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Self-esteem and changes in heart rate during laboratory-based stress

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The relationship between self-esteem (SE), type of stressor, and fluctuations in heart rate was assessed in a sample of 59 college students (40 females, 19 males; with a mean age of 23.98 years (SE_M = 1.0)). SE was measured using the Rosenberg Self-Esteem Scale. The study assessed whether SE buffers the cardiovascular response to stress by comparing responses to two types of stressor: mental arithmetic and verbal memory. As predicted, an SE x stressor interaction was found (p = 0.039). High-SE participants found both stressors moderately stressful but low-SE participants found the mental arithmetic task particularly stressful. This is consistent with the view that mental arithmetic elicits a specific fear that exceeds that associated with other domains of performance. The present study suggests that such fear affects low-SE participants more strongly than high-SE participants. The interaction was statistically independent of potential physiological contaminants such as gender, age, smoking, and caffeine consumption.

Self-esteem (SE), broadly defined, refers to the extent to which individuals value themselves (Reber & Reber, 2001), and has long been identified as an important predictor of adjustment to stress (Lazarus & Folkman, 1987). It is inversely related to psychological indicators of stress and strain (Kivimäki & Kalimo, 1996), and accordingly may have a role in attenuating physiological responses to stress. If this is so, then a case can be made for the implication of low SE in the etiology of diseases that are affected by physiological responsivity to stress (such as, for example, hypertension). In addition, the issue of SE would be an important consideration for experimenters whose research depends on being able to predict physiological responses to challenging stressors. Direct experimental assessments of the physiological link between SE and stress have been rare, and have produced conflicting results. Men who report unstable SE over time appear to exhibit elevated cardiovascular responses to

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stress (Rasmussen, Willingham, & Glover, 1996); and elevated cardiovascular reactivity is characteristic of stressors that are designed to undermine participants' feelings of SE (Gendolla, 1999; Greenberg, Sheldon, Pyszczynski, Rosenblatt, Burling, Lyon, Simon, & Pinel, 1992). However, other studies have failed to establish any systematically predictive relationship between SE and cardiovascular reactivity, either in women (Rasmussen et al., 1996) or in mixed-gender samples (de Geus, Van Doornen, & Orlebeke, 1993).

Most of the evidence supporting an association between SE and cardiovascular variables is indirect, but nonetheless quite suggestive. For example, clinical groups characterized by high or low levels of SE, such as people with bulimia nervosa (Koo-Loeb, Pederson, & Girdler, 1998), subclinical eating-disorder symptomology (Koo-Loeb, Costello, Light, & Girdler, 2000), narcissism (Kelsey, Ornduff, McCann, & Reiff, 2001), psychopathy (Ogloff & Wong, 1990), and antisocial personality disorder (Raine, Venables, & Williams, 1995), each exhibit abnormal patterns of cardiovascular reactivity to stress. Furthermore, constructs closely related to SE, such as self-efficacy (Bandura, Cioffi, Taylor, & Brouillard, 1988; Bandura, Reese, & Adams, 1982; Wiedenfeld, O'Leary, Bandura, Brown, Levine, & Raska, 1990), hardiness (Maddi & Kobasa, 1984) and mastery (Karasek & Theorell, 1990) have also been associated with reduced physiological stress responses. In summary, there would appear to be some justification for suspecting that SE influences participants' responses to stressful situations in ways that are manifest in cardiophysiological reactivity. However, the relationship is yet to be tested directly.

