Psychology and Aging
2012, Vol. 27, No. 1, 190–198

0882-7974/11/$12.00 DOI: 10.1037/a0025570

Loneliness Accentuates Age Differences in Cardiovascular Responses to Social Evaluative Threat

Anthony D. Ong
Cornell University

Jeremy D. Rothstein
Cornell University and Yale University

Bert N. Uchino
University of Utah

The effects of aging and loneliness on cardiovascular stress responses were examined in 91 young (18–30 years) and 91 older (65–80 years) normotensive adults. Participants completed the revised UCLA Loneliness Scale and a modified version of the Trier Social Stress Test. Piece-wise linear growth-curve analysis was used to model group differences in resting, reactivity, and recovery levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP). Replicating and extending prior research, analyses revealed age-related increases in resting SBP and DBP. Adjusting for demographics and health covariates, interactions were found for SBP in which age differences in stress reactivity and recovery were greater among lonely than nonlonely participants. Findings provide further evidence that loneliness interacts with age to augment cardiovascular risk to social evaluative threat.

Keywords: aging, cardiovascular, loneliness, reactivity, recovery, stress

Changes in cardiovascular functioning pervade the aging process and may confer increased risks for morbidity and mortality (Pastor–Barriuso, Banegas, Damian, Appel, & Guallar, 2003; Port, Demer, Jennrich, Walter, & Garfinkel, 2000; Kannel, 2000). Although not all of the mechanisms connecting aging to cardiovascular diseases are known, exaggerated stress responsivity has been implicated as a risk factor for the development of a broad array of coronary conditions, including stroke, myocardial ischemia, and hypertension (Pickering & Gerin, 1990; Krantz et al., 1991; Everson et al., 2001). Of importance, stress-induced alterations in cardiovascular responses are not uniform across individuals (see Krantz & Manuck, 1984; Blascovich & Katkin, 1993), but are thought to be influenced by individual difference factors that precipitate adverse reactions to negative social stimuli (Kemeny, 2009; Hawkley & Cacioppo, 2007).

Accruing empirical evidence from longitudinal (Uchino, Holt–Lunstad, Bloor, & Campo, 2005) and well-controlled laboratory (Uchino, Uno, Holt–Lunstad, & Flinders, 1999; Steptoe, Moses, & Edwards, 1990) studies indicates that aging is associated with increased cardiovascular reactivity to psychosocial stress. A recent meta-analytic review of 31 laboratory studies provides converging support for the hypothesis that psychological activation elicited by evocative or stressful laboratory tasks contributes to, and hastens, age-related declines in cardiovascular health (Uchino, Birmingham, & Berg, 2010). The analysis revealed a robust link between age and greater systolic blood pressure (SBP) reactivity, an association that was moderated by the degree of psychological arousal elicited by laboratory tasks. Given that elevated SBP is the primary underlying predictor of hypertension in adults over the age of 65 (Kannel, 2000; Izzo, Levy, & Black, 2000), it is important to understand what individual difference factors may place older adults at greater risk for the development and progression of cardiovascular disease.

One key factor that appears to account for important variation in cardiovascular risk is loneliness, the chronic feeling of distress that accompanies perceived deficits in the quality of social interactions (Cacioppo & Patrick, 2008). Empirical studies show that although loneliness levels are relatively stable across the adult life span (Cacioppo, Hughes, Waite, Hawkley, & Thristed, 2006; Cacioppo et al., 2006), individual differences in loneliness confer increased vulnerability to depressive symptoms (Cacioppo, Hughes, et al., 2006), cognitive decline (Wilson et al., 2007), impaired sleep (Hawkley, Preacher, & Cacioppo, 2010), and mortality in older adults (Shiovitz–Ezra & Ayalon, 2010). Although the data on age-differences in associations between loneliness and cardiovascular responses is more limited (see Hawkley & Cacioppo, 2007), the available evidence supports a link between loneliness and higher SBP, an effect that is augmented in aging adults (Hawkley, Thisted, Masi, & Cacioppo, 2010; Hawkley, Masi, Berry, & Cacioppo, 2006; Cacioppo et al., 2002).

Despite findings of an association between loneliness and stress-induced alterations in cardiovascular responses, the existing data are limited in several important ways. First, although age differences in resting cardiovascular function are well documented (Lakatta, 1998; Ferrari, Radaelli, & Centola, 2003), prior evidence

This article was published Online First October 17, 2011.

Anthony D. Ong, Department of Human Development, Cornell University; Jeremy D. Rothstein, Department of Psychology, Cornell University, and Yale University School of Medicine; and Bert N. Uchino, Department of Psychology, University of Utah.

Preparation of this article was supported by grants from the National Institute on Aging (R21AG026365) and the Bronfenbrenner Life Course Center (College of Human Ecology, Cornell University).

Correspondence concerning this article should be addressed to Anthony D. Ong, Department of Human Development, G77 Martha Van Rensselaer Hall, Cornell University, Ithaca, NY 14853-4401. E-mail: ado4@cornell.edu

for age-related differences in cardiovascular reactivity to stress has been inconsistent, with some studies reporting lower physiological reactivity to emotional stimuli (Levenson, Carstensen, & Gottman, 1994; Tsai, Levenson, & Carstensen, 2000), and others reporting no (Kunzmann & Gruhn, 2005) or higher reactivity (Uchino et al., 1999; Steptoe et al., 1990; Jennings et al., 1997; Uchino, Berg, Smith, Pearce, & Skinner, 2006). Uchino and colleagues (2010) suggested that contextual factors (i.e., stress stimuli type, degree of personal relevance) may play a role in the adequacy of stress manipulation, and that these factors may have a reliable impact on age-related differences in cardiovascular stress responses. This account is consistent with an emerging literature indicating that specific eliciting conditions are necessary to trigger age-related changes in stress physiology (Charles, 2010; Charles & Piazza, 2009), with exposure to social evaluation representing a necessary ingredient in the stress-eliciting process (Dickerson & Kemeny, 2004; Kemeny, 2009; Taylor et al., 2010). Second, prior investigations of loneliness and health have generally examined middle-aged and older adults and, to date, have not compared young and older adults in the same study, thereby providing limited support for life-span hypotheses regarding greater physiological effects of loneliness with age (Hawkley & Cacioppo, 2007). Third, with few exceptions (Steptoe, Owen, Kunz–Ebrecht, & Brydon, 2004; Grant, Hamer, & Steptoe, 2009; i.e., Cacioppo et al., 2002), there has been little laboratory research on the influence of loneliness on cardiovascular stress regulation. Much of the existing literature is based on the reactivity hypothesis that suggests heightened cardiovascular reactivity may signal early signs of coronary dysfunction (Krantz et al., 1991; Treiber et al., 2003). However, rate of cardiovascular recovery from acute stress may also be important. Recent research suggests that impaired or delayed poststress SBP recovery may portend cardiovascular risk in older adults (Steptoe & Marmot, 2005, 2006; Chida & Steptoe, 2010).

