Age, Daily Stress Processes, and Allostatic Load: A Longitudinal Study

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Abstract
Objective: The present study examined age differences in the association between daily stressors and allostatic load. Method: Participants consisted of 317 adults (34-84 years) who participated in Waves 1 (1996-1997) and 2 (between 2005 and 2009) of the Midlife Development in the United States Survey. During Wave 1, participants reported the stressors they encountered across eight consecutive days. Within-person affective reactivity slopes indexing change in negative affect from a nonstressor day to a stressor day were calculated for each participant. Affective reactivity and stressor exposure scores at Wave 1 were used to predict allostatic load at Wave 2. Results: Heightened levels of affective reactivity at Wave 1 predicted elevated levels of allostatic load at Wave 2 but only among older adults who also reported high levels of stressor exposure. No significant associations emerged for younger adults. Discussion: Daily stress processes may be one pathway through which age-related physical health declines occur.

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Aging is inevitable, but there is immense variability in this process. With unprecedented growth of the 65 and older population over the next few decades (United States Census Bureau, 2014), it is imperative to examine factors that predict improved health and well-being in later adulthood. One factor that has been linked with adverse health outcomes across the life-span is psychosocial stress (DeLongis, Folkman, & Lazarus, 1988; Rohleder, 2014). Research on this topic has largely focused on the health consequences of chronic stressors (e.g., caregiving) and major life events (e.g., divorce; Adler & Snibbe, 2003; Kendall-Tackett, 2009). Adding to this important work is research indicating that daily stressors are also associated with adverse health outcomes (Almeida, McGonagle, & King, 2009).

Daily stressors, such as a lost cellphone or a missed deadline, are minor, but continual adaptation to the perturbations of daily life could result in cumulative biological dysregulation, referred to as allostatic load (Almeida, 2005). The relationship between daily stressors and allostatic load may be even more pronounced with increasing age. According to the model of Strength and Vulnerability Integration (SAVI), age-related decreases in physiological resiliency make it difficult for older adults to recover from stressful events. This is in contrast with younger adults, whose more resilient systems are hypothesized to recover relatively quickly from the physiological effects of stressors (Charles, 2010; Charles & Piazza, 2009). Using SAVI as a framework, the present study examines whether daily stressors, in conjunction with age, prospectively predict allostatic load.

Daily Stressors

Daily stressors refer to the anticipated (e.g., a long daily commute) and unanticipated challenges (e.g., a flat tire during the commute) encountered in daily life. Although minor, daily stressors are not without consequence. On days they occur, they are associated with worse self-reported health (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; Zarski, 1984), more physical health symptoms (Gil et al., 2004; Zohar, 1999), and an exacerbation of physical health conditions (Cathcart & Pritchard, 2008; Sarid et al., 2017; Twisk, Snel, Kemper, & van Mechelen, 1999). For example, external stress from daily hassles is associated with worse physical well-being for both partners in a marital dyad (Falconier, Nussbeck, Bodenmann, Schneider, & Bradbury, 2015), and self-rated health is worse on days work-related stressors occur (Dahlgren, Kecklund, Theorell, & Akerstedt, 2009; Repetti, 1993).
Studies have also begun to link daily stressors with long-term physical health outcomes. In a study of individuals with rheumatoid arthritis, for example, daily stressors predicted more fatigue during the ensuing month, and worrying predicted increased symptomology and self-reported disease activity (Evers et al., 2014). In another study, metabolic syndrome was higher among people who, two years earlier, had reported intense negative social interactions (Ross, Martin, Chen, & Miller, 2011). Daily stressors have even been predictive of mortality, with high levels of daily hassle intensity predicting mortality among men between 53 and 85 years (Jeong, Aldwin, Igarashi, & Spiro, 2016), and greater affective reactivity to daily stressors (operationalized as greater decreases in positive affect on stressor days) predicting mortality among men between 58 and 88 years (Mroczek et al., 2013).

