Emotional Reactivity and Mortality: Longitudinal Findings From the VA Normative Aging Study

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Objectives. Evidence suggests a predictive association between emotion and mortality risk. However, no study has examined dynamic aspects of emotion in relation to mortality. This study used an index of emotional reactivity, defined as changes in positive or negative affect in response to daily stressors, to predict 10-year survival.

Methods. An 8-day daily diary study was conducted in 2002 on 181 men aged 58–88. Multilevel models were employed to estimate emotional reactivity coefficients, which were subsequently entered into a Cox proportional hazards model to predict mortality.

Results. Results indicated that positive emotional reactivity, that is, greater decreases in positive affect in response to daily stressors, increased mortality risk. Negative emotional reactivity did not predict mortality.

Discussion. Findings highlight the potential importance of dynamic aspects of positive affect in prediction of physical health outcomes such as mortality.

Key Words: Emotion—Emotion Regulation—Longevity—Personality—Quantitative Methods.

Psychological characteristics have emerged as important predictors of mortality and other health outcomes among older adults (Mroczek, Spiro, & Turiano, 2009; Turiano et al., 2012). Yet, only a small number of studies have considered emotion as predictors of mortality (e.g., Carstensen et al., 2010; Ong, Mroczek, & Ruffin, 2011; Wilson, Bienes, Mendes de Leon, Evans, & Bennett, 2003), and none have considered dynamic aspects of emotion such as emotional reactivity. Emotion in older adulthood is a topic of great importance (Charles, 2010; Charles, Reynolds, & Gatz, 2001; Steptoe & Wardle, 2011), but little is known about the relationship between dynamic aspects of emotion and critical health outcomes, such as all-cause mortality.

Negative and positive affect are not fixed traits but are moving targets. They go up and down on momentary and daily bases and are often yoked to external events, particularly daily stressors. One way of capturing the (within-person) stress–affect relationship is through emotional reactivity (e.g., Almeida, 2005; Bolger & Schilling, 1991; Mroczek & Almeida, 2004; Suls, Green, & Hillis, 1998). In this paper, we used emotional reactivity for both positive and negative affect (as well as mean level of each) to predict all-cause mortality in a sample of older adults.

Affect and Mortality

Both negative and positive affect are associated with physical health (Chida & Steptoe, 2008; Gallo & Matthews, 2003; Ong, 2010). A great deal of evidence connects negative affect (NA) with various physical health indicators, such as all-cause mortality (Wilson et al., 2003), coronary events (Rozanski, Blumenthal, & Kaplan, 1999), and activation of the hypothalamic-pituitary-adrenal (HPA) axis, which itself is associated with poorer physical health outcomes (Adam, Hawkey, Kudielka, & Caccioppo, 2006; Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Piazza, Almeida, Dmitrieva, & Klein, 2010). Positive affect (PA) has been linked to lower heart rate, well-regulated levels of cortisol, decreased pain, and better immune function (Marsland, Pressman, & Cohen, 2007; Ong, 2010; Steptoe, Wardle, & Marmot, 2005; Zautra et al., 2005; Zautra, Johnson, & Davis, 2005), and as noted earlier, higher positive emotional experience is associated with lower mortality risk (Carstensen et al., 2010; Ong et al., 2011; Steptoe & Wardle, 2011). Moreover, PA may play the
crucial role of blunting the ill physical health effects of NA. Zautra’s “dynamic model of affect” (Zautra, Smith, Affleck, & Tennen, 2001) theorizes that PA can mitigate the potential detrimental effects of NA on physical health. The dynamic model of affect posits that PA plays an important role in preserving well-being when stressors occur by minimizing NA, via a strengthened inverse association between PA and NA. The model posits that physical health resilience flows from the capacity to maintain PA in the face of stressors. Our use of within-person reactivity coefficients (e.g., smaller decreases in PA on stress days) aligns with Zautra coworkers’ dynamic model of affect.

Emotional Reactivity and Mortality

Most studies of emotion and health utilize static or fixed measures of PA and NA. However, affect fluctuates over time and is responsive to occurrences in everyday life such as daily stressors (Bolger & Schilling, 1991). Such emotional reactivity gauges individual variation in the “preparedness to respond [to stimuli] with stronger negative affect” (Larsen & Ketelaar, 1991, p. 138). It is well established that some people experience greater increases in NA on stressor days than other people (Almeida, Piazza, Stawski, & Klein, 2011; Bolger & Schilling, 1991; Mroczek & Almeida, 2004). This NA-based emotional reactivity captures wide individual differences in people’s responsiveness to stress. Emotional reactivity can also refer to decreases in PA in response to a stimulus such as stress (Mroczek, Spiro, Griffin, & Neupert, 2006). Such PA emotional reactivity gauges decreases in PA when stressors occur. Much less is known about PA emotional reactivity. However, PA emotional reactivity may be important as the inability to maintain positive mood in the face of stressors may reflect a lack of emotional resources to cope with stress (Almeida, 2005; Ong, Mroczek, & Riffin, 2011).

