

Blood pressure reactivity predicts somatic reactivity to stress in daily life

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Abstract The purpose of the present study was to examine whether stress-somatic symptom associations may be more pronounced among individuals whose bodies exhibit higher levels of cardiovascular reactivity to a laboratory social stress task. During an initial laboratory session, participants delivered a 5-min speech and individual differences in cardiovascular reactivity were quantified. The same participants subsequently completed a 15-day experience sampling protocol, in which daily levels of stress and somatic symptoms were assessed. Multi-level modeling was used to assess associations among laboratory cardiovascular reactivity, daily stress and somatic symptoms. Daily symptom reports included a set of commonly experienced physical symptoms reflective of general bodily dysfunction. Individuals displaying high levels of laboratory systolic blood pressure reactivity experienced more somatic symptoms on high-stress days, but this was not the case for individuals low in systolic blood pressure reactivity. The results bridge two hitherto distinct health psychology literatures showing that cardiovascular and somatic reactivity to stress are associated. Stress reactivity individual differences in one system may indicate more general differences in bodily reactivity across systems.

Keywords Stress · Reactivity · Psychosomatic · Cardiovascular reactivity · Somatic symptoms

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Introduction

According to the *psychosomatic hypothesis* of stress and health, psychological stress undermines optimal bodily functioning, ultimately leading to disease occurrence and greater mortality likelihood (Kiecolt-Glaser et al. 2002; McEwen 1998). From a behavioral medicine perspective, it is crucial to focus on stress-associated processes that may be precursors to later disease and dysfunction. The extent to which stressors precipitate somatic symptoms is one important model in this regard. Higher levels of environmental stress have been linked to somatic symptoms (Alfven et al. 2008; Murberg and Bru 2007), disabilities due to such symptoms (Carbone et al. 2000), and earlier deaths (Kiecolt-Glaser et al. 2002). Thus, stress-symptom associations are of value in diagnosing disease processes and pre-clinical medical conditions (Barsky 2000; Herbert and Cohen 1993).

However, several lines of investigation have raised concerns in viewing self-reports of somatic symptoms as veridical. The trait of neuroticism, reflecting a general predisposition toward psychological stress reaction (McCrae et al. 2002), predicts somatic symptom reports seemingly independent of objectively assessed health-related problems (Watson and Pennebaker 1989). Individuals diagnosed with somatization disorders often contact medical care-givers with somatic symptom concerns that cannot be verified by medical procedures or diagnoses (Fink et al. 2005). Individual differences such as alexithmia (Luminet et al. 2006) may be similarly linked to somatic symptom reporting and treatment seeking that are independent of physiological causes.

Other sources of data, however, highlight the diagnostic value of somatic symptom reports (Barsky 2000). On the basis of an extensive research program, Kroenke (2003)

found that the majority (67%) of patients presenting with somatic complaints were subsequently diagnosed with precursors to medical disease. Further, Kisely and Simon (2006) distinguished patients according to whether their somatic symptoms could be verified or not by medical procedures. In either case, patients had similarly poor outcomes, including equal levels of psychosocial dysfunction, physical dysfunction, and health-care service use. From this perspective, somatic symptom reports should be taken seriously, as they indicate significant dysfunction in general terms (Fink et al. 2005; Kroenke 2003).

Given these divergent points of view, support for the psychosomatic hypothesis would benefit from using procedures designed to minimize reporting biases in somatic symptom reports. Such procedures were used in the present research. First, because neuroticism is viewed as a confound in symptom reporting (Watson and Pennebaker 1989), controlling for its influence would be desirable. Second, reports of somatic symptoms over long-retrospective time periods have been viewed as suspect (Robinson and Clore 2002). Such retrospective biases can be limited by the use of experience-sampling procedures and within-subject designs (Charles and Almeida 2006; Tennen et al. 2000). Of particular interest was whether we could predict individual differences in stress-symptom associations using an objective physiological measure that would be immune to self-reported biases.