Different causal mechanisms could explain the link between daily stressors and physical health. Some research, for example, indicates that health behaviors may mediate the link between daily stressors and adverse health outcomes (e.g., O’Connor, Jones, Conner, McMillan, & Ferguson, 2008). For example, on days stressors occur, people are more likely to engage in worse health behaviors, such as smoking (Stubbs et al., 2017) and unhealthy eating (O’Connor, Armitage, & Ferguson, 2014), both of which could negatively affect health over time. Other research suggests that daily stressors directly elicit changes in physiological biomarkers that could ultimately result in biological dysregulation (for review, see Piazza, Almeida, Dmitrieva, & Klein, 2010). For example, on days they occur, daily stressors are associated with elevated levels of diurnal cortisol (Stawski, Cichy, Piazza, & Almeida, 2013), catecholamines, and prolactin (Chandola, Heraclides, & Kumari, 2010), as well as interleukin-6 (IL-6) and C-reactive protein (Gouin, Glaser, Malarkey, Beversdorf, & Kiecolt-Glaser, 2012; Sin, Graham-Engeland, Ong, & Almeida, 2015). They are also associated with elevated blood pressure and changes in heart rate variability (Vrijkotte, Van Doornen, & De Geus, 2000). Over time, these physiological changes are hypothesized to lead to increased allostatic load (Almeida, 2005).

**Stressor Exposure Versus Affective Reactivity**

The strength of the association between daily stressors and health outcomes may be dependent on two closely linked yet separate processes: stressor exposure and affective reactivity. Stressor exposure refers to whether a stressor is encountered, whereas affective reactivity refers to the strength of a person’s affective (i.e., emotional) response to the stressor experienced (Charles, Piazza, Mogle, Sliwinski, & Almeida, 2013). Cumulative stressor exposure is a well-known risk factor for adverse health outcomes (McEwen & Stellar,
1993), and a growing body of research indicates that affective reactivity may also have health-related consequences (e.g., Piazza, Charles, Sliwinski, Mogle, & Almeida, 2013; Sin et al., 2015). Among people exposed to a laboratory stressor, for example, those who showed greater reactivity and poorer recovery were more likely to have adverse cardiovascular outcomes over time (Chida & Steptoe, 2010). Similarly, heightened reactivity to daily stressors has been associated with elevated inflammatory biomarkers (Sin et al., 2015). Moreover, whereas affective reactivity was predictive of future health conditions in one study, stressor exposure was not (Piazza, Charles, Sliwinski, et al., 2013). Together, this work suggests that highly reactive individuals may eventually show greater allostatic load than their less reactive counterparts.

Allostasis and Allostatic Load

The concepts of allostasis and allostatic load are closely linked with homeostasis. To achieve homeostasis, the body must actively adapt to changing stimuli. For example, an infection necessitates a fever, low body temperature results in shivering, and the threat of bodily harm elicits the fight-or-flight response. The process by which the body adjusts to meet external demands and subsequently suppresses these responses to return to baseline is termed *allostasis* (McEwen, 2001; McEwen & Seeman, 1999). Allostasis is vital for survival; however, continual adaptation to internal and external milieu may eventually cause biological systems to wear down. This wearing down, referred to as allostatic load (McEwen, 1998), is associated with worse cognitive and physical functioning over time, and increased mortality risk (Seeman, McEwen, Rowe, & Singer, 2001). Most of the research examining predictors of allostatic load has focused on chronic stressors, such as caregiving (Roepke et al., 2011), job stress (Sun, Wang, Zhang, & Li, 2007), and lack of social support (Brooks, Andrade, Middleton, & Wallen, 2014; Horan & Widom, 2015). Unlike daily stressors, which are the “irritating, frustrating, distressing demands” that characterize daily hassles within the environment (Kanner, Coyne, Schaefer, & Lazarus, 1981, p. 3), chronic stressors are enduring, tend to persist over time (Pearlin & Skaff, 1996), and may alter or impede normal social functioning (Sandi & Haller, 2015). Although chronic stressors are more persistent than daily stressors, both forms of stress require similar physiological adaptations that could result in increased allostatic load. A large body of research has demonstrated a link between chronic stress and allostatic load, but few studies to date have explicitly examined the relationship between daily stressors and allostatic load. Thus, although theoretically plausible, it remains unclear if daily stressors are predictive of allostatic load.
The Role of Age