The present investigation examined the impact of loneliness on cardiovascular responses to laboratory stress in young and older adults. Consistent with prior research, we predicted that chronological age would be associated with increases in resting SBP and DBP. Moreover, given previous findings, we hypothesized that as compared with young adults, older adults would show greater SBP reactivity and delayed SBP recovery to laboratory stress. Furthermore, we predicted that stress-induced changes in cardiovascular responses would be moderated by individual differences in loneliness. Finally, given the documented wear and tear on physiological systems produced by loneliness and advancing age, we tested the hypothesis that loneliness would interact with age to influence cardiovascular stress reactivity and recovery.

Method

Participants

Ninety-one young (53 women and 38 men ranging in age from 18 to 30 years) and 91 older (45 women and 46 men ranging in age from 65 to 80 years) adults participated in this study. Young adults were recruited from a university participant pool, while older participants were recruited from the community. The majority of the sample identified as White/Caucasian (73.1%), with the remainder identifying as Asian/Asian American (11.0%), Hispanic/Latino (7.1%), Black/African American (5.5%), or other (3.3%). Table 1 shows levels of household income and years of education did not vary significantly between young and older adults. Eligibility criteria included (a) English speaking, (b) nonsmoker, and (c) no systemic glucocorticoids use (e.g., prednisone, prednisolone, and hydrocortisone) in the past 6 months. Additionally, to avoid potential confounds between health- and age-related processes, only participants who rated their health as "good as or better" than the health of most people their age were included. Exclusionary criteria included self-reported active coronary artery disease, uncontrolled hypertension, or current insulin-dependent diabetes; reported use of β-blockers, inhaled β-agonists, or oral or parenteral corticosteroids within 3 months of study; and reported psychiatric hospitalization within the past year.

Of the 202 individuals who initially enrolled into the study, 20 did not meet the inclusionary criteria and were not included in analyses, leaving a sample of 182. These 182 participants were representative of the larger group of individuals who started the study. Comparisons between the group of 182 versus those omitted from analysis showed no differences in gender, ethnicity, level of household income, years of education, or baseline loneliness or depressive symptoms. Moreover, the excluded older adult sample ($n = 18$) did not differ systematically from the included older adult sample ($n = 91$) on any of these variables. Participants who met eligibility criteria provided verbal consent. All participants were instructed to cease alcohol consumption, heavy exercise, use of nonprescription medication (e.g., antihistamines or pain killers that

Table 1
Means and Standard Deviations of Study Variables by Age Group

	Younger ($N = 91$)		Older ($N = 91$)	
Variable	*M*	*SD*	*M*	*SD*
Age	19.64	1.45	71.73	3.16
Education	14.89	3.51	15.35	2.46
Income	$53,641	$64,257	$55,169	$66,514
Loneliness	32.60	7.76	32.52	7.39
Depressive symptoms	8.15	4.89	7.98	4.81
Emotion report				
Anxious	2.18	1.21	2.34	1.48
Embarrassed	1.39	0.84	1.45	0.74
Disgusted	1.48	0.74	1.35	0.84
Frustrated	1.93	1.47	1.85	1.53
Nervous	1.81	1.04	1.78	1.41
Sad	1.02	0.59	1.14	0.81
Sluggish	1.08	0.43	1.03	0.58
SBP				
Baseline	114.78	22.55	122.23	21.47
Speech preparation	119.21	18.67	125.97	20.45
Speech task	123.49	19.63	131.91	19.43
Mental arithmetic	131.27	20.47	140.84	22.57
Recovery	124.59	19.81	134.96	21.71
Postrecovery	115.64	18.94	131.98	20.76
DBP				
Baseline	62.11	10.58	71.47	12.54
Speech preparation	67.72	11.45	72.59	11.47
Speech task	68.41	11.68	74.94	10.58
Mental arithmetic	73.37	12.24	81.61	12.32
Recovery	67.68	11.53	75.98	11.08
Postrecovery	64.55	11.03	73.95	11.49

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure.

have caffeine) for 24 hours, and caffeine beginning at 10 p.m. the evening prior to reporting for testing.

Procedure

Pretask. During an initial 15-min pretask period, participants completed an informed-consent form and received instructions to deliver a 5-min speech and perform mental arithmetic. All participants consented. After providing informed consent, participants completed preexperimental questionnaires containing demographic variables and measures of loneliness and depressive symptoms, and rested quietly. Baseline blood pressure (BP) measurements were assessed at 2-min intervals in the 10-min period. Each participant then performed a variation of the Trier Social Stress Test (TSST), a widely used laboratory psychosocial stressor known to elicit cardiovascular responses in young and older adults (cf. Kudielka, Hellhammer, & Kirschbaum, 2007; Cacioppo et al., 2002). The modified TSST protocol consisted of a 3-min preparation period, a 5-min free speech, and a 6-min mental arithmetic task.

Speech preparation. In the speech task, participants were asked to imagine that they are in a department store shopping when a security guard falsely accuses them of shoplifting. They were then instructed to prepare a speech to (a) tell their side of the story, (b) tell the manager what the security guard did wrong and why the security guard may have suspected them of shoplifting, (c) say how they can prove they did not steal the item, (d) specify what should happen to the security guard for the mistake, and (e) summarize their points. Participants were told that the speech would be videotaped, that trained psychologists would monitor their nonverbal behavior, that a voice–frequency analysis of nonverbal behavior would be performed, and that the speech would be critiqued on content and presentation style.

Stressor period. After the 3-min preparation period, participants were given 5 min to present their speeches. The speech was delivered to an unresponsive experimenter who behaved as if the speech was lacking in quality. Following the speech task, participants performed out loud six 1-min serial subtraction problems continuously for 6 min. Participants were instructed to begin with a specified number and subtract by a particular number (i.e., the subtrahend). For Minute 1, participants were instructed to subtract from the number 297; for Minute 2, the number was 688; for Minute 3, it was 955; for Minute 4, it was 593; for Minute 5 it was 1,200; and for Minute 6, it was 1,741. For the first minute, the subtrahend was 3. The number subtracted for each subsequent minute was contingent on the participant's performance in the previous minute. To increase task relevance, participants were encouraged to work as quickly and as accurately as possible. In previous work, this task has been found to maintain maximal task involvement and induce reliable endocrine and cardiovascular responses in young as well as older adults (Kudielka, Kirschbaum, Hellhammer, & Kirschbaum, 2004).