Individual differences in cardiovascular reactivity

Hines and Brown (1932) first proposed that individuals displaying large cardiovascular responses to a standard laboratory task (in this case, a cold-pressor task) were those who were more likely to develop hypertension later in life. Manuck et al. (1988) later articulated the cardiovascular reactivity hypothesis, proposing that greater cardiovascular reactivity to environmental stress contributes to the development of cardiovascular disease. Work of this type has resulted in a relatively solid body of findings: Individual differences in cardiovascular reactivity are reliable and longitudinally predict the development of hypertension and coronary heart disease (Stewart et al. 2006; Treiber et al. 2003).

In understanding processes and pathways, though, there is much to learn about how individual differences in cardiovascular reactivity function in more proximal terms (Kamarck and Lovallo 2003; Treiber et al. 2003). For example, attempts to link individual differences in cardiovascular reactivity to personality traits and clinical assessments of temperament have resulted in findings that are somewhat inconsistent (Chida and Hamer 2008; Smith 1992). Laboratory assessments of cardiovascular reactivity have also been questioned. Specifically, Schwartz et al.

(2003) suggest that the conditions instantiated in the laboratory may fail to adequately model the manner in which stressors are encountered and reacted to in everyday life. These authors did, however, acknowledge that more lab-to-life research is needed to better evaluate the laboratory paradigm as a model of disease risk.

Cardiovascular and somatic symptom reactivity

Evidence indicates that stress undermines bodily functioning across several physiological systems including the cardiovascular and somatic systems (Herbert and Cohen 1993; McEwen and Stellar 1993; Watkins and Maier 2005). Excessive stress-reactivity, including cardiovascular reactivity, may contribute to wear-and-tear on stress response systems (allostatic load), in turn leading to system dysfunction and disease (McEwen 1998; McEwen and Stellar 1993). This physiological wear-and-tear has been implicated in the experience of pain and discomfort (McEwen 2001) and stress-related diseases like hypertension have been associated with an increase in reports of somatic symptoms (Kristal-Boneh et al. 1998; Milne et al. 1985). Furthermore, several biological mechanisms linking systems' stress reactions have been identified. These include cytokine-mediated vascular inflammation (Lovallo and Gerin 2003), cortisol's attenuating effects on immune function (Kunz-Ebrecht et al. 2003), and stress-related influences on cell aging (Epel et al. 2006). Accordingly, there were reasons for thinking that reactivity to stress in terms of blood pressure increases might predict reactivity to stress in terms of somatic symptoms.

There are also suggestions that some individuals are more or less vulnerable to the effects of stress on bodily functioning (Funk 1992; Peterson et al. 1988). If so, individuals who are shown to be more physiologically reactive to stress in one bodily system may be more reactive to stress in other bodily systems as well. Few relevant studies have been conducted, particularly from a lab-to-life generalizability perspective (Schwartz et al. 2003). We hypothesized that individuals exhibiting higher levels of cardiovascular reactivity to a laboratory stress task would exhibit a greater degree of covariation between daily stress and daily somatic symptoms in an experience-sampling protocol. This is precisely so because a high degree of physiological reactivity to stress in one system should be observed in other systems as well. Results of this type would extend our knowledge of the correlates of laboratory cardiovascular reactivity measures, while also establishing a firmer biological basis for individual differences in stress-symptom reactivity.

Because neuroticism is viewed as a biasing factor in somatic symptom reporting (Pennebaker 1982, 2000), we also assessed this trait among a subset of participants in our

study. Following the results of a recent meta-analysis (Chida and Hamer 2008), we had no reason to predict that neuroticism would be associated with higher cardiovascular reactivity. Also, Brown and Moskowitz (1997) found that the use of an experience-sampling protocol eliminated symptom reporting biases due to the trait of neuroticism; we thus predicted a similar result in the present study. If so, and to the extent that results remain significant when controlling for neuroticism, the findings could not be viewed as resulting from the perceptual biases associated with neuroticism. In sum, we viewed the study as one capable of integrating hitherto distinct health literatures concerned with individual differences in cardiovascular reactivity and physical symptom responses to environmental stress.

Method

Participants

Fifty-six undergraduate participants first completed a laboratory cardiovascular reactivity session for course credit. Participants were asked to avoid drinking caffeinated beverages and to avoid smoking for at least 1 h prior to arriving for the study session. Participants were screened for cardiovascular diseases including hypertension. An average of 1 month after the cardiovascular reactivity portion of the study, participants completed daily diaries over the course of 15 days for monetary compensation (\$30) or additional course credit. About 1 week after the diary portion of the study a subset of participants returned to the laboratory in return for \$10 so that trait levels of neuroticism could be assessed.¹ The North Dakota State University institutional review board approved these study procedures.

