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Author manuscript

Psychol Aging. Author manuscript; available in PMC 2019 December 13.

Published in final edited form as:

Psychol Aging. 2014 March ; 29(1): 72–83. doi:10.1037/a0035500.

Reactivity to Stressor Pile-Up in Adulthood: Effects on Daily Negative and Positive Affect

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Abstract

This study used data from a 30-day diary study with 289 adults (age range 18–89 years) to model the effects of stressor pile-up on individuals' daily negative affect (NA) and positive affect (PA) and to test for age differences in these effects. Specifically, we developed a new approach to operationalize and model stressor pile-up and evaluated this approach using generalized mixed models, taking into account the gamma response distribution of the highly skewed daily NA data. Findings showed that pile-up of stressors over a 1-week period was significantly coupled with increases in individuals' daily NA above and beyond the effect of concurrent stressors. Findings also showed that the effects of stressor accumulation and concurrent stress were additive rather than multiplicative. Age interacted significantly with stressor accumulation so that a higher age was associated with less NA reactivity to stressor pile-up. Yet, we did not find such an age-related association for NA reactivity to concurrent daily stressors. Daily PA was not associated with daily stress or with stressor pile-up. The operational definition of stressor pile-up presented in this study contributes to the literature by providing a new approach to model the dynamic effects of stress, and by providing new ways of separating the effects of acute stressors from the effects of stressor pile-up. The age differences found in the present study suggest that older adults develop effective emotion regulation skills for handling stressor pile-up, but that they react to acute daily stressors in a similar way than younger adults.

Keywords

stressor pile-up; daily affect; age differences; generalized mixed models

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[6]It should be noted here that in light of these considerations we based the computation of R^2s on the residual variance obtained from testing the null model including the observations that were excluded in the respective M1-M5 model (i.e., observations with non-missing NA or PA along with a missing value of S_{ti} and/or $A_{(6)ti}$). That is, we did not apply a listwise deletion in the ad hoc R^2 computations and used all available information to estimate the total intraindividual variation of daily NA (or PA), which served as the unique baseline of the residual variance reduction obtained with each of the models.

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It is well established that exposure to daily stress is negatively associated with physical and psychological well-being (Bolger, DeLongis, Kessler, & Schilling, 1989; Bolger & Schilling, 1991; DeLongis, Folkman, & Lazarus, 1988; Serido, Almeida, & Wethington, 2004; Stawski, Sliwinski, Almeida, & Smyth, 2008). Although great progress has been made in understanding how humans cope with daily stressors (Aldwin, 2007), a persisting challenge is to gain a better understanding of how stress processes unfold and persist over time and how individual difference variables contribute to the great observed variability in reactivity to and recovery from stress (Williams, Smith, Gunn, & Uchino, 2011). One such aspect of reactivity to stress that is not well understood is the effect of *stressor accumulation* or *stressor pile-up*.[1]

Research suggests that stressor pile-up is common in adults and has negative consequences for physical and psychological well-being above and beyond those of discrete stressors (Bolger et al., 1989; Bolger & Schilling, 1991; Fuller et al., 2003; Grzywacz, Almeida, & McDonald, 2002; Serido et al., 2004). For example, data from the National Study of Daily Experiences, a representative sample of US adults, showed that individuals reported stressful events on approximately 40% of days and multiple stressors on 10% of days (Almeida, Wethington, & Kessler, 2002). Thus, adults appear to regularly experience multiple stressors within and across days, making stressor pile-up a frequent everyday phenomenon.

However, despite the potential importance of stressor pile-up for individuals' well-being, there is a lack of research on its effects and a lack of well-defined methodological approaches to model this phenomenon. Thus, research is needed that examines operational definitions and methods of measuring and modeling stressor pile-up. Such research is an important methodological prerequisite for the study of psychological vulnerabilities to stressor pile-up. Moreover, developing approaches to model stressor pile-up is important for gaining a better understanding of how episodes of acute and chronic stress contribute to the development and manifestation of stress-related health problems (Almeida, 2005; Miller, Chen, & Zhou, 2007). Thus, the present study had two major objectives. First, using data from a 30-day diary study, we introduce a new approach to model reactivity to stressor pile-up separately from reactivity to concurrent daily stressors. Second, we examined potential age differences in these reactivities to address several inconsistencies in the literature on stress reactivity across the adult lifespan.

In addressing these objectives, *reactivity* refers to the within-person covariation between daily stress and daily affect, hence following the definition widely used in the pertinent literature (e.g., Bolger & Schilling, 1991; Sliwinski, Almeida, Smyth, & Stawski, 2009). However, the term reactivity implies a notion of directionality of the stress-affect link, in that intraindividual changes in affect across the observation period are triggered by preceding stressors. In a strict sense, intraindividual covariations cannot confirm such a directionality. Thus, the empirical observations, strictly speaking, reveal the within-person coupling of stress and affect, which is viewed *theoretically* as an indicator of affect reactivity to the occurrence of stressors. In this study, we will use the terms *reactivity to concurrent daily stress* and *reactivity to stressor pile-up*. The former refers to the within-person covariation of

[1]Throughout this article, we use the terms "stressor accumulation" and "stressor pile-up" interchangeably.

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daily stressors and measures of affect as analyzed in other studies, whereas the latter refers to the within-person covariation between the stress accumulation at the end of the day preceding a given study day, and the affect measured on that day.

Reactivity of Negative and Positive Affect to Daily Stress

In research on daily stress, negative affect (NA) has been the primary outcome and a considerable body of evidence has accumulated showing substantial reactivity of NA to daily stress (e.g., Bolger et al., 1989; Serido et al., 2004; Sliwinski et al., 2009; Stawski et al., 2008). Thus, from a theoretical viewpoint, it can be expected that stressful events or situations induce primarily negative emotions. NA has been proposed to be part of a basic biobehavioral inhibition system, which keeps the organism from danger and risks by inhibiting actions that may result in unpleasant or harmful outcomes (Fowles, 1987; Quilty & Oakman, 2004; Watson, Wiese, Vaidya, & Tellegen, 1999). In short, NA is an internal reaction to stimuli signaling potential threat and harm and is especially sensitive to subjectively aversive experiences, such as stressful situations encountered in everyday life.

Thus, NA may be viewed as the domain of choice to assess affective reactivity to stressful experiences. This reasoning also suggests that NA should be particularly sensitive to the accumulation of stressors. Therefore, it seems reasonable to expect that stressor pile-up should have significant effects above and beyond those captured by the within-person association between daily measures of stress and NA (see also reports on lagged-effects of daily stressors on daily NA; Ong, Bergeman, Bisconti, & Wallace, 2006).

In contrast to NA, positive affect (PA) is directed towards the behavioral activation of experiences of pleasure and reward (Fowles, 1987; Quilty & Oakman, 2004; Watson et al., 1999) and is the result of positive experiences. Therefore, PA would be expected to be affected by stressful situations only to the extent to which daily stressors impede the engagement in prohedonic activities. Not all daily stressors, however, create such an impediment. For example, some individuals may neither gain nor seek positive feelings from their work, but may have developed routines to experience pleasure and positive emotions while engaging in out-of-work activities (e.g., playing in a band or engaging in physical exercise). For these individuals, daily stressors at work may therefore cause mostly NA, but may not interfere with their experience of PA. Moreover, negative covariation of PA with daily stress may be reduced or even reversed if some individuals engage in hedonic activities that generate and/or enhance PA and, hence, counteract and potentially overwrite NA responses to stressors (e.g., Folkman & Moscowitz, 2000; Ong, Bergeman, & Bisconti, 2004; Ong et al., 2006). The crucial point for our study is that stressful experiences may trigger more diverse reactions in daily PA, varying in terms of direction, or across stressors and between individuals. Thus, the strength and direction of the within-person coupling of PA and daily stress would be expected to be less pronounced compared to NA reactivity.