Recovery period. Following the arithmetic stressor task, participants were taken to another room where they were asked to sit quietly during a 15-min recovery period and complete a postexperimental questionnaire that included a final emotion report.

Posttask. At the end of the experimental session, participants were debriefed and paid $40.00.

Measures

Revised UCLA Loneliness Scale. The R-UCLA is a measure of general loneliness and degree of satisfaction with one's social network and has good construct validity (Russell, Peplaue, & Cutrona, 1980). Examples of the items are, "I lack companionship" and "I feel in tune with the people around me" (reversed scored). Each of the 20 items is rated on a scale of 1 (never), 2 (rarely), 3 (sometimes), and 4 (often). After reverse scoring appropriate items, loneliness scores are calculated by summing all items. The range of possible scores is 20 to 80, with higher scores signifying greater loneliness. Cronbach's alpha for this measure was .92.

Emotion report. Subjective experiences during the testing session were assessed using emotion reports. Specifically, immediately following the TSST, participants rated the amount felt of seven negative emotions (anxious, embarrassed, disgusted, frustrated, nervous, sad, sluggish).

Ratings were made on 6-point Likert scales, ranging from 0 (*not at all*) to 5 (*extremely*). The internal consistency reliability was .89.

Cardiovascular activity. Systolic and diastolic blood pressure (in mmHg) were measured using a Dinamap ProCare Model 420 monitor (GE Medical Systems, Milwaukee, WI). BP assessments were obtained via a properly sized occluding cuff positioned over the brachial artery of the participant's nondominant arm. SBP and DBP was measured in 2-min intervals while participants were seated during the following experimental phases: baseline, anticipation, speech, arithmetic, and recovery periods. To maximize measurement reliability, SBP and DBP assessments within each phase were averaged to produce a single value for each experimental phase. In all, six average SBP and DBP values were calculated across the laboratory session: once at baseline, three times during the stressor task (preparation, speech, math), and twice during recovery.

Covariates. Demographic and health behavior covariates included gender, cardiovascular medication use, and depressive symptoms. In addition, socioeconomic status indicators (i.e., education level and household income) were also included as covariates due to their known associations with cardiovascular stress responses. Medication use was determined by participants reporting their current use of BP medication. A dichotomous variable was created to indicate whether a participant reported currently taking any cardiovascular medication (0 = not currently using medications, 1 = currently using medications). Depressive symptoms were measured using the Center for Epidemiological Studies Depression (CES-D) scale. To reduce measurement overlap, the loneliness item was removed before calculating a total depressive symptoms score.

Analytic Strategy

Piece-wise linear growth-curve analysis (Raudenbush & Bryk, 2002) was used to model separate BP growth trajectories for each person, and to examine predictors of the parameters defining individual differences in rates of BP reactivity and recovery. The piece-wise linear growth-curve approach is particularly appropriate when one wishes to estimate linear change across two or more distinct time periods. In addition to allowing a time series to be divided into meaningful periods (e.g., stressor period; follow-up period), a piece-wise linear growth ap-

proach also permits examination of variation among individuals in the rates of linear growth within each period of interest (Llabre, Spitzer, Saab, & Schneiderman, 2001; see Kristjansson, Kircher, & Webb, 2007). In the current investigation, piece-wise linear growth-curve modeling included two pieces (periods), one representing the reactivity period (when the TSST is introduced) and the other piece representing the recovery period (following conclusion of the TSST). In these models, levels of BP for each person at each measurement occasion was the criterion variable, and was predicted by piece-wise predictors (Level 1) and person-level predictors (Level 2).

The individual linear growth of BP at time i for person j is shown below in:

$$\text{Level 1: } BP_{ij} = \pi_{0j} + \pi_{1j}(\text{Reactivity}_{ij}) + \pi_{2j}(\text{Recovery}_{ij}) + r_{ij},$$

where BP_{ij} is the mean BP measure for person j at time i; π_{0j}, the intercept, is the predicted mean BP value at baseline for person j; π_{1j} is person j's mean rate of linear BP change during the reactivity period; π_{2j} is person j's mean rate of linear BP change during the recovery period; and r_{ij} is the residual of person j's score at time i from the overall model predicted score.

The level-2 model represents the variability in each of the level-1 intercept and growth parameters across individuals:

$$\begin{aligned}\text{Level 2: } \pi_{0j} &= b_{00} + b_{01}(\text{Gender}) + b_{02}(\text{Education}) \\ &\quad + b_{03}(\text{Income}) + b_{04}(\text{Medication}) \\ &\quad + b_{05}(\text{Depression}) + b_{06}(\text{Age}) \\ &\quad + b_{07}(\text{Loneliness}) + b_{08}(\text{Age} \\ &\quad \times \textit{Loneliness}) + u_{0j}, \\ \pi_{1j} &= b_{10} + b_{11}(\text{Gender}) + b_{12}(\text{Education}) \\ &\quad + b_{13}(\text{Income}) + b_{14}(\text{Medication}) \\ &\quad + b_{15}(\text{Depression}) + b_{16}(\text{Age}) \\ &\quad + b_{17}(\text{Loneliness}) + b_{18}(\text{Age} \\ &\quad \times \textit{Loneliness}) + u_{1j}, \\ \pi_{2j} &= b_{20} + b_{21}(\text{Gender}) + b_{22}(\text{Education}) \\ &\quad + b_{23}(\text{Income}) + b_{24}(\text{Medication}) \\ &\quad + b_{25}(\text{Depression}) + b_{26}(\text{Age}) \\ &\quad + b_{27}(\text{Loneliness}) + b_{28}(\text{Age} \\ &\quad \times \text{Loneliness}) + u_{2j},\end{aligned}$$

where b_{00} is the intercept or mean baseline level of BP for person j; b_{10}, b_{20} are the reactivity and recovery parameters for person j; b_{01}, b_{11}, b_{21} represent the difference between the magnitude of the intercept, reactivity, and recovery coefficients for men and women, where gender is dummy coded (women equals 0); b_{02}, b_{12}, b_{22} represent the intercept, reactivity and recovery coefficients for the effect of education (in years); b_{03}, b_{13}, b_{23} represent the intercept, reactivity and recovery coefficients for the effect of household income; b_{04}, b_{14}, b_{24} represent the difference between the magnitude of the intercept, reactivity, and recovery coefficients for medication use, where medication use is dummy coded (not currently using medication equals 0); b_{05}, b_{15}, b_{25} represent the intercept, reactivity and recovery coefficients for the effect of depressive symptoms; b_{06}, b_{16}, b_{26} represent the difference between the magnitude of the intercept, reactivity, and recovery coefficients for young and older adults, where age is dummy coded (young group equals 0); b_{07}, b_{17}, b_{27} represent the intercept, reactivity and recovery coefficients for the effect of loneliness; b_{08}, b_{18}, b_{28} represents the difference in the magnitude of the loneliness effect on the intercept, reactivity, and recovery coefficients for young and older adults; and u_{0j}, u_{1j}, u_{2j}, represent the deviation of person j's intercept and slope (reactivity, recovery) from the overall intercept and slope. In the present study, all of the person-level predictors were standardized (i.e., mean centered and divided by their sample standard deviation) so that each coefficient reflects differences in the outcome per unit of change in the predictor variable. Separate piece-wise linear growth-curve models were estimated for SBP and DBP.