Not all of the participants provided usable data. Three participants were dropped from further consideration due to incomplete blood pressure data and 6 were excluded because of poor compliance with the daily diary protocol, defined in terms of completing less than 60% of the daily reports. The final sample thus consisted of 47 individuals (28 female), who had an average age of 19.73 years ($SD = 2.34$), an average body mass index of 23.91 kg/m^2 ($SD = 3.56$), were primarily white/Caucasian (89%; Asian 9% and Black or African American 2%), and completed on average 87% of the daily surveys (i.e., 616 total reports). Thirty-five of the 47 participants returned for the second laboratory assessment session and for these individuals, then, we also had scores for the trait of neuroticism. The

¹ Other measures not relevant to the current study were assessed at this second laboratory session.

size of this sample and number of diary days is sufficiently powered for multivariate modeling (Tabachnik and Fidell 2007) and is comparable to the amount of data collected in past diary studies involving dispositions and health outcomes (Baker 2007).

Individual differences in cardiovascular reactivity

Speech task

The initial session assessed individual differences in cardiovascular reactivity in the first author's psychophysiology laboratory. All participant sessions were conducted individually by trained experimenters. Participants were told that the study involved bodily reactions to performing challenging tasks. The physiological recording equipment was explained to the participant, who then provided informed consent. After a participant was fitted with the recording equipment s/he was asked to sit and relax for a 10-min resting baseline period. The experimenter then left the room during this period to facilitate relaxation.

After the baseline period, the experimenter returned and informed participants that they were going to give a 5-min speech on an assigned topic to another purported undergraduate volunteer. The stressor, then, was social in nature, which has been advocated in understanding psychological contributors to cardiovascular reactivity (Kamarck and Lovallo 2003). To enhance the evaluative nature of the task and thereby increase its stressful nature (Hilmert et al. 2002), participants were told that the audience member would evaluate the speech and that experts in communication would evaluate a video recording of the speech at a later time. The participant was then given 5-min to mentally prepare a speech on the topic of euthanasia (Glynn et al. 1999).

The audience member arrived following the 5-min preparation period. This audience member, who was a research confederate, was seated directly in front of the participant, with a small table in between. The confederate was given a clipboard to take evaluative notes during the speech. In addition, the video camera was activated and pointed at the participant. The participant was then asked to deliver the prepared 5-min speech while the experimenter stood behind his/her left shoulder.

The experimenter and audience member responded during the speech in carefully coached ways. The confederate was instructed to periodically take notes and to exhibit signs of active evaluation, including showing facial expressions of interest and concern while occasionally muttering quietly. At 3 min into the speech, the experimenter told the participant to "move onto a new point" or "give more examples," depending on which admonition was viewed as more plausible at the time. Subsequent to

the speech, participants completed several self-report measures not of direct relevance to this report and were then debriefed.

Blood pressure reactivity

Systolic blood pressure and diastolic blood pressure were measured throughout the laboratory session using a Medwave Non-Invasive Blood Pressure Amplifier and Acq-Knowledge Software supplied by Biopac Systems, Inc. (Goleta, CA). The Medwave Amplifier measures systolic blood pressure and diastolic blood pressure approximately every 15 s using a wrist cuff placed on the participant's non-dominant arm. Participants were instructed to keep their wrist on the table in front of them at all times and to move it as little as possible in order to help ensure uninterrupted recording. The reliability and validity of the Medwave Amplifier has been established (Belani et al. 1999).

As is customary in cardiovascular reactivity research, to determine baseline blood pressure levels independent of any orienting effects, baseline systolic blood pressure and diastolic blood pressure values were calculated by averaging readings across the last 4 min of the 10-min baseline period. Stress-linked blood pressure levels were obtained by averaging readings across the 5 min of the speech task. In all cases, blood pressure data were inspected visually and then averaged using Mindware Technologies Software (Gahanna, OH). Reactivity scores were created by subtracting average systolic blood pressure and diastolic blood pressure baseline values from corresponding average stressful speech-task values. Because systolic blood pressure is considered the more reactive component of blood pressure (Obrist 1981), we considered it our primary measure of cardiovascular reactivity, although we also report results for diastolic blood pressure reactivity.