The association between daily stressors and allostatic load may vary not only according to amount of stressor exposure and degree of affective reactivity but also by age. Research over the last few decades reveals a somewhat paradoxical finding—Despite multiple losses associated with age, older adults report stable or even improved affective well-being compared with their younger counterparts (Reed, Chan, & Mikels, 2014). These gains are due to shifting motivations (Carstensen, Isaacowitz, & Charles, 1999), and greater expertise in the ability to employ emotion regulation strategies, acquired from a lifetime of experiences (Charles & Piazza, 2009). There are, however, several vulnerabilities associated with aging, most notably decreases in physiological resiliency. The strengths and vulnerabilities of aging work in tandem to influence mental and physical health outcomes, according to the model of SAVI (Charles, 2010). SAVI posits that older adults’ increased ability to regulate their emotions is adaptive, because when they experience affective distress, they are less able than younger adults to modulate their physiological response. Research examining this tenet of SAVI indicates that older adults are, indeed, less able to physically recover from the negative affect (NA) they experience across the day (Piazza, Charles, Stawski, & Almeida, 2013). They also show delayed blood pressure recovery in comparison with their younger counterparts when asked to ruminate over a laboratory stressor (Robinette & Charles, 2014). If, as SAVI hypothesizes and research attests, older adults do in fact experience greater health-related ramifications from stressful events, then age differences in physiological outcomes should be magnified when stressor exposure is frequent and reactivity to these stressors is high.

The Current Study

The goal of the present study is to examine whether daily stressor exposure and affective reactivity prospectively predict allostatic load, and, if so, whether this association varies by age. Based on SAVI, we predict that age-related increases in allostatic load at Wave 2 will be most pronounced among people who reported frequent stressor exposure and heightened levels of affective reactivity at Wave 1.

Method

Participants and Procedures

Data for the present study comes from the Midlife Development in the United States (MIDUS) Survey, which is a study designed to understand factors that
influence health and well-being across adulthood. The MIDUS consists of a main project and various subprojects across three waves of data. Initial data collection (Wave 1) occurred in the mid-1990s, with four distinct samples: a national sample recruited through random digit dial (RDD; \( n = 3487 \)); siblings of the national RDD sample (\( n = 950 \)); twins, recruited from the Twin Screener Project (\( n = 1914 \)); and participants from oversampled metropolitan areas (\( n = 757 \)), for a total of 7,108 participants. A subset of participants from the RDD (\( n = 1031 \)) and twin sample (\( n = 469 \)) also completed the National Study of Daily Experiences (NSDE). Participants from the sibling sample and the oversampled metropolitan areas did not complete NSDE. The NSDE consisted of eight daily telephone interviews, designed to gain understanding of participants’ daily lives. During each interview, participants were asked a series of questions about their day, such as how they spent their time, the emotions they felt, the physical symptoms they experienced, and the stressors they encountered. At Wave 1, participants ranged from 25 to 74 years of age.

Wave 2 of MIDUS was collected approximately 10 years after initial data collection, when participants were between 34 and 84 years old. Of the original 7,108 participants, 4,693 completed Wave 2. Of these, 1,054 completed the Biomarker project. For the Biomarker project, participants were flown to one of three General Clinical Research Centers (University of California, Los Angeles [UCLA]; University of Wisconsin; or Georgetown University), where they underwent extensive physical exams. During the two days of testing (beginning in mid-afternoon on Day 1 and ending at noon on Day 2), participants’ medical history was obtained, and blood, saliva, and urine samples collected. All samples and anthropometric data were collected and processed according to standardized instructions (for details, see Love, Seeman, Weinstein, & Ryff, 2010). In total, 365 participants completed the Biomarker project at Wave 2 and the NSDE project at Wave 1.

**Measures**

*Daily stressors.* Daily stressors were assessed using the Daily Inventory of Stressful Experiences, an interview protocol consisting of seven stem questions about the stressors participants may have encountered during the previous 24 hours (Almeida, Wethington, & Kessler, 2002). Specifically, participants were asked whether they experienced any arguments, non-argument tensions, work- or school-related stressors, home-related stressors, discrimination-related stressors, network events, and any other stressor not mentioned. For each day, participants were assigned a 1 if they reported experiencing a stressor and a 0 if they did not. Of the 365 participants, six were missing daily stressor data, bringing the final sample to 359. Participants
reported experiencing stressors on 44% of days, which is slightly higher than the 40% reported by the full NSDE sample (Almeida, 2005).

**Daily emotional experience.** The Non-Specific Psychological Distress Scale was used to assess daily emotional experience. On each interview day, participants reported on a 5-point scale, anchored at 1 (*none of the time*) and 5 (*all of the time*), how often they had experienced six emotions or emotion descriptors: restless or fidgety, worthless, hopeless, nervous, so sad that nothing could cheer you up, and that everything was an effort. The mean of these emotions was computed, resulting in a daily NA score, from which affective reactivity scores were computed.

