

Aging and Cardiovascular Reactivity to Stress: Longitudinal Evidence for Changes in Stress Reactivity

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Although age differences in cardiovascular function are well documented, little research has provided longitudinal evidence for age-related changes in cardiovascular reactivity to stress. In this study, the authors report such data from a follow-up of their prior work (B. N. Uchino, D. Uno, J. Holt-Lunstad, & J. B. Flinders, 1999) with participants between the ages of 30 to 70 ($n = 108$, mean follow-up = 10 months, range = 7 to 16 months). Results revealed longitudinal evidence for an age-related increase in systolic blood-pressure reactivity and parasympathetic withdrawal to acute stress. The implications of these findings are discussed in light of the increased cardiovascular disease risk with age, as well as the links between aging, emotions, and physiology.

Age differences in cardiovascular function are well documented and may confer increased vulnerability to physical health problems (Ferrari, Radaelli, & Centola, 2003; Kart, Metress, & Metress, 1992; Timiras, 1994). For instance, chronological age is associated with increases in resting blood pressure that may then influence cardiovascular disease risk (Timiras, 1994). However, much less is known about how older adults respond physiologically to psychological stress and how these reactions may change over time (Boutcher & Stocker, 1996; Jennings et al., 1997; Uchino et al., 1999). Although there is still debate about the health significance of cardiovascular reactivity (Linden, Gerin, & Davidson, 2003; Manuck, 1994; Treiber et al., 2003), preliminary evidence exists that stress-induced changes in cardiovascular function may be independent predictors of the development and exacerbation of cardiovascular disease (Kamarck et al., 1997; Krantz et al., 1991; Light, Dolan, Davis, & Sherwood, 1992; Matthews, Woodall, & Allen, 1993). An examination of such age-related changes in cardiovascular reactivity to stressful circumstances may help researchers further understand the increased cardiovascular disease risk with age. Thus, the major aim of this study was to examine

longitudinal age-related changes in cardiovascular reactivity during stress.

Much evidence exists on age differences in resting cardiovascular function. For instance, resistance to blood flow is increased because of structural, hormonal, and receptor-level changes over time (Elliot, Sumner, McLean, & Reid, 1982; Palmer, Ziegler, & Lake, 1978; Pfeifer et al., 1983). There is also a decrease in β -adrenergic responsiveness with age (Bertel, Buhler, Kiowski, & Lutold, 1980; Feldman, Limbird, Nadeau, Robertson, & Wood, 1984). However, estimates of resting cardiac output are only slightly reduced in older individuals because of little if no age difference in resting heart rate (Folkow & Svanborg, 1993; Lakatta, 1993). Nevertheless, the net result of these processes is that chronological age is typically associated with increases in systolic blood pressure (SBP) and diastolic blood pressure (DBP) at rest (Fleg, Tzankoff, & Lakatta, 1985; Garwood, Engel, & Capriotti, 1982; Landahl, Bengtsson, Sigurdsson, Svanborg, & Svardsudd, 1986; Steptoe, Moses, & Edwards, 1990). Aging also tends to be associated with decreased resting parasympathetic control of the heart as indexed by respiratory sinus arrhythmia (RSA; DeMeersman, 1993; Hrushesky, Fader, Schmitt, & Gilbertsen, 1984).

Considerably less is known about the association between age and cardiovascular reactivity during psychosocial stress. The existing studies have mostly examined cross-sectional associations between age and stress-induced blood pressure or heart-rate changes (Fauchaux, Dupuis, Baulon, Lille, & Bourliere, 1983; Garwood et al., 1982; Ginter, Hollandsworth, & Intriери, 1986; Steptoe et al., 1990). These prior studies suggest that older adults have greater SBP reactivity than their younger counterparts, with no apparent difference in DBP reactivity (Fauchaux, Bourliere, Baulon, & Dupuis, 1981; Garwood et al., 1982; Johansson & Hjalmarson, 1988; Steptoe et al., 1990; but see Ginter et al., 1986). The data for heart rate are more equivocal, but some studies suggest lower heart-rate reactivity during stress in older individu-

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This research was generously supported by James A. Shannon Director's Award 1 R55 AG13968 and RO1 AG018903 from the National Institute on Aging, as well as Grant R01 HL68862 from the National Heart, Lung, and Blood Institute. We thank David Lozano, Daniel Litvack, John T. Cacioppo, Robert Kelsey, and William Guethlein for their expert technical assistance and for providing us with copies of their data acquisition and reduction software (i.e., ANS suite and ENSCOREL).

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als (e.g., Barnes, Raskind, Gumbrecht, & Halter, 1982; Ginter, Hollandsworth, & Intrieri, 1986; but see Steptoe et al., 1990).

Only a few studies have attempted to further characterize such age differences in reactivity to stress by examining more detailed measures of cardiovascular function. These more detailed measures include impedance-derived measures of cardiac output and total peripheral resistance (TPR), along with estimates of sympathetic (preejection period [PEP]) and parasympathetic (RSA) control of the heart (see chapter 12 of Stern, Ray, & Quigley, 2001; Sherwood, 1993, for reviews of these indices). An examination of these measures is important because the measures may (a) shed light on the more specific processes underlying general age differences in cardiovascular reactivity (Uchino et al., 1999) and (b) have implications for cardiovascular disease risk in their own right (Julius, 1993; Binkley, Nunziata, Haas, Nelson, & Cody, 1991).