Stress and somatic symptoms in daily life

Overview

To establish that individual differences in cardiovascular reactivity have predictive value the daily diary portion of the study was conducted subsequent to assessing individual differences in cardiovascular reactivity. Individuals were instructed to log onto a website every night and complete daily reports for 15 consecutive days. Surveys were posted each evening (8 p.m.) and removed promptly the following morning (9 a.m.) to ensure that participants completed the surveys during the required time period. Daily e-mail reminders, sent each morning, sought to maximize compliance with the protocol. Somatic symptom reports were

collected before asking individuals about their daily stressors. This order is preferred because it precludes the possibility that relatively subjective somatic symptom reports could be biased by a prior consideration of the stressful events participants reported on the day in question. All scales were necessarily brief to ensure high levels of compliance given the demanding nature of the daily protocol.

Daily somatic symptoms

Somatic symptoms are diverse in nature, pertaining to difficulties related to fatigue, aches and pains, respiratory difficulties, digestive problems, etc. However, there are both psychometric and physiological reasons for thinking that many of these symptoms co-occur (Pennebaker 1982) and can be linked to poorer bodily functioning in general terms (Sharpe and Bass 1992). Accordingly, we sampled a subset of somatic symptoms that we viewed as commonly experienced, diverse in nature, and reflective of general bodily dysfunction. This brief symptom scale had been validated in previous research in our lab (Compton et al. 2008). Participants were specifically asked to rate the extent (1 = not at all; 5 = extremely) to which they felt each of four symptoms (faintness, headache, muscle aches, and nausea) on the day in question. The scale was reliable ($\alpha = 0.76$).

Daily stressors

Stress can be defined in event-related terms or in terms of subjective experiences (Lazarus 1991). There are benefits to defining stress in terms of events, particularly because doing so mitigates potentially tautological relations between stressful events and psychological reactions to them (Lazarus 2000). We assessed daily stressors in terms of a subset of items chosen from the Inventory of College Students' Recent Life Experiences (Kohn et al. 1990). The subset of items selected were those that we viewed as common in the everyday life of college students and was validated in previous research (Compton et al. 2008). Individuals were asked to indicate the extent (1 = not at all true today; 5 = very much true today) to which they encountered five stressors on the day in question (a lot of responsibilities, had a deadline to worry about, health problems or fatigue, not enough time to meet obligations, and too many things to do at once).² The scale was reliable ($\alpha = 0.87$).

² One of the daily stressors involved "health problems or fatigue". This stressor item clearly refers to bodily symptoms and might therefore inflate stress-symptom associations, though it is difficult to see how this would affect the moderation effects of systolic blood pressure reactivity observed. In any case, it was deemed important to

Trait differences in neuroticism

A subset of the participants ($n = 35$) returned to the fourth author's laboratory and, in this context, reported on their levels of the trait of neuroticism. Goldberg's (1999) neuroticism scale was used, as it has been associated with reliable and valid neuroticism scores in many previous studies (Robinson et al. 2006; Tamir and Robinson 2004). In specific terms, individuals were asked the extent to which 10 items reflective of high levels of neuroticism (e.g., "worry about things") generally characterize the self (1 = very inaccurate; 5 = very accurate). Goldberg's scale correlates highly with other neuroticism scales, such as the NEO-PI (Costa and McCrae 1992). Alpha was 0.86 in the present study.

Statistical analyses and their rationale

We first examined bivariate correlations among the study variables, including average levels of daily stress and somatic symptoms. We then used multilevel modeling procedures to examine associations between individual differences in cardiovascular reactivity and stress-linked somatic symptoms in daily life. Multilevel modeling techniques are more appropriate than typical linear regression procedures for examining hypotheses of the present type as such procedures appropriately correct for missing data (i.e., missing daily reports) and also partition within-subject and between-subjects sources of variance optimally (Fleeson 2007). In specific terms, we used the SAS PROC MIXED procedure (Singer 1998) to examine whether the slope of the relationship between the two within-subject level 1 variables—daily stress and daily somatic symptoms—changed as a function of the three between-subjects level 2 variables (i.e., systolic blood pressure reactivity, diastolic blood pressure reactivity, and the trait of neuroticism), each examined in separate analyses.

