudio.com/>) document also includes the output of the analysis code and is included in Supplemental Method, R Markdown. Our hypothesis is that the within-participant 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-participant association and an individual difference variable. First, one can derive for each participant an indicator of the strength of the within-participant association (e.g., compute a within-participant correlation coefficient between subjective stress and heart rate) and then correlate this indicator with the individual difference variable. Second, one can estimate a linear mixed-effects model (LMEM) to examine if 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 difference variable. If, for example, the effect of subjective stress on heart rate is stronger for participants high in psychological well-being, then the within-participant association is positively related to psychological well-being. The second approach is preferable from a statistical standpoint (Hox, Moerbeek, & Van de Schoot, 2017) but somewhat less intuitive. We therefore report the LMEM in the main text, but include the within-participant correlation coefficient approach in Supplemental Results, part I. Findings were consistent across the two approaches, with the exception of CRP, which had 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 and the non-independence due to participants and families (Brauer & Curtin, 2017). Our model thus includes five fixed effects: self-reported stress (level-1), the well-being indicator of interest (level-2), their interaction, age (level-2), and the interaction of self-reported stress and age. The model includes a by-participant random intercept, a by-participant random slope for heart rate, and a by-family random intercept. The two by-participant random effects were allowed to correlate.

This model was represented in R as:

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

Our focus is on the interaction effect in this model, which represents the degree to which within-participant 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 due to the broad age range of the sample extending from early to late adulthood, and because older participants had lower stress-heart rate coherence ($b = -0.008$, $F(1, 867.0) = 7.757$, $p = .005$). Gender was not associated with stress-heart rate coherence ($b = .051$, $F(1, 876.8) = .560$, $p = .455$), and so was not included as a covariate in the analyses. We fit a separate model for each of the five well-being indicators of interest and denial coping (six total tests). Kenward-Roger approximation via the ‘modelSummary()’ method in lmSupport package version 2.9.13 provided estimates of F , error df , 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.

	<i>M(SD)</i>	<i>b</i>	SE	<i>F</i>	error <i>df</i>	<i>p</i>	<i>p-adjusted</i>
Psychological Well-Being							
PWB	23.28(3.52)	0.050	0.010	26.70	822.8	<.0001***	<.0001***
Depression	.86 (.81)	-0.249	0.041	36.77	783.7	<.0001***	<.0001***
Anxiety	3.42 (.90)	-0.211	0.037	32.49	769.4	<.0001***	<.0001***
Physical Well-Being							
IL-6 (log2)	2.96 (2.89)	-0.145	0.031	22.20	762.3	<.0001***	<.0001***
CRP (log10)	2.85 (4.26)	-0.175	0.065	7.16	827.2	0.008*	0.008*
Denial coping							
	6.09 (2.22)	-0.069	0.015	20.69	853.3	<.0001***	<.0001***

Table 2. The relationships between stress-heart rate coherence and the well-being indicators as well as denial coping, adjusting for age. Values listed in ‘*M(SD)*’ column represent means(standard deviations) for each well-being indicator or denial coping. *b* is the interaction term in the model, representing the extent to which the stress-heart rate relationship is associated with the well-being indicator (or denial coping). Note that error *df*, *F*, and *p* are approximated via the Kenward-Roger method. ^aHolm-Bonferroni adjusted

