

# Genetic and Environmental Effects on Daily Life Stressors: More Evidence for Greater Variation in Later Life

Susan Turk Charles  
University of California, Irvine

David M. Almeida  
Pennsylvania State University

People vary in the occurrence and perceived severity of stressors experienced in their daily lives. In the current study, the authors examined the extent to which individual differences in these relatively minor occurrences are determined by genetic endowment as well as environmental influences and how these effects vary by age. Identical ( $n_{\text{pairs}} = 111$ ) and fraternal ( $n_{\text{pairs}} = 99$ ) twin adults ranging from 25 to 73 years old reported the occurrence and severity of their daily stressors on 8 consecutive evenings. Both genetic and unique environmental effects accounted for the variance in stressor occurrence, whereas shared family and unique environmental effects accounted for the variance in the perceived severity of these stressors. In addition, the influence of unique environment on perceived stress exerted a stronger influence among the older adults than the younger adults.

*Keywords:* aging, stressors, perceived severity

People vary from one another in the number and severity of stressors reported over a typical week. These stressors refer to minor events that disrupt daily life, such as unpleasant social encounters or unexpected work deadlines. Environmental factors undoubtedly play a strong role, but we hypothesize that they are not sufficient to explain variation in the experience and perceptions of daily stressors. In the following study, we examined relative genetic and environmental influences accounting for individual differences in the occurrence and severity of daily stressors among a nationwide sample of same-sex male and female twin pairs ranging from 25 to 73 years old. In addition, we examined the effects of age on overall levels of stressor occurrence and perceived severity as well as its potential moderating effects on relative genetic and environmental variances for each of these experiences.

## Daily Stressors

Minor daily stressors influence emotional and physical functioning on the day they occur (see reviews by Pearlin, Lieberman, Menaghan, & Mullan, 1981; Serido, Almeida, & Wethington,

2004; Zautra, 2003) and create aggregated effects that increase vulnerability to problems, including anxiety and depression (Lazarus, 1966, 1999; Lazarus & DeLongis, 1983; Lazarus & Folkman, 1984; Pearlin & Schooler, 1978; Zautra, Guarnaccia, & Dohrenwend, 1986). Since Pearlin and his colleagues first used the term *the stress process* (Pearlin et al., 1981), a growing number of researchers in sociology and psychology have examined the effects of daily stressors on mental and physical well-being. Researchers studying stressor occurrence have predominantly focused on potential social and environmental casual agents, such as the busyness of the environment (Aspinwall & Taylor, 1997).

Behavioral geneticists have long recognized that individuals seek or create their environment partially on the basis of genetic proclivities (e.g., Plomin, DeFries, & Loehlin, 1977). Few researchers, however, have discussed genetically based mechanisms responsible for these individual differences in exposure to diverse types of environments, including variation in exposure to daily stressors. Scientists recognize that personal factors may be responsible for the occurrence of daily minor but stressful events, such as “disorganized, provocative, and generally maladaptive behavior” (Epstein & Katz, 1992, p. 814), but to date the heritability of these occurrences has not been examined. Moreover, the relationship between stressor occurrence and correlates with possible biological origins have been examined in only a handful of studies that have focused on neuroticism, a trait influenced by both genetic and environmental effects. People with higher levels of neuroticism report more frequent daily stressors (Bolger & Zuckerman, 1995; Suls & Martin, 2005).

## Insight From Research on Life Events

The idea that life experiences are partially heritable has not been examined in the context of daily stressors, but it has been studied in relation to major life events. Major events include infrequent but significant events such as the death of a family member, the loss of a job, or divorce. Although daily stressors are conceptually distinct from life events in their predictive ability and effects on

---

Susan Turk Charles, Department of Psychology and Social Behavior, University of California, Irvine; David M. Almeida, Department of Human Development and Family Studies, Pennsylvania State University.

The research reported in this article was supported by Grants AG16731 and NIH R01-AG19239 from the MacArthur Foundation Research Network on Successful Midlife Development and the National Institute on Aging awarded to David M. Almeida and Grant NIH R01 AG-23845 awarded to Susan Turk Charles. We thank Wendy Johnson, Robert Krueger, and Bo Cleveland for their helpful comments on drafts of this article and to Shaun Purcell for sharing his script and his helpful comments in running these models.

Correspondence concerning this article should be addressed to Susan Turk Charles, Department of Psychology and Social Behavior, University of California, Irvine, 3340 Social Ecology II, Irvine, CA 92697-7085. E-mail: scharles@uci.edu

health and well-being, they are related to one another (Rowlison & Felner, 1988). In addition, daily stressors are often the sequelae to life events and the reason why people experience psychosocial distress in response to major life events (Wagner, Compas, & Howell, 1988). As such, the same factors that increase vulnerability to a major event may also increase vulnerability to daily stressors. Both genetic and environmental effects are necessary to explain individual differences in number of life events (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1993; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990), with genetic effects accounting for 28% to 40% of the variance (for a review, see Plomin, DeFries, McClearn, & Rutter, 1997). Using the life events literature to inform this study, we hypothesized that genetic and environmental effects would exert similar influences on variation in daily stressors. Also from the life events literature, we examined the role of neuroticism in the occurrence of these stressors.

Neuroticism was originally conceptualized to assess nervous system activity, with high levels of neuroticism indicating a nervous system that becomes easily disrupted and is characterized by physiological instability (Eysenck, 1963/1998). Higher levels of neuroticism are related to a greater number of negative life events (Headey & Wearing, 1989). Similarly, higher neuroticism is related to a greater amount of stressor exposure (Gunthert, Cohen, & Armeli, 1999) and stressor reactivity (Mroczek & Almeida, 2004). Because the genetic effects on a number of life events are mediated by neuroticism (Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997), we examined whether neuroticism may also mediate the genetic variation for the occurrence of minor events of daily life as well.

### Perceptions of Daily Stressors

How an individual appraises these stressors is associated with well-being and is sometimes a more powerful predictor of health than the actual stressor occurrence (Carver, Scheier, & Pozo, 1992; Epel et al., 2004; Gunthert, Cohen, & Armeli, 1999; Mittelman, Roth, Haley, & Zarit, 2004). Researchers have posited that both innate temperament and the environment shape these appraisals (Aspinwall & Taylor, 1997; Bolger & Schilling, 1991; Lazarus, 1991). Regarding environmental influences, Lazarus (1991) discussed unique aspects idiosyncratic to each day and to each person, but he also mentioned childhood experiences that may be shared by siblings. He stated that childhood consists of formative years when a person's most important and stable beliefs are established. Other researchers have emphasized the role of parents in modeling and influencing children's appraisals to emotional events. For example, parents model behavior that predicts how children later respond to everyday stressors (e.g., Eisenberg et al., 2001; Patterson, DeBaryshe, & Ramsey, 1989). For these reasons, genetic as well as environmental (both shared and unique) influences may contribute to variation in the perceived severity of daily stressors.

