

Cardiovascular and endocrine reactivity in older females: Intertask consistency

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Abstract

Age-related structural and functional changes in the cardiovascular, sympathoadrenomedullary (SAM), and hypothalamic-pituitary-adrenocortical (HPA) systems may affect the ability to reliably identify individual differences in response to stress. Heart rate, preejection period, respiratory sinus arrhythmia, respiratory rate, norepinephrine, epinephrine, adrenocorticotrophic hormone, and cortisol were assessed in 64 healthy older women (mean = 67 years) in response to a mental arithmetic and public-speaking task. All cardiovascular and endocrine measures changed significantly during the tasks. All measures were consistent across the two tasks ($r_{s,s} = .50$ to $.97$). Moreover, a majority of women in this sample exhibited cross-task consistency in the relative activation of the autonomic, SAM, and HPA systems (i.e., response profiles). Further research is recommended to examine the significance of consistent individual differences in response profile.

Descriptors: Cardiovascular reactivity, Endocrine reactivity, Intertask consistency, Older women, Response profiles

Individual differences in cardiovascular reactivity to acute laboratory stressors have been shown to predict health risk. In an important prospective study, Light, Dolan, Davis, and Sherwood (1992) reported that heart rate (HR) reactivity to a reaction time task predicted blood pressure levels 10 to 15 years later even after taking into account traditional risk factors such as resting blood pressure levels and parental history of hypertension. However, HR changes may arise from increased sympathetic activity, decreased vagal activity, or a combination of sympathetic and vagal activity. Moreover, adverse cardiovascular consequences (e.g., hypertension, ischemias, and arrhythmias) have been associated specifically with elevated sympathetic activity (Pagani et al., 1991),

whereas protection against arrhythmias is afforded by increased vagal activity (Kristal-Boneh, Raifel, Froom, & Ribak, 1995).

Prior research employing young adult participants has demonstrated consistent individual differences in HR reactivity across mild psychological stressors (Berntson et al., 1994; Gramer & Huber, 1993). In an extension of these findings, young adults exposed to three psychological tasks (mental arithmetic, speech, and a reaction time task) were observed to differ reliably in the autonomic origins of HR reactivity (Berntson et al., 1994). That is, although the relative activation of the sympathetic and parasympathetic nervous systems differed across individuals, the autonomic response mode was consistent within individuals across tasks (mean intertask correlation, $r = .76$).

Among the elderly, the intertask consistency of individual differences in autonomic response mode is less well established than among young adults. In the current study, we were interested in differentiating elderly individuals on the basis of noninvasive measures of sympathetic and vagal activity, namely HR, preejection period (PEP) and respiratory sinus arrhythmia (RSA). An important consideration in the use of PEP as an index of sympathetic activity is its sensitivity to nonneural input such as changes in preload and afterload. In the elderly, arterial and venous compliance decrease due to a thickening of the endothelial linings and a decline in the smooth muscle mass of blood vessels (Berne & Levy, 1997). The consequent elevations in systolic and diastolic blood pressure levels contribute to increased afterload. In addition, thickening of the ventricular myocardium and shrinking of the

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cardiac ventricles among the elderly diminishes cardiac output; the consequent decrease in venous return to the heart contributes to decreased preload (Berne & Levy, 1997). Both increased afterload and decreased preload lengthen PEP independent of neural input, thereby posing a potential challenge to detecting the covariation of PEP changes with sympathetically mediated changes in HR.

PEP is also affected by changes in the sensitivity of the end-organ to neural stimulation. Despite increased sympathetic nerve activity, as reflected in elevated plasma norepinephrine in response to stress (Esler et al., 1995), heart rate reactivity to mental stress, orthostasis, and exercise is reduced in the elderly (Berne & Levy, 1997; Esler et al., 1995). Diminished adrenergic responsiveness has been proposed as at least partially responsible for this effect (Esler et al., 1995). To the extent that stress-induced PEP changes are constrained by reduced responsivity to sympathetic activity, the probability of differentiating the elderly on the basis of PEP responsiveness is likewise limited.

In the current study, the consistency of individual differences in stress-induced changes in HR, PEP, and RSA among older women was assessed across a mental arithmetic and a public-speaking task. In previous research, these two tasks have been shown to evoke significant increases in heart rate and blood pressure (Becker et al., 1996; Berntson, Cacioppo, & Fieldstone, 1996; Saab, Matthews, Stoney, & McDonald, 1989), with most participants exhibiting an underlying autonomic response consisting of sympathetic activation and vagal withdrawal (Berntson et al., 1994; Cacioppo et al., 1995). We extended our evaluation of the intertask consistency of sympathetic and vagal activity by assessing cross-task consistency of autonomic response profile to examine whether individuals exhibited consistency in relative activation of the sympathetic and vagal branches of the ANS.