If it is considered that SE constitutes the individual's representation of his or her ability to meet the demands of a given situation, then there exists the possibility that it is a somewhat *valid* representation of such ability. Indeed to date, authors have adopted the theoretical stance that SE buffers the effects of stress (e.g., Lazarus & Folkman, 1984), and researchers have accumulated a modicum of empirical support for the view (e.g., Rasmussen et al., 1996). Accordingly, therefore – assuming SE to be a valid indicator of the individual's coping abilities – its effect on the stress response should be contingent on the stressfulness of the situation being experienced. In other words, if the individual is faced with a situation that is very stressful then SE should be a strong predictor of the stress response. However, if the individual is faced with a situation that is less stressful, then SE should bear less of a relationship with the individual's physiological response. In this way, by indicating the individual's appraisal of available coping resources, SE should operate as a buffer against appraised stress. Two common stressors used in laboratory studies of physiological responsivity are mental arithmetic and verbal memory tasks. If these tasks differ in perceived

stressfulness, then this should emerge in an examination of the relationship between SE and the cardiovascular response elicited by either task.

Previous research suggests that most people possess a specific fear of mathematical performance that exceeds the fear associated with other domains of cognitive performance (Ashcraft & Kirk, 2001; Bandalos, Yates, & Thorndike-Christ, 1995; Schneider & Nevid, 1993; Wigfield & Meece, 1988). This is somewhat consistent with previous research showing mathematics tasks to elicit stronger physiological stress responses than other tasks (e.g., Hughes, 2001). Contrary to stereotype, mathematics anxiety does not appear to be related to gender when previous exposure to tuition is controlled for (e.g., Flessati & Jamieson, 1991; Hunsley & Flessati, 1988). Thus, for both men and women, a comparison of factors that differently influence stress responses to mathematics and other cognitive tasks should allow for the demonstration of a stress-buffering effect.

The present study was thus designed to assess the potential cardiovascular stress-buffering effects of SE. Cardiovascular stress responsivity was chosen as the target of the present research, as much previous work has implicated cardiovascular reactivity in the etiology of heart disease. The resulting theoretical position, referred to as the "reactivity hypothesis" (Light, Sherwood, & Turner, 1992) is not uncontroversial, but has received ongoing support from both animal (e.g., Kaplan, Manuck, Williams, & Strawn, 1993) and human (e.g., Blascovich & Katkin, 1993) studies.

In a between-groups design, participant's heart-rate responses to two stressors (a mental arithmetic task and a verbal memory task) were compared. Three predictions were made. Firstly and secondly, main effects for SE and type-of-stressor were predicted (*viz.*, that participants with low SE would exhibit greater heart-rate reactivity than those with high SE, and that heart-rate responses to the mental arithmetic task would be higher than responses to the verbal memory task). Thirdly, an SE × stressor interaction was predicted, whereby the difference in reactivity between the mental arithmetic and memory tasks would be more pronounced among participants with low SE than those with high SE.

METHOD

Participants. Sixty-two undergraduate psychology students (41 females, 21 males), who were attending an urban third-level college, were enlisted to take part in the experiment. The distribution of gender in the sample was proportional to that of the college population from which the

sample was drawn. One woman and two men who took part in the cardiovascular trials did not return their questionnaires, and so were deemed to have withdrawn from the experiment. This yielded a final study sample of 40 females and 19 males. All participants participated as part of an undergraduate course in experimental psychology, and accordingly received course credit. The ages of these participants ranged from 18 to 48 years, with a mean of 23.98 years (SE_M = 1.0). The sample contained 21 smokers, and 33 participants who identified themselves as coffee-drinkers. A male experimenter was used in 40 of the 59 trials, resulting in 18 trials where the genders of the experimenter and participant were the same and 41 trials where they were different. It was decided to assess the possibility that experimenter-participant gender congruence exerted a systematic effect on responses. Mean resting cardiovascular levels were compared as follows: male participant trials versus female participant trials, male experimenter trials versus female experimenter trials, and gender congruent trials (where the participant and experimenter were of the same gender) versus gender incongruent trials (where they were different). The only significant difference detected was in resting systolic blood pressure between male and female participants ($t = 4.44$, $df = 57$, $p = 0.009$). The relevant means were as follows: males = 132.11 mmHg (SE_M = 2.7), females = 117.31 (SE_M = 1.9). (The Bonferroni procedure was used to adjust p-values to allow for the number of comparisons.) This difference in systolic blood pressure (and the absence of a difference in heart rate) is consistent with norms established in previous research. More importantly, the absence of an effect of experimenter-participant gender congruence, although contrary to some speculation, is also consistent with previous research on the matter (e.g., Kleinke & Williams, 1994).