### Possible Influences of Age

Researchers have indicated that aging does not represent a monolithic predictable trajectory and that heterogeneity in cognitive, physical, and psychosocial functioning increases the number of individual differences in later life (e.g., Baltes & Baltes, 1990; Rowe & Kahn, 1987, 1997). As a result, variation in the occur-

rence and perception of stressors may increase with age, with implications for the relative genetic and environmental influences responsible for this variability. With age, the accumulated effects of decisions and behaviors over a lifetime create greater heterogeneity among older adults. For example, patterns of exercise, weight control, and other health behaviors practiced over the years create greater differences in health status among older adults than among younger adults (Rowe & Kahn, 1987). Thus, the influence of unique environmental effects may increase with age.

Unique environmental variance may also increase with age for the perceived severity of these stressors. Lazarus (1991) maintained that although the foundation for appraisals is laid during the formative years, alterations occur as a result of life experiences and aging. Goals, motivations, and ways of thinking are altered as a result of a "challenging world and our changing functional abilities" (Lazarus, 1991, p. 346). Greater variability in reports of affect (e.g., Charles, Reynolds, & Gatz, 2001) may reflect greater variability in appraisals and responses to daily events. For this reason, unique environmental effects may exert stronger influences on variation in perceived severity of stress among older adults relative to younger adults.

Although older age may be associated with greater interindividual variability for stress processes, researchers in prior studies have found that aging is nonetheless associated with benefits regarding stressor occurrence and perceived severity when examining group mean levels. Older adults report fewer undesirable daily events than do younger adults (Almeida & Horn, 2004; Zautra, Finch, Reich, & Guaranaccia, 1991). When stressors do occur, older age is related to reductions in perceived severity and affective distress (Almeida & Horn, 2004; Phifer, 1990; Uchino, Berg, Smith, Pearce, & Skinner, 2006). For these reasons, we predicted greater variation but lower overall mean levels of stressor occurrence and perceived severity among older adults compared with younger adults.

### The Present Study

In the present study, we examined the average occurrence of daily stressors over a week and their perceived severity among a sample of adult male and female same-sex twins ranging from 25 to 73 years old. Every evening over 8 consecutive days, people reported daily stressors they had experienced and rated their severity. Extrapolating from the life events literature, we hypothesized that the variance in average number of stressors experienced over the course of the week is accounted for by genetic and unique environmental effects, with genetic effects being additive and contributing somewhere between 28–40% of the variance. On the basis of the writings of Lazarus (1991), we proposed that both shared and unique environmental influences as well as genetic influences account for variation in perceived severity of these stressors. Additionally, for both the occurrence and perceived severity of daily stressors, we examined the influence of age on overall mean levels and as a moderator of relative genetic and environmental effects on the variance of these stressor characteristics. Finally, on the basis of the life events literature and prior studies that have established relationships between neuroticism and daily stressors, we explored whether neuroticism would mediate any observed genetic effects.

## Method

### Sample and Procedure

Data for the analyses are from the National Study of Daily Experiences (NSDE), one of the in-depth studies from the National Survey of Midlife in the United States Survey (MIDUS) carried out under the auspices of the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife. The total NSDE sample of 1,483 comprises 1,031 randomly selected respondents from the MIDUS random-digit-dialed subsample and 452 MIDUS twins from the larger MIDUS twin sample (Johnson & Krueger, 2005; Kessler, Gilman, Thornton, & Kendler, 2004). We selected twin pairs to participate in this study if they had high self-reported certainty of zygosity, ascertained from a series of questions regarding physical similarities and validated using a subsample of respondents whose zygosity was confirmed using DNA analysis (for further information on the sample, refer to Kessler et al., 2004). Both members of 210 twin pairs participated in the study, including 111 identical, or monozygotic (MZ), twin pairs and 99 same-sex fraternal, or dizygotic (DZ), twin pairs.

Twins ranged in age from 25 to 73 years old ( $M = 43.4$  years,  $SD = 11.8$  years) and all were reared together. Forty-seven percent of the respondents were men and 53% were women. Most of the respondents (76%) were married, 9% were divorced, 2% were separated, 2% were widowed, and 11% were never married. Ninety-two percent of the respondents were European American, 6% were African American, and 2% reported another ethnicity. Seventy-seven percent of the respondents were currently working and 6% were retired. On an assessment of education level that used a 12-point scale ranging from 1 = *no education* to 12 = *doctorate degree*, participants averaged a little more than a high school degree ( $M = 7.03$ ,  $SD = 2.35$ ). Many of the twins were relatively young, with approximately half the sample ranging from 25 to 41 years old ( $n = 58$  MZ twin pairs,  $n = 49$  DZ twin pairs) and half the sample ranging from 42 to 73 years old ( $n = 53$  MZ twin pairs,  $n = 50$  DZ twin pairs).

Over the course of 8 consecutive evenings, respondents completed short telephone interviews about their daily experiences. On the final evening, respondents also answered several questions evaluating this previous week. To aid in the independence of reporting, we interviewed cotwins at least 2 weeks apart from each other. The initiation of interviews was staggered across the day of the week to control for possible confounding between day of study and day of week. Retention rate for the twin sample was 88%, and 90% of the respondents completed at least 6 interviews. The average number of interviews completed across the twin sample was 7.12, providing 2,990 daily interviews.

### Measures

**Daily stressor occurrence.** Daily stressor occurrence was assessed through a semistructured Daily Inventory of Stressful Experiences (Almeida, Wethington, & Kessler, 2002). The inventory consisted of a series of stem questions asking whether specific types of daily stressors had occurred in the past 24 hours and a set of interviewer guidelines for probing affirmative responses. Participants were asked about the occurrence of six specific stressors: an argument or a disagreement with someone, a time when they could have engaged in a disagreement but decided to let it pass,

something happened at work that most people would consider stressful, something happened at home that most people would consider stressful, an experience of discrimination, something happened to a close friend that was upsetting. Participants were also asked to describe any other stressors that might have occurred but were not previously mentioned. Notably, all of these events do not represent major life events but instead the minor annoyances of daily life. The most often reported occurrence in the sample was an avoided disagreement, followed by a problem at work, a problem at home, and an actual argument. Respondents completed interviews each evening of the 8-day protocol. Because people varied in the number of days they participated in the study, the total number of stressors mentioned across the 8 days was divided by the number of recorded days. Thus, the total score represented an average stressor rating across all participant days, such that 3 stressors across 8 days yielded a score of 0.375. When asked to rate whether the number of stressors experienced across the week was typical of their daily life, the majority of adults reported the week was typical (62%), with the rest equally distributed between stressors being more frequent or less frequent than usual.