Results

Descriptive Analyses

Descriptive statistics of the main study variables are described in Table 1. Analyses of the emotion reports completed immediately following the stressor tasks confirmed that the tasks elicited higher levels of anxiety ($M = 2.22$, $SD = 1.38$) and frustration ($M = 1.84$, $SD = 1.59$) than any other emotion. Reports of anxiety and frustration did not differ by gender, age, or loneliness. As predicted, the stressor tasks were associated with increases in SBP and DBP in both young and older adults. Consistent with prior research (Cacioppo et al., 2002), mean levels of loneliness did not differ by age or gender. Beyond the expected effect of stressor task, no main effects for, or interactions with, age and loneliness emerged as significant for any of the self-report emotion items.

Preliminary growth-curve analyses examined the distribution of BP values across occasions and persons. These analyses are referred to as *fully unconditional* (Raudenbush & Bryk, 2002) because no predictor variables are specified at any level. Comparison of the variance components between this fully unconditional model and the partially unconditional model (described in the Level 1 equation above) revealed that 54% of the true variance in SBP was accounted for by the linear reactivity coefficient. A test of deviance between the linear reactivity model and the linear recovery model indicated that the time-varying recovery parameter accounted for significant overall model variance, $\chi^2(3) = 1{,}084.88$, $p < .001$. Specifically, the inclusion of the linear recovery parameter in the model accounted for an additional 34% of the SBP variance (for a total of 88%). Similarly, 26% of the variance in DBP was accounted for by the linear reactivity coefficient, with the linear recovery coefficient accounting for an additional 31% of the DBP variance, $\chi^2(3) = 485.134$, $p < .001$ (for a total of 57%). The variance estimates for each of the parameters in the model indicate that there was significant variation between individuals in the intercepts and slopes for the baseline SBP (τ_{00}) = 37.19, $p < .001$; DBP (τ_{00}) = 17.09, $p < .001$, reactivity, SBP

(τ_{11}) = 2.11, $p < .001$, and recovery, SBP (τ_{22}) = 3.31, $p < .001$, periods that warrant the inclusion of person-level predictors.

Piece-Wise Growth-Curve Analyses

A conditional piece-wise growth-curve model was estimated to determine the extent to which age and loneliness explained between-person differences in resting BP levels, average BP reactivity, and average rate of BP recovery. An initial fully conditional model (intercepts and slopes) was run with all of the covariates (b_{01}–b_{05}, b_{11}–b_{15}, b_{21}–b_{25}) and a final model was estimated with only the significant covariates and higher order predictors (two- and three-way interactions) from the initial model.[1] Table 2 shows that parameter estimates and standard errors for the final covariate-adjusted piece-wise growth-curve model.

Resting cardiovascular function. Consistent with prior literature, age was significant predictor of resting SBP and DBP. Examination of the partial effect parameters indicated that the average resting SBP of older adults was 8.91 mmHg higher than that of young adults. Similarly, on average, the resting DBP of older adults were 7.03 mmHg higher than that of young adults. Moreover, with each 1-*SD* increase in loneliness, resting levels of SBP and DBP increased by an additional 3.34 mmHg and 2.13 mmHg, respectively. Finally, there was a significant interaction involving age and loneliness for both resting SBP ($b = 1.49$, $p < .001$) and DBP ($b = 0.79$, $p < .05$). A multiparameter hypothesis test comparing young and older adults confirmed that the age-related differences in SBP and DBP were significant for those high (1 *SD* above the mean) in loneliness.

Cardiovascular stress reactivity. Table 2 shows parameter estimates for SBP and DBP reactivity. As shown in the table, during the stressor or reactivity period, mean SBP and DBP levels increased at a rate of 5.53 mmHg and 3.34 mmHg, respectively. Replicating previous research, older adults exhibited greater SBP reactivity ($b = .61$, $p < .001$), but not DBP reactivity, to acute social stress. Furthermore, as compared with individuals low in loneliness (1 *SD* below the mean) those high in loneliness (1 *SD* above the mean) showed greater SBP ($b = 1.19$, $p < .001$) and DBP ($b = .38$, $p < .001$) reactivity to stress. Of importance, the effect of loneliness on SBP reactivity differed for young and older adults, as indicated by the significant interaction of loneliness and age ($b = .48$, $p < .01$). Inspection of the covariate-adjusted trajectories in Figure 1a clearly shows that lonely older adults (1 *SD* above the mean in loneliness) experienced the steepest increase in SBP reactions to acute stress. To confirm this, we conducted multiparameter hypothesis tests (Raudenbush & Bryk, 2002, pp. 58–61) to test the null hypothesis that young and older adults were similar. We conducted these tests for both lonely (1 *SD* above the mean) and nonlonely (1 *SD* below the mean) individuals. The results from a mutiparameter hypothesis test comparing young and older lonely adults indicated that the reactivity growth patterns were significantly different, $\chi^2(2) = 6.12$, $p < .05$. However, in a multiparameter hypothesis test comparing young and older nonlonely adults, the reactivity growth patterns were not significantly different, $\chi^2(2) = 1.92$, $p < .50$.

Cardiovascular stress recovery. Piece-wise linear growth-curve analyses also examined the influence of age and loneliness on SBP and DBP recovery. As shown in Table 2, during poststressor or recovery period, mean SBP and DBP levels decreased at a rate of 7.16 mmHg and 4.42 mmHg, respectively. As compared with young adults, older adults evidenced more delayed SBP ($b = 3.33$, $p < .001$) and DBP ($b = 1.29$, $p < .001$) recovery following social stress. Furthermore, the significant effect of loneliness indicates that with each 1 *SD* increase in loneliness, the rate of recovery decreased by an additional .42 units. This rate of SBP decline was further qualified by a significant interaction of loneliness and age ($b = 1.16$, $p < .001$). The results from a multiple parameter hypothesis tests comparing young and older adults at high levels of loneliness confirmed that the recovery growth patterns were significantly different, $\chi^2(3) = 8.84$, $p < .05$. In contrast, in a multiple hypothesis test comparing young and older adults at low levels of loneliness, the recovery growth-curve patterns were not significantly different, $\chi^2(3) = 0.69$, $p < .50$. Figure 1a and Figure 1b depict covariate-adjusted SBP recovery trajectories for lonely (1 *SD* above and the mean) and nonlonely (1 *SD* below the mean) young and older adults. It can be seen in Figure 1b that nonlonely older adults experienced a recovery curve similar to (i.e., almost parallel with) the recovery curve for nonlonely young adults. Conversely, in comparison to lonely young