Mental arithmetic and public speaking, as well as other similar psychological tasks, have also been shown to elicit significant increases in plasma levels of the catecholamines epinephrine and norepinephrine (Becker et al., 1996; Cacioppo et al., 1995; Oleshansky & Meyerhoff, 1992; Saab et al., 1989). In some studies, brief psychological stressors have also elicited hypothalamic-pituitary-adrenocortical (HPA) activity manifest in elevated levels of ACTH (Cacioppo et al., 1995) or cortisol (McCann et al., 1993). Although glucocorticoid secretion is essential to the mobilization and regulation of bodily resources, the increased cortisol levels seen with social stress and aging may exacerbate atherosclerosis and diabetes, depress immune function, and potentiate brain damage and cognitive impairments (McEwen & Stellar, 1993). Furthermore, cortisol has been shown to inhibit the reuptake of catecholamines, thereby potentiating the effect of norepinephrine on heart rate and contractility (Berne & Levy, 1997; Kvetnansky et al., 1995). The health implications of exaggerated or prolonged activity of the HPA axis thus reinforce the importance of identifying individual differences in HPA reactivity to acute psychological stressors. Nevertheless, to date, evidence of stable HPA (ACTH, cortisol) and sympathoadrenomedullary (SAM; epinephrine, norepinephrine) responses across brief psychological stressors is lacking (e.g., Berger et al., 1987). In accord with our evaluation of autonomic response consistency, we examined not only the intertask consistency of separate endocrine responses but also the consistency of individual differences in endocrine response profile (i.e., relative activation of SAM and HPA systems) across the two tasks.

Finally, an additional goal was to explore the nature and pattern of interrelationships among indices of sympathetic, vagal, SAM, and HPA responses to acute psychological stressors in a sample of

older women. Specifically, we attempted to replicate prior research linking sympathetic and HPA activity (Cacioppo et al., 1995; Uchino, Cacioppo, Malarkey, & Glaser, 1995) by evaluating the relationship between changes in HR and PEP with changes in cortisol and ACTH in an older sample. Further, we extended our evaluation of response profile consistency by examining the consistency of individual differences in the activation of the sympathetic system relative to activation of the HPA and SAM systems.

Method

Participants

Sixty-four women (50 Caucasian, 11 African American, 3 not designated) who were part of a longitudinal project participated in this study over a 3-month period. Women qualified to participate (a) were in good psychological and physical health and were normotensive; (b) had no history of psychological disorder or chronic illness;

(c) were not taking beta-blockers; (d) consumed, on average, less than 10 alcoholic beverages per week; (e) exercised, on average, less than 10 hours per week; and (f) were not math, speech, or needle phobic. Participants were paid \$75.00 for 3.5 hours of participation in the study.

The women ranged in age from 47 to 83 years ($M_{\text{age}} = 67.0$, $SD = 8.2$). Fifty (82%) were Caucasian, 11 (18%) were African American, and three women did not provide race information. All women were postmenopausal (menopausal status was confirmed in the younger women if they had had a hysterectomy) and 31% ($n = 20$) were receiving estrogen replacement therapy (ERT). The average height of the participants was 159.8 cm ($SD = 21.3$), the average weight was 70.4 kg ($SD = 16.8$), and the average body mass index (BMI) was 27.1 ($SD = 5.2$).

Procedure

In preparation for the study, participants were asked (a) to reschedule their appointment if they became ill or experienced a major negative life event (e.g., death in the family); (b) to refrain from ingesting anti-inflammatory agents, antihistamines, or alcohol during the 24 hr preceding the test day; (c) to refrain from exercise the day before the study; and (d) to refrain from eating, drinking tea or coffee, or smoking after midnight the night preceding their scheduled appointment.

Testing began for all participants at approximately 8 a.m. in the General Clinical Research Center of the Ohio State University Hospital. A respirometer was secured around the participants' lower thorax, and spot electrodes were attached for impedance cardiography. The two outer current electrodes were placed over the fourth cervical vertebra and the ninth thoracic vertebra. The two inner recording electrodes were placed 4 cm above the clavicle and over the sternum at the fourth rib. (See Cacioppo et al., 1994, for details on electrode placement.) With the participant in a supine position, a 20-gauge, indwelling catheter was inserted into an antecubital vein. During an ensuing 30-min adaptation period, an innocuous set of questionnaires was verbally administered. At the end of the adaptation period, participants were seated in an upright position and a blood sample was collected to assess baseline endocrine activity. This was immediately followed by the recording of 6 min of baseline cardiovascular and respiratory activity.

After the baseline measures, participants received instructions for both tasks, and any questions about the tasks were answered. Cardiovascular and respiration measures were collected continuously throughout each 6-min task. A blood sample was collected

for endocrine assays directly after each task. The order of the tasks was counterbalanced across participants.