**Perceived severity.** Perceived severity was measured by one question asking participants to rate the severity of each stressor on a 4-point Likert-type scale, from 1 = *not at all severe* to 4 = *extremely severe*. The average score across all reported stressors throughout the 8-day interview was used in the analyses.

**Neuroticism.** Neuroticism was assessed in the MIDUS survey using a short form developed specifically for this survey and validated in pilot studies (see Lachman & Weaver, 1998). Participants responded to statements asking them to “please indicate how well each of the following describes you” on a scale from 0 = *not at all* to 4 = *a lot*, and their responses were averaged. The adjectives were *moody*, *worrying*, *nervous*, and *calm* (which was reversed scored). Items were derived from other personality scales, including Goldberg’s (1992) Big Five markers, and have been used in prior research to document associations between health and affect measures (Lachman & Weaver, 1998; Mroczek & Kolarz, 1998). Cronbach’s alpha was .79.

### Analytic Technique

Biometric models consisted of two sets of analyses. The first set of analyses partitioned the variance for each of the stressor characteristics—their occurrence and perceived severity—into genetic and environmental components. These components include additive genetic (A), nonadditive genetic (D), shared environmental (C), and unique or nonshared environmental (E) effects. Additive genetic effects represent the sum of the average effect of the alleles that contribute to the phenotype: in this study, stressor occurrence and stressor severity. Nonadditive effects include the effects of dominance (allelic interaction within a gene) or epistasis (gene-gene interaction). The probability of DZ twins sharing two necessary alleles for nonadditive effects is 25%, and thus nonadditive effects increase the MZ intraclass correlation relative to the DZ intraclass correlation to a greater extent than do additive effects, where the probability of DZ twins having the same genes is approximately 50%. Shared environmental effects represent the environment shared between twin pairs in their rearing as well as later in life. Similar parental discipline and similar religion are examples of aspects that are commonly shared by twin pairs who

are reared together. Unique environmental effects are those not shared by twins. These effects would make twins no more similar to each other than they are to others in their population. Subtracting the intraclass correlations of MZ twin pairs for a specific phenotype from 1.00 provides a rough estimation of the effects of unique environmental influences. Models examining these variance components assume that the variance of a phenotype, such as stressor occurrence, is a linear function of these effects.

Stressor occurrence and perceived severity were each examined separately in univariate analyses that calculated the variance components using maximum likelihood estimation of the raw data using Mx (Neale, 1997). Prior to these analyses, MZ twins and DZ twins were placed in separated groups. Within these groups, the firstborn twin (discerned using self-reported questionnaire data) was always included as Twin 1 in the analyses, and the second-born twin was labeled Twin 2. Differences in means and variance for both stressor measures were examined between Twin 1 and Twin 2 within each of the MZ and DZ groups to ensure that all people were sampled from a similar population.

For each stressor measure, a full model estimated the genetic, shared environment and unique environmental effects (i.e., the A, D, C, and E effects). For both stressor occurrence and stressor severity, the ACE model resulted in a lower  $-2$  log likelihood fit estimation ( $-2LL$ ) than did the ADE model, and for this reason, only results from the ACE model are presented in this article. Next, reduced models were compared with the full ACE model to examine whether parameters—that is, A and/or C—could be dropped without resulting in a model that had a significantly worse fit, as indicated by a significantly increased chi-square fit of the overall model. Akaike's information criterion (AIC), calculated as chi-square divided by twice the degrees of freedom, takes into account the complexity of the model and was used to identify the most parsimonious model from among the reduced models, with a lower AIC representing a more parsimonious model (Akaike, 1987).

A second set of analyses examined the moderating effects of age on the variance components for the occurrence of stressors and for

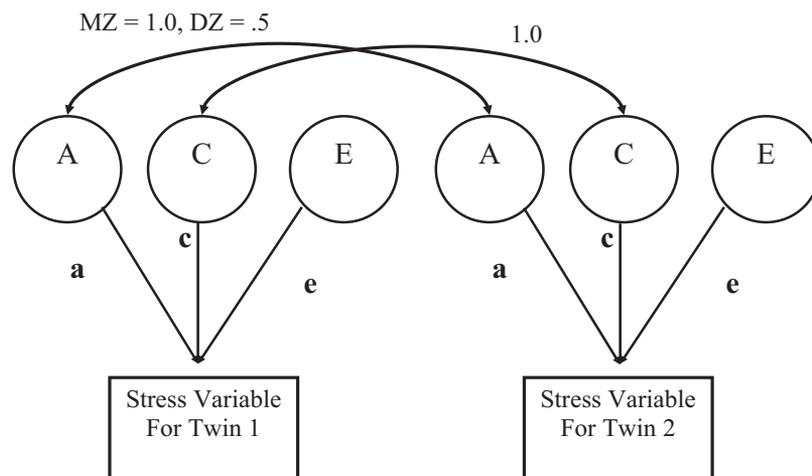
stressor severity. These models used Mx script from Purcell (2002) that allowed the estimation of the moderating effects of age on variance components and on the overall mean of each stressor variable. The difference between the model described above and this moderation model is seen when comparing Figures 1 and 2. Figure 1 displays the univariate ACE model that estimates the variance components of a phenotype, such as stressor occurrence, for Twin 1 and Twin 2. Figure 2 shows the same basic model with the inclusion of an additional phenotype (age), which exerts a main effect on the mean ( $\beta_M$ ), and moderating effects on the genetic ( $\beta_T$ ), shared environment ( $\beta_U$ ), and unique environment ( $\beta_V$ ) variance components. In this way, the model tests whether variance components were present and to what degree for each level of age. Moderation is evidenced as the degree to which these variance components differ across age levels.