Assessments of Cardiovascular Reactivity and Self-Esteem. Cardiovascular parameters were measured using an Omron R1 Digital Blood Pressure Monitor, with a wrist-mounted cuff. Measures were obtained from participants' non-dominant arms. In general, readings were taking during two stages of the experiment: before, and during, the performance of a computer-based laboratory task. Two different tasks were presented to participants as laboratory stressors. In one group, participants (n = 23; 13 women, 10 men) were exposed to a verbal memory task, which was a version of the Digit Span sub-test from the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981). In each trial of this task, participants were presented with a string of digits, one at a time, on a computer monitor and immediately afterwards were required to enter the numbers they had seen, using a number-keypad. As in the Wechsler original, participants were first presented with three-digit strings; in every

second succeeding trial the presented string was increased in length by one digit. In the second group (n = 36; 27 women, 9 men), participants were exposed to a mental arithmetic task (Hughes, 2001), which involved a series of two- to five-digit number subtraction problems. After the presentation of each problem, participants were required to enter the correct answer using the keypad. Participants were first presented with a problem involving a three-digit number minus a two-digit number. The difficulty of the task was systematically altered (by increasing or decreasing the lengths of the problem numbers) as a function of the participant's success in performance. In both the verbal memory and mental arithmetic tasks, participants were given feedback regarding their success or failure after each test-item. In neither task was a forced-time response paradigm employed: participants were free to use as much time as they needed to respond to each item. This was intended to maximise participant-involvement in the task in question, by reducing the extent to which they were hurried into guessing.

The experiment required participants to enrol for one of two independent groups. The incidental nature of group formation led to a slight imbalance of participants between the two groups. The distribution of gender across the two groups was also imbalanced, but not to a degree that deviated from chance levels ($\chi^2 = 2.195$, df = 1, $p > 0.13$).

Heart-rate reactivity was defined as the arithmetic difference between the mean level of heart rate observed before the stressor and that measured during the stressor. Measures of systolic blood pressure (SBP) reactivity and diastolic blood pressure (DBP) reactivity were also computed, in the same way.

SE was assessed using Rosenberg's (1965) Self-Esteem Scale. The scale, consisting of ten self-descriptive statements requiring agreement ratings on a four-point scale from the respondent, is extremely widely used (Andrews & Brown, 1993). Although early factor analyses appeared to extract two separate underlying factors (e.g., Kaplan & Pokorny, 1969; Zeller & Carmines, 1980), more recent studies have identified a single common factor (e.g., Gray-Little, Williams, & Hancock, 1997), lending the instrument factorial validity. Scores can range from 10 to 40, with higher scores indicating higher SE. Although authors sometimes query the usefulness of the concept of SE (e.g., Street & Isaacs, 1998) or the feasibility of its psychometric assessment (e.g., Eiser, Eiser, & Havermans, 1995; Forster & Schwartz, 1994), many have found evidence of factorial validity in SE-measurement scales (e.g., Tomas & Oliver, 1999) and SE remains one of the most studied concepts in social psychology. Indeed, the Rosenberg scale has been shown to demonstrate sound psychometric qualities in several languages, including Estonian (Pullmann & Allik, 2000),

German (Ferring & Filipp, 1996), Spanish (Banos & Guillen, 2000), and Swedish (Forsman & Johnson, 1996).