**Operationalization of affective reactivity.** Affective reactivity, in daily diary studies of stress and affect, is typically defined as the time-varying (within-person) slope between stress and affect (Mroczek et al., 2013). To calculate an affective reactivity slope for each participant, we estimated ordinary least squares (OLS) regression models for each individual, where their affect score was regressed on their daily stress. Daily stress was a dichotomous variable, indicating whether the participants’ day had any stressors or was stressor free. The resulting slope, which was retained for subsequent analyses, reflects the difference in affect associated with the reported experience of any daily stressors, and serves as the operant index of affective reactivity. Slopes were set to missing for individuals who reported stressors on either none (0%) or all (100%) of the days (n = 37), as a valid affective reactivity score could not be calculated. In addition, the intercept for these models, reflecting the participant’s level of NA on nonstressor days, was retained for use as a covariate in subsequent analyses, to ensure that any effects reflected affective reactivity and not NA, per se. We opted for retaining slopes from OLS regression, as opposed to multilevel models using full-information maximum likelihood as individual slope estimates to avoid bias in estimates due to shrinkage (Snijders & Bosker, 2012).

**Physiological biomarkers.** All biomarkers were collected during an overnight (5:00 p.m. to 11:00 a.m.) visit to one of three General Clinical Research Centers (for details regarding biomarker measurement, including collection protocols and assay procedures, see Gruenewald et al., 2012). Biomarkers were collected from seven physiological systems. *Indicators of cardiovascular functioning* included systolic blood pressure, diastolic blood pressure, and resting pulse. *Indicators of sympathetic nervous system (SNS) functioning* included overnight measures of urinary epinephrine and norepinephrine. *Indicators of the parasympathetic nervous system (PNS)* included markers of heart rate
variability, including both low and high spectral power, standard deviation of R-R (heartbeat to heartbeat) intervals, and the root mean square of successive differences. Indicators of hypothalamic pituitary adrenal (HPA) axis activity included overnight urinary cortisol and serum dehydroepiandrosterone sulfate (DHEA-S). Indicators of inflammation included C-reactive protein, fibrinogen, IL-6, e-Selectin, and intracellular adhesion molecule-1 (ICAM-1). Indicators of lipid and general metabolic activity included waist–hip ratio, body mass index (BMI), triglycerides, high density lipoprotein cholesterol, and low density lipoprotein cholesterol. Indicators of glucose metabolism included fasting glucose, glycosylated hemoglobin, and insulin resistance.

_Coventional load scores_. For each of the seven systems, risk indices were computed by calculating the proportion of biomarkers—ranging from two to six per system—for which participants scored in the high-risk quartile ranges (upper or lower quartiles depending on whether low or high values confer a greater risk for health). System risk scores ranged from 0 to 1 and were then added together to result in an allostatic load score that ranged from a minimum of 0 to a maximum of 7. This methodology, which has been successfully used in previous research, has the benefit of ensuring that all systems (regardless of number of biomarkers assessed) are equally represented in the final allostatic load score (Gruenewald et al., 2012). Allostatic load scores were computed on individuals who had information on at least six systems. Of the 1,054 individuals who completed the Biomarker project, nine were missing allostatic load data, bringing the total sample to 1,043. Five of these individuals were in the current analysis, dropping the final study sample from 322 to 317. Scores on allostatic load in the study sample ranged from 0 to 4.88, with a mean of 1.63 ($SD = 1.04$).

_Participant characteristics_. Table 1 provides sample characteristics for the Wave 2 Biomarker project ($n = 1,043$) and the subsample that comprises the present study ($n = 317$). No significant differences were detected on key demographic variables between the two samples. Participants in the present study ranged in age from 34 to 84 years ($M_{age} = 55.7$), were primarily White (93.4%), and were well-educated, with 45.7% having attained a bachelor’s degree or higher. Most participants (73.7%) reported having at least one chronic health condition during the previous year, and 42.3% reported that they had ever smoked cigarettes regularly in their life.