One of the challenges of assessing the consistency of cortisol responsiveness is the tendency of cortisol responses to habituate with repeated exposure to the same stressor (Rose, 1984; but see Kirschbaum et al., 1995). Changes in stimulus context have been shown to diminish the likelihood of cortisol response habituation (Rose, 1984). McCann et al. (1993), for example, found that during a 2-hr experimental session, elevations in plasma cortisol were maintained at levels significantly higher than those of a control session by gradually increasing the difficulty of the task (serial addition). In the current study, two distinct and brief psychological stressors, mental arithmetic and public speaking, were employed to minimize habituation and enhance the ability to detect stable individual differences in HPA responsiveness.

Speech stressor. The speech stressor was based, in principle, on the task developed by Saab et al. (1989). Participants were told to imagine they had been falsely accused by an obnoxious bill collector of failing to pay a medical bill, and that they were to prepare a speech to be given in their defense when speaking with the bill collector's supervisor. They were also told their speech was being recorded and would be compared with the speeches of others in the study. Participants were given 3 min to prepare and were then expected to present their speech for a full 3 min.

Math stressor. The math stressor consisted of serial subtraction for six 1-min epochs customized to each individual's ability as has been described previously (Cacioppo et al., 1995). The minuend and subtrahend were changed at the beginning of each minute in order to maintain task engagement. Participants were asked to work as quickly and accurately as possible, and were prompted at the beginning of even numbered minutes to speed up their responses.

Measures

The electrocardiogram (ECG) signal, basal thoracic impedance (Z_0), and the first derivative of the impedance signal (dZ/dt) were obtained with a Minnesota Impedance Cardiograph (model 304B; Surcom, Inc., Minneapolis, MN). Respiration (BR) was recorded using an EPM systems amplifier (EPM Systems, Midlothian, VA). Digitization rates, filtering information, and data reduction procedures have been described previously (Cacioppo et al., 1995).

Minute-by-minute means were calculated for each cardiovascular measure. HR was quantified as the number of R-spikes in the ECG waveform in each minute. PEP was quantified as the time interval in milliseconds from the onset of the ECG Q-wave to the B-point of the dZ/dt wave (Sherwood et al., 1990). RSA was quantified as the natural logarithm of the variance of the heart period pattern within the respiratory frequency range (i.e., 0.12 to 0.40 Hz). BR was quantified as the number of breaths per minute. The minute-by-minute means were averaged over the 6-min baseline and each 6-min stressor to increase reliability. Based on the recommendation of Berntson et al. (1994), interbeat interval (IBI) data were also acquired (from the ECG waveform) as an alternative measure of cardiac chronotropy.

Epinephrine (EPI), norepinephrine (NEPI), adrenocorticotropic hormone (ACTH), and cortisol (CORT) were assayed from the blood samples drawn at baseline, after the first stressor, and again after the second stressor. Plasma catecholamine concentrations (EPI and NEPI) were determined by high performance liquid chromatography (HPLC) using a Waters system with an electrochemical detector. The sensitivity of this HPLC system for EPI is

10 pg/ml and for NEPI is 20 pg/ml. With this system, intra- and interassay coefficients of variation are 12% for EPI and 7% for NEPI. Plasma ACTH concentrations were determined using an immunoradiometric method (Nichols Institute, Capistrano, CA). This method has intra- and interassay coefficients of variation of less than 10%, and a sensitivity of 1 pg/ml. Plasma cortisol concentrations were determined using a fluorescent polarization technique that measures both free and protein-bound circulating cortisol (TDX-Abbott Lab, Chicago, IL). This method has intra- and interassay coefficients of variation of less than 10%.

Data Analysis

To minimize the impact of measurement errors and extreme scores, values that exceeded three standard deviations from the mean were not included in analyses. To retain statistical power, this procedure was conducted on each variable independently, resulting in varying degrees of freedom for each analysis. Approximately half of the variables did not require any values to be excluded; among remaining variables, an average of 3% of values were excluded for any given measure (mode = 1.6%, range = 1.6–7.8%). Furthermore, degrees of freedom were adjusted for measures in which technical problems resulted in incomplete data.

Repeated measures ANOVAs with three levels for period (baseline, speech task, math task) were conducted to assess the effects of the psychological stressors on autonomic and endocrine activity. (The Huynh-Feldt epsilon was included to adjust the degrees of freedom in the event of violations of the sphericity assumption.) To ascertain that each task evoked a physiological response, pairwise comparisons were performed adjusting the alpha level to .025 to compare each task level to baseline. ANOVAs were conducted using simple change scores (task level minus initial baseline level) to determine whether autonomic and endocrine responses to the two stressors differed significantly. Although simple change scores are easier to interpret, residualized change scores may be more appropriate in the event of baseline dependent responses (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991). Only RSA, respiration, and NEPI measures exhibited significant correlations between baseline levels and change scores ($r_s = -.46, -.66, \text{ and } -.39$, respectively). Because analyses using residualized change scores revealed a similar pattern of results as analyses using simple change scores, and because simple change scores are generally deemed preferable, only simple change score analyses will be discussed further.