***p* < .001. *** *p* < .0001.

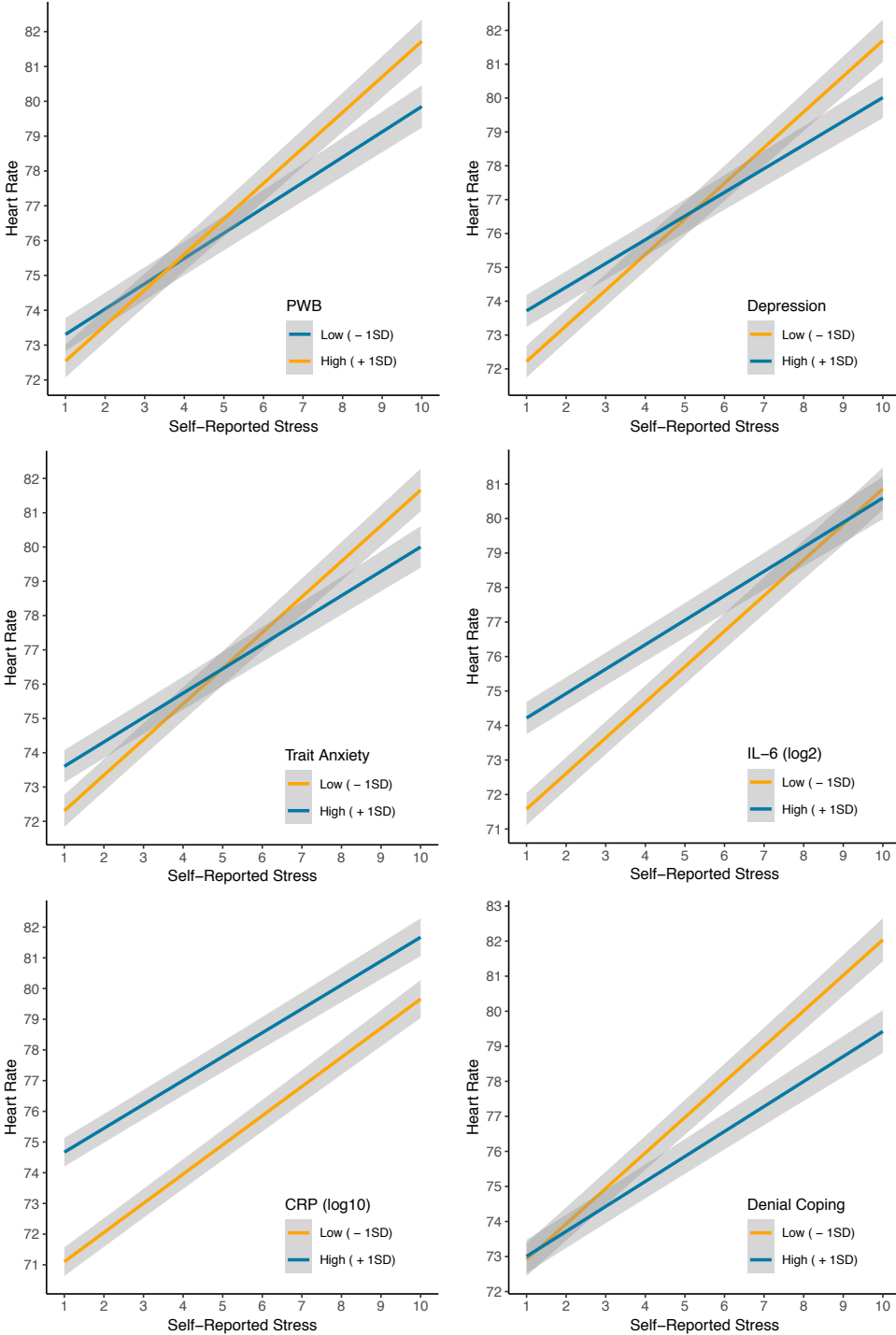


Figure 2. Association between stress and heart rate for high (1 standard deviation above the mean) and low (1 standard deviation below the mean) on each well-being indicator and denial

coping. Orange bars represent higher well-being; blue bars represent lower well-being. Gray shading represents 95% confidence interval.

Psychological well-being. The (statistical) effect of stress on heart rate was found to be moderated by PWB, $b = .050$, $F(1, 822.8) = 26.70$, $p < .0001$, such that 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, $b = -0.211$, $F(1, 769.4) = 32.49$, $p < .0001$, such that individuals with higher stress-heart rate coherence reported fewer depressive symptoms and had lower trait anxiety. Supplemental Results part III describes exploratory analyses investigating PWB subscales.