Tests of moderation were calculated using the maximum likelihood fit function and the raw data option in Mx (Neale, 1997). Full and reduced models tested the effect of dropping the moderating effects on the variance components. If dropping the influence of age resulted in a nonsignificant difference in the overall fit of the model, age was not related to change in variance components. For a comprehensive description of this analytic technique, refer to Purcell (2002). Standardized parameter estimates are presented for the univariate models illustrated in Figure 1, and nonstandardized estimates are presented for the moderation models, illustrated in Figure 2.

## Results

### Occurrence of Stressors

Participants reported on average 4 stressors across the week,  $M = 3.91$ ,  $SD = 3.23$ , range = 0–21. Across the entire sample, participants reported at least 1 stressor on an average of 36% of study days, with 11% of study days including 2 or more stressors. Men and women reported similar numbers of stressors averaged



*Figure 1.* Model for estimating the mean and variance components for one phenotype (stressor variable). A = additive genetic effects; C = shared environmental effects; E = unique or nonshared environmental effects; a = value of the pathway coefficient for latent variable A; c = value of the pathway coefficient for latent variable C; e = value of the pathway coefficient for latent variable E; MZ = monozygotic twin pairs; DZ = dizygotic twin pairs.

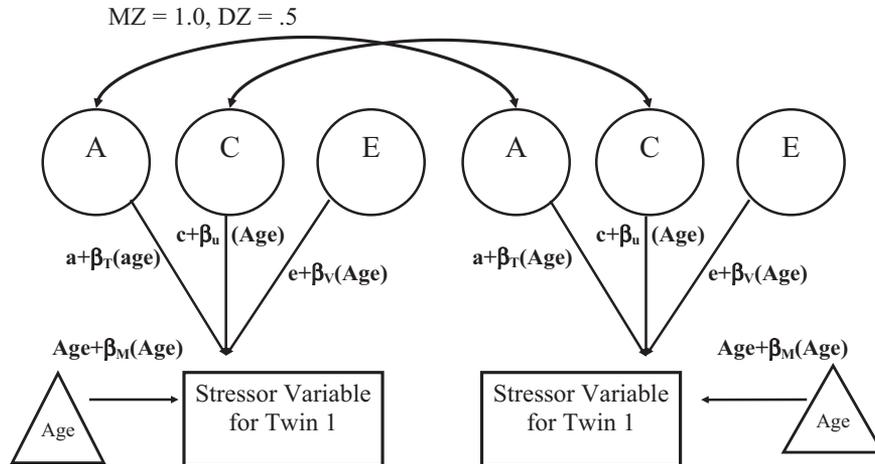


Figure 2. Model for estimating the moderating effects of age on the overall mean genetic and environmental variance components of another phenotype (stressor variable). A = additive genetic effects; C = shared environmental effects; E = unique or nonshared environmental effects;  $\beta_M$  = the effect of age on this overall intercept;  $\beta_T$  = the effect of age on the genetic variance component;  $\beta_U$  = the effect of age on the shared environmental variance component;  $\beta_V$  = the effect of age on the unique environmental variance component; MZ = monozygotic twin pairs; DZ = dizygotic twin pairs.

across all interview days, although women trended in the direction of more stressors per day compared with men:  $M = 0.53$ ,  $SE = 0.03$ , for women versus  $M = 0.40$ ,  $SE = 0.03$ , for men,  $t(418) = -1.78$ ,  $p = .07$ . Age was related to stressor occurrence, in that older adults reported fewer stressors than did younger adults,  $r = -.15$ ,  $p = .003$ ,  $n = 420$  individuals. To illustrate, people aged 25–35 years reported an average of 0.56 ( $SD = 0.39$ ) stressors per day, whereas people aged 65 to 74 years reported an average of 0.40 ( $SD = 0.38$ ) stressors per day. In addition, higher levels of neuroticism were related to greater numbers of stressors reported,  $r = .15$ ,  $p = .002$ ,  $n = 420$ .

Prior to biometric modeling, assumptions of equal means and variances were tested. Ten outliers for the occurrence of stressors were rescored to equal the next highest value in a Winsorized mean procedure. After this recoding, means and variance were similar when comparing Twin 1 and Twin 2 within zygosity. MZ and DZ twins also did not vary in the overall means or variances of their reported stressors. Comparing intraclass correlations of MZ ( $r = .37$ ,  $p < .01$ ,  $n_{\text{pairs}} = 111$ ) with those of DZ ( $r = .16$ ,  $ns$ ,  $n_{\text{pairs}} = 99$ ) twin pairs suggested the presence of genetic effects. The results of the full ACE model are presented in Table 1. The additive genetic effects accounted for 37% of the variance, and unique environmental effects accounted for the remaining variance (63%). A model estimating only unique effects (the E model) proved a worse fit than the full model, and the most parsimonious model was the AE model.

We also examined possible effects of age on the genetic and environmental influences on stressor occurrence. These effects, depicted by the beta weights associated with the variance components in Figure 2, did not contribute to the overall fit of the model. Thus, age did not interact significantly with either the genetic or the environmental effects on stressor occurrence. In addition, we examined the effects of neuroticism. Neuroticism was correlated

with stressor occurrence but did not account for the genetic variance in this phenotype.<sup>1</sup>

### Subjective Severity

Respondents rated their stressors, on average, between 2 (*not very stressful*) and 3 (*somewhat stressful*;  $M = 2.65$ ,  $SD = 0.64$ ). Consistent with prior research (Almeida & Horn, 2004; Almeida et al., 2002), mean levels of subjective severity varied by gender and by age. Women rated stressors as being more severe than did men (for women,  $M = 2.81$ ,  $SE = 0.04$ ; for men,  $M = 2.47$ ,  $SE = 0.05$ ),  $t(379) = -5.36$ ,  $p = .0001$ , and older age was related to lower perceived severity ( $r = -.15$ ,  $p = .003$ ,  $n = 381$  individuals). To illustrate, people aged 25–41 years ( $n = 205$  individuals) reported, on average, a rating of 2.73 ( $SD = 0.57$ ), whereas adults 42 years of age and older ( $n = 176$  individuals) reported an average rating of 2.57 ( $SD = 0.67$ ). Of note, people who reported

<sup>1</sup> We had examined whether neuroticism may explain the genetic effects in daily stressors as was found for negative life events (Saudino et al., 1997). Comparing the cross-correlation (i.e., the correlation between the neuroticism score for Twin 1 and stressor occurrence for Twin 2, and vice versa) between MZ twins ( $r = .13$ ) and DZ twins ( $r = .28$ ), however, suggested that variation shared between neuroticism and stressor occurrence most likely stems from the effects of unique environment and not genetic influences. We also examined other personality traits not discussed in this article that have been examined in the life events literature (openness to experience and extraversion), but they also did not explain the genetic variance in the occurrence of daily stressors. Of note, analyses examining these relationships used bivariate models that were not described in the *Analytic Technique* section, as findings were not significant and not the main focus of the article. Because genetic effects were not observed for stressor severity, we did not test for a shared genetic component between stressor severity and neuroticism.