Procedure. The experimental trials took place a small biofeedback laboratory in the psychology department of the participants' college. Participants, who were tested individually, sat with a computer, monitor, and blood pressure equipment on a table approximately 30 cm in front of them. In each trial, the experimenter sat alongside the participant operating the cardiovascular monitoring equipment. After an initial 10-minute resting period during which no cardiovascular readings were taken, the participant was asked to sit quietly for a three-minute (pre-test) period, during which two sets of readings were taken (after minute one and minute two respectively). The participants were then asked to perform the laboratory task for another three minutes, during which two more sets of readings were taken (at the same intervals). After the experimental trial, the participant was asked to complete a brief questionnaire, which contained the Rosenberg Self-Esteem Scale, together with questions on relevant biographical and health information. The overall experimental sequence (ten-minutes rest, three-minutes baseline, and three-minutes testing) was intended to provide a standardized measure of cardiovascular reactivity. It was typical of methodologies used in similar studies in the area, and was successful in eliciting strong increases heart rate.

RESULTS

Descriptive Statistics and Manipulation Checks. The mean SE score across the sample was 32.42 ($SE_M = 0.5$), with a median of 33 (scores ranging from 23 to 40). There was no significant difference in SE between the two stressor groups ($t = 1.15$, $df = 57$, $p > 0.25$). Mean levels of heart rate before and during each stressor are presented in Table 1. An initial manipulation check revealed that both the mental arithmetic ($t = 4.77$, $df = 35$, $p < 0.001$) and verbal memory ($t = 4.54$, $df = 22$, $p < 0.001$) tasks elicited significant increases in heart rate. However, the verbal memory task failed to elicit significant responses in either SBP or DBP. This may have reflected the nature of this task as an active (or myocardial), rather than passive (or vascular), stressor (the mathematics task would appear to have both active and passive qualities). As such, these cardiovascular dependent variables were excluded from subsequent inferential analyses.

Table 1. Mean cardiovascular levels before and during each laboratory stressor.

	Mental Arithmetic (n = 36)		Verbal Memory (n = 23)	
	Before	**During**	**Before**	**During**
Pulse[a]	76.25	88.56	74.46	83.67
	(1.92)	(3.17)	(3.53)	(3.50)
SBP[b]	120.79	133.19	124.09	124.50
	(1.99)	(2.78)	(3.40)	(3.18)
DBP[b]	76.61	84.18	76.54	78.63
	(1.58)	(1.93)	(1.97)	(2.17)

Note: [a] bpm; [b] mmHg; numbers in parentheses indicate standard errors

There were no significant differences between smokers and non-smokers with respect to baseline levels of heart rate ($p > 0.3$). There were also no significant differences in heart-rate baselines between coffee-drinkers and non-drinkers ($p > 0.2$ in each case). In addition, there were no significant differences in SE between smokers and non-smokers ($p = 0.269$) or between coffee-drinkers and non-drinkers ($p = 0.075$). Finally, given that a strong positive (Pearson) correlation existed between baseline and elevated levels of heart rate ($r = +0.67$, $df = 57$, $p < 0.001$), it was decided to enter this baseline as a covariate in subsequent analyses of heart-rate reactivity.

Self-Esteem and Stressor Differences in Heart-Rate Reactivity. A median split was performed on the Rosenberg Self-Esteem Scale scores, creating a dichotomous SE variable (High SE [n = 31; 19 women, 12 men] and Low SE [n = 28; 21 women, 7 men]). The distribution of gender across the two SE groups revealed no significant association between SE and gender ($\chi^2 = 1.27$, $df = 1$, $p > 0.25$). Independent t-tests revealed no significant differences between participants of high and low SE in baseline heart rate, elevated heart rate, or heart-rate reactivity ($p > 0.35$ in each case). Finally, a comparison of heart-rate reactivity elicited by the two stressors revealed no significant between-group differences ($t = 0.95$, $df = 59$, $p > 0.05$).

Self-Esteem, Stressor, and Heart-Rate Reactivity. In order to assess the effects of SE and condition on heart-rate reactivity, data were entered into a two-way (SE × Stressor) Analysis of Covariance (ANCOVA), with baseline heart rate entered as the covariate. The ANCOVA confirmed the absence of significant main effects for either SE or stressor, but did

reveal a significant SE × stressor interaction ($F(1,54) = 4.48$, $p = 0.039$; see Table 2). To further test the validity of this finding, a series of additional ANCOVAs was conducted, adding the following variables to baseline heart rate as covariates in each case: age, gender, smoking status (yes/no), and coffee drinking status (yes/no). Each of these analyses corroborated the observed effects, yielding significant SE × stressor interactions, but no main or interaction effects involving covariates.