For multilevel model testing, variables were transformed in a manner recommended by the multivariate literature. Daily stress was person-centered prior to analyses (Enders and Tofighi 2007). Thus, an individual's stress score for a given day represented how much more/less stressful the particular day was relative to his/her average levels of daily stress. Additionally, the level 2 variables were z-scored prior to model testing. Finally, intercepts and slopes were treated as random effects in the multilevel

models, as they were hypothesized to vary between persons (Tabachnick and Fidell 2006).

Results

Descriptive statistics and zero-order correlations

Descriptive statistics and correlations among the measures (including average levels of daily stress and somatic symptoms) are reported in Table 1. The scores for neuroticism were close to previously reported norms (Goldberg et al. 2006), indicating good variability in the neuroticism measure. Average daily stress was near the midpoint of its response scale and somatic symptoms fell below the midpoint of its response scale. Both scales exhibited reasonable variability across participants and days, however (Table 1). Average levels of systolic blood pressure increased from baseline ($M = 131$ mmHg) to stressor ($M = 163$ mmHg), as did average levels of diastolic blood pressure (M 's = 76 and 97 mmHg, respectively), both P 's < .01.

Table 1 reveals that neuroticism was not associated with cardiovascular reactivity to stress. Neuroticism was a significant predictor of daily stress, but not daily somatic symptoms, replicating Brown and Moskowitz (1997). Individuals experiencing higher levels of daily stress reported higher levels of somatic symptoms, consistent with stress-related models of somatization (Sharpe and Bass 1992). Finally, in zero-order terms, cardiovascular reactivity measures did not predict average levels of somatic symptoms in daily life. The more important question, though, is whether individual differences in cardiovascular reactivity predict higher levels of somatic symptoms on stressful days.

Within-subject associations between stress and somatic symptoms

Consistent with the zero-order correlations reported above, multilevel modeling analyses revealed that none of the level 2 variables (i.e., systolic blood pressure reactivity, diastolic blood pressure reactivity, and the trait of neuroticism) significantly predicted average levels of daily somatic symptoms, P 's > .20. The within-subject association between daily stress and daily somatic symptoms was marginally significant, however [$b = .10$, $t(608) = 1.94$, $P = .05$]. In other words, high stress days were generally associated with the experience of more somatic symptoms. Thus, the present findings are consistent with the idea that stress does appear to play some role in potentiating somatic upset on a day-to-day basis (Brown and Moskowitz 1997; Charles and Almeida 2006). This is particularly true for certain individuals, however, as further results will show.

Footnote 2 continued

replicate the key multi-level model interaction with this item dropped from the computation of daily stress scores. Indeed, the interaction remained significant when doing so [$b = .08$, $t(567) = 2.26$, $P < .05$].

Table 1 Means and correlations among study variables

	1.	2.	3.	4.	5.	Average	SD
1. Neuroticism	–					2.64	0.73
2. Daily Stress	0.47*	–				2.24	0.62
3. Daily Somatic Symptoms	0.11	0.41*	–			1.74	0.55
4. Systolic Blood Pressure Reactivity (mmHg)	–0.20	–0.03	0.08	–		31.52	18.87
5. Diastolic Blood Pressure Reactivity (mmHg)	–0.19	0.05	0.15	0.90*	–	21.65	14.50

Note: Daily outcomes were averaged across daily reports for purposes of these analyses. All daily measures were made with reference to 1–5 response scales. Neuroticism analyses $n = 35$ and all others $N = 47$

* $P < .05$

Cardiovascular reactivity as a predictor of stress-symptom reactivity

We then tested our primary hypothesis, which involved a predicted cross-level interaction between cardiovascular reactivity and daily stress-symptom reactivity. For this analysis, systolic blood pressure reactivity was entered as a predictor of the within-subject relation between daily stress and daily somatic symptoms. As hypothesized, a significant interaction was found (Table 2). The positive nature of the association indicates that individuals who exhibited greater systolic blood pressure reactivity in a laboratory context also experienced a greater increase in somatic symptoms on high-stress days. Vice versa, lower levels of systolic blood pressure reactivity were associated with weaker stress-symptom relations.