Empirical studies of PA reactivity in response to stress have yielded mixed findings and have provided support for impeding as well as facilitating effects of daily stress. For example, whereas Uchino, Berg, Smith, Pearce, and Skinner (2006) reported that PA was positively associated with daily stress, Röcke, Li, and Smith (2009) found PA reactivity to be restricted

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to negative daily events among young, but not old individuals. In contrast, Stawski et al. (2008) reported a negative association between daily stress and PA only among older adults, but not among young individuals. On the basis of these findings and the fact that to date PA reactivity has been less frequently studied than NA reactivity, it is still an open question to what extent PA shows at all reactivity to individuals' exposure to daily stressors.

Given this general background, it is difficult to predict how the accumulation of daily stressors might impact individuals' PA. On the one hand it seems plausible that if individuals increase their engagement in behaviors that generate PA to counteract negative responses to stressors, then they may do this even more so when stressors pile-up. That is, a single stressful event may not necessarily trigger such prohedonic behaviors and the experienced stress may simply subside as time passes. In contrast, continued or repeated exposure to stressors may motivate deliberate engagement in activities that create positive emotions. On the other hand, it may also be the case that continued exposure to stressor pile-up may result in the obstruction of prohedonic engagement, hence pushing the level of PA downward. Given these possible scenarios, both impeding as well as facilitating effects could be expected with regard to PA reactivity to stressor pile-up. Across individuals and/or situations, these counteracting effects could potentially cancel each other out, resulting in very little or zero reactivity of PA to stressor pile-up.

In conclusion, these considerations result in the assumption that the system of NA should be the primary behavioral domain in which reactivity to daily stress should be assessed. For this reason, we focused primarily on the effects of stressor pile-up on NA, yet also examined the reactivity in PA to stressor pile-up.

Age Differences in Affect Reactivity to Stressor Pile-Up

Coping successfully with life stress is indicative of psychological resilience and a person's reactivity to daily stress is such an indicator of resilience. The extent to which reactivity to daily stressors is subject to age-related change is, thus, an important issue for research on adult development and aging. Several theories suggest that older individuals may be less reactive to stressors than younger adults due to improved emotion regulation skills (e.g., Carstensen et al., 2011; Hay & Diehl, 2011; John & Gross, 2004). In addition, familiarity with stressors that have been repeatedly encountered across the adult lifespan may reduce reactivity (e.g., Frijda, 1988; Röcke et al. 2009), and older individuals may also show less reactivity because they tend to be overall exposed to fewer stressors in their daily lives (Stawski et al., 2008). In contrast, it could be argued that old age comes with loss of resources needed to cope successfully with stressors (e.g., declining health, loss of social support resources), such that vulnerability to stress may increase with age, resulting in greater variability in NA in response to daily stress. In particular, it has been proposed that, rather than habituation, sensitivity to stress may increase across the lifespan, due to kindling effects in the aging brain (Mroczek & Almeida, 2004).

To date, evidence of age differences in daily stress reactivity has been mixed. With respect to the coupling of daily stress and NA, some studies found less reactivity in older compared to younger adults (Hay & Diehl, 2010; Uchino, et al. 2006), whereas others reported young

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adults to be less reactive (Mroczek & Almeida, 2004) or equally reactive than older adults (Röcke et al., 2009; Stawski et al., 2008). Although these studies provide cross-sectional evidence of age differences of NA reactivity to daily stress, Sliwinski et al. (2009) reported longitudinal increases of stress-NA reactivity. Only few of these studies also provided age group comparisons of PA reactivity, overall resulting in rather inconsistent findings (Röcke et al., 2009; Stawski et al., 2008).

Focusing on daily NA, it remains an open question whether and to what extent there are age differences in reactivity to stressor pile-up. It seems very reasonable to differentiate the previously presented arguments for or against reduced stress reactivity in old age with respect to reactivity to *acute daily stressors* versus reactivity to *stressor accumulation* across multiple days. For instance, it may well be that the immediate effects of stressors on the affective system operate on a more biological level, resulting in the instantaneous and less regulated affective responses that are measured in the intraindividual association between measures of daily stress and concurrent daily NA. In contrast, repeated exposure to multiple stressors within a short time period may prolong the post-stress activation of the affect system in a way that under increasing stress accumulation higher levels of NA are experienced over several consecutive days. However, such a longer lasting stress reaction may also be more likely to be affected by efforts of emotion regulation, compared to the more immediate NA activation.

This reasoning is consistent with the model of strength and vulnerability integration (SAVI) proposed by Charles (2010). SAVI posits that increasing age comes with improvements in emotion regulation strategies, including positive reappraisal or reduced recall of negative events. Key to SAVI is the prediction that these strengths work *after* negative events and hence get increasingly effective as time passes. With respect to the *concurrent* affective reactions to negative events, SAVI predicts that age-related emotion regulation strengths are counterbalanced by age-related vulnerabilities in terms of reduced physiological flexibility which decreases a person's ability to down-regulate arousal caused by stressors. Thus, better emotion regulation skills may result in less reactivity to stressor pile-up among older compared to younger adults, leading to the hypothesis that reactivity to stressor accumulation in terms of NA should decrease with age. On the other hand, the association between daily stress and daily NA should increase or should stay stable across the adult lifespan (e.g., if increasing physiological vulnerability and improved emotion regulation cancel each other out in short-term affective responses to stressful experiences).

The Present Study

The main objective of the present study is to present a new approach for operationalizing and modeling stressor pile-up as a source of intraindividual variability in outcomes. Specifically, in this study we examined the outcomes of daily NA and addressed the following two questions: First, we examined whether stressor accumulation contributed significantly to the individual variation of daily NA above and beyond the effects of concurrent stressors. We hypothesized that stressor pile-up would be significantly associated with daily NA above and beyond the effect of concurrent stressors. In addition, we examined whether the state of stressor pile-up on a given day moderated individuals' reactivity to

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stressors experienced on that day. That is, we examined whether the effects of stressor pile-up and daily stress on NA were additive or multiplicative.

Second, we examined age differences in affect reactivity to stressor pile-up. With respect to NA, we expected that older adults would be less reactive to stressor pile-up than younger adults. Support for this expectation would be suggestive of age-related improvements in emotion regulation.

Third, the same questions were also examined with regard to PA. However, because of the lack of clear theoretical expectations concerning daily PA reactivity to stressor pile-up no specific hypotheses were put forward for these analyses.

Method

Participants

The sample consisted of 147 men and 142 women from North Central Florida. The study included healthy community-residing adults without major sensory impairments, concurrent depression, or a history of mental illness. Also, participants had to be physically able to come to the testing location and have the cognitive abilities to complete the study protocol without any difficulty. To ensure an even distribution of age, participants were recruited from three age groups: young adults (n = 107; age range 18–39 years), middle-aged adults (n = 94; age range 40–59 years), and older adults (n = 88; age 60 and older). The sample ranged in age from 18–89 (M = 48.6 years, SD = 19.9 years). Men and women were evenly distributed within each age group. Recruitment strategies did not differ across the age groups.