Table 2. Summary of Two-Way Analysis of Covariance for effects of stressor and self-esteem on heart-rate reactivity, controlling for baseline heart rate.

Source	SS	df	MS	F	p
Baseline heart rate (covariate)	260.03	1	260.03	1.50	ns
Stressor	196.34	1	196.34	1.14	ns
Self-Esteem	56.47	1	56.47	0.33	ns
Stressor × Self-Esteem	775.46	1	775.46	4.48	0.039
Error	9341.35	54	172.99		

The nature of the SE x stressor interaction is illustrated in Figure 1. Whereas heart-rate reactivity in response to the mental arithmetic stressor was much higher for participants with low SE ($M = 18.00$ bpm, $SE_M = 4.53$) than for those with high SE ($M = 8.68$ bpm, $SE_M = 2.91$), heart-rate reactivity levels in response to the memory stressor in low- and high-SE participants were much closer (although, were higher for participants with high SE [$M = 12.11$ bpm, $SE_M = 2.69$] than for those with low SE [$M = 7.36$ bpm, $SE_M = 2.81$]). Independent t-tests conducted on these two pairs of means revealed that the latter difference between stressors among the high SE group was clearly non-significant ($t = 0.70$, $df = 29$, $p = 0.489$), whereas the difference between stressors among low SE participants approached significance ($t = 2.00$, $df = 29$, $p = 0.056$). To further explore the SE × stressor interaction, equivalent independent t-tests were conducted on elevated levels of heart rate (rather than heart-rate reactivity). These tests revealed that – again – the difference between stressors among participants with high SE was clearly non-significant ($t = 0.92$, $df = 29$, $p = 0.37$). However, in this analysis the between-stressor differences among participants in the low SE group was statistically significant ($t = 2.41$, $df = 29$, $p = 0.02$). Finally, the corresponding analysis using baseline levels of heart rate (rather than elevated levels or heart-rate reactivity) showed no

significant between-stressor differences in either SE group ($p > 0.3$ in both cases). In combination, these results strongly imply that the observed SE × stressor interaction resulted mainly from the large between-stressor differences in heart-rate reactivity among low SE participants, and not from differences among high SE participants.