To further clarify the nature of this interaction, somatic symptom means were estimated for individuals low (-1 SD) versus high ($+1$ SD) in systolic blood pressure reactivity as a function of low (-1 SD) versus high ($+1$ SD) levels of daily stress. These estimated means are displayed in Fig. 1. The figure suggests that individuals high, but not low, in systolic blood pressure reactivity experienced more somatic symptoms on more stressful days. To confirm this interpretation of the findings, simple slope comparisons (Aiken and West 1991; Fleeson 2007; Nezlek 2008) were performed. Among individuals high in systolic blood

pressure reactivity, there was a significant association between daily stress and daily somatic symptoms [$b = .22$, $t(567) = 3.23$, $P < .01$]. However, no such association existed among individuals low in systolic blood pressure reactivity [$b = -.02$, $t(567) = -0.23$, $P > .80$]. Thus, somatic responses to daily stress were exclusive to individuals displaying high systolic blood pressure reactivity in a laboratory stressor context.

When systolic blood pressure reactivity was replaced with diastolic blood pressure reactivity in a subsequent multilevel modeling analysis, the resulting interaction was not significant (Table 2). Thus, although systolic blood pressure and diastolic blood pressure reactivity were strongly correlated, systolic blood pressure reactivity was the stronger predictor of stress-symptom relations. Differential results of this type are consistent with prior suggestions that systolic blood pressure reactivity is the more sensitive measure of stress-reactivity when stressors are defined as short-term, episodic occurrences (Obriest 1981).

Recall that there was no zero-order correlation between systolic blood pressure reactivity and neuroticism. Thus, multilevel modeling findings involving systolic blood pressure reactivity cannot be due to shared variance with neuroticism. Additional analyses sought to further substantiate this point in within-subject terms. In a multilevel model, neuroticism did not interact with daily stress to predict daily somatic symptoms [$b = -0.10$, $t(462) = -1.61$, $P > .10$].

Table 2 Fixed effects for the moderating role of systolic (top panel) and diastolic (bottom panel) blood pressure in the daily stress-somatic symptom relationship

	Parameter estimate	Standard error	t -value
Results involving systolic blood pressure reactivity			
Intercept	1.74	0.08	21.62*
Stress	0.10	0.05	1.94†
Systolic blood pressure reactivity	.04	0.08	0.59
Stress \times systolic blood pressure reactivity	.11	0.05	2.41*
Results involving diastolic blood pressure reactivity			
Intercept	1.74	0.08	21.80*
Stress	0.10	0.05	1.98*
Diastolic blood pressure reactivity	0.08	0.08	1.03
Stress \times diastolic blood pressure reactivity	0.07	0.05	1.54

* $P < .05$; † $P = .05$

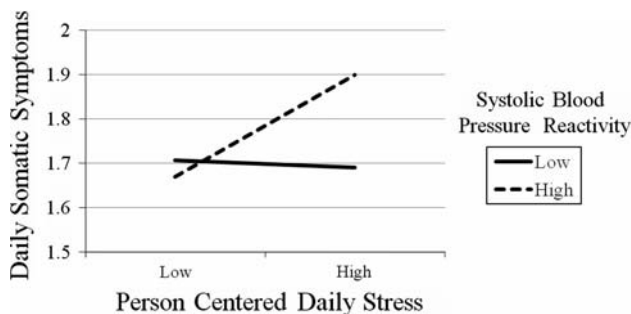


Fig. 1 Daily somatic symptoms as a function of daily stress and systolic blood pressure reactivity

Furthermore, controlling for neuroticism did not alter systolic blood pressure's moderation of stress-symptom associations, $P < .05$, despite the reduced sample size of this test. Thus, systolic blood pressure reactivity predicted stress-related somatization in daily life independent of neuroticism-linked influences.