The possibility that race, ERT, and BMI may have exerted a differential influence on physiological activity was evaluated in a series of analyses. Although our predominantly Caucasian sample may have limited our ability to detect race differences, the only race differences noted were a lower basal cortisol level among the African American than the Caucasian women, $M_s = 7.9$ vs. 11.1 , $p < .01$, and a greater decrease in PEP to the tasks among the African American than the Caucasian women, $M_s = -9.9$ vs. -3.1 , $p < .01$. The use of estrogen replacement therapy yielded only two significant effects. Analyses of variance revealed that ACTH levels across all periods (baseline, 2 tasks) were lower among women using ERT, $F(1,50) = 4.46$, $p < .05$, than those not using ERT. In addition, EPI increased to a greater extent in those on ERT than those not, $F(1,54) = 6.07$, $p < .05$. Finally, with one exception, cardiovascular and endocrine measures were not significantly influenced by body mass index: analyses of covariance revealed a significant effect of BMI on norepinephrine levels across all periods, $F(1,56) = 4.56$, $p < .05$. This was reflected in significant negative correlations of BMI with baseline norepinephrine level,

$r = -.27, p < .05$, and average norepinephrine task level, $r = -.29, p < .05$. Because race, ERT, and BMI had minimal impact on the physiological measures, these variables will not be discussed further.

Pearson correlations were used to assess the consistency of autonomic and endocrine response to the two stressors, followed by Spearman's correlations to determine the consistency of the rank ordering of individuals across tasks on the magnitude of reactivity. To compare these consistency estimates with intratask reliability estimates, Cronbach's alpha was used to estimate the reliability of the cardiovascular measures across the six 1-min means in each task. Responses to the two tasks were then averaged, and additional Pearson correlations were performed on the aggregate change scores as well as the task-specific change scores to assess the degree and nature of the association among measures of cardiovascular activity, among measures of endocrine activity, and between measures of cardiovascular and endocrine activity.

To evaluate the consistency of response profiles, a median split of reactivity scores on each task and measure was used to create "relatively activated" (scores above the median) and "relatively deactivated" (scores below the median) response categories. (An exception to the labeling scheme was made for PEP, in which case scores above the median were labeled "relatively deactivated," and scores below the median were labeled "relatively activated.") Response categories for each measure were paired to generate response profiles. Pairing of response categories resulted in four response profiles for each task. For example, the four autonomic response profiles based on PEP and RSA, were (1) relatively activated PEP, relatively deactivated RSA, (2) relatively activated PEP, relatively activated RSA, (3) relatively deactivated PEP, relatively deactivated RSA, and (4) relatively deactivated PEP, relatively activated RSA. To minimize the probability of Type I errors, a select set of profile pairings were formed: PEP-RSA to evaluate sympathetic-parasympathetic profiles; PEP-CORT to evaluate sympathetic-HPA profiles; and EPI-CORT to evaluate SAM-HPA profiles. Cohen's K coefficient was used to evaluate the cross-task consistency of response profiles as cross-tabulated in a 4 (pairings

of response categories in Task 1) \times 4 (pairings of response categories in Task 2) matrix.

All statistical tests were performed using SPSS for Windows (Version 8.0, SPSS, Inc.) with an alpha level of .05.

Results

Initial ANOVAs were performed to assess whether the psychological stressors elicited significant changes in cardiovascular and neuroendocrine activity in these older women. All analyses were collapsed across task order because in no case did order produce a significant main effect, all $ps > .10$.

Task-Induced Autonomic Activity

Effect of period. The results for cardiovascular activity are summarized in Table 1. Significant main effects for period (baseline, speech task, math task) were observed for HR, RSA, PEP, and BR. Pairwise comparisons confirmed that each task evoked an overall pattern of reciprocal sympathetic activation and vagal withdrawal as evidenced by significantly increased HR, diminished RSA, and diminished PEP relative to baseline levels. The decrease in RSA was not simply attributable to vocalizing, because it decreased an equivalent amount during speech preparation, $-.51$, as during speech delivery, $-.55, t(55) = .38, p = .71$. Furthermore, to ensure that the observed changes in RSA were not merely secondary to alterations in respiratory frequency during the tasks, independent of changes in vagal tone (Hirsch & Bishop, 1981), we applied an adjustment based on the Hirsch and Bishop data (see Berntson et al., 1997, footnote 7). After adjusting for any task-related changes in respiration that might affect RSA, RSA was nevertheless found to decrease during the tasks, $F = 7.522, df = 2, 102, p = .001$.