_Covariates_

Covariates, the data for which was collected at Wave 2, included sex (coded male or female), race (coded White or other, due to the limited number of
ethnic minorities in the sample), education (coded as less than a high school education, a high school diploma or general educational development [GED], some college, or a bachelor’s degree or higher), history of smoking (coded as ever smoked or nonsmoker), BMI (measured continuously, winsorized at the 99th percentile, and mean-centered), prescription medication use (coded yes or no), over-the-counter medication use (coded yes or no), and presence of chronic health conditions (which included 23 self-reported physical health conditions, coded yes or no, that were subsumed into 19 categories: lung conditions, consisting of asthma, bronchitis, emphysema, other lung conditions, tuberculosis; bone-related conditions, including arthritis and backaches; digestive conditions, consisting of recurring stomach trouble, indigestion, or diarrhea; HIV/AIDS; autoimmune disorders; high blood pressure; diabetes; neurological problems; history of heart trouble; stroke; trouble with mouth, teeth, or gums; history of cancer; thyroid disease; hay fever; bladder-related conditions; gall bladder problems; migraines; hernia; anxiety, depression, or other emotional disorder). We also included as a covariate Wave 1 chronic health conditions for a general baseline measure of physical health. Finally, we included a comprehensive measure of cumulative stress burden from Wave 2 that assessed participants’ history of stress across several domains of life (Slopen et al., 2012; Slopen et al., 2013). These domains included early life stress, stressful life experiences, problems with the family during the past year, current financial stress, discrimination, neighborhood stress, relationship stress, perceived inequality, work–family spillover, physical work stress, and psychological work stress. Each domain was standardized and summed for a

### Table 1. Demographic Characteristics of the Biomarker Sample and the Study Sample.

<table>
<thead>
<tr>
<th>Demographic characteristics</th>
<th>Biomarker sample</th>
<th>Study sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(N = 1,043)</td>
<td>(n = 317)</td>
</tr>
<tr>
<td>Age (SD)</td>
<td>55.2 (11.8)</td>
<td>55.7 (12.23)</td>
</tr>
<tr>
<td>Sex (% female)</td>
<td>54.6%</td>
<td>56.5%</td>
</tr>
<tr>
<td>Race (% White)</td>
<td>92.8%</td>
<td>93.4%</td>
</tr>
<tr>
<td>Education (% with bachelor’s degree or higher)</td>
<td>46.7%</td>
<td>45.7%</td>
</tr>
<tr>
<td>Marital status (% married)</td>
<td>72.3%</td>
<td>74.5%</td>
</tr>
<tr>
<td>Current work status (% yes)</td>
<td>54.5%</td>
<td>54.9%</td>
</tr>
<tr>
<td>Chronic conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 conditions</td>
<td>29.0%</td>
<td>26.3%</td>
</tr>
<tr>
<td>1 condition</td>
<td>29.7%</td>
<td>27.9%</td>
</tr>
<tr>
<td>2 or more conditions</td>
<td>41.3%</td>
<td>45.8%</td>
</tr>
</tbody>
</table>
total chronic stress score. Our goal in including a measure of chronic stress was to examine whether daily stressors predicted allostatic load above and beyond that predicted by chronic stress exposure. Because this comprehensive assessment includes questions regarding work, children, and marital partners, we also included statistical adjustment for whether a person currently works, has a child, and has a marital partner, all coded as yes/no.

Results

Prior to hypothesis testing, basic descriptive statistics and graphics for all variables were examined for outliers, out of range/influential values, and distributional properties. Outliers were winsorized at approximately the 99th percentile to minimize the influence of extreme values. Square-root or logarithmic transformations were then employed to normalize skewed variables. Analyses were conducted using both transformed and nontransformed data. The pattern of results was identical when using transformed and nontransformed data; thus, for ease of interpretation, we present our findings in their original, nontransformed metric.

To account for dependency in the data (due to the inclusion of twins), data were analyzed using general estimating equations (GEE) in Proc Gen Mod (Liang & Zeger, 1986). Predictor variables included age, stressor exposure, affective reactivity, and their interactions. Covariates, assessed at Wave 2, included sex, race, education, BMI, history of smoking, current medication use, chronic health conditions, and history of cumulative stress burden. We also included as a covariate Time 1 chronic health conditions. We ran two models predicting allostatic load: the first including only covariates and the second including age, daily stressor variables, and the two- and three-way interactions testing our hypothesis (see Table 2).

Test of the Hypothesis

Results of the final model revealed a significant main effect of age \((p < .001)\) but not stressor exposure \((p = .667)\) or affective reactivity \((p = .790)\). These main effects, however, were qualified by a significant three-way interaction between stressor exposure, affective reactivity, and age \((p = .022)\). To decompose this interaction, we plotted the predicted allostatic load scores at ±1 standard deviation \((SD)\) for stressor exposure, affective reactivity, and age (see Figure 1). At younger ages \((-1 SD)\), at low levels of exposure \((-1 SD)\), allostatic load scores were similar, regardless of levels of affective reactivity. This was confirmed by a test of the simple slope for reactivity at −1 SD age and −1 SD exposure \((.036, SE = 0.532, 95\% CI [−1.007, 1.079], p = .95)\).
Similarly, at younger ages (−1 SD), at higher levels of exposure (+1 SD), no statistically significant differences emerged as a function of affective reactivity (−.662, *SE* = 0.367, 95% CI [−1.381, −0.057], *p* = .071).