Discussion

As hypothesized, individuals displaying greater systolic blood pressure reactivity to a laboratory stressor also exhibited a greater covariation of stress and somatic symptoms in daily life. Reactivity effects of this type were independent of the trait of neuroticism, which has been viewed as a nuisance factor in understanding the physiological basis of somatic symptoms (Pennebaker 2000). Diastolic blood pressure reactivity did not predict the extent to which stress and somatic symptoms covaried in daily life. This is not surprising given that systolic blood pressure reactivity has been shown to more closely track state-related variations in stress and active coping in such contexts (Obrist 1981). Additionally, individual differences in systolic blood pressure reactivity have been shown to be more reliable over time and more predictive of preclinical and clinical cardiovascular disease (Treiber et al. 2003). Thus, our findings converge with others in suggesting that individual differences in cardiovascular stress-reactivity may be better assessed in terms of systolic rather than diastolic blood pressure (Kamarck and Lovallo 2003). The present results therefore have significant implications for understanding the correlates of cardiovascular reactivity, somatic symptoms, and their interface.

A proximal perspective on individual differences in cardiovascular reactivity

Large cardiovascular reactions to stress can cause damage to the cardiovascular system, in turn contributing to the development of cardiovascular disease (Lovallo and Gerin

2003; Manuck et al. 1988; Treiber et al. 2003). In fact, even singular assessments of cardiovascular reactivity have predicted higher resting blood pressure and the development of hypertension a number of years later (Matthews et al. 1993; Ming et al. 2004). Given the successes of the cardiovascular reactivity model of cardiovascular health, it would seem useful and informative to identify proximal correlates of cardiovascular reactivity (Kamarck and Lovallo 2003), including those in the realm of daily stress-reactivity (Schwartz et al. 2003), to address issues of mechanism and process.

The present study is informative along such lines because we were able to show that individuals high in cardiovascular reactivity in the laboratory also exhibited stronger somatic symptom stress-reactivity in daily life. The present results may be viewed as providing support for a multi-systems view of individual differences in stress reactivity. That is, reactivity may involve a broad interface of body systems concerned with threat mitigation, likely involving the cardiovascular (Lovallo 2005), neuroendocrine, and immune systems (Watkins and Maier 2005). Indeed, the convergence of multiple physiological indicators of stress-reactivity has been shown to be especially predictive of adverse health outcomes (Cole et al. 2003). The covariation of cardiovascular and somatic symptom stress-reactivity observed in our study could be particularly problematic from the latter perspective.

As to why cardiovascular reactivity is linked to greater stress-symptom associations, several possibilities can be considered. This link could be due to common genetic factors. In favor of this idea, individuals clearly differ in their genetic predisposition to cardiovascular reactivity and to cardiovascular disease (De Geus et al. 2007; Selby et al. 1991). There also appears to be some genetic contribution to somatization responses to stress (Guze 1993; Kendler et al. 1995). For such a constitutional perspective to make sense in the present context, though, the relevant factor(s) would have to underlie cardiovascular reactivity tendencies in the laboratory and stress-symptom reactivity in everyday life. Our findings encourage investigation of this potential genetic interface.

The observed link between cardiovascular reactivity and somatic symptom stress-reactivity could be due to a negative emotional temperament, which has predicted stress-reactivity in self-report studies (Suls and Martin 2005). Arguing against this view, our assessment paradigms should be free of such influences. First, cardiovascular reactivity is an objective physiological measure. Second, we used a daily protocol assessing somatic symptoms to minimize the problems that can occur when somatic symptoms are reported in retrospective terms (Brown and Moskowitz 1997; Robinson and Clore 2002). Finally, and most importantly, the stress-reactivity trait of neuroticism

was not a significant predictor of systolic blood pressure reactivity in the present study, nor was it a significant predictor of stress-symptom relations in daily life.

Independent of emotional temperament, state emotional responses to stress may influence cardiovascular and physical symptom responses to stress. State negative affect including reports of anxiety and depression has been linked to somatic symptoms (Charles and Almeida 2006; Cohen et al. 1995). However, emotional states and physiological stress reactivity, particularly cardiovascular reactivity (Feldman et al. 1999) have not been strongly correlated in past research. Cardiovascular reactivity has been linked to effort exerted during a task (Hilmert et al. 2002; Obrist 1981; Wright and Kirby 2001). Thus, participants who have tendencies to exert more effort in response to stress would exhibit greater systolic blood pressure reactivity and perhaps experience more somatic symptoms. An important question for future research is whether cardiovascular reactivity and somatic stress reactivity are associated with common psychological stress responses.