All participants spoke English as their primary language and 86% identified themselves as Caucasian, 9% as Black, and 3% as Hispanic. On average, participants reported 16.2 years of education (SD = 2.9). The median reported annual income was $35,000–50,000. Most of the young adults (72.9%) were single, whereas most middle-aged (62.8%) and older adults (63.6%) were married. Participants described themselves as being in good health (M = 5.11, SD = .89; 1 = *very poor*, 6 = *very good*) and being satisfied with their lives (M = 4.61, SD = .72; 1 = *extremely unhappy*; 6 = *extremely happy*).

Procedure

Participants first attended a two to three hour individual baseline session. The day following the baseline session, participants began 30 consecutive daily assessments, consisting of a self-administered diary and a phone interview in the evening. To keep the time intervals between diaries as even as possible, participants were instructed to complete diaries each evening at approximately the same time. To verify that participants complied with this request, we asked them to fill in the starting and end time each day in a designated space on the diary, and these “time stamps” were checked when diaries were returned. Participants who deviated from these instructions, were reminded during the next phone call and were monitored to assure compliance. All testing was performed by trained research assistants.

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In total, data from 8,521 study days were available, which is 98.3% of the maximally possible 8,670 days (i.e., 289 participants × 30 days). In our analyses only days with non-missing data on NA (or PA) were included in the analyses. This resulted in 7,795 days from 285 participants (*M* = 27.4 days per person).

Measures

Daily stress—During the phone interviews, participants completed the Daily Inventory of Stressful Events (DISE; Almeida et al., 2002). The DISE consists of seven stem questions assessing the occurrence of stressors, including having or avoiding arguments, as well as stressors that occur in various life domains (e.g., at work/school/volunteering, in personal health). The timeframe for asking about stressful events was the past 24 hours (i.e., the occurrence of stressors since the last interview). The number of stressful events participants experienced each day was summed to create an index of daily stress, ranging from 0 to 7. Participants also rated the severity of each daily stressor on a 5-point scale (0 = *not at all stressful*; 4 = *very stressful*). These severity ratings were summed to create an index of daily *stress severity* (range 0–28).

All analyses presented in this study were performed using both the number of stressors and the daily stress severity score. However, the presentation and discussion of findings will focus on the results obtained from the stress severity scores. Our preference for the severity measure is grounded in Lazarus and Folkman's (1984) theory of stress, appraisal, and coping and empirical evidence. Findings from numerous studies have shown that it is the perceived severity, based on primary and secondary appraisal processes, that makes a stressor more or less stressful. Also, both measures were highly correlated (see Table 4), rendering the results virtually identical when we used the number of stressors as the measure of choice.

Daily negative and positive affect—Each day, participants completed the Positive Affect and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The NA and PA scales each consist of 10 affect items, such as angry (NA) or enthusiastic (PA). Respondents indicated how often they had felt this way during the past 24 hours on a 5-point scale (1= *very slightly or not at all*; 5 = *extremely*). Thus, daily NA and PA scores can range from 10 to 50, with higher scores being indicative of a higher level of NA and PA, respectively. The PANAS has high internal consistency and test-retest reliability (Watson et al., 1988). We estimated the internal consistency coefficients on each day of measurement separately. The resulting coefficients ranged from .80 to .89 for NA and from .88 to .94 for PA.

Statistical Analyses

Operationalization of stressor pile-up—To determine the effect of stressor pile-up on the within-person variability in any daily outcome *above and beyond* the effect of concurrent daily stress, it must be specified how the *amount of pile-up* on a given study day is quantified. Consider a person who reported daily stressors for two weeks, starting on a Monday. The individual did not report any stressors from Monday through Thursday of the first week. The person then had four days with high stress (i.e., multiple and severe stressors

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each day) from Friday through Monday of the next week, and for the remainder of the observation period the individual again did not report any stressors (i.e., six days of no stress). Using this example, even if this individual experienced about the same amount of stress within each day from Friday to Monday, the stress pile-up increases across these four days, and the amount of pile-up reached by the end of Monday is expected to "reverberate" across the subsequent stressor free days. That is, stressor accumulation denotes a *dynamic process* unfolding over time and a measure is needed to capture this dynamic time-varying aspect of stressor pile-up from day to day.

A measure indicating the stressor accumulation on a given day within an observation period may be defined in terms of the amount of stress experienced in close temporal proximity. Daily stressor accumulation could then be measured by summing a person's daily stress measures up to the respective day and weighting them according to their temporal proximity. This general idea is expressed in the following equation:

$$A_{(k)ti} = \frac{\sum_{j=1}^{k} (k-j+1) S_{(t-j)i}}{\sum_{j=1}^{k} j},$$

where $A_{(k)ti}$ is the index of stressor accumulation across k days on study day t for individual i, and S_{ti} is the stress measured on day t of individual i (i.e., in our study the stress severity score or the number of stressors). Note that k specifies the number of study days that precede day t, which are included in the computation. Thus, the numerator contains the sum of daily stress measures over k preceding days, weighted by temporal proximity to study day t.[2] Division by the sum of weights (denominator) serves to scale the index to the same range of values as S_{ti}.

For a computational example, the daily stress data from one selected respondent are shown in Table 1. Both daily stress measures, the stress severity scores and the number of stressors reported on each day, and the respective accumulation indices computed from these stress measures across $k = 6$ days are listed. For example, applying the above formula, the accumulation value at study day 7 is computed as follows: $A_{(6)7i} = (S_{1i} + 2S_{2i} + 3S_{3i} + 4S_{4i} + 5S_{5i} + 6S_{6i})/(1 + 2 + 3 + 4 + 5 + 6)$; thus, the accumulation value based on the severity scores is $(1\times2 + 2\times2 + 3\times4 + 4\times2 + 5\times0 + 6\times2)/21 = 38/21 = 1.810$. Thus, the stressors reported on the 6 days preceding day 7 have been weighted according to their reversed distance to day 7, with the most distant day multiplied by 1 and the closest day multiplied by 6. Note that the divisor 21 is the sum of these weights, which serves only to scale the index in a more convenient way. Rewriting the formula as $A_{(6)7i} = (1/21)S_{1i} + (2/21)S_{2i} + (3/21)S_{3i} + (4/21)S_{4i} + (5/21)S_{5i} + (6/21)S_{6i}$, the weights given to the daily stress scores

[2]Refinement of $A_{(k)ti}$ may be achieved by weighting the stress intensities S_{ti} with the temporal closeness to day t not linearly as presented, but quadratically by replacing $(k - j)$ with $(k - j)^2$. Such a replacement implies a stronger weighting of stressors more proximal to day t, following the logic of the Euclidian distance measure. However, this may also imply an underweighting of the more distant stressors, such that the stress accumulation index $A_{(k)ti}$ would possibly indicate mostly the carry-over effects from this previous day stress, instead of a longer stress accumulation period. For that reason, we decided to use the linear weighting. Checking also the quadratic weighting empirically, we obtained in the NA models R^2 values about .009-.012 below their linearly weighted counterparts. Similarly, we checked if weighting was needed at all by using equal weights for all k study days included in the accumulation index. This revealed R^2s about .010-.015 lower than the linearly weighted $A_{(k)ti}$.

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sum up to 1, such that the accumulation score indicates the average stress per day, weighted by temporal proximity across the preceding 6-day period.