There appear to be only a few studies that examined age difference in cardiovascular reactivity during psychosocial stress that used such detailed measures (Boutcher & Stocker, 1996; Jennings et al., 1997; Uchino et al., 1999). All are cross-sectional studies and have produced conflicting results. Boutcher and Stocker (1996) examined cardiovascular reactivity during a 2-min Stroop task in a sample of 15 young (mean age = 21) and 15 older (mean age = 59) men. Results revealed that age was associated with decreases in TPR reactivity and no differences in cardiac output reactivity. Inconsistent with prior research, Boutcher and Stocker found no age difference in SBP reactivity. However, the relatively small sample size and short task may have limited the ability of their study to detect some of these age differences.

Jennings et al. (1997) used a large sample of 902 men (ages 46 to 64) from the Kuopio Ischemic Heart Disease Risk Factor Study. Multiple task assessments to stress were obtained by using the Pittsburgh Reactivity Battery and aggregated to increase the reliability and generalizability of the cardiovascular reactivity assessment (Kamarck et al., 1992). Results revealed that older men had greater stress-induced reactivity on both indices of cardiac and vascular reactivity. Consistent with Boutcher and Stocker (1996), Jennings et al. did not find any age differences in parasympathetic reactivity to stress, at least as indexed by a task known to elicit a vagal-mediated heart-rate deceleration (i.e., anticipatory response to shooting task). These authors interpreted these data as primarily reflecting an age-related increase in sympathetic control of the cardiovascular system. This interpretation is consistent with Esler et al. (1995), who found an increase in cardiac sympathetic activity during mental stress in older individuals.

In our prior study, which examined age differences in cardiovascular reactivity, we tested 133 men and women (ages 30 to 70; Uchino, Uno, Holt-Lunstad, & Flinders, 1999). Similar to Jennings et al. (1997), we specifically designed our protocol to increase the reliability and generalizability of our assessments (averaged across multiple tasks over multiple minutes). However, in comparison to prior research, we examined a wider age range and included both men and women. Replicating prior research, we found age to predict an increase in SBP reactivity to stress. Consistent with Jennings et al., we also found evidence for increased cardiac and vascular reactivity to stress in older adults. However, inconsistent with their findings, we found age to predict increased parasympathetic withdrawal during stress as indexed by RSA. These findings were consistent for both men and women and could not be ex-

plained by demographic factors or task-specific affective changes and performance.

In the present study, we report data from a follow-up of our prior sample (Uchino et al., 1999). To date, no study that we are aware of has reported longitudinal evidence on age-related changes in cardiovascular reactivity to stress. This study provides an opportunity to examine the replicability of prior cross-sectional findings and to determine whether older adults show greater or lesser reactivity over time to psychological stress. Prior research on age, emotions, and physiological function suggests that older adults may show decreased physiological responding to general emotional stimuli (Levenson, Carstensen, & Gottman, 1994). However, one of the largest and most comprehensive studies on age and stress reactivity to date (Jennings et al., 1997) showed increased cardiac and vascular reactivity to stress as a function of age (also see Uchino et al., 1999). These findings are consistent with broader research, suggesting that older adults may have difficulty in regulating negative emotions, possibly because of age-associated declines in cognitive resources (Labouvie-Vief, 1999). Thus, we predicted that older adults are more likely to evidence greater cardiovascular reactivity to acute stress over time. Consistent with our prior research, these results are also predicted to be independent of other demographic factors, task-specific affect/performance, and health behaviors.

Given our longitudinal design, an ancillary aim of this study was to examine the test-retest reliability of the cardiovascular reactivity assessments. Prior research that used reactivity protocols has been criticized for being relatively poor in reliability (see Kamarck et al., 1992). If the reactivity assessments are characteristic of an individual's stress response over time, then it should be characterized by some temporal consistency. In a meta-analysis conducted by Swain and Suls (1996), changes in heart rate and SBP had moderate reproducibility, whereas DBP reactivity reliability was low. However, many of these studies were not designed to increase reliability, and much less research exists on the temporal reliability of the more detailed cardiovascular assessments (i.e., cardiac output, TPR, PEP, RSA; see Burleson et al., 2003). In the present study we report our attempts to increase the reliability of stress reactivity assessments and the resulting test-retest reliability of our cardiovascular reactivity assessments.

Method

Participants

In our original study, we tested 64 men and 69 women between the ages of 30 and 70 (see Uchino et al., 1999). Approximately equal numbers of men and women were recruited from each decade group (e.g., 30 to 39) through advertisements placed in local newspapers. Individuals were paid \$35 for approximately 2.5 hr of participation. The following self-reported inclusion criteria were used to select healthy participants: (a) no existing hypertension, (b) no cardiovascular prescription medication use, (c) no past history of chronic disease with a cardiovascular component (e.g., diabetes), (d) no recent history of psychological disorder (e.g., major depressive disorder), (e) no tobacco use, and (f) no consumption of more than 10 alcoholic beverages a week (see Cacioppo et al., 1995).