Effect of task. Table 1 includes mean change scores for each autonomic measure and task. Consistent with some previous research (Berntson et al., 1994), ANOVAs comparing the magnitude of the changes induced by each task revealed that the speech task

Table 1. Mean (SEM) Cardiovascular Activity as a Function of Task and Period

Measure ^a	df	Base	Math	Speech	F-ratio	H-F Epsilon
HR (beats/minute)	2, 102	64.42 (1.18)	72.47† (1.39)	74.84† (1.54)	82.829**	.913
HR change	1, 50		8.01 (0.71)	9.71 (0.79)	5.337*	
PEP (ms)	2, 94	94.45 (2.86)	90.03† (3.01)	87.63† (3.26)	18.885**	.853
PEP change	1, 43		-2.90 (0.70)	-4.68	5.143*	
RSA (log units)	2, 112	5.244.79† (0.14)	4.70†	18.209** (0.14)	.999 (0.14)	
RSA change	1, 55		-0.50 (0.09)	-0.56 (0.10)	0.449, n.s.	
BR (breaths/minute)	2, 116	14.98 (0.37)	18.84† (0.34)	17.13† (0.30)	56.842**	.843
BR change	1, 58		3.86 (0.44)	2.15 (0.34)	33.298**	

^aHR = heart rate; RSA = respiratory sinus arrhythmia; BR = respiratory rate; PEP = pre-ejection period. SEM = standard error of the mean.

** $p < .01$, †individual task significantly different than baseline ($p < .05$), n.s. = not statistically significant ($p > .05$).

Table 2. Mean (SEM) Endocrine Activity as a Function of Task and Period

Measure ^a	df	Base	Math	Speech	F-ratio	H-F Epsilon
EPI (pg/ml)	2, 112	20.40 (1.03)	26.65† (1.69)	27.19† (1.59)	23.816**	1.000
EPI change	1, 55		5.14 (0.89)	6.32 (1.15)	1.649, n.s.	
NEPI (pg/ml)	2, 116	585.20 (28.43)	542.37† (26.70)	560.86 (24.39)	6.070*	.991
NEPI change	1, 57		-48.10 (11.65)	-28.78 (12.68)	2.973, n.s.	
ACTH (pg/ml)	2, 102	10.53 (0.75)	14.67† (1.28)	15.08† (1.29)	25.559**	.890
ACTH change	1, 57		4.03 (0.76)	4.45 (0.88)	0.521, n.s.	
CORT (pg/ml)	2, 118	10.16 (0.44)	11.30 (0.51)	12.06† (0.50)	9.569**	.870
CORT Change	1, 62		1.34 (0.49)	2.28 (0.50)	7.352**	

^aEPI = epinephrine; NEPI = norepinephrine; ACTH = adrenocorticotrophic hormone; CORT = cortisol. SEM = standard error of the mean.

* $p < .05$, ** $p < .01$, † individual task significantly different than baseline ($p < .05$), n.s. = not statistically significant ($p > .05$).

elicited greater HR and PEP activity than the math task, whereas the math task elicited greater respiratory increases than the speech task. RSA changes did not vary as a function of task.

Task-Induced Endocrine Activity

Effect of period. The results for endocrine activity are summarized in Table 2. Significant main effects for period were observed for EPI, NEPI, ACTH, and CORT. Pairwise comparisons confirmed that each task significantly increased EPI, ACTH, and CORT relative to baseline levels. Unexpectedly, NEPI levels decreased significantly overall.

Effect of task. Table 2 includes mean change scores for each neuroendocrine measure and task. The speech task elicited greater increases in cortisol than did the math task; the remaining neuroendocrine measures did not differ as a function of task.

Intertask consistency. Given that the math and speech tasks were effective in eliciting significant changes in cardiovascular and endocrine parameters, our primary goal was to assess the consistency of individual differences in responses to these tasks.

Cardiovascular measures. Each cardiovascular measure displayed high levels of intratask reliability: Cronbach’s alpha for HR_(math) = .99, HR_(speech) = .99; PEP_(math) = .99, PEP_(speech) = .99; and RSA_(math) = .94, RSA_(speech) = .92.

As shown in Table 3, task levels and change scores were consistent across the mental arithmetic and speech task. Pearson correlations were significant for each cardiovascular measure, although task levels were more highly correlated than change scores, as expected given the summing of baseline and task measurement errors in the change score measurements. For task levels, correlations ranged from .80 for RSA to .97 for PEP. For change score levels, correlations ranged from .48 for HR to .63 for PEP. All correlations were statistically significant. Spearman correlations

confirmed that the reactivity-based rank ordering of individuals remained fairly constant across the two tasks. All Spearman correlations were statistically significant, ranging from .50 to .97. Spearman correlations are included in Table 3.

Autonomic response profile. Recall that individuals were classified as relatively activated in terms of PEP changes whether they showed relative sympathetic activation and relative vagal deactivation or relative sympathetic activation and relative vagal activation. These two autonomic response patterns may differ in health consequences, however. Response profiles distinguish between these two autonomic response patterns, enabling analysis of consistency in individual’s autonomic pattern of cardiac activation.