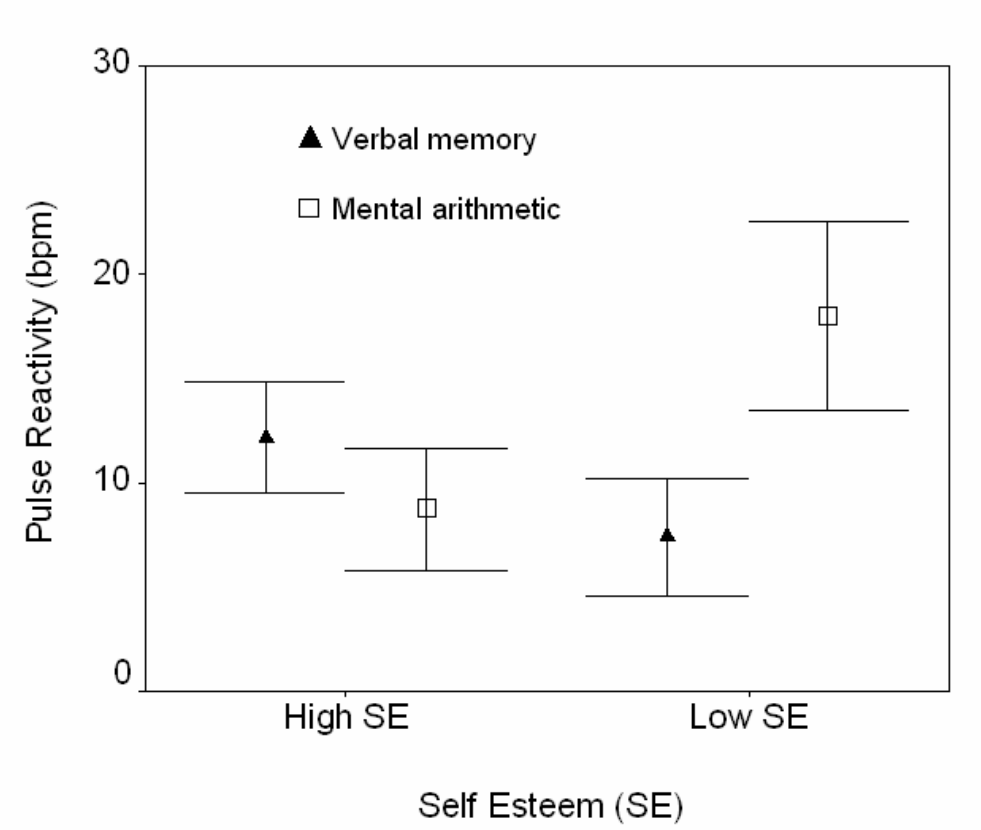

Figure 1. Mean heart-rate reactivity to each stressor for participants high and low in self-esteem, demonstrating a statistically significant SE x stressor interaction (error bars indicate the range ± 1 standard error). Triangles represent the means for the verbal memory task; squares represent those for the mental arithmetic task.

DISCUSSION

The key finding of the present study suggests vivid individual differences – associated with SE – in people's cardiovascular responses to psychological stressors. Clear empirical support was found for the

prediction that SE would moderate the apparent stressfulness of different stressors. Specifically, the difference in elevations in heart rate between mental arithmetic and memory was particularly exaggerated in low SE participants, but was indiscernible in high SE participants. A thorough scrutiny of the results reveals that the observed effects were independent of potential contaminating factors such as gender, smoking, caffeine consumption, and age. Contrary to predictions, neither SE nor type-of-stressor exerted an independent effect on heart-rate reactivity, thereby highlighting the importance of their inter-relationship. Such findings are important for two reasons. Firstly, they shed light on the interaction between feelings of self-worth and the cardiovascular stress response, which is in turn important for clarifying aspects of the psychosomatic etiology of heart disease. The heart-rate responses of participants in this study were modest and were not in themselves likely to precipitate a psychosomatic illness. However, if SE were found to buffer the effects of chronic environmental stressors, then an impact on lifetime cardiovascular disease risk could be anticipated. Accordingly, continuing research in the direction (as well as the extent) of effects of psychological variables on physiological stress responses will be important in contributing to a fuller understanding of psychosomatic etiological mechanisms.

Secondly, the present findings draw attention to specific extraneous variables that impinge on the design of research studies in cardiovascular reactivity. By elaborating on what individual difference characteristics influence participant's physiological responses to laboratory-based stressor tasks, the present findings help researchers decide what factors might be important to consider as possible target variables or covariates.

A number of features in the design of the present study should be borne in mind when attempting to evaluate the usefulness of its findings. Firstly, a between-participants design was used, which fails to exclude the possibility that idiosyncratic between-group differences aversely influenced the results. The observed interaction between SE and laboratory stressor would be more explicitly examinable in future research if high- and low-SE participants perform both types of stressor on different occasions. However, the extent to which the participant's familiarity with the laboratory procedure in a repeated-measures design would serve to contaminate their physiological stress responses is unclear. Secondly, information on potential covariates was based on self-report data, which calls into the question the validity of information gathered on participants' smoking, coffee-drinking, and age, each of which could be distorted due to social desirability effects. However, it is unlikely that social desirability distortions of these variables systematically operated to eliminate their effects on the key relationships under scrutiny.