Physical well-being. The (statistical) effect of stress on heart rate was found to be significantly moderated by IL-6 and CRP, such that participants with higher stress-heart rate coherence also 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, such that higher stress-heart rate coherence were associated with less tendency towards the use of denial as a coping strategy, $b = -.069$, $F(1, 853.3) = 20.69$, $p < .0001$.

Reactivity and Recovery

Stress reactivity and recovery from stress are distinct theoretical constructs that may share overlapping variance with stress-heart rate coherence and be associated with well-being.

Thus, exploratory analyses investigated whether the associations between stress-heart rate coherence and well-being markers and denial coping may be due to shared variance with reactivity and recovery indices.

We computed heart rate reactivity for each subject by taking the difference in heart rate from baseline to each stressor task, then averaging for each subject 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 heart rate or subjective stress from each recovery period to the previous stressor task, then averaging across the two recovery periods. This resulted in two more measures: heart rate recovery and subjective stress recovery.

Briefly, we fit the same LMEM interaction model, 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.

	<i>M(SD)</i>	<i>b</i>	SE	<i>F</i>	error <i>df</i>	<i>p</i>
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	1318.70	752.5	< .0001***
Recovery						
Subjective stress	-2.46 (1.66)	0.046	0.020	5.25	711.3	0.022*
Heart rate	-3.06 (3.33)	-0.216	0.006	1306.21	672.1	< .0001***

Table 3. Relationship between reactivity and recovery measures and stress-heart rate coherence.

M(SD) are means and standard deviations of each reactivity and recovery measure; *b* represents the interaction term in the model, or the extent to which the stress-heart rate relationship is associated with, the reactivity or recovery measure.

***p* < .001. *** *p* < .0001

We also fit our original LMEM model 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 fit a LMEM regressing each well-being indicator on the two reactivity and two recovery measures, adjusting for age and non-independence due to families (see Supplemental Method, R Markdown for complete details on the model). Heart rate recovery was significantly associated with PWB, $b = -1.239$, $F(1, 1009.1) = 6.15$, $p = .013$, as well as trait anxiety, $b = .284$, $F(1, 974.5) = 5.04$, $p = .025$, such that greater decreases in heart rate from stressor to recovery periods was associated with higher psychological well-being, and lower trait anxiety. Heart rate reactivity was significantly associated with CRP, $b = -.017$, $F(1, 985.5) = 7.12$, $p = .008$, such that greater increases in heart rate from baseline to stressor periods was associated with lower CRP. All other results were not significant, (see Supplemental Method, R Markdown section ‘Reactivity and Recovery’ for full model results).

	<i>b</i>	SE	<i>F</i>	error <i>df</i>	<i>p</i>
Psychological Well-Being					
PWB (divided by 10)	0.050	0.010	26.94	814.3	< .0001***
Depression (divided by 10)	-0.249	0.041	36.52	775.5	< .0001***
Anxiety (divided by 10)	-0.210	0.037	31.88	761.2	< .0001***
Physical Well-Being					
IL-6 (log2)	-0.150	0.031	23.14	754.1	< .0001***
CRP (log10)	-0.183	0.066	7.68	815.5	.006**
Denial coping	-0.070	0.015	20.92	844.9	< .0001***

Table 4. Relationship between stress-heart rate coherence and well-being indicators and denial coping, when adjusting for stress and heart rate reactivity and recovery. *b* represents the interaction term of the model, representing the extent to which the stress-heart rate relationship is associated with the well-being indicator (or denial coping).

** $p < .001$. *** $p < .0001$

Variability in stress-heart rate coherence

We also examined variability in stress-heart rate coherence in the sample. We estimated a linear mixed-effects model 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 only extracted to plot their distribution, and were not used in any models. Computed this way, participants who reported their level of stress such that it was associated with their heart rate will have larger EBLUPs; participants who reported their level of stress such that it was not strongly associated with their heart rate will have EBLUPs closer to 0, and participants who reported their level of stress such that increases in self-reported stress coincide with decreases in heart rate, or vice versa (self-reported stress decreases when heart rate increases), will have EBLUPs less than 0. Figure 3 depicts associations between heart rate and self-reported stress for different individuals in the sample to graphically display the variability in stress-heart rate coherence.