Table 2
Fixed and Random Estimates for SBP and DBP Baseline, Reactivity, and Recovery

	SBP		DBP	
Fixed effects	Estimate	*SE*	Estimate	*SE*
Baseline, π_0				
Intercept	130.67***	0.21	72.89***	0.28
Depression	0.39*	0.17	−0.01	0.18
Age	8.91***	0.36	7.03***	0.37
Loneliness	3.34***	0.17	2.13***	0.26
Age × Loneliness	1.49***	0.32	0.79*	0.38
Reactivity, π_1				
Intercept	5.53***	0.11	3.34***	0.09
Depression	0.16*	0.08	0.03	0.26
Age	0.61***	0.17	0.23	0.15
Loneliness	1.19***	0.08	0.38***	0.08
Age × Loneliness	0.48**	0.14	0.27†	0.14
Recovery, π_2				
Intercept	−7.16***	0.17	−4.42***	0.20
Age	3.33***	0.23	1.29***	0.26
Loneliness	−0.42**	0.12	−0.21	0.17
Age × Loneliness	1.16***	0.20	0.43	0.27

	SBP		DBP	
Random effects	Variance	χ^2	Variance	χ^2
Baseline, u_{0j}	$\tau_{00} = 1.76$***	249.58	$\tau_{00} = 1.35$	178.48
Reactivity, u_{1j}	$\tau_{11} = 0.15$	179.47	$\tau_{11} = 0.07$	83.80
Recovery, u_{2j}	$\tau_{22} = 0.10$	158.09	$\tau_{22} = 0.06$	142.86
Level, r_{ij}	$\sigma^2 = 5.72$		$\sigma^2 = 9.29$	

† $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

[1] To explore possible moderating effects of age, we also examined whether depression would interact with age to influence cardiovascular stress responses. None of the cross-level interactions between depression and age and linear changes in BP responses were significant.

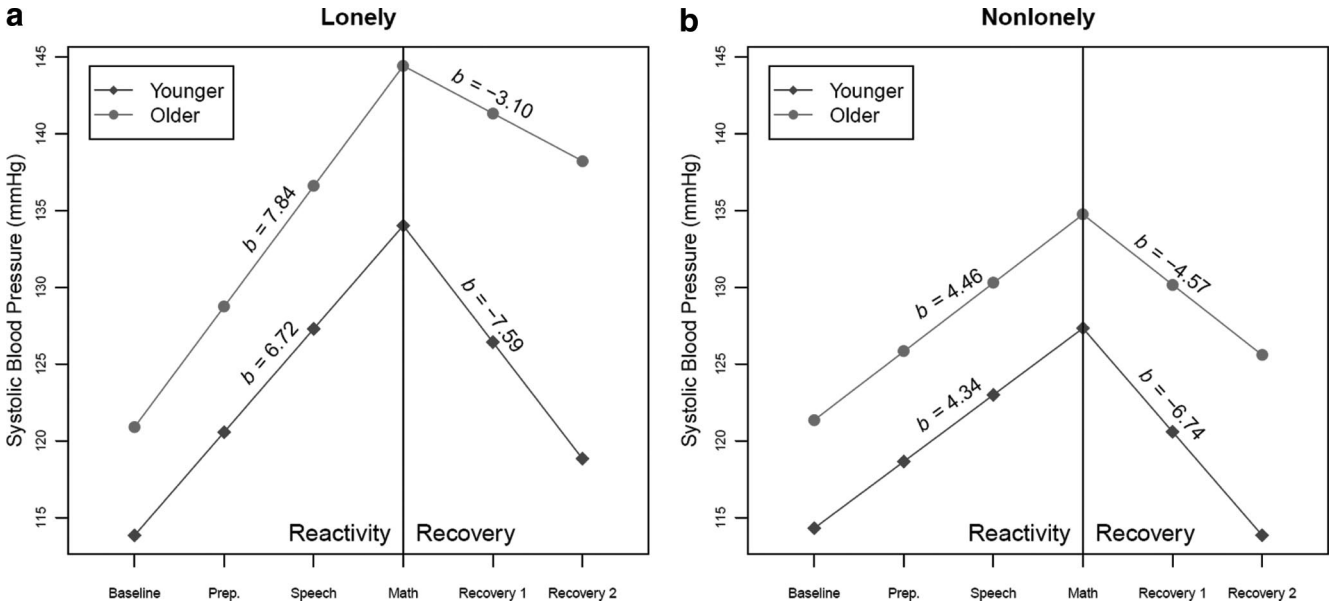

Figure 1. (a) Predicted linear SBP growth trajectories by stressor phase and age for lonely adults. Regression lines represent covariate-adjusted SBP values for young and older adults set at 1 *SD* above the mean in loneliness. (b) Predicted linear SBP growth trajectories by stressor phase and age for older nonlonely adults. Regression lines represent covariate-adjusted SBP values for young and older adults set at 1 *SD* below the mean in loneliness.

adults, lonely older adults (see Figure 1a) experienced more dampened SBP recovery.

Discussion

Although age and perceived loneliness have been implicated in the pathophysiology of cardiovascular disease, the underlying pathways by which aging and loneliness affect cardiovascular health are not well understood. The current study suggests that stress may represent an important pathway by which the health effects associated with loneliness in later life can be explained and predicted. Results of the present study suggest that as compared with young adults, older adults exhibited higher levels of resting SBP and DBP, greater SBP reactivity, and more delayed SBP recovery following laboratory-induced social evaluation. In addition, loneliness was associated with greater SBP reactivity and diminished SBP recovery. Furthermore, the effect of loneliness on stress-induced changes in SBP reactivity and recovery differed for young and older adults.