Two additional noteworthy aspects are illustrated in Table 1. First, the stress accumulation indices, both severity-based and number-based, peak at day 22, the day after this respondent's most stressful day. After that peak, the accumulation declines slowly, keeping a relatively high value across the subsequent days. This illustrates the desired quality of the accumulation measure to reflect the "reverberation" of stressful experiences across the time period of k days. Second, $A_{(k)ti}$ could not be computed validly for the first $k = 6$ study days. If, for example, the 6-day accumulation at study day 6 were to be computed, the stress value S_{0i} would be needed. This value, however, was not measured because the respective day $t = 0$ did not belong to the observation period. Therefore, there is a need to find a time frame k large enough for valid measurement of stressor accumulation, but not too large to lose too much of the early study days for measurement.

Finally, note also that $A_{(k)ti}$ was defined in a way that the computation of stressor accumulation reached on study day t does not include the stress experienced on day t. We did this because we aimed to model the effect of the k-day accumulation *above and beyond* the effect of concurrent stress S_{ti} measured on study day t. Therefore, it was more appropriate to compute the stressor accumulation reached at the beginning of that day, excluding S_{ti} from the computation.

Modeling reactivity of daily affect to stressor pile-up—To examine whether stressor pile-up contributed to the intraindividual variation of daily NA, we used the stressor accumulation index as described above and performed Generalized Linear Mixed Models (GLMM; Hedeker, 2005). The models which were tested are listed in Table 2. GLMM is a method to fit multilevel mixed models to a wide range of response variable distributions, not restricted by the normality assumption.[3] In our data, NA was skewed substantially, suggesting that the gamma response distribution was more appropriate to represent the data.

Model 1 (M1) and Model 2 (M2) examined the absolute stand-alone effects of $A_{(k)ti}$ and S_{ti} on the daily affect outcome, respectively, and were computed mainly for R^2 comparison with Model 3 (M3). M3 included both $A_{(k)ti}$ and S_{ti} as predictors. Thus, M3 is the crucial model with respect to our first research aim examining how stressor pile-up accounts for the daily variation in NA or PA above and beyond the stress experienced on that day. In M3 also the within-person mean of S_{ti} across study day k to 30 (i.e. the period of daily NA or PA modeled in these analyses) was included as predictor (denoted SP_i), in order to control for interindividual differences in the overall level of stressful experiences across the observation period (Hoffman & Stawski, 2009). Hence, β_2 and β_3 indicate uniquely the effects of

[3]The formal specification of the GLMM is: $Y_{ti} = \mu_{ti} + \varepsilon_{ti}$; and $g(\mu_{ti}) = \boldsymbol{x}'_{ti}\boldsymbol{\beta} + \boldsymbol{z}'_{ti}\boldsymbol{u}_i$. The right-hand part of the second equation is called the linear predictor, including the design vectors (regressors) $\boldsymbol{x}'_{ti}$ of fixed effects β and $\boldsymbol{z}'_{ti}$ of the random effects u_i (for individual i at time t). Thus, the response variable Y_{ti} is decomposed in the so-called marginal mean μ_{ti}, predicted by the model, and the residual ε_{ti}. However, Y_{ti} is linked with the linear predictor not directly, but only after transformation by a link function $g(\cdot)$, which usually depends on the distribution of Y_{ti} (see Hedeker, 2005). In our case, $g(\cdot)$ was either the gamma function, in order to fit the gamma response distribution, or the identity function, which leads to conventional mixed modeling based on the normality assumption (for details, see SAS Institute Inc., 2009, pp. 2080–2430).

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intraindividual variation of stressor pile-up and concurrent stress, respectively. Note that the within-person mean of $A_{(k)ti}$ was not added to M3, because this nearly equals SP_i (e.g., for $k = 6$ it can be derived from the above formula that the individual mean $AP_{(6)i} \approx SP_i + (.04S_{6i} + .03S_{5i} + .02S_{4i} + .01S_{3i} + .006S_{2i} + .002S_{1i}) - (.03S_{30i} + .02S_{29i} + .01S_{28i} + .006S_{27i} + .002S_{26i})$).[4]

Model 4 (M4) extended M3 by adding the $A_{(k)ti} \times S_{ti}$ interaction to examine if the stressor accumulation up to day t moderated individuals' reactivity to daily stress in a way that with increasing stressor accumulation the impact of the daily stress severity on the daily NA or PA increased or decreased. It may be noted here that we also tested the $SP_i \times A_{(k)ti}$ and $SP_i \times S_{ti}$ interactions in models not shown in Table 2, to check whether the individual mean level of stress experienced across the observation period moderated the effects of daily stressor pile-up and/or concurrent daily stress on the affect outcomes. However, these interactions were not statistically significant; hence, we do not report these models. Also, the fixed and random effects of the linear time trend included in the models were nonsignificant; hence, these were left out in the models reported for reasons of parsimony.

Finally, to examine whether reactivity to stressor accumulation was related to age across the adult lifespan, we modeled the interactions of age with $A_{(k)ti}$ and S_{ti} by extending M3 to Model 5 (M5). We analyzed linear, quadratic, and cubic effects of age on the outcome and on the reactivity to stressor accumulation and daily stressor severity. That is, we reasoned that across the large age range covered in our study, age-related development of reactivity may not simply occur in a linear fashion. For instance, if reactivity would peak or bottom out in middle adulthood, or if decrease (or increase) in reactivity would accelerate (or decelerate) in late adulthood, such patterns would be detected by significant quadratic or cubic age effects.

M1 to M5 were computed using the procedure GLIMMIX in SAS 9.2 (SAS Institute Inc., 2009), which fits GLMM for a wide range of response variable distributions, including gamma and normality. Doing so, we applied the Laplace integral approximation method, which enables the computation of likelihood-based fit statistics (for computational details refer to the GLIMMIX manual, SAS Institute Inc., 2009, pp. 2080–2430). The variables $A_{(k)ti}$, S_{ti}, SP_i, and age_i were grand-mean centered prior to estimating the mixed models. To evaluate the portion of intraindividual variance explained by the GLIMMIX estimates, we followed Xu's (2003) rationale to operationalize R^2 in terms of the reduction of residual variance compared to the random-intercept-only null model. Thus, we computed for each model $R^2 = 1 - \sigma^2/\sigma_0^2$, where σ^2 and σ_0^2 denote residual variances of the model tested and the null model, respectively.

[4]Because of the extreme multicollinearity between the two within-person means, we used the more conventional approach to separate the within-person and between-person effects of each predictor by using of person-mean centered scores together with their respective person means. However, inclusion of only one of the two person means $AP_{(k)i}$ and SP_i together with the person-mean centered scores of $A_{(k)ti}$ and S_{ti} was a possible modeling alternative (Hoffmann & Stawski, 2009), which we also examined, but do not report here to avoid confusion about the meaning of the results. This analysis yielded the same results in terms of $A_{(k)ti}$ and S_{ti} effects and R^2s. In the daily stress and affect literature, both approaches have been used. For example, Röcke et al. (2009) used person-mean centering of the daily stress scores, whereas Sliwinski et al. (2009) and Stawski et al. (2008; though with dichotomous indicators of daily stress) included person means together with the stress scores not centered on the person mean.

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Results

Preliminary Analyses

Response distribution of daily NA and PA—Descriptive statistics of the sample distribution of daily NA across respondents and study days indicated substantial non-normality: $M = 12.7$, $SD = 3.9$, *skewness* = 2.3, *kurtosis* = 7.1. NA showed a large right-skew, with low levels of NA reported on most of the study days (i.e., the NA mode was at the minimum of 10). The χ^2 goodness-of-fit values indicated a much better fit of the gamma ($\chi^2 = 257.1$, $df = 36$) as compared to the normal distribution ($\chi^2 = 435{,}338.3$, $df = 36$), though none fitted perfectly (i.e., both χ^2 with $p \leq .001$, indicating lack of fit).[5] Thus, we present the findings from the gamma-based mixed model analyses on daily NA.