The median values (ranges) used in the PEP/RSA profile were as follows: PEP_(speech) = -4.33 (-18.33-2.33), PEP_(math) = -2.33 (-18.67-13.93), RSA_(speech) = -0.52 (-2.15-1.04), RSA_(math) = -0.50 (-2.52-1.12). Nonparametric analyses revealed significant cross-task consistency of the individual differences in the profile of relative sympathetic and vagal reactivity, Cohen’s $K = .41$, $p < .01$. Overall, 56% (range = 50-64%) of the

Table 3. Intertask Correlations of Levels of Cardiovascular Activity (Math vs. Speech)

	N	Pearson	Spearman
Task HR	52	.83**	.84**
HR change	51	.51**	.63**
Task PEP	48	.97**	.97**
PEP change	44	.62**	.60**
Task RSA	57	.80**	.74**
RSA change	56	.61**	.54**

** $p < .01$.

ejection period associated with an increase in HR. In addition, HR changes were negatively, although nonsignificantly, correlated with RSA changes, $r = -.11$, n.s.), with a diminution of RSA denoting decreased parasympathetic input to cardiac chronotropy. (Ancillary statistical analyses revealed that the pattern of results did not change when IBI rather than HR was employed as a measure of cardiac chronotropy.) Although these stressors produced an overall reciprocal pattern of sympathetic activation and vagal withdrawal, RSA changes were minimally and nonsignificantly correlated with PEP changes, $r = -.04$, n.s., suggesting individual differences in the mode of autonomic response to psychological stress.

Regarding the endocrine measures, CORT changes exhibited an anticipated positive correlation with changes in ACTH, $r = .62$, $p < .01$. Neither ACTH changes nor CORT changes were significantly correlated in this sample with sympathetic activation as indexed by PEP changes.

The only significant interrelationships among the cardiovascular and endocrine systems involved changes in EPI. Consistent with sympathetic influence over both cardiac and adrenal activity, task-aggregated EPI changes were positively correlated with changes in HR and PEP, $r_s = .34$ and $-.36$, respectively, $p_s < .05$.

Interrelations Among Response Profiles

Individuals were classified as relatively activated in SAM responses whether they showed high SAM activation and low HPA activation or high SAM activation and high HPA activation. Response profiles distinguish between these two response patterns, enabling analysis of consistency in an individual's pattern of endocrine activation. If individuals display consistency in response patterning, then the latter response pattern (high SAM and high HPA) may signify unique health concerns due to the more likely and/or more frequently evoked stress-related increases in cortisol.

The median values (ranges) used in the EPI/CORT profile were as follows: $EPI_{(speech)} = 6.36$ (-9.00 – 31.00), $EPI_{(math)} = 5.16$ (-4.00 – 26.00); $CORT_{(speech)} = 2.14$ (-4.60 – 12.20), $CORT_{(math)} = 1.16$ (-5.70 – 12.40). Examination of response profiles across the SAM and HPA systems revealed that individual differences in the level of EPI reactivity relative to the level of CORT reactivity appeared stable across tasks. Approximately 49% (range = 38–71%) of participants could be correctly classified into the appropriate EPI-CORT response profile (chance = 25%) in the second task on the basis of response profile in the first task (Cohen's $K = .32$, $p < .01$). Of the participants who were misclassified, the majority (approximately 90%) were misclassified based on only one of the two measures. Moreover, among the 24% of participants incorrectly classified on the basis of EPI response category only; in slightly more than half of these cases (54%), misclassification resulted on the basis of minimal task differences that nevertheless produced differences in categorization as defined by a median split (i.e., at least one task response was <2 pg/ml from the median). Similarly, among the 22% of participants incorrectly classified on the basis of CORT response category only, about 33% of the CORT misclassifications were obtained despite minimal differences in CORT reactivity between tasks. Thus the 49% of participants that were correctly classified may represent a conservative estimate of cross-task EPI-CORT response profile consistency. This consistency in EPI-CORT response profile was found despite the independence of EPI and CORT reactivity across participants. That is, EPI response categories for the math and speech tasks did not predict CORT response categories ($\Pi^2(1) < 0.48$, n.s.). Substituting ACTH for CORT in all of the above analyses produced similar results.

The median values (ranges) used in the PEP/CORT profile were as follows: $PEP_{(speech)} = -4.38$ (-18.33 – 2.33), $PEP_{(math)} = -2.5$ (-18.67 – 13.93); $CORT_{(speech)} = 1.56$ (-4.60 – 8.30), $CORT_{(math)} = 0.98$ (-5.70 – 10.20). Examination of response profiles across the sympathetic and HPA systems revealed that PEP-CORT response profiles were consistent across tasks, Cohen's $K = .42$, $p < .01$, even though PEP response category was independent of CORT response category in both tasks ($\Pi^2(1) < 2.7$, n.s.). Approximately 50% (range = 39–88%) of participants could be correctly classified into one of four PEP-CORT response profiles in the second task on the basis of their response profile in the first task. (The PEP-ACTH response profiles produced similar results.) Of the participants who were misclassified, the majority (approximately 94%) were misclassified based on only one of the two measures. Twenty-six percent of the misclassifications were due to PEP miscategorizations only, and 21% were due to CORT miscategorizations only. Given the small task differences that nevertheless resulted in category differences between tasks, response profile consistency may be considerably greater than suggested by the 50% who were correctly classified on the basis of median splits. Thus, using PEP as an index of sympathetic activity, these data suggest that individuals display consistent sympathetic-HPA response profiles across the math and speech tasks.