Findings from the current study are consistent with research showing an age-related increase in SBP reactivity to psychosocial stress (Uchino, Kiecolt Glaser, & Cacioppo, 1992; Uchino et al., 1999). Thus, our results support a model of increased cardiovascular reactivity with advancing age (Uchino et al., 1999; Jennings et al., 1997; Uchino et al., 2006). At least two factors may account for discrepancies between our findings and those reported in previous research (Tsai et al., 2000; Levenson, Carstensen, Friesen, & Ekman, 1991). First, cardiovascular responses were elicited in this study by having participants deliver a speech and perform mental arithmetic, arguably a more active and psychologically evocative task than those used in previous research. Thus, it is possible that age-related increases in physiological stress responses emerge only when a critical threshold of task activation is reached (Labouvie–Vief, 2003; Labouvie–Vief, Lumley, Jain, & Heinze, 2003). Uchino et al. (2005) argued for such a threshold effect whereby older adults showed attenuated stress reactivity at low levels of task activation but greater reactivity at higher levels of activation. Second, the magnitude of age-related physiological reactivity may be influenced by the degree of task self-relevancy (Teachman & Gordon, 2009). Consistent with this view, Kunzmann and Grühn (2005) found no age-related differences in autonomic reactivity when the emotion induction task included age-relevant content (e.g., film clip depicting the loss of a loved one). Thus, further studies examining age-relevant moderators (e.g., physical stressors, negative stereotypes) of the age-SBP reactivity association have the potential to advance work in this area.

Analyses of cardiovascular reactivity responses suggested a significant and unique association between loneliness and SBP responses in young and older adults. In the current study, lonely individuals exhibited greater SBP reactivity to social evaluative stress than nonlonely individuals. Furthermore, the effect of loneliness on SBP reactivity was stronger in older than in young individuals. These results extend prior research on the relationship between loneliness and SBP differences previously observed in middle-aged and older individuals (Hawkley et al., 2006; Cacioppo et al., 2002). Although this pattern is consistent with previous research, there are at least two reasons for why SBP, but not DBP, was affected by the age by loneliness interaction. First, SBP is increasingly seen as a more health-relevant endpoint in older adults (Lloyd–Jones, Evans, Larson, O'Donnell, & Levy,

1999). Moreover, SBP is typically measured with greater measurement reliability as compared wth DBP (e.g., test–retest reliability), so it may be a more sensitive assessment of cardiovascular reactivity (Kamarck et al., 1992).

To the best of our knowledge, our tests of age and loneliness differences in cardiovascular recovery are entirely new and, as such, must be interpreted conservatively. Analyses of blood pressure recovery responses suggested SBP differences related to age and loneliness, with age-differences in SBP recovery emerging in lonely, but not in nonlonely adults. Accumulating evidence suggests that the rate of cardiovascular recovery following acute stress is as important as the magnitude of cardiovascular reactivity in signaling vulnerability to disease (Steptoe & Marmot, 2005, 2006; Chida & Steptoe, 2010). Indeed, there is suggestive evidence (e.g., Borghi, Costa, Boschi, Mussi, & Ambrosini, 1986) that among older adults, recovery effects may be even more diagnostic of disease than reactivity differences. Future experimental studies building on this work are needed to confirm the extent to which impaired stress recovery represents a unique pathway that links age and loneliness with cardiovascular disease.

Our conclusions are necessarily limited by some features of our methods and analyses. First, our results are cross-sectional, and longitudinal data are required before any age-related changes in loneliness and cardiovascular stress responses can be inferred. Nonetheless, our findings are consistent with longitudinal research indicating that the cardiovascular manifestations of loneliness accrue in later life to produce greater increases in SBP (Hawkley, Thisted, et al., 2010). Second, it is unclear to what extent our age findings reflect normative structural changes in the cardiovascular system with age (Uchino et al., 1999), stable individual differences in loneliness (Cacioppo, Hughes, et al., 2006), or cumulative exposure to stressors across the life span (McEwen & Seeman, 1999). Alternatively, the cardiovascular effects of loneliness may be stronger in older adults due to the centrality of emotionally supportive ties in later life (Carstensen, 1992; Lang & Carstensen, 1994). This interpretation is consistent with the recent model of strength and vulnerability integration (SAVI; Charles, 2010; Charles & Piazza, 2009), which posits that age-related vulnerabilities in physiological flexibility may result from situations that involve threats to social belonging. The SAVI model suggests that age may interact with perceptions of social belonging (i.e., perceived loneliness) to influence individual differences in physiological stress responses (Hawkley & Cacioppo, 2007). Additional studies that include young, middle-aged, and older adults are needed to investigate this possibility in more detail. Third, our sample consisted of a cross-section of relatively healthy adults. Thus, future studies should explore whether these effects are robust beyond the contribution of health and cognitive variables that likely covary with age. Finally, the current study assessed patterns of cardiovascular stress reactivity and recovery using laboratory measures of SBP and DBP. However, future studies should include more sensitive and more comprehensive assessments of cardiovascular function (e.g., impedance cardiography) that continuously record cardiovascular activity during psychosocial stress testing. Such assessments may provide additional specificity to psychophysiological data as they have been linked to differential appraisal patterns (e.g., threat/challenge; Tomaka, Blascovich, Kibler, & Ernst, 1997), and self-regulatory processes more generally (e.g., Thayer, Hansen, Saus–Rose, & Johnsen, 2009). In prior work, age differences in blood pressure reactivity appear to be due to a combination of increases in both cardiac output (CO) and total peripheral resistance (TPR; Uchino et al., 2010). However, the effects of loneliness on blood pressure appear to reflect increases in TPR but decreases in CO (Hawkley, Burleson, Berntson, & Cacioppo, 2003). Given these differential patterns it would be difficult to make strong statements as to what may be driving the age by loneliness interactions observed in the present study. Identifying the operative pathways underlying the loneliness effects on age-related differences in cardiovascular stress responses, thus, remains an important task for future work.

Conclusion

Gradual declines in cardiovascular functioning contribute to increased risks for morbidity and mortality in later adulthood. Importantly, alterations in cardiovascular processes are not invariant with age. Recent research suggests that individual differences in perceived loneliness may play a key role in modulating the magnitude of age-related differences in cardiovascular responses to psychosocial stress. Consistent with this research, findings from the present study support the hypothesis that loneliness accentuates age-related differences in cardiovascular stress reactivity and recovery.

References

Blascovich, J., & Katkin, E. S. (Eds.). (1993). *Cardiovascular reactivity to psychological stress and disease.* Washington, DC: American Psychological Association. doi:10.1037/10125-000

Borghi, C., Costa, F., Boschi, S., Mussi, A., & Ambrosini, E. (1986). Predictors of stable hypertension in young borderline subjects: A five-year follow-up study. *Journal of Cardiovascular Pharmacology, 8,* 138–141. doi:10.1097/00005344-198608005-00030

Cacioppo, J. T., Hawkley, L. C., Crawford, E., Ernst, J. M., Burleson, M. H., Kowalewski, R. B., . . . Berntson, G. G. (2002). Loneliness and health: Potential mechanisms. *Psychosomatic Medicine, 64,* 407–417.