PA showed no pronounced non-normality ($M = 26.6$, $SD = 8.4$, *skewness* = .15, *kurtosis* = -.47) and the normal distribution revealed the best fit to the data in terms of the χ^2; although again significant deviation from perfect normality was indicated, $\chi^2 = 404.3$, $df = 38$, $p \leq .001$. Thus, the assumption of normality of the response variable was considered suitable for performing M1 to M5 with the PA variable.

For comparability reasons we performed all M1-M5 analyses for NA and PA using both response distributions. Notably, modelling the gamma versus normal response distributions, we found virtually the same patterns of statistical significance of the parameter estimates in any of the models tested, and the gamma-based estimates of the R^2s did not differ by more than .001 from their normality-based counterparts. However, the BIC and other likelihood-based fit statistics indicated worse fit of the normality-based analyses of NA, whereas the gamma-based analyses provided poorer fit with daily PA.

Selection of the accumulation time frame *k*—Prior to testing models M1 to M5, the time period k of study days to be included in the computations of the stress accumulation index $A_{(k)ti}$ had to be chosen. A value of k suited for the stressor accumulation analyses may be selected in a data-driven way such that $A_{(k)ti}$ accounts for as much of the intraindividual variance in NA as possible. Thus, we applied an ad-hoc strategy and tested a sequence of M1 computations with $k = 2, 3, \ldots, 10$, in order to compare the respective R^2s. As can be seen in Table 3, the R^2 peaked at $k = 6$, providing support for the choice of the 6-day period for the computation of the stress accumulation measure. Thus, the maximum of about 10% of the intraindividual daily NA variation was explained by $A_{(6)ti}$. Note that Table 3 also contains the R^2s obtained when the daily *number of stressors* instead of stressor severity was used in the $A_{(k)ti}$ computations, resulting virtually in identical findings.

In addition, $A_{(6)ti}$ was considered theoretically reasonable, as it covers the one-week period preceding the respective study day t, such that this includes stressors that occur with some regularity at certain weekdays in adults' everyday lives (e.g., work- and family-related

[5]We also checked other candidates of a right-skew distribution (lognormal, Weibull, exponential), which all revealed χ^2 larger than gamma. Thus, it may be concluded from these checks that gamma was the best response distribution model for the mixed model analyses.

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stressors, see also Chui, Diehl, Lumley, & Labouvie-Vief, 2013; Larsen & Kasimatis, 1990). Thus, $A_{(6)ti}$ was used in all subsequent analyses.

Note again that $A_{(6)ti}$ cannot be computed for respondents' first 6 study days, hence only data from study days 7 to 30 were included in all analyses, yielding 6,139 data points of NA from 276 respondents. For these data points, 4,774 values of $A_{(6)ti}$ could be computed (i.e, $A_{(6)ti}$ was missing at 22.2% of person-days). Across individuals and days, the stress accumulation index $A_{(6)ti}$ ranged from 0 to 8.10, the grand mean was 1.46 (SD = 1.17), and the intraindividual mean values of $A_{(6)ti}$ ranged from 0.04 to 6.73. On average, $A_{(6)ti}$ values could be computed for 17.3 study days for each of the 276 respondents (including 5 with all $A_{(6)ti}$ values missing).

Like for NA, we estimated M1 for daily PA with $A_{(k)ti}$ computed with varying time frames k = 1 to 10. For PA, the R^2 peaked at k = 7, but was only marginally lower for k = 6 in the gamma-based (.090 vs. .089, respectively), as well as in the normality-based analyses (.088 vs. .087). Notably, the effect of stressor accumulation was not significant in any of the estimated models. Thus, in view of negligible differences in the R^2 and non-significant effects obtained from the 7-day and 6-day stress accumulation index, we chose to stay with the k = 6 time frame established for NA. This preserved the comparability of the results obtained for both dimensions of daily affect.

Finally, having chosen $A_{(6)ti}$, Table 4 shows basic statistics for all variables analyzed. For the variables measured daily, the means, standard deviations and between-person correlations were computed on the aggregated intraindividual mean scores. The overall correlations computed across persons and study days are also shown. Note the close-to-perfect correlations between the individual mean scores of number, severity, and pile-up of stressors. Age was not associated with any of the aggregated stress variables. Also, neither age, nor age-group as used in the sampling procedure was a significant predictor of the aggregated stress scores (regression analysis) or the daily scores of stressor severity or pile-up (random-intercept mixed model). In addition, we checked for age differences in the intraindividual range or maximum of $A_{(6)ti}$ (indicating, in relative or absolute terms, the individual peaks of stressor pile-up), but again no significant effect of age was found.

Does Stress Accumulation Increase Daily Negative Affect?—Table 5 shows the results of testing models M1 to M4 with NA as the dependent variable. Reading this table, note with respect to the meaning of the absolute values of the fixed regression coefficients and random intercept variances that in the GLMM with the gamma response distribution these do not directly refer to the scale of the data analyzed. That is, the fixed regression coefficients do not refer to the predicted value μ_{ti} conditional on the predictors as in conventional linear regression analyses, but to the so-called linear predictor $g(\mu_{ti})$ (see Footnote 3). However, the signs of the effects and the significance tests can be interpreted as usual.

With respect to our research question, the following findings were particularly relevant. First, there was a significant effect of stressor accumulation on NA above and beyond concurrent daily stress. That is, after controlling for the perceived severity of stressors that

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occurred on a given day in M3, the fixed effect of $A_{(6)ti}$ on NA measured on that day was significant. This indicated that, for the total sample, daily NA was higher when stressors accumulated across days. Also, the $A_{(6)ti}$ effects varied significantly across individuals (i.e., significant random effects). In M1, the absolute "stand-alone" fixed and random effects of $A_{(k)ti}$ were also statistically significant, but it should be mentioned here that including the within-person mean of stressor severity SP_i (or of stress accumulation) in that model, the fixed effect was reduced and no longer significant, whereas the random variance of the $A_{(k)ti}$ slopes, as well as the R^2 remained the same. Thus, not controlling for concurrent daily stressor severity promotes a kind of shift of the stressor pile-up slopes towards lower and negative values (but still the same interindividual variation). This makes sense, as is illustrated with the data shown in Table 1: Because increases and declines of the daily stressor severity S_{ti} are reflected with a 1-day time-lag by respective changes of $A_{(k)ti}$, there were several study days with elevated daily stressor severity but a decreased stressor accumulation value (e.g., study days 9, 16, and 21), On these days the daily stressor severity elevation may have promoted a NA increase while at the same time the stressor accumulation index decreased. Similarly, on days following the S_{ti} peaks, when concurrent daily stress goes down again, $A_{(k)ti}$ goes up due to the previous day's high stress value. Therefore, in a model that does not control for S_{ti}, a tendency towards a lower and more negative stressor accumulation slope may be produced, and it is crucial to model the effect of $A_{(k)ti}$ above and beyond the concurrent daily stress, such as in M3.

Second, the comparison of the findings from M1 and M2 showed that stressor pile-up as single predictor of daily NA accounted for about half the variance in daily NA as compared to concurrent daily stress (i.e., 10.5% vs. 20.4% of the variance in daily NA), and adding $A_{(6)ti}$ to the daily NA stress reactivity model increased the amount of variance accounted for in daily NA by about 12% (cf. M2 vs. M3). Third, the results from testing M4 failed to show a significant interaction of stressor accumulation with daily stress. That is, the stress accumulated until a given study day did not moderate individuals' reactivity to the stress they experienced on that day.