In our follow-up, individuals were rescreened according to the inclusion criteria listed above and paid \$35 for approximately 2.5 hr of participation. We were able to recontact and schedule 108 (81%) of our original partic-

ipants on average 10 months later ($SD = 1.6$, range = 7 to 16 months).¹ The main reason for attrition was related to our inability to contact and in a small minority of cases problems in scheduling individuals for the follow-up. As a result, we conducted preliminary analyses to determine any differences between the individuals who were not retested in terms of basic demographics factors. A one-way analysis of variance (Time 1 sample: not retested, retested) on age, gender, income, and education level revealed a nonsignificant group difference on education level, $F(1, 131) = 3.62$, $p < .06$ ($R^2 = .027$). Individuals who were retested had a slightly higher education level than those who were not retested ($M = 3.56$ vs. 4.13; 3 = graduated from college, 4 = partial graduate/professional school). It is important to note that there were no differences as a function of age ($p > .57$). No other differences approached significance. Thus, there did not appear to be any systematic differences between individuals not retested versus those that were retested. Basic demographic information on the retested sample, along with information on several covariates, is included in Table 1.

Procedure

Individuals were first recontacted by telephone and rescreened according to the inclusion criteria detailed under the *Participants* section. Qualifying individuals were scheduled for an appointment, and participants' self-reports were again checked for reliability against the inclusion criteria. On arrival, participants completed an informed consent document and a demographic background questionnaire. Following completion of these questionnaires, the participants' height and weight were obtained by using a standard medical scale from which body mass index was calculated (i.e., weight in kg/height² in meters).

Participants were then escorted to a separate sound-attenuated room where four Mylar bands were placed in the tetrapolar configuration for impedance cardiograph recordings according to published guidelines (Sherwood et al., 1990). To maximize the reliability of the assessments, we used the same Mylar band distances from Time 1 for Time 2. An occluding cuff of appropriate size was also placed on the upper left arm. Individuals were seated in a comfortable chair and instructed to relax for the next 12 min while resting measures of cardiovascular function were obtained. During the final 5 min of the resting assessment, cardiovascular assessments of SBP and DBP were obtained once every 90 s, whereas impedance cardiograph readings were recorded continuously. Participants also com-

pleted the State-Trait Anxiety Scale (Marteau & Bekker, 1992) at the end of the rest period as a baseline measure of state anxiety.

Following the resting assessments, participants performed a speech and mental arithmetic protocol developed by Cacioppo et al. (1995). The same basic stressor tasks were used for the Time 2 reactivity protocol with slight changes to minimize habituation (e.g., different speech topic and different set of serial subtractions). The order of the stressors was counterbalanced, and all verbal instructions were standardized. For the speech task, participants were asked to formulate a speech about their reactions to the following scenario:

They have received a visit from a bill collector that has falsely accused them of not paying a debt. When talking to the bill collector, they were instructed to present the following points in their defense: (a) tell the bill collector their side of the story, (b) tell the bill collector what the credit card company may have done wrong in turning the debt over, (c) tell the bill collector why the credit card company may have turned over the debt, (d) explain how they could prove the debt was paid, (e) explain what should happen to the credit card company for their mistake, (f) tell the bill collector what they will do to clear up this situation, and (g) provide a summary of all their points.

Any questions from participants about the upcoming task were answered prior to the preparation period. To increase the relevance of the task for participants, we told the participants that it was very important that they imagine that this was actually happening to them because their speech would be recorded and compared with the speeches of others in the study. Participants were given 3 min to prepare and 3 min to present their speeches while impedance cardiography measures were recorded continuously throughout the task. Blood-pressure readings were obtained during Min 1 and Min 3 of the preparation and actual speech periods. As a measure of task performance, the number of speech prompts given to participants was recorded. State anxiety was again assessed at the end of the speech task.

The mental arithmetic task consisted of six 1-min serial subtraction problems that participants performed out loud. Participants were given a new subtraction problem each minute. If participants made a mistake, then they were told that they would be corrected by the experimenter with an answer sheet and to resume the serial subtraction from the corrected number. Any questions about the subtraction tasks were answered prior to the start of Min 1. After Min 1, each subsequent problem was adjusted for difficulty so that effort was relatively constant across participants (i.e., approximately 10 serial subtractions per minute; see Cacioppo et al., 1995). To increase the relevance of the task, we encouraged participants to work as quickly and as accurately as possible because their responses would be recorded and compared with the performance of others in the study. Impedance cardiography measures were recorded continuously throughout the task, whereas blood pressure was assessed during Min 1, 3, and 5 of the task. Performance during the task was measured as the percentage of incorrect serial subtractions. At the end of the math task, participants again completed the state anxiety measure. On completion of both psychological stressors, participants were debriefed, compensated, and thanked for their participation.