Cacioppo, J. T., Hawkley, L. C., Ernst, J. M., Burleson, M., Berntson, G. G., Nouriani, B., & Spiegel, D. (2006). Loneliness within a nomological net: An evolutionary perspective. *Journal of Research in Personality, 40,* 1054–1085. doi:10.1016/j.jrp.2005.11.007

Cacioppo, J. T., Hughes, M. E., Waite, L. J., Hawkley, L. C., & Thristed, R. A. (2006). Loneliness as a specific risk factor for depressive symptoms: Cross-sectional and longitudinal analyses. *Psychology and Aging, 21,* 140–151. doi:10.1037/0882-7974.21.1.140

Cacioppo, J. T., & Patrick, W. (2008). *Loneliness: Human nature and the need for social connection.* New York, NY: Norton.

Carstensen, L. L. (1992). Social and emotional patterns in adulthood: Support for socioemotional selectivity theory. *Psychology and Aging, 7,* 331–338. doi:10.1037/0882-7974.7.3.331

Charles, S. T. (2010). Strength and vulnerability integration: A model of emotional well-being across adulthood. *Psychological Bulletin, 136,* 1068–1091. doi:10.1037/a0021232

Charles, S. T., & Piazza, J. R. (2009). Age differences in affective well-being: Context matters. *Social & Personality Psychology Compass, 3,* 1–14.

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55,* 1026–1032. doi:10.1161/HYPERTENSIONAHA.109.146621

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research.

Psychological Bulletin, 130, 355–391. doi:10.1037/0033-2909.130.3.355

Everson, S. A., Lynch, J. W., Kaplan, G. A., Lakka, T. A., Sivenius, J., & Salonen, J. T. (2001). Stress-induced blood pressure reactivity and incident stroke in middle-aged men. *Stroke, 32,* 1263–1270. doi:10.1161/01.STR.32.6.1263

Ferrari, A., Radaelli, A., & Centola, M. (2003). Aging and the cardiovascular system. *Journal of Applied Physiology, 95,* 2591–2597.

Grant, N., Hamer, M., & Steptoe, A. (2009). Social isolation and stress-related cardiovascular, lipid, and cortisol responses. *Annals of Behavioral Medicine, 37,* 29–37. doi:10.1007/s12160-009-9081-z

Hawkley, L. C., Burleson, M. H., Berntson, G. G., & Cacioppo, J. T. (2003). Loneliness in everyday life: Cardiovascular activity, psychosocial context, and health behaviors. *Journal of Personality and Social Psychology, 85,* 105–120. doi:10.1037/0022-3514.85.1.105

Hawkley, L. C., & Cacioppo, J. T. (2007). Aging and loneliness: Downhill quickly? *Current Directions in Psychological Science, 16,* 187–191. doi:10.1111/j.1467-8721.2007.00501.x

Hawkley, L. C., Masi, C. M., Berry, J. D., & Cacioppo, J. T. (2006). Loneliness is a unique predictor of age-related differences in systolic blood pressure. *Psychology and Aging, 21,* 152–164. doi:10.1037/0882-7974.21.1.152

Hawkley, L. C., Preacher, K. J., & Cacioppo, J. T. (2010). Loneliness impairs daytime functioning but not sleep duration. *Health Psychology, 29,* 124–129. doi:10.1037/a0018646

Hawkley, L. C., Thisted, R. A., Masi, C. M., & Cacioppo, J. T. (2010). Loneliness predicts increased blood pressure: Five-year cross-lagged analysis in middle-aged and older adults. *Psychology and Aging, 25,* 132–141. doi:10.1037/a0017805

Izzo, J. L., Levy, D., & Black, H. R. (2000). Importance of systolic blood pressure in older Americans. *Hypertension, 35,* 1021–1024.

Jennings, R., Kamarck, T., Manuck, S., Everson, S. A., Kaplan, G. A., & Salonen, J. T. (1997). Aging or disease? Cardiovascular reactivity in Finnish men over the middle years. *Psychology and Aging, 12,* 222–238. doi:10.1037/0882-7974.12.2.225

Kamarck, T. W., Jennings, J. R., Debski, T. T., Glickman-Weiss, E., Johnson, P. S., Eddy, M. J., & Manuck, S. B. (1992). Reliable measures of behaviorally-evoked cardiovascular reactivity from a PC-based test battery: Results from student and community samples. *Psychophysiology, 29,* 17–28. doi:10.1111/j.1469-8986.1992.tb02006.x

Kannel, W. B. (2000). Elevated systolic blood pressure as a cardiovascular risk factor. *American Journal of Cardiolology, 85,* 251–255. doi:10.1016/S0002-9149(99)00635-9

Kemeny, M. E. (2009). Psychobiological responses to sociat threat: Evolution of a psychological model of psychoneuroimmunology. *Brain, Behavior, and Immunity, 23,* 1–9. doi:10.1016/j.bbi.2008.08.008

Krantz, D. S., Helmers, K., Bairey, N., Nebel, L., Hedges, S., & Rozanski, A. (1991). Cardiovascular reactivity and mental stress-induced myocardial ischemia in patients with coronary artery disease. *Psychosomatic Medicine, 53,* 1–12.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin, 96,* 435–464. doi:10.1037/0033-2909.96.3.435

Kristjansson, S. D., Kircher, J. C., & Webb, A. K. (2007). Multilevel models for repeated measures research designs in psychophysiology: An introduction to growth curve modeling. *Psychophysiology, 44,* 728–736. doi:10.1111/j.1469-8986.2007.00544.x

Kudielka, B. M., Hellhammer, D. H., & Kirschbaum, C. (2007). Ten years of research with the trier social stress test-revisited. In E. Harmon–Jones & P. Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 56–83). New York, NY: Guilford Press.

Kudielka, B. M., Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). Differential heart rate reactivity and recovery after psychosocial stress (TSST) in healthy children, younger adults, and elderly adults: The impact of age and gender. *International Journal of Behavioral Medicine, 11,* 116–121. doi:10.1207/s15327558ijbm1102_8

Kunzmann, U., & Gruhn, D. (2005). Age differences in emotional reactivity: The sample case of sadness. *Psychology and Aging, 20,* 47–59. doi:10.1037/0882-7974.20.1.47

Labouvie–Vief, G. (2003). Dynamic integration: Affect, cognition, and the self in adulthood. *Current Directions in Psychological Science, 12,* 201–206. doi:10.1046/j.0963-7214.2003.01262.x

Labouvie Vief, G., Lumley, M. A., Jain, E., & Heinze, H. (2003). Age and gender differences in cardiac reactivity and subjective emotion responses to emotional autobiographical memories. *Emotion, 3,* 115–126. doi:10.1037/1528-3542.3.2.115

Lakatta, E. (1998). Cardiovascular regulatory mechanisms in advanced age. *Physiological Reviews, 73,* 413–467.