Another possibility is that individual differences in the regulation of emotional responses could underlie our findings. Gross (1998) has long shown that the suppression of negative emotion is associated with physiological costs. Conversely, reappraising the meaning of a stressor reduces the physiological consequences of that stressful event (Gross and Thompson 2007). Gross and John (2003) have also shown that there are robust individual differences in tendencies toward emotional suppression versus reappraisal. Thus, it may well be that high levels of cardiovascular reactivity and somatic reactivity can be linked to an emotion regulation strategy of suppression, whereas low levels of cardiovascular reactivity and somatic reactivity can be linked to an emotion regulation strategy of reappraisal. On the basis of these considerations, it would be of particular value to link individual differences in bodily stress reactivity to the manner in which stressors are appraised, handled, and regulated in daily life (Schwartz et al. 2003; Smith et al. 2004).

Implications for understanding somatic symptom reports

There have long been two views of somatic symptom reports. One emphasizes the value of such reports in understanding a person's bodily state and potential dysfunctions (Barsky 2000). A second perspective views such symptom reports as belief-driven and potentially problematic indicators of underlying physiology (Pennebaker 2000). At least two features of the present study lead us to suggest that symptom reporting had a physiological basis. First, symptom reporting was stress-contingent in nature, in that we controlled for biases that could affect mean

reporting, but not within-subject changes across days (Charles and Almeida 2006). Second, we used an objective measure of physiological reactivity to stress—cardiovascular reactivity—to predict such symptoms. The proportion of symptom reactivity to stress predicted by cardiovascular reactivity, then, should be viewed in terms of a common biological substrate.

Stress-symptom associations, though replicated well enough, are not especially large in within-person daily process studies (Charles and Almeida 2006). The present results allow us to understand why this is the case. Specifically, our results suggest that there are many individuals—here, those low in cardiovascular reactivity—for whom stress and physical symptoms appear to be quite independent in daily life. Finally, the utility of cardiovascular reactivity as a pre-clinical probe of disease risk (Treiber et al. 2003) appears to extend into the somatic symptom realm as well.

Limitations and extensions

Our interest in daily stress and somatic symptomatology was of a molar type and it is important to use short scales in experience sampling protocols. Therefore, we were not able to identify discrete stressors or symptoms contributing to the patterns reported. Owing to the use of a brief but valid stressor scale, we cannot be sure which particular stressors resulted in somatic symptom reactivity among individuals high in cardiovascular reactivity. This concern is mitigated somewhat by the fact that daily hassles of diverse types seem to function similarly in terms of their cumulative impact (Chamberlain and Zika 1990). Owing to the use of a brief but valid somatic symptom scale, we cannot be sure which particular symptoms were elicited by stress among individuals high in cardiovascular reactivity. Although stress impacts multiple bodily systems, effects may be usefully differentiated in terms of whether they involve symptoms of pain, gastrointestinal problems, or respiratory issues (Charles and Almeida 2006).

In understanding the correlates of individual differences in cardiovascular reactivity, many studies have adopted a trait approach. That is, they have sought to determine whether self-reported traits like hostility or neuroticism can predict who will exhibit larger cardiovascular responses to laboratory stressors. The fruit of such studies has arguably been quite limited (Chida and Hamer 2008; Smith 1992). Instead, we may learn a lot more about individual differences in cardiovascular reactivity—including mechanisms and consequences—by examining its correlates outside of the laboratory (Kamarck and Lovallo 2003) and in daily life (Schwartz et al. 2003). From this perspective, our findings support the idea that cardiovascular reactivity is a bona fide individual difference variable quite independent of whether it correlates with self-reported traits or not.

Conclusions

Experiences of pain, nausea, and other somatic symptoms are pervasive among both clinical and non-clinical populations (Kroenke 2003). Our results indicate that stress is associated with such symptoms, but more importantly shows that this is particularly true among individuals exhibiting large cardiovascular responses to laboratory stress. The findings extend our understanding of cardiovascular reactivity, somatic symptoms, and what may be viewed as a more general individual difference tendency toward bodily reactivity to stress. In this respect, the findings integrate two health psychology literatures on stress-reactivity that were previously largely independent.

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