Measures

Cardiovascular measures. A Minnesota Impedance Cardiograph Model 304B (Bio-impedance Technology, Chapel Hill, North Carolina) was used to measure the electrocardiogram (ECG), basal thoracic imped-

Table 1
Follow-Up Sample Characteristics

Variable	Sample
Age frequency distribution (%)	
30–39	26
40–49	28
50–59	29
60–70	17
<i>M</i> (<i>SD</i>) age	47.9 (10.9)
<i>Mdn</i> education level	Partial college– graduated college
<i>Mdn</i> yearly income	20,000–29,000
<i>M</i> (<i>SD</i>) change in	
Body mass index	0.00 (0.00)
Weekly sleep	–1.85 (7.75)
Weekly exercise	–0.62 (4.59)
Weekly alcohol consumption	–0.10 (1.70)
Weekly caffeine consumption	–0.05 (1.21)
Reported health status	–0.06 (0.60)
State anxiety	0.00 (0.62)
Speech prompts	–0.24 (1.41)
Percentage of wrong math problems	0.00 (0.06)

¹ It is important to note that the follow-up time period did not differ as a function of age. In addition, none of our findings regarding age and cardiovascular function were changed when statistically controlling for variations in time to follow-up.

ance (Z_0), and the first derivative of the impedance signal (dZ/dt). Four Mylar bands were placed in the tetrapolar configuration according to published guidelines (Sherwood et al., 1990). A 4-mA AC current at 100 kHz was passed through the two outer bands, and Z_0 and dZ/dt were recorded from the two inner bands. The ECG, Z_0 , and dZ/dt signals were digitized at 500 Hz. The impedance data were ensemble averaged within 1-min epochs. Ensemble averaging uses the R point of the ECG as a reference for the successive averaging of the ECG and subsequent dZ/dt signals. This procedure reinforces both signals while random movement artifacts and lower frequency respiratory influences are effectively filtered out (Kelsey & Guethlein, 1990). Each ensemble-averaged waveform was verified or edited prior to analyses by a trained scorer.

Stroke volume was estimated using the Kubicek equation (see Sherwood et al., 1990) and the subsequent cardiac output in liters/minute was calculated by multiplying heart rate \times (stroke volume/1000). TPR was measured in resistance units ($\text{dynes} - \text{second} \times \text{cm}^{-5}$) based on mean arterial pressure and cardiac output (i.e., $\text{TPR} = \text{mean arterial pressure} \div \text{cardiac output} \times 80$). PEP was calculated as the time interval in milliseconds between the Q point of the ECG and the B point of the dZ/dt signal. These minute-by-minute impedance-derived measures were averaged across minutes within epochs (e.g., baseline, stress tasks) to increase the reliability of these assessments (Kamarck et al., 1992).

RSA provides a noninvasive measure of parasympathetic control of the heart and was calculated on the basis of the digitized interbeat intervals (IBI) that were checked and edited for artifacts, using the detection algorithm of Berntson, Quigley, Jang, and Boysen (1990). After linear detrending, the heart-period time series was bandpass filtered from .12 to .40 Hz, using an interpolated finite impulse response filter (Neuvo, Cheng-Yu, & Mitra, 1984). RSA was then calculated as the natural log of the area under the heart-period spectrum (calculated by a fast fourier transform and scaled to ms^2/Hz). RSA was calculated on a minute-by-minute basis and aggregated across minutes within each epoch to increase reliability.

A Dinamap Model 8100 monitor (Critikon corporation, Tampa, Florida) was used to measure blood pressure. The Dinamap used the oscillometric method to estimate blood pressure (see Epstein, Huffnagle, & Bartkowski, 1991; Gorbach, Quill, & Lavine, 1991, for validation studies). Blood-pressure assessments were obtained through a properly sized occluding cuff positioned on the upper left arm of the participant according to the manufacturer's specifications. Mean SBP and DBP for each epoch was averaged across minutes to increase the reliability of these assessments.

State Anxiety. A short form of the Spielberger State-Trait Anxiety Scale was administered to participants at the end of the resting baseline and following completion of each of the psychological stressors (Martean & Bekker, 1992). Participants were asked to rate their current feelings on a four-point scale, ranging from 1 (*not at all*) to 4 (*very much*). Consistent with prior work, the internal consistency of the scale in our study was high (Cronbach's $\alpha = .77$ to $.85$).

Health Behavior Assessment. Self-reported weekly sleep, exercise, alcohol consumption, and caffeine consumption were measured with a health behavior scale used in prior research (Kiecolt-Glaser, Dura, Speicher, Trask, & Glaser, 1991). Participants were asked the following questions for an average week: (a) How many total hours of sleep do you get? (b) How many total hours do you spend exercising strenuously (e.g., jogging, sports)? (c) How many alcoholic beverages do you consume (12-oz. beer equivalents)? and (d) How many caffeinated beverages do you consume (coffee, tea, soda pop)?

Results

Preliminary Analyses

We first verified that the stressor tasks were again adequate to alter cardiovascular reactivity to the follow-up protocol. The cardiovascular reactivity protocol was designed through pilot testing

to increase reliability through aggregation across multiple tasks and minutes (Cacioppo et al., 1995; Uchino et al., 1999). As a result, we first averaged minute-by-minute cardiovascular measures within each epoch (i.e., baseline, speech, math). The speech and math task scores were then averaged, and change scores were computed relative to baseline (see Cacioppo et al., 1995; Kamarck et al., 1992). As noted earlier our protocol was also designed to minimize habituation from Time 1 to Time 2. Consistent with our prior work, the stressor tasks at time two led to significant changes in cardiovascular reactivity (see Table 2). Acute stress was associated with an increase in SBP, $F(1, 106) = 255.40, p < .001$; DBP, $F(1, 106) = 215.10, p < .001$; cardiac output, $F(1, 102) = 15.61, p < .001$; and heart rate, $F(1, 103) = 273.40, p < .001$. In addition, PEP was shortened (indicating greater sympathetic cardiac control), $F(1, 102) = 23.93, p < .001$, and RSA was decreased (indicating greater vagal withdrawal), $F(1, 103) = 15.45, p < .001$. Thus, the stressor was associated with significant changes in cardiac function.²