One related issue that might be important is that participants were classified as smokers and non-smokers, or coffee-drinkers and non-drinkers, without regard for the relative extents of either habit where they occurred. The nature and significance of the effects of SE and stress on heart rate may well be influenced not only by the presence or absence of users of tobacco and caffeine in the population, but also by the relative amounts of tobacco and caffeine used by different people. Furthermore, such categorisations do not reflect the varying personal histories of smoking and coffee-drinking in the population. People's statuses as former smokers or former coffee-drinkers may well proved relevant to interactions involving SE. This is particularly true with regard to smoking, where levels of SE may determine the success of an individual's attempts to quit smoking. As such, there may be a fundamental and relevant difference in personality (and SE) between participants classified as non-smokers and participants classified as former smokers. Such a difference may prove fruitful in future studies of SE and physiological stress responses relevant to health.

A similar question can be raised regarding the appropriate measurement of SE. Some authors have questioned whether the Rosenberg scale accurately measures the trait aspect of SE, and suggest that its measurements might be confounded by participant mood variables (Robertson & Simons, 1989). There is some evidence that interview-based assessments of SE are less vulnerable to state-type mood effects (Andrews & Brown, 1993). However, other studies have found the Rosenberg scale to be a highly stable measure of trait-SE (e.g., Miller, Kreitman, Ingham, & Sashidharan, 1989). This issue is important given that in this study, SE was measured shortly after the participant had taken part in the stressful laboratory task. This was to prevent the pre-test priming of participants as to the purpose of the experiment. If the Rosenberg scale is influenced by situational factors, it is possible that participant's SE scores were affected by their performance on the task. However, the apparent differential in stressfulness between the two stressors was not reflected in participants' SE scores in the present study, where no significant between-group difference was observed. This suggests that task performance did not influence self-reported SE.

On a technological level, future research might better explore cardiovascular stress responsivity through the assessment of hemodynamic profile, given that some researchers have raised significant questions about the cardiac and vascular nature of the stress response (e.g., Gregg, James, Matyas, & Thorsteinnson, 1999).

In conclusion, the findings of the present study are consistent with research that has observed associations between physiological reactivity and indirect markers of SE (such as hardiness, self-efficacy, and mastery). They

are also consistent with laboratory experiments that have attempted to manipulate participants' SE through the provision of false feedback with regard to task performance, subsequently noting corresponding changes in cardiovascular reactivity (e.g., Gendolla, 1999; Greenberg et al., 1992). Furthermore, they are consistent with research that shows that personality and psychiatric categories characterised by low or unstable SE are also characterised by irregular patterns of psychophysiological responsivity (e.g., Kelsey et al., 2001). Such an accumulation of evidence is important to consider when seeking to explain the onset of cardiovascular disease in the context of biopsychosocial mechanisms. The conceptualisation of the stress response as a process wherein the individual weighs up their own capacity to deal with the challenges that lie ahead (e.g., Lazarus & Folkman, 1984), is one where a systematic analysis of SE and cardiovascular reactivity can make a direct contribution. An individual's SE may predict the severity of their response to stress, and so may be of central importance (under the cardiovascular reactivity hypothesis; Light et al., 1992) to their susceptibility to coronary atherosclerosis.

In addition, these findings may also help explain the failure of some previous studies to detect relationships between SE and cardiovascular reactivity (e.g., de Geus et al., 1993), by introducing the possibility that these authors' choices of laboratory stressors in themselves diminished the chances of detecting statistically significant effects. They may also be helpful in explaining the inconsistency in cardiovascular reactivity research based on a wide range of psychological and psychosocial variables. Although the internal validity of each study may well be appropriate (presumably the variations in participants' levels of SE were evenly distributed among experimental groups by virtue of random sampling), the influence of SE $\times$ stressor interactions becomes relevant when the results of a series of studies using different laboratory stressors are compared. The results of those studies that appear to upset the trend of other research may stem from the choice of a relatively unusual laboratory stressor, or some other methodological aspect to which SE is relevant. A thorough understanding of the relationships between SE, stressors, and target variables may help bring order to what currently appears to be a confused epidemiological picture.

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