**Table 2. Age and Daily Stress Processes as Predictors of Allostatic Load (n = 317).**

<table>
<thead>
<tr>
<th></th>
<th>Model 1 Covariates only</th>
<th>Model 2 Final model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unstandardized coefficients <em>b</em> (<em>SE</em>)</td>
<td>Unstandardized coefficients <em>b</em> (<em>SE</em>)</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.984 (0.251)***</td>
<td>1.431 (0.233)***</td>
</tr>
<tr>
<td>Sex (ref = Women)</td>
<td>−0.111 (0.118)</td>
<td>−0.230 (0.099)*</td>
</tr>
<tr>
<td>Race (ref = White)</td>
<td>−0.318 (0.207)</td>
<td>−0.169 (0.202)</td>
</tr>
<tr>
<td>Education (ref = bachelor’s degree)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than HS degree</td>
<td>1.294 (0.427)**</td>
<td>1.519 (0.389)***</td>
</tr>
<tr>
<td>HS diploma/GED</td>
<td>0.117 (0.141)</td>
<td>0.040 (0.130)</td>
</tr>
<tr>
<td>Associate’s degree/some college</td>
<td>0.169 (0.141)</td>
<td>0.191 (0.127)</td>
</tr>
<tr>
<td>W2 chronic conditions</td>
<td>0.051 (0.048)</td>
<td>−0.002 (0.042)</td>
</tr>
<tr>
<td>W2 smoking (ref = never smoked)</td>
<td>0.025 (0.110)</td>
<td>0.006 (0.101)</td>
</tr>
<tr>
<td>W2 body mass index</td>
<td>0.057 (0.011)***</td>
<td>0.067 (0.010)***</td>
</tr>
<tr>
<td>W2 prescription medication use (ref = none)</td>
<td>0.386 (0.128)**</td>
<td>0.227 (0.113)*</td>
</tr>
<tr>
<td>W2 over-the-counter medication use (ref = none)</td>
<td>0.052 (0.150)</td>
<td>−0.122 (0.134)</td>
</tr>
<tr>
<td>W1 chronic conditions</td>
<td>0.061 (0.048)</td>
<td>0.055 (0.041)</td>
</tr>
<tr>
<td>W2 cumulative lifetime stress</td>
<td>−0.026 (0.060)</td>
<td>−0.014 (0.054)</td>
</tr>
<tr>
<td>W2 work status (ref = employed)</td>
<td>−0.018 (0.129)</td>
<td>−0.027 (0.115)</td>
</tr>
<tr>
<td>W2 has child (ref = has children)</td>
<td>0.089 (0.170)</td>
<td>0.068 (0.165)</td>
</tr>
<tr>
<td>W2 marital partner (ref = marriage or marriage-like setting)</td>
<td>0.083 (0.133)</td>
<td>0.085 (0.116)</td>
</tr>
<tr>
<td>NA on nonstressor day</td>
<td></td>
<td>−0.212 (0.329)</td>
</tr>
<tr>
<td>Age</td>
<td>0.035 (0.004)***</td>
<td></td>
</tr>
<tr>
<td>DSE</td>
<td>0.115 (0.267)</td>
<td></td>
</tr>
<tr>
<td>AR</td>
<td>0.065 (0.244)</td>
<td></td>
</tr>
<tr>
<td>DSE × Age</td>
<td>0.009 (0.020)</td>
<td></td>
</tr>
<tr>
<td>AR × Age</td>
<td>0.031 (0.019)</td>
<td></td>
</tr>
<tr>
<td>DSE × AR</td>
<td>1.709 (1.242)</td>
<td></td>
</tr>
<tr>
<td>DSE × AR × Age</td>
<td>0.274 (0.119)*</td>
<td></td>
</tr>
</tbody>
</table>

*Note. W2 = Wave 2; W1 = Wave 1; NA = negative affect; DSE = stressor exposure; AR = affective reactivity.