Age-Related Differences of Daily Negative Affect Reactivity to Stress Accumulation—To analyze whether the effects of stressor accumulation and/or daily concurrent stress on NA differed across age groups, we tested three versions of M5, modeling linear, quadratic, and cubic effects of age. The BIC indicated that the linear age-effect model fitted best and no curvilinear age-effects were needed (BIC_{linear} = 20,976.8; $BIC_{quadratic}$ = 20,991.4; BIC_{cubic} = 20,998.7; for a rationale of BIC-based model selection, see Kass & Raftery, 1995). Notably, the linear age-effect M5 showed a markedly better BIC than its no-age-effects counterpart M3.

Table 6 shows the results from testing M5 with linear effects of age. A significant negative main effect of age indicated a general tendency of lower daily NA being associated with a higher age. However, with respect to our research questions the interaction effects were of greatest interest. First, the interaction of daily stress severity S_{ti} and age was not significant, indicating that there were no age-related differences in NA reactivity to concurrent daily stress. Second, the interaction of age with stressor accumulation, however, was significant. The negative sign of this interaction indicated that older adults tended to show less NA

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reactivity to stressor accumulation compared to younger adults. M5 had virtually the same R^2 as M3. However, because age was a between-subject predictor, it was expected that it might explain between-person random variance of the daily stress and stress accumulation slopes, rather than within-person residual variation. Comparison of the M3 and M5 random effect variances suggested that about 5% of the between-person variance in NA reactivity to stressor accumulation was accounted for by age.

Stress Reactivity in Daily Positive Affect—Testing of the models M1 to M5 on PA yielded no significant fixed effects of stressor accumulation or concurrent daily stress severity or the within-person mean of stressor severity. However, random slope variances were significant with $p \leq .001$ in all models. The R^2 computed from the normality-based solution were .093, .052, .136, and .136 for models M1 to M4, respectively. Thus, despite the non-significant fixed effects, $A_{(k)ti}$ reduced the intraindividual residual variation of daily PA by a small, but more than marginal portion, suggesting that PA reactivity to stressor pile-up varied significantly between individuals. Finally, checking for age effects by testing M5, we found no significant interaction of age with $A_{(6)ti}$ or S_{ti}. Again, the linear age-effect model was selected based on BIC comparison, and in this model age showed the only significant fixed effect (aside from the intercept) of $\beta = .087$ ($p \leq .001$), indicating a general age-related increase in daily PA.

Discussion

To date, research on the effects of stressor accumulation on individuals' short- and long-term physical and psychological health has been rather limited. This is surprising given that theorists (Lazarus, 1999; Selye, 1956; Zautra, 2003) have emphasized for a long time that distinguishing the effects of acute, time-limited stressors from the effects of cumulative stressor burden is essential for understanding processes of stress and coping in an ecologically valid way (Aldwin, 2007; Almeida, 2005). The present study contributes to this literature by presenting a new approach for modeling the effects of stressor pile-up on individuals' daily affect measures, testing these effects on daily NA and PA, and examining whether these effects were different for younger and older adults.

Main Findings

The findings from this study contribute to the literature on coping with daily stress in four ways. First, we established a stress accumulation index which provides a time-varying continuous measure of the amount of stressor pile-up reached at the time when a hypothesized outcome, such as daily NA, is measured. Hence, the effects of stressor pile-up can be investigated as a dynamic process over time, with the predictor changing intraindividually within a continuous range. This is advantageous compared to operationalizations that simply distinguish the dichotomous states of pile-up vs. no pile-up. Also, compared to previous operationalizations (see Grzywacz & Almeida, 2008), our approach permits the incorporation of longer periods of days in the computation of the magnitude of stressor accumulation. In doing so, it seems reasonable, and is implied by the definition of the term "accumulation," that stressors should contribute more to the cumulative stress burden the closer they were encountered to the day of interest. The stress

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accumulation index presented in this study applies this rationale in that it sums up the severity of daily stressors and assigns weights according to the stressors' temporal proximity to the time point at which the state of pile-up is assessed.

Second, using the stress accumulation index, we found in mixed models of daily NA a significant effect of stressor accumulation above and beyond the effect of concurrent daily stress. That is, the more stressors piled up prior to a given study day, the more NA was experienced on that day. This finding was consistent with findings reported by Grzywacz and Almeida (2008) who examined the effects of stressor pile-up on adults' inclination to engage in binge drinking. Stressor accumulation accounted for about 10%, and together stressor pile-up and daily stress accounted for about one third of the intraindividual variance in individuals' daily NA.

Moreover, the effects of concurrent daily stress and stressor pile-up on individuals' daily NA were additive in nature. That is, the stress accumulated until a given study day did not moderate individuals' reactivity to the stress they experienced on that day, but had an independent effect on their NA. Although our analytical technique was different from the approach chosen by Bolger et al. (1989), this finding was consistent with their results which also failed to support a multiplicative effect of (same-day) stress accumulation in a sample of 166 married couples. Thus, we conclude that, in general, there is no strong effect of stressor pile-up amplifying the susceptibility to concurrent daily stress. It may be that such an effect does not show up except under extreme pile-up conditions which overburden the individual so that additional stress cannot be regulated any more. Alternatively, it seems reasonable to speculate whether the effects of stress accumulation and concurrent stress may only be multiplicative in populations who suffer from severe chronic stress burden, such as long-term caregivers for individuals with a serious illness. We suggest that these are fruitful questions to pursue in order to gain a clearer understanding of the role of stressor pile-up in the overall stress process and with regard to health and well-being.

Third, our findings indicated a moderating effect of age on daily NA reactivity to stressor pile-up. That is, a higher age was associated with a smaller increase in daily NA due to the stressor pile-up. Although this effect was small in terms of effect size (i.e., about 5% of the between-person variation of NA reactivity), it seems noteworthy nevertheless. Taking into account (a) that emotion regulation is influenced by a number of biological, psychological, and social conditions, with some of these only weakly related to age, and (b) that the age-related processes that impact emotion regulation also vary a great deal interindividually, it can be concluded that in cross-sectional analyses a large amount of age-related variability should not be expected. Rather, if this small age-related variability of NA reactivity to stressor accumulation found in our study is due to age-related within-person *changes*, it suggests that such change across the adult lifespan is crucial.

More importantly, our findings showed a negative age difference in NA reactivity to stressor accumulation, but no age difference in terms of NA reactivity to concurrent daily stress. This result is consistent with our reasoning that it is theoretically and empirically meaningful to distinguish the immediate activation of the NA system caused by acute stress from the affect regulation efforts that unfold in the aftermath of a series of stressful experiences. The

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argument that increasing age may be associated with less reactivity to stressors due to increased emotion regulation skills (e.g., Hay & Diehl, 2011; John & Gross, 2004) may apply particularly to the effects of stressor pile-up, but not to reactivity solicited by an acute stressor. Of course, this conclusion is currently only based on the findings from our study and more research is needed to confirm these results. However, as we already argued above, this result is consistent with the SAVI model proposed by Charles (2010). Following the SAVI rationale with respect to stressor pile-up, individuals may have developed skills to distance themselves from *past* stressful experiences that had accumulated across the previous days (e.g., by reinterpreting these in a more positive way), whereas immediate affective reactivity to the stressors encountered at the same day cannot benefit from these regulation strategies, because they take time to work effectively.