Table 1  
*Model Fit Results for Stressor Occurrence*

Effect	A	C	E	$\Delta df$	$\Delta\chi^2$	$p$	AIC
ACE	.37	.00	.63				
AE	.37		.63	1	0.00	1.00	-2.00
CE		.26	.74	1	3.49	.06	1.49
E			1.00	2	18.219	.000	14.22

Note. A = genetic effects; C = shared environmental effects; E = nonshared environmental effects;  $\Delta df$  = change in the degrees of freedom compared with the full ACE model;  $\Delta\chi^2$  = change in chi-square fit from the full ACE model;  $p$  = the test of significance when comparing the  $\Delta\chi^2$  with the full  $\chi^2$ ; AIC = Akaike's information criterion; BIC = Bayesian information criterion. Standardized variance components are presented for each parameter. For the full ACE model,  $-2\log$  likelihood = 413.588,  $df = 416$ , AIC = -418.412, BIC = -905.404, age-adjusted BIC = -246.341.

no stressors during the study period did not contribute to these analyses. As a result, participants contributing to these analyses included 103 MZ twin pairs (including 97 complete twin pairs where both members responded to the severity questions) and 89 DZ twin pairs (including 78 complete twin pairs). Thirty adults over the age of 42 years and 9 people younger than the age of 42 years reported no stressors and thus were not included.

Means and variances were not significantly different across groups of twins when comparing Twin 1 with Twin 2 within zygosity and when comparing across MZ and DZ twins. Comparing intraclass correlations of MZ ( $r = .25, p < .05, n_{\text{pairs}} = 97$ ) with those of DZ ( $r = .30, p < .01, n_{\text{pairs}} = 78$ ) twin pairs suggested there were no genetic effects for perceived severity and some effects of shared environment. Because gender and age can artificially inflate shared environmental effects, we included them as covariates in this analysis. Results of full and reduced models estimating genetic, shared, and nonshared effects are presented in Table 2. Estimated parameters for the full model suggest no genetic effects, moderate shared effects (23%), and large nonshared effects accounting for 77% of the variance of perceived severity. Dropping either the genetic or the shared environmental effects did not significantly reduce the fit of the model, although an

Table 2  
*Model Fit Results for Perceived Severity*

Effect	A	C	E	$\Delta df$	$\Delta\chi^2$	$p$	AIC
ACE	.00	.23	.77				
AE	.24		.76	1	2.24	.13	0.24
CE		.23	.77	1	0.00	1.00	-2.00
E			1.00	2	8.55	.01	4.55

Note. Age and gender were included as covariates in this analysis, because both may potentially inflate estimates of C. A = genetic effects; C = shared environmental effects; E = nonshared environmental effects;  $\Delta df$  = change in the degrees of freedom compared with the full ACE model;  $\Delta\chi^2$  = change in chi-square fit from the full ACE model;  $p$  = the test of significance when comparing the  $\Delta\chi^2$  with the full  $\chi^2$ ; AIC = Akaike's information criterion; BIC = Bayesian information criterion. Standardized variance components are presented for each parameter. For the full ACE model,  $-2\log$  likelihood = 692.279,  $df = 375$ , AIC = -57.721, BIC = -652.837, sample size-adjusted BIC = -58.764.

E-only model was significantly worse than the full model, indicating the significant role of familial resemblance.

We next calculated the ACE components for stressor severity and included age as a potential moderator. Results are presented in Table 3. Comparing models with and without the inclusion of age revealed that including age significantly improved the fit of the model: the full model with age,  $-2LL = 706.296, df = 373$ , versus the reduced model with no age effects,  $-2LL = 714.378, df = 376, \Delta C^2 = 8.08, \Delta df = 3, p = .04, AIC = 2.08$ . A comparison between the nested and full models indicated that age significantly moderated the effect of nonshared environment on stressor severity.

The full model suggests several other interesting trends across age groups. For example, genetic effects were not indicated by the MZ and DZ intraclass correlations or the univariate analyses in Table 2, but these effects are suggested when examining intraclass correlations of the youngest MZ twins, aged 25 to 41 years ( $r = .40, p = .003, n = 54$  intact twin pairs) and same-aged DZ twins ( $r = .24, p = .11, n = 45$  intact twin pairs). In addition, shared environmental variance was not influenced by age, but its relative contribution diminished in light of the significantly increasing influence of environmental variance. For example, using the parameter estimates from the moderation model to estimate effects at a specific age, the contributions of genetic, shared, and nonshared environmental effects accounting for variation in perceived severity estimated from this model are 20%, 32%, and 48%, respectively, for people aged 25 years old. For people aged 65 years old, however, the contributions of these same components are estimated at 0%, 18%, and 82%. These estimates are calculated by determining the percentages of genetic, shared, and nonshared environmental effects at different ages, displayed in Figure 3. Taking all of the effects together, the overall variance of this measure increased with age, suggesting that even though the mean rating of perceived severity diminishes with age, older adults exhibit greater variation around the mean than do younger adults. A significant reason for this increased variance is the greater influence of nonshared environmental variance.

Table 3  
*The Full Model and Best-Fitting Model for the Moderation of Age and Gender on Subjective Severity*

Parameter in the model	Estimate	
	Full model	Best-fitting model
ACE		
M	3.02	3.01
Genetic variance	.417	—
Shared environmental variance	.288	.345
Nonshared environmental variance	.246	.294
Age		
Main effect	-.009	-.009
Genetic moderation	-.006	—
Shared environmental moderation	.0003	—
Nonshared environmental moderation	.006	.005
-2log likelihood ( $df$ )	706.296 (373)	706.733 (375)

Note. Parameters are unstandardized estimates. Comparison between the full and best-fitting model;  $\chi^2 = 0.436 (\Delta df = 3), p = .93, AIC = -5.35$ . ACE = additive genetic, shared environmental, and nonshared environmental effects.