Discussion

Among the older women studied, the mental arithmetic and speech tasks were sufficient to elicit heightened autonomic activity (as indexed by changes in HR, PEP, and RSA), increased sympathetic adrenomedullary activity (as indexed by changes in EPI), and increased hypothalamic-pituitary-adrenocortical activity (as indexed by changes in ACTH and CORT). These results are consistent with previous research reporting significantly augmented cardiovascular, SAM, and HPA responses to acute psychological stressors (Cacioppo et al., 1995; Kirschbaum et al., 1995; Oleshansky & Meyerhoff, 1992).

Importantly, given interest in examining the autonomic differentiation of elderly who fall ill, cardiovascular reactivity to the mental arithmetic and speech tasks showed high intertask consistency in this sample of older women. As such, these results replicate previous findings in young adults but in a less resilient population. Spearman correlations confirmed individual consistency in the rank ordering of cardiovascular responses to the two tasks. That is, individuals who exhibited the greatest reactivity to the mental arithmetic task were also most likely to show the greatest reactivity to the speech task. Furthermore, Pearson correlations confirmed that individuals responded consistently in terms of the magnitude of sympathetic and vagal responses across stressors requiring active coping. Thus, even in an older population, PEP and RSA reactivity continue to predict individual differences in task-induced sympathetic and vagal activity.

Moreover, when categorized in terms of level of RSA reactivity relative to level of PEP reactivity, consistency was evident in the autonomic response category across tasks despite the independence of PEP and RSA reactivity across participants. For example, although level of PEP reactivity did not predict level of RSA reactivity across participants, participants who were categorized as low PEP and low RSA responders to one task (i.e., congruous with concurrent sympathetic activation and vagal withdrawal) were likely to be categorized as low PEP and low RSA responders to the other task. These data therefore also provide evidence of consistent individual differences in relative sympathetic and vagal reactivity

across tasks, suggesting potential utility of research into such differences. For example, a response profile consisting of sympathetic activation and vagal withdrawal (reciprocal activation of the sympathetic and parasympathetic branches of the autonomic nervous system, ANS) may represent an efficient mechanism to optimize HR reactivity in coping with a stressful situation. In contrast, coactivation of the two autonomic branches would be expected to yield a greater vascular response, while minimizing effects on HR. In addition to functional differences among these patterns, they may also have distinct health implications. High sympathetic control, for example, is a risk factor following myocardial infarctions, but this risk can be partially offset by concurrent parasympathetic control (Kristal-Boneh et al., 1995). Response profiles may therefore provide insight into how individuals are coping as well as the health consequences on the cardiovascular system, insights not provided when looking at either sympathetic or vagal reactivity alone.

An important caveat to the use of RSA changes to index vagal reactivity is the impact of respiratory changes on RSA amplitude (Berntson et al., 1997; Hirsch & Bishop, 1981). In the current study, respiratory changes may have significantly influenced RSA. Nevertheless, we found that psychological stress evoked vagal withdrawal beyond what would be expected given stress-evoked changes in respiratory frequency, consistent with the results of autonomic blockade studies using young participants (Berntson et al., 1994).

Endocrine measures also showed high intertask consistency across a wide range of SAM and HPA responses in these older women. To date, few studies have attempted to assess the consistency of endocrine responses, and most of these have focused on the consistency of cortisol responses. Berger et al. (1987) were unable to demonstrate stable individual differences in cortisol response when assessed across a variety of physical and psychological challenges. However, in a study involving male and female mono- and dizygotic twins, Kirschbaum, Wüst, Faig, and Hellhammer (1992) found that the salivary cortisol response to bicycle exercise was significantly correlated with the cortisol response to CRH (corticotropin releasing hormone) injection.

In contrast, at least among women, the peak cortisol response to the psychologically stressful tasks of public speaking and mental arithmetic was not significantly correlated with the response to either bicycle exercise or CRH injection. Furthermore, in comparing the cortisol responses of mono- and dizygotic twins, these researchers concluded that genetic factors played a major role in explaining the consistency of individual differences in adrenocortical responsivity to CRH stimulation, but a lesser role in responsivity to psychological stress. The unique effect of psychological stress on HPA activity may reflect the fact that psychological stress involves additional historical and interpretive processes beyond those involved in responses to physical stimulation or CRH administration. The investigation of HPA responses to active coping challenges may therefore offer a rich opportunity to identify individual differences that may contribute in a unique fashion to the identification of health risks associated with a tendency toward exaggerated cortisol reactivity.