An ancillary aim of this study was to examine the test-retest reliability of our reactivity assessments. Because of the importance of stable baseline assessments in reactivity protocols, we first examined the temporal reliability of our resting assessments (see Table 3). In these analyses, we statically controlled for gender and age as these variables are associated with differences in resting cardiovascular function. Consistent with prior research, baseline assessments were associated with significant test-retest reliability for all measures. The highest reliabilities were for SBP ($r = .58, p < .001$), DBP ($r = .65, p < .001$), and heart rate ($r = .65, p < .001$).

Of greater interest were the test-retest reliabilities of the reactivity assessments. In the past, reactivity protocols have been criticized as having low reliability (Swain & Suls, 1996). This protocol was designed to maximize reliability by using aggregation across minutes and multiple tasks. In addition, the stressor tasks and Mylar band placements were relatively consistent across the testing periods. In these analyses, we examined the association between reactivity scores (i.e., average of both tasks minus baseline) during Time 1 and Time 2, while statistically controlling for resting measures, gender, and age. As shown in Table 3, significant test-retest reliabilities were found for all reactivity measures, although variability existed in the magnitude of the correlations. The highest test-retest reliabilities were found for heart rate ($r = .75, p < .001$), SBP ($r = .69, p < .001$), and RSA ($r = .63, p < .001$) reactivity. With some exceptions, these results are consistent with the 1-year test-retest reliabilities reported by Jennings et al. (1997) and more recently by Burleson et al. (2003), who used a similar reactivity protocol. Thus, although some of these correla-

² Although the protocol was associated with sizable and significant increases in reactivity during Time 2, habituation across testing sessions was nevertheless evident for SBP ($p < .01$), DBP ($p < .05$), cardiac output ($p < .001$), heart rate ($p < .001$), and PEP ($p < .01$) reactivity. The magnitude of most of these changes were small (on average, less than 2 mm/Hg for blood pressure and 2 beats per minute for heart rate). Relatively larger average differences in habituation occurred for cardiac output (.30 L/min) and PEP (3.5 ms). No significant habituation, however, was found for measures of TPR and RSA reactivity.

tions are moderate in size, the protocol appeared effective in increasing test–retest reliability compared with prior research.³

Age-Related Changes in Baseline Cardiovascular Function

As a prelude to our main analyses, we first examined age-related changes in baseline levels of cardiovascular function. Using simultaneous regression analyses with age as the predictor variable, we examined Time 2 resting assessments while statistically controlling for changes in body mass index over the testing period, Time 1 resting cardiovascular function, and gender (see Table 4). There was no evidence for an age-related increase in resting blood pressure over the follow-up period ($ps > .57$). However, because of potential differences in the underlying basis of blood pressure (i.e., flow or resistance), we examined potential age-related changes in cardiac output and TPR. It is important to note that analyses of the underlying determinants of blood pressure revealed a nonsignificant age-related decrease in resting cardiac output ($\beta = -.18, p = .07, R^2 = .021$) and a significant age-related increase in resting TPR ($\beta = .19, p < .05$). The net effect of these underlying processes would be to cancel out any overall age-related increase in blood pressure. Consistent with prior research, there was also a trend toward an age-related decrease in resting RSA ($\beta = -.16, p = .09, R^2 = .018$). No other effects approach significance. Thus, across the testing period, some age-related changes in resting cardiovascular function occurred within our sample of participants spanning the ages of 30 to 70.

Age-Related Changes in Cardiovascular Reactivity to Stress

The primary aim of this study was to test for age-related changes in cardiovascular reactivity during stress. We thus examined Time 2 reactivity scores in simultaneous regression analyses where age was the predictor variable while statistically controlling for changes in body mass index, gender, the relevant baseline measures of the cardiovascular assessment, as well as the relevant reactivity score during Time 1.

Table 2
Mean and Standard Deviation Baseline and Task Levels of Time 2 Cardiovascular Activity

Measure	Baseline		Task	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
SBP (mm/Hg)	117.46	11.38	130.68	15.06***
DBP (mm/Hg)	69.52	9.12	76.60	10.02***
Cardiac output (L/min)	2.59	1.31	2.83	1.41***
Total peripheral resistance (Dynes $- s \times cm^{-5}$)	3346.0	1642.4	3385.3	1570.5
Heart rate (BPM)	66.28	10.90	74.26	11.32***
Preejection period (ms)	104.92	19.17	99.79	18.43***
RSA (natural log)	5.82	1.20	5.55	1.09***

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; BPM = beats per minute; RSA = respiratory sinus arrhythmia. For Epoch main effect, *** $p < .001$.