Lang, F. R., & Carstensen, L. L. (1994). Close emotional relationships in late life: Further support for proactive aging in the social domain. *Psychology and Aging, 9,* 315–324. doi:10.1037/0882-7974.9.2.315

Levenson, R. W., Carstensen, L. L., Friesen, W. V., & Ekman, P. (1991). Emotion, physiology, and expression in old age. *Psychology and Aging, 6,* 28–35. doi:10.1037/0882-7974.6.1.28

Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1994). Influence of age and gender on affect, physiology, and their interrelations: A study of long-term marriages. *Journal of Personality and Social Psychology, 67,* 56–68. doi:10.1037/0022-3514.67.1.56

Llabre, M., Spitzer, S., Saab, P., & Schneiderman, N. (2001). Piecewise latent curve modeling of systolic blood pressure reactivity and recovery from the cold pressor test. *Psychophysiology, 38,* 951–960. doi:10.1111/1469-8986.3860951

Lloyd–Jones, D. M., Evans, J. C., Larson, M. G., O'Donnell, C. J., & Levy, D. (1999). Differential impact of systolic and diastolic blood pressure level on JNC-VI staging. Joint national committee on the prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension, 34,* 381–385.

McEwen, B. S., & Seeman, T. E. (1999). Protective and damaging effects of stress mediators: Elaborating and testing the concepts of allostasis and allostatic load. *Annals of the New York Academy of Sciences, 896,* 30–47. doi:10.1111/j.1749-6632.1999.tb08103.x

Pastor–Barriuso, R., Banegas, J. R., Damian, J., Appel, L. J., & Guallar, E. (2003). Systolic blood pressure, diastolic blood pressure, and pulse pressure: An evaluation of their joint effect on mortality. *Annals of Internal Medicine, 139,* 731–739.

Pickering, T. G., & Gerin, W. (1990). Cardiovascular reactivity in the laboratory and the role of behavioral factors in hypertension: A critical review. *Annals of Behavioral Medicine, 12,* 3–16. doi:10.1207/s15324796abm1201_1

Port, S., Demer, L., Jennrich, R., Walter, D., & Garfinkel, A. (2000). Systolic blood pressure and mortality. *The Lancet, 355,* 175–180. doi:10.1016/S0140-6736(99)07051-8

Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks, CA: Sage.

Russell, D., Peplaue, L. A., & Cutrona, C. E. (1980). The revised UCLA Loneliness Scale: Concurrent and discriminate validity evidence. *Journal of Personality and Social Psychology, 39,* 472–480. doi:10.1037/0022-3514.39.3.472

Shiovitz–Ezra, S., & Ayalon, L. (2010). Situational versus chronic loneliness as risk factors for all-cause mortality. *International Psychogeriatrics, 22,* 455–462. doi:10.1017/S1041610209991426

Steptoe, A., & Marmot, M. G. (2005). Impaired cardiovascular recovery following stress predicts 3-year increases in blood pressure. *Journal of Hypertension, 23,* 529–536. doi:10.1097/01.hjh.0000160208.66405.a8

Steptoe, A., & Marmot, M. G. (2006). Psychosocial, hemostatic, and

inflammatory correlates of delayed postress blood pressure recovery. *Psychosomatic Medicine, 68,* 531–537.

Steptoe, A., Moses, J., & Edwards, S. (1990). Age-related differences in cardiovascular reactions to mental stress tests in women. *Health Psychology, 9,* 18–34. doi:10.1037/0278-6133.9.1.18

Steptoe, A., Owen, N., Kunz–Ebrecht, S., & Brydon, L. (2004). Loneliness and neuroendocrine, cardiovascular, and inflammatory stress responses in middle-aged men and women. *Psychoneuroendocrinology, 29,* 593–611. doi:10.1016/S0306-4530(03)00086-6

Taylor, T., Seeman, T. E., Eisenberger, N., Kozanian, T., A., M., & Moons, W. (2010). Effects of a supportive or an unsupportive audience on biological and psychological responses to stress. *Journal of Personality and Social Psychology, 98,* 47–56. doi:10.1037/a0016563

Teachman, B., & Gordon, T. (2009). Age differences in anxious responding: Older and calmer, unless the trigger is physical. *Psychology and Aging, 24,* 703–714. doi:10.1037/a0016813

Thayer, J. F., Hansen, A. L., Saus–Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine, 37,* 141–153. doi:10.1007/s12160-009-9101-z

Tomaka, J., Blascovich, J., Kibler, J., & Ernst, J. M. (1997). Cognitive and physiological antecedents of threat and challenge appraisal. *Journal of Personality and Social Psychology, 73,* 63–72. doi:10.1037/0022-3514.73.1.63

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65,* 46–62.

Tsai, J. L., Levenson, R. W., & Carstensen, L. L. (2000). Autonomic, subjective, and expressive responses to emotional films in older and younger Chinese Americans and European Americans. *Psychology and Aging, 15,* 684–693. doi:10.1037/0882-7974.15.4.684

Uchino, B. N., Berg, C. A., Smith, T. W., Pearce, G., & Skinner, M. (2006). Age-related differences in ambulatory blood pressure during daily stress: Evidence for greater blood pressure reactivity with age. *Psychology and Aging,* 231–239. doi:10.1037/0882-7974.21.2.231

Uchino, B. N., Birmingham, W., & Berg, C. (2010). Are older adults less or more physiologically reactive? A meta-analysis of age-related differences in cardiovascular reactivity to laboratory tasks. *Journal of Gerontology: Psychological Sciences, 65*B, 154–162. doi:10.1093/geronb/gbp127

Uchino, B. N., Holt–Lunstad, J., Bloor, L., & Campo, R. (2005). Aging and cardiovascular reactivity to stress: Longitudinal evidence for changes in stress reactivity. *Psychology and Aging, 20,* 134–143. doi:10.1037/0882-7974.20.1.134

Uchino, B. N., Kiecolt Glaser, J. K., & Cacioppo, J. T. (1992). Age-related changes in cardiovascular response as a function of a chronic stressor and social support. *Journal of Personality and Social Psychology, 63,* 839–846. doi:10.1037/0022-3514.63.5.839

Uchino, B. N., Uno, D., Holt–Lunstad, J., & Flinders, J. (1999). Age-related differences in cardiovascular reactivity during acute psychological stress in men and women. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences, 54*B, P339–P346. doi:10.1093/geronb/54B.6.P339

Wilson, R. S., Krueger, K., Arnold, S., Schneider, J., Kelly, J., & Barnes, L. (2007). Loneliness and risk of Alzheimer's disease. *Archives of General Psychiatry,* 234–240. doi:10.1001/archpsyc.64.2.234

Received October 4, 2010
Revision received August 10, 2011
Accepted August 15, 2011 ■