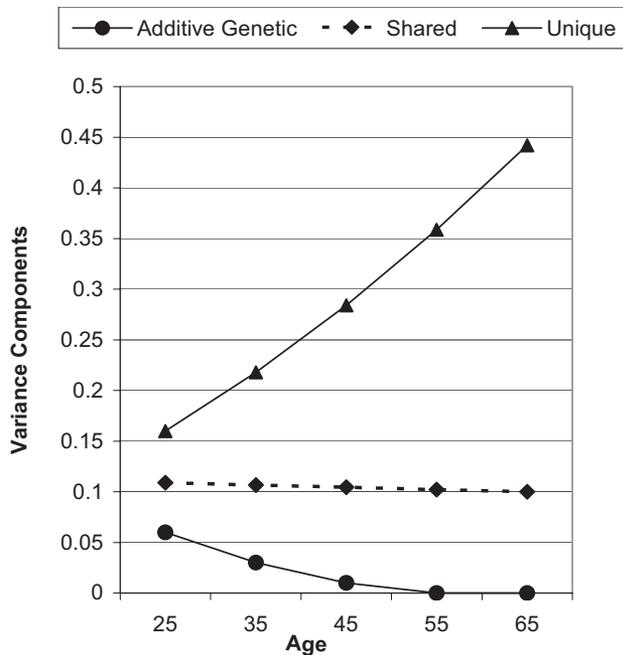


Figure 3. Estimated unstandardized genetic and environmental (shared and nonshared) variance components of daily stressors as a function of age (in years).

## Discussion

Minor strains and problems are natural occurrences in the rhythm of daily life: an unpleasant social encounter, a problem at work, an unexpected and unwelcome expense. With the present study, we are the first to examine the extent to which relative genetic and environmental effects (both those shared by siblings and those unique to an individual) accounted for the number and perceived severity of these relatively minor occurrences. Analyses yielded estimates for both genetic and environmental effects explaining individual differences in the occurrence of stressors regardless of age. Once these stressors occurred, however, only environmental effects influenced the perceived severity of these experiences. Moreover, age moderated the influence of unique environmental effects on perceived severity, indicating that older adults are more variable from each other than younger adults are on reports of perceived severity as a result of nonshared experiences.

### *Stressor Occurrence: Genetic and Environmental Influences*

We predicted that proportions of genetic and environmental influences documented in life events research would be similar to those giving rise to the unpleasant annoyances of daily life, and the current findings are generally consistent with this view. The percentages of genetic and nonshared environmental effects contributing to individual differences in daily stressors—37% and 63%, respectively—were similar to the percentages of genetic and nonshared environmental variance in life events—40% and 60%, respectively—found in a previous study (Plomin et al., 1990).

Similarities were not found, however, for factors accounting for the genetic variance. Whereas personality traits completely account for genetic variation in major life events (Saudino et al., 1997), these same traits did not account for the genetic variance for daily stressors.

Specific mediators for the genetic influences on daily stressor occurrence are speculative at this time. These factors may include characteristics such as distractibility and forgetfulness that may lead to minor mishaps or misunderstandings leading to daily stressors. Other possibilities include traits such as impulsiveness or novelty seeking, where quick action may result in creating situations that could have been prevented with a little forethought. Further research examining individual characteristics that predispose some people to more stressors than others would illuminate this issue, because current research is limited. Most studies have focused on neuroticism, and neuroticism failed to account for the genetic effects on stressor occurrence in the current study.

Factors responsible for nonshared environmental variance, also speculative, may include environmental influences that have been discussed in prior research, such as the busyness of the environment (Aspinwall & Taylor, 1997). Shared environmental variance, in contrast, did not predict stressor occurrence. Shared environmental effects are also not present in studies focused on life events (e.g., Plomin et al., 1990), suggesting that these effects are not present for exposure to negative events, either major events or minor daily occurrences, in adulthood.

### *Stressor Severity: Genetic and Environmental Variance*

The current study suggested, however, that shared environmental effects influence how people perceived the severity of these stressors. Shared environmental effects have been found in prior studies of beliefs (Koenig, McGue, Krueger, & Bouchard, 2005), and the current findings suggest that beliefs about how to respond to minor stressors in life are also imparted partially from familial environmental influences. Lazarus (1991) emphasized the importance of beliefs in appraising events in one's life. In addition, prior research of parental transmission has shown that the manner in which a mother expresses her emotions is related to her child's emotion regulation (Eisenberg et al., 2001). Family influences, then, may include skills that parents learn regarding emotion regulation and parenting that will then affect their children, a family transmission that intervention studies have illustrated (Buggental et al., 2002).

Although shared environmental effects were present, nonshared environmental variance accounted for the majority of the overall variance. Some of these unique environmental effects may stem from differences in the actual severity of what people encounter. Lazarus's (1991) appraisal theory recognizes that stressors vary in their severity, and this objective environmental threat plays a role in how people appraise negative situations. People's perceptions correspond to a moderate degree to more objective criteria (Almeida et al., 2002), so some of these ratings may stem from the idiosyncratic qualities of the immediate situation. Additional factors may be unique experiences in childhood and adulthood such as social support that may buffer the stressfulness of negative events (see the review by Cohen & Wills, 1985) and instill self-efficacy that may enhance coping responses (Sandler, Miller, Short, & Wolchik, 1989). Of course, nonshared environmental

influences include error variance in the model, so the actual influences are most likely less than they appear. Still, results indicate that nonshared environmental influences are most important for perceived stressor severity.

Genetic effects, in contrast, were minimal for variation in perceived severity. This finding was unexpected, given theoretical supposition that innate characteristics influence appraisals (Lazarus, 1991). One possible explanation may lie in the patterns observed in the models testing the moderating effects of age. The pattern of results suggested that genetic effects are present for younger adults, but these influences disappear among the older adults. Perhaps over time, people learn to appraise their world from a stance of experience as opposed to a position more aligned with innate, temperament-based characteristics. Researchers using larger samples in future studies will be able to examine this possible explanation.

### *The Role of Age*

The main effects of fewer stressors and lower perceived severity of these stressors with older age are consistent with prior findings (Almeida & Horn, 2004). Regarding stressor occurrence, older adults often have fewer restrictions on their time as a result of retirement, and freedom in structuring their day may enable them to avoid stressors more effectively than younger adults can (Hogas, Wilms, & Baltes, 1998). In addition, researchers have posited that older adults are better at avoiding situations that create negative distress (Carstensen, Gross, & Fung, 1997), consistent with experiencing fewer stressors with age. Regarding subjective severity, younger adults, particularly women, are more likely to see daily stressors as threatening to their self-image in the eyes of others (Almeida & Horn, 2004). In contrast, older adults are less interested in how other people—particularly strangers—see them. Instead, their definition of the self fluctuates less on a daily basis (Charles & Pasupathi, 2003) and is believed to be more resilient to external influences as a result of their lower degree of concern about the opinions of others.