Table 3
Test–Retest Reliabilities of Baseline and Changes in Cardiovascular Reactivity to Acute Stress

Measure	Baseline	Reactivity
SBP (mm/Hg)	.58***	.69***
DBP (mm/Hg)	.65***	.49***
Cardiac output (l/min)	.30**	.40***
Total peripheral resistance (Dynes $- s \times cm^{-5}$)	.49***	.22*
Heart rate (BPM)	.65***	.75***
Preejection period (ms)	.55***	.59***
RSA (natural log)	.51***	.63***

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; BPM = beats per minute; RSA = respiratory sinus arrhythmia. * $p < .05$. ** $p < .01$. *** $p < .001$.

As shown in Table 5, in our analyses of age-related changes in SBP reactivity during stress, the covariates of resting SBP ($\beta = .29, p < .01$) and SBP reactivity ($\beta = .67, p < .001$) during Time 1 were strong predictors of Time 2 SBP reactivity. Nevertheless, age was still an independent predictor of Time 2 SBP reactivity ($\beta = .20, p < .01$), indicating that older adults showed a greater increase in SBP reactivity to acute stress over the follow-up period. Similarly, the covariates of resting RSA ($\beta = .70, p < .001$) and RSA reactivity ($\beta = .66, p < .001$) during Time 1 were strong predictors of Time 2 RSA reactivity. It is important to note that age, again, emerged as a significant predictor of RSA reactivity during Time 2 ($\beta = -.21, p < .01$). Moreover, the negative beta weight indicates that older adults showed greater parasympathetic withdrawal during stress over the testing periods. No other age effects approached significance.

Potential Alternative Explanations for Age-Related Changes in Reactivity

We conducted analyses to examine whether these age-related changes in SBP and RSA reactivity might be because of the influence of other confounding factors. One possibility is that these results reflect the influence of other basic demographic factors. Inconsistent with this possibility, these effects were unchanged while statistically controlling for family income and education level. It is also possible that these age effects might be because of various task-specific variables, such as affective changes or performance. It is important to note that statistically controlling for changes (i.e., Time 2 minus Time 1; see Table 1) in state anxiety, number of speech prompts, or the percentage of wrong math

³ Because of differences in how researchers examine the test–retest reliability of cardiovascular assessments, we replicated our analyses by using Spearman rank-order correlations that emphasize the consistency of the rank ordering of reactivity scores over time. Results were largely comparable with the results reported in the text. The largest difference occurred for TPR reactivity in which the Spearman rank correlation coefficient was .37 ($p < .05$). We also examined the possibility of age and gender differences in the reliability of our reactivity assessments. Results of moderated regression analyses aimed at examining this question showed that neither age nor gender moderated any of the associations between Time 1 and Time 2 reactivity.

Table 4
Age-Related Changes in Resting Cardiovascular Measures by Using Simultaneous Regression Analyses (β)

Variable	Time 2 resting cardiovascular assessments						
	SBP	DBP	CO	TPR	HR	PEP	RSA
Time 1 resting assessment	.62***	.65***	.31**	.49***	.64***	.53***	.54***
Change in BMI	.11	.07	-.01	-.01	.02	-.13	-.02
Gender	-.03	-.12	.01	-.04	.10	-.06	-.07
Age	.05	.01	-.18	.19*	.04	-.07	-.16

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; TPR = total peripheral resistance; HR = heart rate; PEP = preejection period; RSA = respiratory sinus arrhythmia; BMI = body mass index.

* $p < .05$. ** $p < .01$. *** $p < .001$.

problems did not alter any of the age effects on SBP or RSA reactivity.

We also examined the influence of changes in health behaviors, such as average weekly reported sleep, exercise, alcohol consumption, and caffeine consumption (Time 2 minus Time 1 assessment, see Table 1). Although an increase in average sleep ($\beta = .12$, $p = .05$) and exercise ($\beta = .15$, $p < .03$) over the follow-up period were independent predictors of less parasympathetic withdrawal during stress, none of the reported age effects on reactivity were altered by the inclusion of these health behaviors.

The potential influence of health status was also examined by statistically controlling for standard ratings of self-rated health (*bad to excellent*). Statistically controlling for self-rated health did not influence any of the age-related changes in cardiovascular reactivity reported in the *Age-Related Changes in Cardiovascular Reactivity to Stress* section. Instead, our previously nonsignificant age-related decline in resting cardiac output was now significant ($p < .04$). Finally, our age effects were unchanged while statistically controlling for parental history of hypertension.

Exploratory Analyses

We conducted several analyses to examine more exploratory questions related to age-related changes in cardiovascular reactivity. Given the potential for nonlinear effects of age on health outcomes, we examined potential curvilinear associations by modeling the age-squared term in predicting changes in cardiovascular reactivity. None of the age-squared terms approached significance in these analyses, suggesting the lack of nonlinear effects in our sample.