Fourth, we found, in general, no PA reactivity to daily stressors. This was the case for concurrent daily stressors as well as for stressor pile-up. Noticing however that the mixed models on daily PA also revealed significant random slope effects for daily stressors and stressor accumulation, the findings support the reasoning that daily PA may be linked to stressful events in more complex and interindividually diverse ways. That is, some stressors may interfere with the generation of PA in some individuals, but not so in others; and some stressors may in some individuals even promote activities that generate PA.

Additional Findings

Two other results from our study are noteworthy. First, we found that daily NA data were highly skewed and did not meet the normality assumption. Therefore, we chose a more appropriate mixed model response distribution and estimated GLMM based on the gamma distribution. This method yielded results in terms of significant effects and R^2s that were similar to those obtained under the normality assumption, though the latter yielded poorer model fit. Thus, although model misspecification due to non-normal response data may have increased the residuals, those model parameters relevant with respect to our study objectives seemed not crucially affected (see also McCulloch & Neuhaus, 2011). This finding is quite reassuring with regard to many studies on daily NA reactivity which have used mixed model analyses based on the normality assumption (e.g., Hay & Diehl, 2010; Mroczek & Almeida, 2004; Röcke et al., 2009; Sliwinski et al., 2009; Uchino et al., 2006). The fact that our findings regarding the effect of concurrent daily stress on NA would not have differed in any significant way if we had tested conventional normality-based mixed models suggests that the normality-based estimates of daily stress effects in other studies were not biased in any major way due to potential NA non-normality.

Second, based on simple comparisons of the R^2s obtained for several choices of the accumulation period k, we found that stressor pile-up was appropriately modeled over a period of 6 days. To the extent that this time frame permits the inclusion of stressors that occur regularly on certain weekdays in adults' lives, it seems also suited for future studies of stressor pile-up effects. However, noticing the R^2 values shown in Table 3, the six-day accumulation period should not be carved in stone. In particular, in studies based on daily data across shorter observation periods, a shorter accumulation period may be chosen to avoid loss of too many of the early study days for modelling the within-person covariance of

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the stressor pile-up with the study outcome. Moreover, our findings suggest that accumulation periods much larger than 6 days may not be needed to improve the predictability of $A_{(k)ti}$ for daily NA. For future studies of reactivity to stressor pile-up, we propose that investigators determine on a case by case basis which pile-up time k is best suited to optimally predict the study outcome of interest.

Limitations and Caveats

Several limitations of the present study need to be acknowledged. First, we used in our operationalization of stressor pile-up a linear time-only decay function to weight the effect of past stressful experiences on daily affect. However, it is reasonable to assume that these effects may vary due to the effects of other variables than time. For instance, different types of stressors may differ in terms of their effects across days, such that a time-decay function moderated by stressor type may better predict daily affect measures. Moreover, interindividual difference variables related to physiology and health, personality, and social resources may promote varying susceptibilities to different stressor types. For instance, getting quickly over a health-related stressor may be obstructed by hypochondriac concerns, such that for individuals with high health-related anxiety health stressors become more relevant than others in terms of pile-up. Overall, interindividual difference characteristics may moderate the relevance of stressful experiences within the process of stressor accumulation, hence promoting interindividual differences in the pile-up dynamics. Although we are aware of these general issues, we regard the time-only decay function implied in Equation 1 as a reasonable starting point to gain better insight into stressor pile-up processes. We also believe that this equation can serve as foundation for further refinements that examine the inclusion of theoretically and practically relevant moderator variables.

Second, the time-related linearity of the weights given to the stress values in the pile-up function also deserves consideration. Note again that we also examined the effectiveness of a quadratically time-related weighting, as well as a non-weighted computation of the accumulation index. Both of these solutions performed slightly worse than the linear weighting. However, this does not rule out that another nonlinear function could outperform the linear time decay in modeling the effects of stressor pile-up. Thus, further refinements of the function denoted in Equation 1 may also comprise the search for a non-linear time-related weighting of the daily stress values that improves the performance of the accumulation index in predicting daily affect or other outcomes.

Third, with regard to the estimates in M1 to M5 for the effects of daily stress and stressor accumulation a note of caution is in order concerning missingness in S_{ti} and more specifically in $A_{(k)ti}$. If individuals did not have S_{ti} measures on some study days, this resulted in a larger number of missing values in $A_{(k)ti}$. For $k = 6$ a missing value of S_{ti} on a given study day t implies missingness of the stress accumulation index at the next 6 study days following day t, (as S_{ti} is included in the computation of $A_{(6)t+1\cdot i}$ to $A_{(6)t+6\cdot i}$). Thus, if missingness in S_{ti} occurs not at random, this may particularly bias the estimation of the accumulation effects: If high stress experienced on a given day or piled-up across several days causes participants to miss a daily stress interview, individual peaks of stressor

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accumulation and/or the stress accumulation values on days after such peaks (at periods of recovery) may be missed. This means a "cut off" of a crucial part of the accumulation variation which may co-vary with daily affect, such that the share of NA (or PA) variance explained by stressor accumulation could be underestimated. Also, a scenario could be considered that might overestimate the portion of variance accounted for by $A_{(k)ti}$.[5]

Fourth, we also acknowledge that the severity ratings that were provided by the study participants perhaps underestimated the severity of the stress at the time of actual occurrence. This may have been due to the fact that the daily stress interviews were conducted roughly in 24-hour intervals and usually later in the day. This design inevitably resulted in a time lag between stressor occurrence and the report of the incidence, and we cannot rule out that a certain degree of "cooling down" had occurred during that time, which may have resulted in consistently lower severity ratings. Additional work is needed to address this issue in a systematic and conclusive way.

Finally, a general issue in sampling older adults deserves particular attention when it comes to studies of daily stress, namely the possibility of sampling bias in favor of healthy and socio-economically advantaged individuals. Such individuals may not only have lower exposure to daily stressors, but they may also have better resources and skills for emotion regulation. Thus, the age advantage in our study in terms of reactivity to stressor pile-up may reflect positive selectivity in the older study participants rather than effects of age. Although this argument is, in principle, a reasonable one, we do not believe that it applies to our study because unlike other studies (e.g., Almeida & Horn, 2004, Mroczek & Almeida, 2004; Stawski et al., 2008) we failed to find any age differences in exposure to daily stressors. That is, despite strong empirical and theoretical reasons (e.g., Charles, 2010) suggesting that older individuals should be exposed to fewer daily stressors than younger persons, we found these groups equally "stressed" in our study. This at least speaks against pronounced selectivity of older adults living under particular low stress conditions in our sample.

Overall Conclusion

We believe the present study makes a valuable contribution to the literature on stress and coping. In particular, the presented operational definition of stressor pile-up allows a more dynamic assessment of these processes and their effects on relevant outcomes than other operationalizations currently available in the literature. Furthermore, our findings based on data from a 30-day diary study suggest that this approach holds promise to further our understanding of the time-related dynamics of stress and coping, as well as the lifespan development of resilience in terms of coping with daily stressors. Thus, we view the present study and the proposed operational definitions of stress accumulation as a starting point for further refinements and extensions. These may particularly concern conditions and predictors of interindividual differences in the reactivity to stressor pile-up.

Acknowledgments

This research was supported by grant R01 AG21147 from the National Institute on Aging.