Although the current study demonstrated intertask stability of ACTH and cortisol responses, this assessment must be tempered by the fact that both ACTH and cortisol response have a relatively long latency and time course (Richter et al., 1996). Consequently, evidence regarding the consistency of individual differences in HPA reactivity to psychological stress will require test-retest studies. Nevertheless, the fact that significantly elevated ACTH and

cortisol were evident after 6 min of the first psychological stressor suggests that these endocrine responses were sufficiently rapid to assess task-specific effects. Thus these results are encouraging in light of analyses currently under way assessing temporal stability in ACTH and cortisol responsivity to these tasks in these same participants over a 3-year period.

The current demonstration of stable epinephrine responses to the two tasks is one of the first to identify stable individual differences in the magnitude of adrenomedullary response to psychological stressors, and the first to do so in a sample of older women. Among highly reactive Type A men, Seraganian et al. (1985) reported temporal stability of EPI and NEPI responses to a variety of psychological tasks over a period of several weeks. In the current study of older women, EPI responses revealed stable individual differences in SAM reactivity across tasks. Individual differences in SAM responsivity could be reflected in the type and magnitude of interactions between the catecholamines and the immune system (Ader, Cohen, & Felten, 1995; Miller, 1998), interactions that may lead to distinct immune consequences.

NEPI responses were also stable across the two tasks, but were decreased rather than increased relative to baseline levels. Although this finding was unexpected, Seraganian et al. (1985) reported a similar phenomenon among some adult men in whom NEPI was seen to decrease during acute stressors. A postural change may explain our results. Baseline NEPI readings were taken shortly after a long adaptation period, which ended with a change from a supine to a sitting position. Although postural changes typically elicit minimal adrenomedullary activity (Kohrt, Spina, Ehsani, Cryer, & Holloszy, 1993), central noradrenergic activity is sensitive to orthostatic stress. Plasma NEPI, which reflects primarily the neurally derived neurotransmitter (Esler et al., 1990), increases with shifts to upright posture, and those increases are enhanced in the elderly (Kohrt et al., 1993; Sowers, Rubenstein, & Stern, 1983). Therefore, orthostatically induced increases in basal NEPI in the current study may have obscured the expected stress-induced increases in NEPI. Alternatively, or in addition, a 30-min adaptation period after insertion of an indwelling catheter may be insufficient for NEPI levels to return to baseline levels, helping to explain why NEPI did not appear to increase during either math or speech.

In contrast with previous reports linking HPA activation with sympathetic activity (Cacioppo et al., 1995; Uchino et al., 1995), cortisol changes were not significantly correlated with either HR or PEP reactivity in these older women. A possible explanation relates to the differential loss of beta- and not alpha-adrenergic sensitivity with age. Because the sympathetic effects on the PEP are mediated by beta-adrenoceptor activation, whereas CRH release is stimulated by alpha-1 adrenoceptor activation, and because the degree of beta-adrenergic sensitivity loss may have been inconsistent among the women in this study, the likelihood of detecting covariation between PEP and CRH-stimulated ACTH and cortisol may also have been diminished. Alternatively, age-related structural and mechanical changes in the cardiovascular may influence PEP to a greater extent than in the young, contributing to an inability to detect covariation between neurally based changes in PEP and HPA activity in the elderly. This issue also warrants further study.

On the other hand, nonparametric analyses revealed significant cross-task consistency in PEP-CORT response profiles. Thus, even though sympathetic activity did not predict HPA activation across participants, individual differences in the relative activity of the sympathetic and HPA systems were stable across tasks. Similarly,

EPI-CORT response profiles demonstrated cross-task consistency. These results warrant further research into the significance of consistent individual differences in sympathetic-HPA and SAM-HPA response profiles. Sympathetic-HPA response profiles may be of particular interest here. As noted earlier, exaggerated or prolonged activity of the HPA axis may contribute to atherosclerosis and diabetes, as well as potentiate brain damage and cognitive impairments. Excessive sympathetic activation may have comparably dismal implications for cardiovascular health. The combination of increased sympathetic activation and elevated cortisol therefore bodes poorly for an individual's health, especially if this stress response profile is characteristic of an individual across stressors.

In summary, the psychological characteristics of the speech and arithmetic tasks employed in the current study evoked physiolog-

ical responses of considerable consistency among these older women. Despite any age-related changes in the cardiovascular, the SAM axis, and the HPA axis, this older population exhibited consistent individual differences in cardiovascular and endocrine reactivity to the two acute stressors. Furthermore, a majority of women in this sample exhibited cross-task consistency in response profiles within the autonomic, SAM, and HPA systems, as well as across these systems. Although the significance of consistent individual differences in response profile remains to be explored, the induction of consistent autonomic and endocrine responses across acute psychological stressors may have implications in the development and/or progression of diseases such as ischemias and arrhythmias (Pagani et al., 1991), as well as autoimmune and infectious diseases (Ader et al., 1995).

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