We also examined the potential moderation of age-related changes in cardiovascular reactivity through important health behaviors. To examine this question, we conducted moderated regression analyses by testing the same models reported earlier but with the Age \times Change in Health Behavior (e.g., exercise) cross-product term based on the centered main effects (Aiken & West, 1991). No significant interactions occurred between age and changes in (a) sleep patterns, (b) alcohol consumption, or (c) caffeine consumption. However, changes in exercise patterns interacted with age to predict SBP ($\beta = -.18$, $p < .05$) and RSA ($\beta = -.14$, $p < .05$) reactivity. Follow-up simple slope analyses (Aiken & West, 1991) revealed that increases in exercise were associated with lower SBP reactivity in the older ($p < .05$) but not

younger group of participants (*ns*). In addition, increases in exercise were associated with lower RSA reactivity to stress in the younger ($p < .001$) but not in the older group of individuals (*ns*).⁴

Discussion

The main aim of this study was to provide longitudinal evidence for age-related changes in cardiovascular reactivity during acute stress by using more detailed measures of cardiovascular function. We found that older adults were more likely to show an age-related increase in SBP and RSA reactivity to acute stress. These changes were independent of other demographic factors, task-specific state anxiety, task-specific effort/performance, health behaviors, and self-rated health. Exploratory analyses also showed that these age-related changes in cardiovascular reactivity were moderated by changes in exercise patterns. In addition, initial analyses of changes in resting assessments revealed that older individuals were more likely to show an increase in resting TPR, an association that was not reflected in resting blood-pressure assessments because of an age-related decrease in cardiac output. There was also a trend toward an age-related decrease in resting parasympathetic activity as indexed by RSA. Overall, these data are consistent with the age-related increase in cardiovascular disease risk and suggest that particular cardiovascular "profiles" may be valuable to further understand this risk.

Our study also found that reliable assessments of cardiovascular reactivity to stress may be obtained by careful measurement and protocol design. The test-retest reliabilities revealed in this study were relatively high for reactivity assessments. The increased reliabilities may be attributable to (a) the use of similar tasks that adjusted for habituation, (b) the use of aggregation across multiple minutes and tasks, and (c) controlling for other sources of variability in the assessments context (e.g., consistent electrode band placements across time). Our findings are consistent with Kamarck et al. (1992), who have argued that the reliability of reactivity protocols can be enhanced considerably by proper attention to such measurement issues.

⁴ It is important to note that the significant interaction between age and changes in exercise also renders the earlier analyses in which we use this variable as a covariate invalid (i.e., homogeneity of regression slopes assumption, see Cohen & Cohen, 1983).

Table 5
Age-Related Changes in Cardiovascular Reactivity by Using Simultaneous Regression Analyses (β)

Variable	Time 2 cardiovascular reactivity						
	SBP	DBP	CO	TPR	HR	PEP	RSA
Time 2 resting assessment	-.17	-.46***	.07	-.31*	-.30***	-.57***	-.86***
Time 1 resting assessment	.29**	.50***	-.01	.17	.28**	.43***	.70***
Time 1 react. assessment	.67***	.51***	.41***	.30*	.73***	.59***	.66***
Change in BMI	-.01	.06	-.04	.05	.04	-.02	-.02
Gender	-.08	.09	.01	-.06	.07	.02	-.06
Age	.20**	.04	.13	-.04	.02	.02	-.21**

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; TPR = total peripheral resistance; HR = heart rate; PEP = preejection period; RSA = respiratory sinus arrhythmia; react. = reactivity; BMI = body mass index.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Although not a primary aim, this study provided evidence for changing hemodynamic processes potentially underlying resting blood pressure as a function of age. Aging was associated with an increase in resting TPR and a decrease in cardiac output. Over the follow-up period, the net result was to maintain resting blood-pressure levels. However, over longer periods of time, resting blood-pressure levels are likely to increase as the stronger trend toward increased vascular resistance continues (Julius, 1993). These data highlight the dynamic adjustments characteristic of the aging cardiovascular system.

One of the most consistent findings in the age and cardiovascular reactivity literature is a cross-sectional difference in stress-induced SBP changes. It is important to note that this study provides one of the first longitudinal validations for this association. Although the health relevance of cardiovascular reactivity to stress is still a topic of debate (Linden, Gerin, & Davidson, 2003; Treiber et al., 2003), some evidence is consistent with the role of blood-pressure changes in the development of cardiovascular disease risk (Manuck, 1994). Moreover, these associations may have implications for the exacerbation of disease in older adult populations. For instance, Krantz et al. (1991) found that SBP reactivity predicted greater myocardial ischemia in cardiovascular patients. In fact, recent research is documenting the importance of SBP more generally in understanding cardiovascular disease risk (Lloyd-Jones, Evans, Larson, O'Donnell, & Levy, 1999).

The other important finding in this longitudinal study relates to an age-related increase in parasympathetic withdrawal during stress. This replicates our prior cross-sectional finding (Uchino et al., 1999) and is consistent with animal models that used graded stimulation of the vagus nerve (Ferrari, Daffonchio, Gerosa, & Mancina, 1991). Ferrari et al. (1991) found that the effects of electrical stimulation were stronger in older compared with younger rats. This may be important as parasympathetic withdrawal is thought to be an important predictor of cardiovascular disease outcomes (Binkley et al., 1991; Bigger, Kleiger, & Fleiss, 1988). Our findings also suggest that although age-related changes in cardiovascular reactivity during psychosocial stress may be because of structural changes in the cardiovascular system (Fleg, 1986; Lakatta, 1993), neural-receptor level processes are also important. A decrease in RSA appears to be a sensitive index of cardiac parasympathetic withdrawal as shown by pharmacological blockade studies (Berntson et al., 1994; Cacioppo et al., 1994).