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Table 1

Example Computation of Stressor Pile-Up Across 6-Day-Periods Based on Daily Stressor Severity Ratings and on Daily Numbers of Stressors

	Stressor Severity		Number of Stressors	
Study day *t*	**Severity at day *t* (S_{ti})**	**Pile-up ($A_{(6)ti}$)**	**Stressors at day *t* (S_{ti})**	**Pile-up ($A_{(6)ti}$)**
1	2	*n*	1	*n*
2	2	*n*	1	*n*
3	4	*n*	2	*n*
4	2	*n*	1	*n*
5	0	*n*	0	*n*
6	2	*n*	1	*n*
7	0	1.810	0	.905
8	0	1.238	0	.619
9	4	.762	2	.381
10	1	1.524	1	.762
11	0	1.429	0	.857
12	0	1.095	0	.667
13	1	.762	1	.476
14	0	.810	0	.619
15	0	.524	0	.429
16	2	.238	1	.238
17	2	.714	1	.429
18	2	1.143	1	.619
19	2	1.476	1	.762
20	1	1.714	1	.857
21	7	1.619	3	.952
22	0	3.190	0	1.571
23	2	2.429	1	1.190
24	0	2.333	0	1.143
25	1	1.667	1	.810
26	2	1.381	1	.810
27	0	1.429	0	.810
28	0	.857	0	.524
29	2	.619	1	.381
30	0	.952	0	.524

Note. Data from 1 selected respondent; *n* = Stress pile-up not computed.

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Table 2

Generalized Linear Mixed Models of Daily Negative and Positive Affect.

Model	GLMM linear predictor
M1:	$g(\mu_{ti}) = \beta_0 + \beta_1 SP_i + \beta_2 A_{(k)ti} + u_{0i} + u_{2i} A_{(k)ti}$
M2:	$g(\mu_{ti}) = \beta_0 + \beta_1 SP_i + \beta_3 S_{ti} + u_{0i} + u_{3i} S_{ti}$
M3:	$g(\mu_{ti}) = \beta_0 + \beta_1 SP_i + \beta_2 A_{(k)ti} + \beta_3 S_{ti} + u_{0i} + u_{2i} A_{(k)ti} + u_{3i} S_{ti}$
M4:	$g(\mu_{ti}) = \beta_0 + \beta_1 SP_i + \beta_2 A_{(k)ti} + \beta_3 S_{ti} + \beta_4 A_{(k)ti} \times S_{ti} + u_{0i} + u_{2i} A_{(k)ti} + u_{3i} S_{ti}$
M5:	$g(\mu_{ti}) = \beta_0 + \beta_1 SP_i + \beta_2 A_{(k)ti} + \beta_3 S_{ti} + u_{0i} + u_{2i} A_{(k)ti} + u_{3i} S_{ti} + \sum_{j=1}^{m} (\beta_{3+j} age_i^j + \beta_{3+m+j} A_{(k)ti} \times age_i^j + \beta_{3+2m+j} S_{ti} \times age_i^j)$

Note. μ_{ti} = predicted value (marginal mean) of the response variable (daily NA or PA) of individual i at study day t; SP_i = mean of stressor severity (or numbers of stressors) of individual i; $A_{(k)ti}$ = k-day stressor accumulation; S_{ti} = stressor severity (or numbers of stressors); $A_{(k)ti} \times S_{ti}$ = interaction of stressor accumulation with daily stressor severity (or number of daily stressors); age_i = age of participant; β's denote fixed effects; u's denote the random effects (indices, respectively).

M5 specified for m = 1, 2, 3; i.e. linear, quadratic, cubic age effects, respectively.

g(·) = GLMM link function, i.e. gamma or identity (assuming normality) (for details, see SAS Institute Inc., 2009, pp. 2080-2430).

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Table 3

Intraindividual Variation of Daily Negative Affect Explained by the Stress Accumulation Index $A_{(k)ti}$ based on Different Choices of the Accumulation Period k.

k	1	2	3	4	5	6	7	8	9
Stressor Severity R^2	.050	.073	.095	.096	.095	.105	.087	.083	.095
Number of Stressors R^2	.043	.064	.088	.091	.093	.104	.090	.091	.097

Note. $R^2 = 1 - \sigma^2/\sigma_0^2$; where σ^2 and σ_0^2 denote variance of residuals on the data scale of M1 and random intercept null model, respectively, estimated with SAS PROC GLIMMIX by use of the gamma distribution link function (SAS Institute Inc., 2009).

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Table 4

Descriptive Statistics: Between-Person Means, Standard Deviations, and Correlations, and Correlations across Persons and Study Days.

			Correlations				
	M	***SD***	**NA**	**PA**	**NStr**	**SevStr**	**$A_{(6)ti}$**
NA	13.17	3.08		−.10***	.32***	.38***	.29***
PA	26.90	6.70	−.12*		.02	.03*	.05***
NStr	0.70	.47	.31***	.06		.94***	.46***
SevStr	1.47	1.03	.36***	.05	.97***		.47***
$A_{(6)ti}$	1.47	1.03	.39***	.05	.96***	.97***	
Age	48.58	19.85	−.32***	.25***	−.03	−.03	−.02

Note. Means, standard deviations, and correlations below the main diagonal denote between-person statistics (i.e. computed across individuals from individual means of NA, PA, NStr, SevStr, and $A_{(6)ti}$). Correlations above the main diagonal were computed across individuals and study days.

NA = negative affect; PA = positive affect; NStr = number of stressors; SevStr = daily stressor severity; $A_{(6)ti}$ = 6-day stress accumulation index.

* *p* q .05;

*** *p* q .001.

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Table 5

Results from Generalized Linear Mixed Model Analyses of Daily Negative Affect Predicted by 6-Day Stress Accumulation $A_{(6)ti}$, Daily Stress Severity S_{ti}, and Within-Person Mean of Stress Severity SP_i (Models M1-M4).

	Model 1		Model 2		Model 3		Model 4	
	Estimate	**SE**	**Estimate**	**SE**	**Estimate**	**SE**	**Estimate**	**SE**
Fixed effects:								
Intercept	2.520	.011**	2.529	.010**	2.520	.010**	2.521	.010**
$A_{(6)ti}$	.017	.006*			.021	.005**	.021	.006**
S_{ti}			.049	.003**	.053	.003**	.053	.003**
SP_i					.024	.012*	.025	.012*
$A_{(6)ti} \times S_{ti}$							.000	.002
Random variances:								
Intercept	.027	.003**	.026	.002**	.022	.002**	.022	.002**
$A_{(6)ti}$	.003	.001**			.003	.001**	.003	.001**
S_{ti}			.001	.000**	.001	.000**	.001	.000**
R^2	.105		.204		.327		.326	

Note. Models estimated with SAS PROC GLIMMIX by use of the gamma distribution link function (SAS Institute Inc., 2009).

* $p \leq .05$;

** $p \leq .001$

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Table 6

Results from General Linear Mixed Model Analyses of Age-Effects on Negative Affect Reactivity to Stress Accumulation $A_{(6)ti}$ and Daily Stress Severity S_{ti} (Model M5).

	Estimate	SE
Fixed effects:		
Intercept	2.5234	.0092***
$A_{(6)ti}$	.0221	.0056***
S_{ti}	.0528	.0027***
SP_i	.0220	.0111*
Age_i	−.0032	.0005***
$Age_i \times A_{(6)ti}$	−.0008	.0003**
$Age_i \times S_{ti}$	−.0001	.0001
Random variances:		
Intercept	.0188	.0019**
$A_{(6)ti}$	.0028	.0006**
S_{ti}	.0009	.0002**
R^2	.326	

Note. Model estimated with SAS PROC GLIMMIX by use of the gamma distribution link function (SAS Institute Inc., 2009).

*$p \leq .05$;

**$p \leq .01$;

***$p \leq .001$