We also examined the degree to which age moderated genetic and environmental effects on stressor occurrence and subjective severity. The null findings for stressor occurrence indicate that although we cannot examine whether the same genes and the same environmental influences are exerting effects of people across age groups, the proportions of genetic and environmental variance are similar across these groups. Findings are consistent with studies showing similar influences of genes and environment across age groups for other psychosocial phenomena such as personality (e.g., Read, Vogler, Pedersen, & Johansson, 2006) and suggest that both genes and environment are important even in later adulthood. In contrast, age moderated the degree of nonshared environmental influence on perceived severity. For older adults, the relative influence of environmental effects was much stronger than was the influence of these same effects for younger adults, consistent with Lazarus's (1991) argument that aging is accompanied by the accumulation of life experiences that lead to changes in appraisals.

### *Limitations and Future Directions*

The older adults in this sample represented people in their 60s and early 70s, with people who constitute the middle-old (75–85

years old) and oldest-old (85–95 years old) not represented. Greater variability for these young-old adults may be very different from variability observed in much older samples. For example, dementia increases in prevalence with age, and greater age-related variability in appraisals may arise as a result of the presence of cognitive deficits in this population. Thus, we interpret the greater variability in this sample as being a result of greater experience and adoption of different perspectives, but we recognize that different processes may also be related to variability in very old samples with potentially negative consequences for appraisals.

Studies in which relative genetic and environmental influences are examined require large samples, and statistical power is a limitation in this study. The unique daily diary design of this study, however, permitted us to examine daily processes not often captured in biometric research. In addition, each person's score represents the average response across 8 separate days, which allows for a greater degree of reliability for these responses than had we asked people about their stressors at a single time point. Another concern stems from the cross-sectional nature of these data. The study is guided by theoretical models of development, but longitudinal data are necessary to disentangle developmental processes from cohort and historical effects.

Researchers in future studies can examine factors responsible for the genetic and environmental components that account for stressor occurrence and perceived stressor severity. In addition, larger genetically informed studies with more power can detect if differences present in the research on major life events, such as gender, may also apply to research on the daily stress process, as well as how these patterns may or may not characterize age-related trends for people in their 80s and 90s. Finally, once researchers have examined potential genetic and environmental mechanisms responsible for stressors, they can examine interactions between these genetic and environmental components. For example, someone who is predisposed to be easily distracted and disorganized might do well in a more structured environment but may experience a greater number of stressors in more chaotic situations. These interactions were not examined in the current study but pose interesting possibilities for future research.

### *Conclusion*

Daily stressors represent events that are part of the rhythm of daily life: the minor irritations and frustrations that influence our daily well-being. Psychologists have focused predominantly on how people respond to stressors as opposed to individual differences that may predispose some people to experience more stressors than others. With the current study, we are the first to examine genetic influences in the occurrences of these minor stressors, and findings indicate that stressor occurrence is accounted for primarily by nonshared environmental influences, but genetic effects also play a nontrivial role. Variation in how people perceive these stressors, however, is primarily determined by environmental effects. Moreover, age moderated these environmental influences. Together, findings suggest that age is related to greater heterogeneity not in whether one experiences a stressor but in how these stressors are appraised.