* *p < .05. ** *p < .01. *** *p < .001.
A different pattern, however, emerged for older adults, whose levels of allostatic load varied by degree of stressor exposure and affective reactivity. For older adults reporting lower levels of exposure (−1 SD), allostatic load scores did not differ as a function of affective reactivity, as indicated by a nonsignificant simple slope (−0.624, SE = 0.631, 95% CI [−1.862, 0.614], p = .323). For older adults reporting higher levels of exposure (+1 SD), however, those exhibiting higher reactivity (+1 SD) exhibited significantly higher allostatic load, as evidenced by a significant simple slope for affective reactivity (1.51, SE = 0.538, 95% CI [0.455, 2.564], p = .005). Thus, higher levels of affective reactivity combine with higher levels of stressor exposure to confer preferentially increased levels of allostatic load among older adults. Specifically, for older adults with high stressor exposure, a 1 SD increase in affective reactivity was associated with a .28 increase in allostatic load.

**Adjusting for Time 2 Stressor Variables**

To ensure that our models reflected longitudinal associations between daily stressors and allostatic load, and were not just a proxy for current day stressor...
exposure and reactivity, we ran a separate model that included Wave 2 affective reactivity and stressor exposure. Analyses were identical as above, except for the addition of the Wave 2 stressor variables. Results indicated that Wave 2 affective reactivity ($b = .323, p = .202$) and stressor exposure ($b = - .417, p = .107$) were not significantly associated with allostatic load, and that the interaction between age, stressor exposure, and affective reactivity (both assessed at Wave 1) remained significant ($b = .312, p = .013$).

**Discussion**

For years, scientists have identified multiple factors related to improved health and well-being in later life. Their discoveries have led to increased awareness of health risks, and an understanding of how people can be effective agents in helping to determine how well they age. The present study adds to this literature by revealing that daily stressors—the minor hassles in life—have long-term implications for health outcomes in later adulthood. This study builds on a growing body of research indicating that the minor hassles of life may aggregate over time and eventually result in mental and physical health consequences (e.g., Charles et al., 2013; Chiang, Turiano, Mroczek, & Miller, 2018).

**The Effect of Cumulative Assaults**

Across previous studies, the association between daily stress processes and health outcomes has been largely age-invariant (e.g., Charles et al., 2013; Sin et al., 2015). Previous research has also demonstrated that affective reactivity, and not stressor exposure, per se, is related to adverse health outcomes (e.g., Piazza, Charles, Sliwinski, et al., 2013). In contrast to previous work, the results of the present study indicate that in the context of daily stress processes, it is the combination of multiple assaults—stressor exposure, affective reactivity, and age—that is most predictive allostatic load. Specifically, we found that although increased affective reactivity was associated with elevated allostatic load 10 years after initial assessment, it was only in the context of heightened stressor exposure and older age, a finding that differs from previous research. There are a few potential explanations for these disparate findings.

First, previous studies that examined the link between affective reactivity and subsequent health outcomes largely focused on self-reported mood disorders and chronic health conditions (e.g., Cohen, Gunthert, Butler, O’Neil, & Tolpin, 2005; O’Neil, Cohen, Tolpin, & Gunthert, 2004; Piazza, Charles, Sliwinski, et al., 2013). In contrast, the present study focused on allostatic load, which is a cumulative measure of biological dysregulation. Although
self-reported health and allostatic load are related (Brown, Turner, & Moore, 2016), they are fundamentally different constructs; thus, the factors that predict them may differ. For example, self-reported chronic conditions and health status are largely based on an individual’s knowledge or perception of their physical constitution. Thus, when heightened affective reactivity is experienced, there could be perceptible increases in physical health symptoms, which could explain why the route from affective reactivity to self-reported physical health is direct. In contrast, allostatic load indicators are not necessarily perceptible and comprise multiple biomarkers, many of which may not begin to show significant wear until multiple risk factors are present. Thus, the pathway from daily stress processes to allostatic load may be more indirect and require the presence of additional risk factors.

Another difference between the present study and previous studies that have examined biomarker risk in conjunction with daily stressors is that previous studies examined data collected concurrently, rather than longitudinally, and from one physiological system, as opposed to multiple systems (e.g., Sin et al., 2015). Perhaps several factors, including increased stressor exposure and heightened affective reactivity, are necessary to evoke changes in the physiological systems that allostatic load comprises. In this way, our findings do not diminish the importance of affective reactivity but rather illustrate how it may work in conjunction with other variables when predicting cumulative biological dysregulation. Supporting this hypothesis is a recent study that examined the association between affective reactivity to daily stressors and mortality (Chiang et al., 2018). In this study, heightened affective reactivity to daily stressors predicted mortality across a period of 20 years, but only among those individuals who reported having at least one chronic illness. Thus, it is possible that for longitudinal health outcomes with more severe implications, affective reactivity is most predictive when in the presence of another existing risk factor.