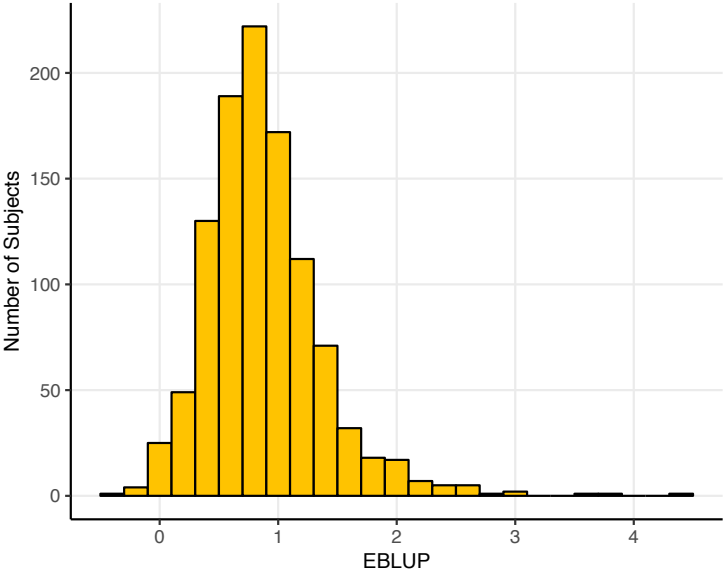
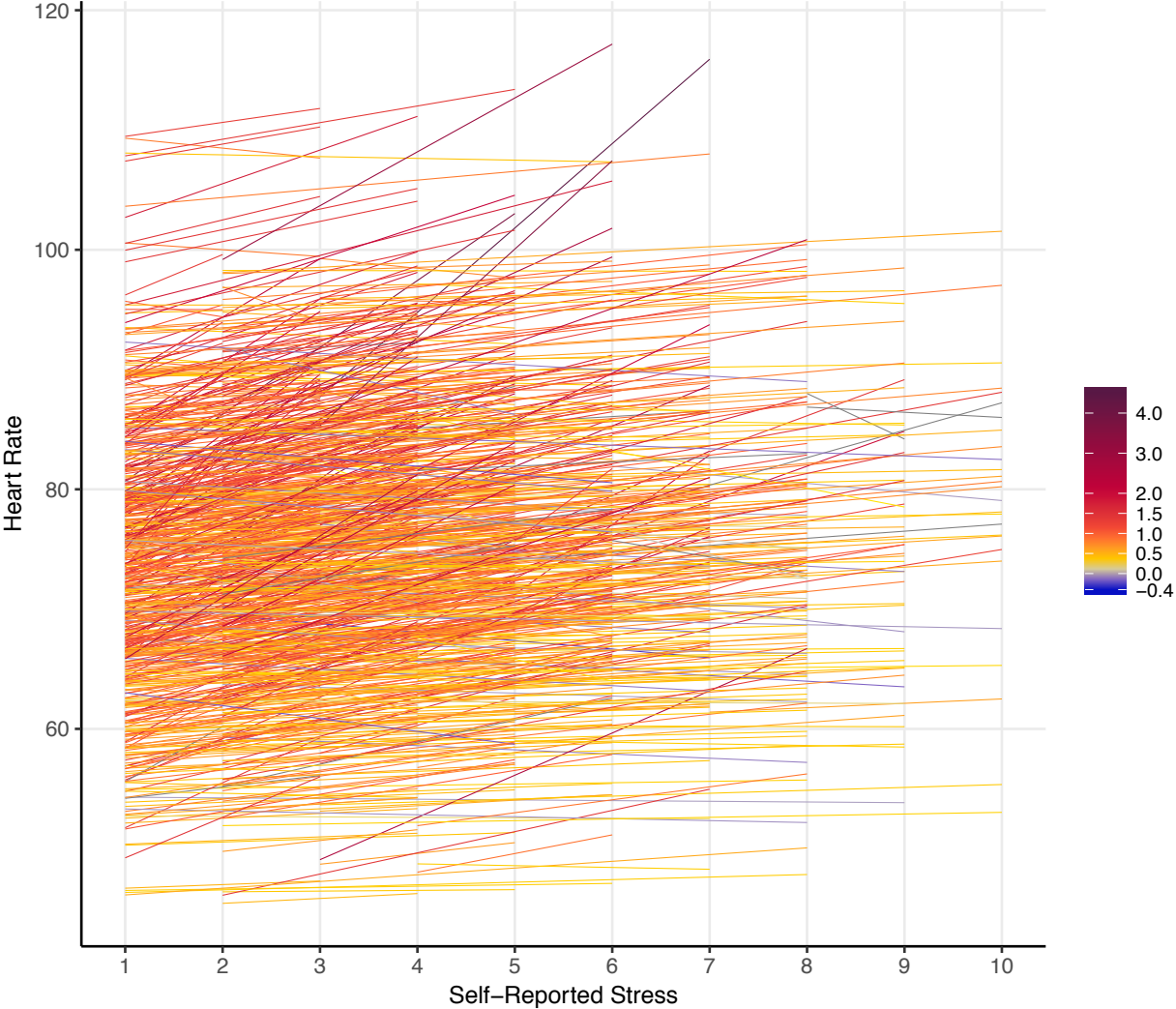