On a more general level it is important to discuss the seemingly conflicting physiological findings in the age and stress reactivity literature compared with the broader age and emotions literature (Jennings et al., 1997; Levenson et al., 1994). Researchers working in the aging and emotions area have found evidence that older adults show dampened physiological responses to emotional tasks (Levenson, 2000; Tsai, Levenson, & Carstensen, 2000). However, our prior cross-sectional and now longitudinal findings are consistent with Jennings et al. (1997) and support more of an age-related increase in stress responsivity (Uchino et al., 1999). These findings are also consistent with developmental research by Labouvie-Vief (2003) who has suggested that age-related declines in cognitive resources may be associated with decrements in regulating negative emotions. If, indeed, age is associated with an increase in stress responsivity, then an important challenge for older adults is to manage negative emotions or situations in a way that maintains positive growth and feelings of control (Baltes, 1997; Carstensen, Isaacowitz, & Charles, 1999; Labouvie-Vief, 2003; Rothermund & Brandtstadter, 2003; Schulz & Heckhausen, 1996). Future research will be needed to link such stress reactivity perspectives with broader developmental models on age, coping, and emotional adjustment.

Nevertheless, there are alternative explanations for these seemingly divergent findings that require further discussion. First, the tasks that are used in stress reactivity studies are typically more evocative than those used by prior researchers in the aging and emotions literature. It is possible that there is a threshold effect whereas such age-related increases in physiological responsivity are only evident under more stressful or threatening situations. In fact, these are the conditions under which older individuals are most likely to show decrements in self-regulation to negative stimuli (Labouvie-Vief, 2003). A second possible reason relates to potentially different patterns of age-related changes in response to emotional stimuli. This issue is salient because reactivity researchers and emotion researchers have typically included different autonomic nervous system assessments given their focus (e.g., reactivity researchers examine cardiovascular measures thought to increase risk for heart disease). These different patterns of physiological changes to emotional stimuli may in turn reflect the complexity of the aging cardiovascular system. Research on age and physiological function reveals a multifaceted set of findings within and across systems (Timiras, 1994). For instance, there are

a number of age-related cardiovascular changes and compensatory adjustments aimed at maintaining functionality throughout the years (Lakatta, 1993). This complexity makes clear the importance of focusing on patterns of reactivity and modeling underlying physiological mechanisms. Thus future studies that include a battery of overlapping, conceptually driven measures can best model what changes might be serving particular functions or health risks in the older adult.

There are several limitations of the present study that need to be discussed. First, we did not find longitudinal evidence for other cross-sectional findings in the age and stress reactivity literature. However, a larger sample size may have resulted in more power to detect smaller longitudinal effect sizes in our study. For instance, prior cross-sectional work would lead us to expect an age-related increase in vascular resistance (i.e., TPR reactivity), along with decreases in vascular compliance, that might be driving greater blood-pressure changes during stress (Ferrari et al., 2003). However, TPR is usually characterized by increased measurement error compared with blood pressure, in part because it combines the measurement error of several variables (e.g., mean arterial pressure, cardiac output). This greater measurement error can compromise the statistical power of a study to find such differences. A longer follow-up period (e.g., 5 years) may have also aided in increasing the effect size for age that could be determined for this study. Indeed, one might expect such age-related changes in cardiovascular function to be especially evident in the older sample over such a time frame (Folkow & Svanborg, 1993). However, this possibility needs to be considered in light of corresponding changes in health status that may complicate the interpretation of such age-related changes.

Second, because of the homogeneous ethnic composition of our sample and its relatively high education level, future research would be needed to establish the generality of these age-related changes in cardiovascular reactivity. It is important to note, however, that we were able to replicate prior research on age-related differences in resting cardiovascular function as well as the well-documented prior cross-sectional finding on age and stress-induced SBP reactivity. This gives us some confidence in the generalizability of our findings. Nevertheless, a broader sampling of different ethnic groups and socioeconomic status would be necessary to address this issue.

Finally, although the focus of this research was on age-related changes in cardiovascular reactivity, the precise mechanisms and moderating processes that might clarify such changes require further study. Part of our findings may reflect simple age-related changes in physiological function that occur over time (Timiras, 1994). However, these physiological changes may also be driven by relevant psychosocial and behavioral processes. For instance, a sedentary lifestyle, control-related processes, and social relationships may all play powerful roles in such age-associated health outcomes (Rowe & Kahn, 1987). A recent intervention with older, chronically stressed caregivers showed that moderate exercise training can decrease blood-pressure responses to acute stress (King, Baumann, O'Sullivan, Wilcox, & Castro, 2002). This finding is consistent with our exploratory analyses suggesting that older individuals were able to benefit from increases in exercise as indexed by lowered SBP reactivity. Thus, although the effect sizes for age in this study were small, the examination of such moderating processes may explain additional variance in these age-

related changes. These findings also highlight the importance of a "successful aging" approach that examines why some individuals seem to maintain greater functionality throughout the years (Baltes, 1997; Rowe & Kahn, 1987; Schulz & Heckhausen, 1996). The challenge of this perspective is to identify key factors that may be protective of such apparent age-related changes in physiological function. Future interdisciplinary research that highlights the links between social, psychological, behavioral, and physiological levels of analyses would greatly aid in this process.

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Received March 11, 2004

Revision received August 30, 2004

Accepted September 2, 2004 ■

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