## References

- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*, 317–332.
- Almeida, D. M., & Horn, M. C. (2004). Is daily life more stressful during middle adulthood? In C. D. Ryff & R. C. Kessler (Eds.), *A portrait of midlife in the United States* (pp. 425–451). Chicago: University of Chicago Press.
- Almeida, D. M., Wethington, E., & Kessler, R. C. (2002). The Daily Inventory of Stressful Experiences (DISE): An interview-based approach for measuring daily stressors. *Assessment*, *9*, 41–55.
- Aspinwall, L. G., & Taylor, S. E. (1997). A stitch in time: Self-regulation and proactive coping. *Psychological Bulletin*, *121*, 417–436.
- Baltes, P. B., & Baltes, M. M. (Eds.). (1990). *Successful aging: Perspectives from the behavioral sciences*. New York: Cambridge University Press.
- Bolger, N., & Schilling, E. A. (1991). Personality and the problems of everyday life: The role of neuroticism in exposure and reactivity to daily stressors. *Journal of Personality*, *59*, 355–386.
- Bolger, N., & Zuckerman, A. (1995). A framework for studying personality in the stress process. *Journal of Personality and Social Psychology*, *69*, 890–902.
- Bugental, D., Ellerson, P., Lin, E. K., Rainey, B., Kokotovic, A., & O'Hara, N. (2002). A cognitive approach to child abuse prevention. *Journal of Family Psychology*, *16*, 243–258.
- Carstensen, L. L., Gross, J., & Fung, H. (1997). The social context of emotion. In M. P. Lawton & K. W. Schaie (Eds.), *Annual review of geriatrics and gerontology* (pp. 325–352). New York: Springer.
- Carver, C. S., Scheier, M. F., & Pozo, C. (1992). Conceptualizing the process of coping with health problems. In H. S. Friedman (Ed.), *Hostility, coping, and health* (pp. 167–182). Washington, DC: American Psychological Association.
- Charles, S. T., & Pasupathi, M. (2003). Age-related patterns of variability in self-descriptions: Implications for everyday affective experience. *Psychology and Aging*, *18*, 524–536.
- Charles, S. T., Reynolds, C. A., & Gatz, M. (2001). Age-related differences and change in positive and negative affect over 23 years. *Journal of Personality and Social Psychology*, *80*, 136–151.
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, *98*, 310–357.
- Eisenberg, N., Gershoff, E. T., Fabes, R. A., Shepard, S. A., Cumberland, A. J., Losoya, S. H., et al. (2001). Mothers' emotional expressivity and children's behavior problems and social competence: Mediation through children's regulation. *Developmental Psychology*, *37*, 475–490.
- Epel, E. S., Blackburn, E. H., Lin, J., Dhabar, F., Adler, N., Morrow, J., & Cawthon, R. (2004). Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Sciences, USA*, *101*, 17312–17315.
- Epstein, S., & Katz, L. (1992). Coping ability, stress, productive load, and symptoms. *Journal of Personality and Social Psychology*, *62*, 813–825.
- Eysenck, H. J. (1998). Biological basis of personality. *Neuropsychiatry and Clinical Neuroscience*, *10*, 230–231. (Original work published 1963)
- Goldberg, L. R. (1992). Development of markers for the Big-Five factor structure. *Psychological Assessment*, *4*, 26–42.
- Gunther, K. C., Cohen, L. H., & Armeli, S. (1999). Role of neuroticism in daily stress and coping. *Journal of Personality and Social Psychology*, *77*, 1087–1100.
- Headley, B., & Wearing, A. (1989). Personality, life events, and subjective well-being: Toward a dynamic equilibrium model. *Journal of Personality and Social Psychology*, *57*, 731–739.
- Horgas, A. L., Wilms, H. U., & Baltes, M. M. (1998). Daily life in very old age: Everyday activities as expression of successful living. *The Gerontologist*, *38*, 556–568.
- Johnson, W., & Kruger, R. F. (2005). Higher perceived life control decreases genetic variance in physical health: Evidence from a national twin study. *Journal of Personality and Social Psychology*, *88*, 165–173.
- Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A., & Eaves, L. (1993). A twin study in recent life events and difficulties. *Archives of General Psychiatry*, *50*, 789–796.
- Kessler, R. C., Gilman, S. E., Thornton, L. M., & Kendler, K. S. (2004). Health, well-being, and social responsibility in the MIDUS twin and sibling subsamples. In O. G. Brim, C. D. Ryff, & R. C. Kessler (Eds.), *A portrait of midlife in the United States* (pp. 124–152). Chicago: University of Chicago Press.
- Koenig, L. B., McGue, M., Krueger, R. F., & Bouchard, T. J. (2005). Genetic and environmental influences on religiousness: Findings for retrospective and current religiousness ratings. *Journal of Personality*, *73*, 471–488.
- Lachman, M. E., & Weaver, S. (1998). The sense of control as a moderator of social class differences in health and well-being. *Journal of Personality and Social Psychology*, *74*, 763–773.
- Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lazarus, R. S. (1999). *Stress and emotion: A new synthesis*. New York: Springer.
- Lazarus, R. S., & DeLongis, A. (1983). Psychological stress and coping in aging. *American Psychologist*, *38*, 245–254.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.
- Mittelman, M. S., Roth, D. L., Haley, W. E., & Zarit, S. H. (2004). Effects of a caregiver intervention on negative caregiver appraisals of behavior problems in patients with Alzheimer's disease: Results of a randomized trial. *Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*, *59*, P27–P34.
- Mroczek, D. K., & Almeida, D. M. (2004). The effect of daily stress, personality, and age on daily negative affect. *Journal of Personality*, *72*, 355–378.
- Mroczek, D. K., & Kolarz, C. M. (1998). The effect of age on positive and negative affect: A developmental perspective on happiness. *Journal of Personality and Social Psychology*, *75*, 1333–1349.
- Neale, M. (1997). *Mx: Statistical modeling* (4th ed.). Richmond, VA: Medical College of Virginia, Department of Psychiatry.
- Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist*, *44*, 329–335.
- Pearlin, L. I., Lieberman, M. A., Menaghan, E. G., & Mullan, J. T. (1981). The stress process. *Journal of Health and Social Behavior*, *22*, 337–356.
- Pearlin, L. I., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behavior*, *19*, 2–21.
- Phifer, J. F. (1990). Psychological distress and somatic symptoms after natural disaster: Differential vulnerability among older adults. *Psychology and Aging*, *5*, 412–420.
- Plomin, R., DeFries, J. C., & Loehlin, J. C. (1977). Genotype–environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*, *84*, 309–322.
- Plomin, R., DeFries, J. C., McClearn, G. E., & Rutter, M. (1997). *Behavioral genetics* (3rd ed.). New York: Freeman.
- Plomin, R., Lichtenstein, P., Pedersen, N. L., McClearn, G. E., & Nesselrode, J. R. (1990). Genetic influence on life events during the last half of the life span. *Psychology and Aging*, *5*, 25–30.
- Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research*, *5*, 554–571.
- Read, S., Vogler, G., Pedersen, P., & Johansson, B. (2006). Stability and change in genetic and environmental components of personality in old age. *Personality and Individual Differences*, *40*, 1637–1647.
- Rowe, J. W., & Kahn, R. L. (1987, July 10). Human aging: Usual and successful. *Science*, *237*, 143–149.

- Rowe, J. W., & Kahn, R. L. (1997). Successful aging. *The Gerontologist*, 37, 433–440.
- Rowlison, R. T., & Felner, R. D. (1988). Major life events, hassles, and adaptation in adolescence: Confounding in the conceptualization and measurement of life stress revisited. *Journal of Personality and Social Psychology*, 55, 432–444.
- Sandler, I. N., Miller, P. A., Short, J., & Wolchik, S. A. (1989). Social support as a protective factor for children in stress. In D. Belle (Ed.), *Children's social networks and social supports* (pp. 277–307). Oxford, England: Wiley.
- Saudino, K. J., Pedersen, N. L., Lichtenstein, P., McClearn, G. E., & Plomin, R. (1997). Can personality explain genetic influences on life events? *Journal of Personality and Social Psychology*, 72, 196–206.
- Serido, J., Almeida, D. M., & Wethington, E. (2004). Chronic stressors and daily hassles: Unique and interactive relationships with psychological distress. *Journal of Health and Social Behavior*, 17, 17–33.
- Suls, J., & Martin, R. (2005). The daily life of the garden-variety neurotic: Reactivity, stressor exposure, mood spillover, and maladaptive coping. *Journal of Personality*, 73, 1–25.
- 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*, 21, 231–239.
- Wagner, B. M., Compas, B. E., & Howell, D. C. (1988). Daily and major life events: A test of an integrative model of psychosocial stress. *American Journal of Community Psychology*, 61, 189–205.
- Zautra, A. J. (2003). *Emotions, stress and health*. New York: Oxford University Press.
- Zautra, A. J., Finch, J. F., Reich, J. W., & Guarnaccia, C. A. (1991). Predicting the everyday life events of older adults. *Journal of Personality*, 59, 507–538.
- Zautra, A. J., Guarnaccia, C. A., & Dohrenwend, B. P. (1986). Measuring small life events. *American Journal of Community Psychology*, 14, 629–655.

Received March 15, 2006

Revision received January 16, 2007

Accepted January 17, 2007 ■