Figure 3. Stress-heart rate coherence across the sample. A. Each individual's slope between self-reported stress and heart rate. Each line represents a separate participant, with colors according to strength of association. B. Distribution of empirical best linear unbiased predictors (EBLUPs) for within-subject associations between stress and heart rate.

Discussion

We examined the functional significance of coherence between the subjective experience of stress and physiology within-individuals and found it to be tied to multiple markers of well-being. While coherence across subjective experience and physiology has often been theorized as important to adaptive functioning (e.g., Darwin 1872; Plutchik, 1980; Lang, 1988; Lazarus, 1991; Ekman, 1992; Levenson, 1994), it has rarely been demonstrated, with little consideration to 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 with psychological well-being, and inverse associations with factors commonly associated with reduced well-being, including anxiety, depression, and pro-inflammatory 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.

As 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 will also be 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 highly on measures of emotional intelligence or Beck's Cognitive Insight scale (Beck et al., 2004) would be expected to have higher 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 greater arousal or engagement with the task. If queried about such experience, they may have reported levels of arousal that tracked strongly with their heart rate, and thus have demonstrated strong associations between physiology and a subjective experience that was not stress. That said, the procedure of sitting at a computer completing 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., using an accelerometer attached to the chair) for more complete examination of this potential confound.

It will be 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 coherence of subjective experience with physiological variables other than heart rate (e.g., corrugator and zygomatic facial EMG, 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 benefitted from extensive and detailed assessments from a large and socio-demographically 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 a more precise scale for measuring subjective stress is needed. Older individuals may have

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 be 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 of measure stress-heart rate coherence. However, Mauss et al. (2005) demonstrated comparable emotion-relevant experiential and physiological responses between participants completing continuous ratings of their emotion while watching emotion-eliciting films, and participants who provided no ratings.

Conclusions

Within-participant stress-heart rate coherence across stress induction and recovery shows promise as a novel characteristic that contributes not only to psychological, but also physical well-being. This work forms 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.

Author Contributions

S. L. Sommerfeldt developed the current study concept. S. L. Sommerfeldt performed the data analysis with help from M. Brauer, S. M. Schaefer and R. J. Davidson. C. Ryff led MIDUS study design. S. L. Sommerfeldt drafted the manuscript, and all other authors provided critical revisions. All authors approved the final version of the manuscript for submission.

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