**SAVI: A Framework for Understanding the Current Findings**

Our findings are also consistent with SAVI, which predicts that age-related gains in affective well-being seen in previous research diminish when older adults are faced with loss of social belonging; encounter chronic, uncontrollable stressors; or experience neurological dysregulation (Charles, 2010). In the present study, heightened affective reactivity was particularly pernicious in the face of frequent stressor exposure, which may have been reflective of older adults’ diminished affective experience when they experienced frequent stressors. SAVI also posits that when older adults encounter a stressor and are unable to disengage from it, they have greater difficulty modulating their
physiological responses, which could have health-related consequences. This is an important point from a developmental perspective, and is one reflected in the present study. Previous studies have demonstrated a positive association between age and allostatic load (Crimmins, Johnston, Hayward, & Seeman, 2006), a finding that was replicated in the present study. Differences in stressor exposure and affective reactivity, however, affected this relationship. Determining the conditions under which some older adults report higher stressor exposure and greater affective reactivity—such as those posited by SAVI—is, thus, an important point for future research.

**Do Daily Stressors Matter for Younger Adults?**

In the present study, stressor exposure and affective reactivity did not predict allostatic load levels in the younger age group. This does not mean, however, that daily stressors are completely innocuous for people in their 20s and 30s. As previous research indicates, daily stressors, regardless of age, are associated with worse self-reported mental and physical health conditions (Cathcart & Pritchard, 2008; DeLongis et al., 1982; Sarid et al., 2017; Zarski, 1984) and short-term changes in physiological biomarkers (Gouin et al., 2012; Sin et al., 2015). Unlike other conceptualizations of health, however, allostatic load is slow to develop (McEwen, 1998), and it may take years for psychological processes to exert an effect on the systems it comprises. The “younger” adults in our sample were approximately 42 years of age when their allostatic load was measured and approximately 32 when daily stressors were assessed. Due to physiological resiliency earlier in life, the 10-year period between the ages of 32 and 42 may simply not be long enough for psychological stress processes to exert influence on the systems that comprise allostatic load. In contrast, the “older” adults in the sample were approximately 57 years of age when their daily stressors were assessed and approximately 67 years of age when their allostatic load was assessed. Physiological resiliency is likely lower during the 10-year period between 57 and 67 and may, thus, be more prone to damage from repeated psychosocial assaults.

It is also important to consider one of the design limitations of the present study when interpreting our results. Daily stress processes and allostatic load were examined across a period of approximately 10 years. Thus, it is important to acknowledge that the present study provided just a glimpse into processes that had been in the works for many years and that will be in the works for many more years. Although daily stress processes do not yet appear to have affected allostatic levels for the younger adults in our sample, additional years of stressor exposure and affective reactivity may alter that trajectory. This is an important question to examine but one that requires more than 10 years of longitudinal data to examine.
Limitations

Although the present study adds to the literature on stress, health, and aging, there are several limitations that should be noted. First, we cannot generalize our results, due to the homogenous nature of the sample. Participants in the present study were highly educated and primarily White. Moreover, the Biomarker project only included those individuals well enough to travel to a clinical research center. Thus, our results may not generalize to a less healthy, more diverse sample, with lower levels of educational attainment. This is an important limitation to note because research indicates that levels of allostatic load are higher in ethnic minorities as compared with Whites (Geronimus, Hicken, Keene, & Bound, 2006), in those with less educational attainment, and among those who report worse health (Juster, McEwen, & Lupien, 2010). If we had had a more diverse sample, it is possible that our results may have extended into younger ages. Future research is necessary to test this possibility.

Another limitation is that the Biomarker project was not included in MIDUS until Wave 2. Because of this, we did not have baseline allostatic load data. Thus, unlike previous studies that examined whether daily stress processes were associated with changes in self-reported chronic conditions (e.g., Charles et al., 2013; Piazza, Charles, Sliwinski, et al., 2013), we could not examine whether daily stress processes predicted changes in allostatic load but rather predicted allostatic load 10 years after initial assessment. Thus, while we consider our study an important first step in examining the link between people’s daily lives and their physiological functioning, our findings must be interpreted within the confines of the data.

Conclusion

Despite these limitations, the present study adds to a growing body of literature documenting that what we do in our daily lives—the situations we are exposed to, the stressors they create, and our reactions to these stressors—has implications for health, particularly in later adulthood. These findings highlight the need for interventions aimed at managing the daily stress in older adults’ lives.

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