Theoretically, why should NA or PA reactivity be associated with physical health outcomes such as mortality risk? We have argued that emotional reactivity (either greater increases in NA or greater decreases in PA) should predict mortality risk as stronger and more repeated emotional responses to stress may overengage HPA axis functioning (Mroczek et al., 2006). Indeed, Repetti, Taylor, and Seeman (2002) and Cacioppo (1998), among others, have theorized that greater responsiveness to stress is likely to have adverse effects on downstream physical health outcomes, via the mechanism of repeated HPA axis activation. More generally, and consistent with the earlier reasoning, emotional reactivity indexes how people react to aversive or stressful events (Charles et al., 2013). Low reactivity implies “taking things in stride” (Piazza et al., 2013) and not overreacting, thereby preventing overactivation of certain physiological processes (e.g., HPA axis function). In fact, a key goal of certain therapeutic techniques such as mindfulness-based stress reduction (MBSR; Kabat-Zinn, 1998; Speca et al., 2000) is to lessen people’s responsiveness to stressors. Indeed, techniques such as MBSR that emphasize lessened reactivity to stressors have been linked to improved immune function (Davidson et al., 2003; Witek-Janusek et al., 2008) and to increased activation in brain regions that promote PA (Davidson et al., 2003). Stronger reactions to stress, whether larger increases in NA or larger decreases in PA, may damage health either through repeated physiological activation (e.g., HPA axis overactivation) as theorized earlier, or via other mechanisms such as eliciting unhealthy behaviors when stressors occur (excessive drinking, stress eating, smoking). Our theoretical assertion (Mroczek et al., 2006) that reactivity should be associated with adverse health outcomes is consistent with recent reports that directly link greater reactivity to both greater general affective distress and a higher risk of having an affective disorder (Charles et al., 2013), worse sleep-related outcomes (Ong et al., in press), and higher likelihood of having a chronic physical disorder 10 years later (Piazza et al., 2013).

Very few empirical investigations have employed dynamic variables such as emotional reactivity as predictors of mortality. At least two prior investigations have demonstrated that dynamic phenomena in certain psychological characteristics can predict mortality. First, greater variability in perceived control over a period of several weeks among older adults predicted mortality 5 years later (Eizenman, Nesselroade, Featherman, & Rowe, 1997). Second, long-term (10 years) increases in the personality trait neuroticism were associated with increases in mortality risk (Mroczek & Spiro, 2007). These two studies show that dynamic aspects of characteristics that are related to positive and negative affect (perceived control and neuroticism) are potentially useful predictors of mortality.

In this study, we similarly used an indicator of a dynamic process, emotional reactivity to daily stressors, to predict mortality. We began by first testing the effect of (mean) daily positive and negative affect as predictors of all-cause mortality for more than 10 years of follow-up. Although this study was primarily focused on emotional reactivity as predictors of mortality, we first tested the effect of mean levels of PA and NA, per theoretical models discussed earlier that indicate level of affect should directly affect health—specifically the dynamic model of affect (Zautra et al., 2001) but also the broad and build model, which has recently been linked to physical health (Fredrickson, 2001; Kok et al., 2013). We then tested the predictive effect of emotional reactivity. We expected mean PA to be associated with lower mortality risk, and we expected three variables to predict higher mortality risk: mean NA, decreases in PA in response to stressors (PA emotional reactivity), and increases in NA in response to stressor (NA emotional reactivity).

Method

Sample

Participants for the analyses were drawn from the Veterans Affairs (VA) Normative Aging Study (NAS), a longitudinal study of more than 2,000 men that began in the 1960s as an investigation of normal aging processes.
in men (see Spiro & Bossé, 2001 for additional information). Starting in August 2002, a subsample of the NAS was recruited for a “measurement burst”—an 8-day daily diary study on daily stressors, physical symptoms, positive and negative affect, memory failures, pain, and social support (see Neupert, Almeida, Mroczek, & Spiro, 2006 for additional information). Between August 2002 and April 2003, more than 300 NAS respondents were contacted and invited to participate in the measurement burst. Of these, more than 200 agreed, and 181 men returned usable surveys. Almost all participants (99%) completed all 8 days of the study, yielding a total of 1,439 person-days of data. Respondents who completed the diary did not differ significantly from those who refused or from NAS participants in a 2001 survey who were not included in the diary subsample in terms of age (diary $M = 73.27$), self-rated health (diary $M = 2.58$), life event stressors (diary $M = 3.36$), or neuroticism (diary $M = 2.21$).

**Mortality**

Vital status of NAS participants is monitored by periodic mailings, and when notified, death certificates are obtained and coded for cause of death. Of the 181 participants, 35 died (19%) during the 10 years (ending in late 2012) following the 2002–2003 diary study period. Within the daily study sample, we compared decedents to survivors on a number of variables. Decedents were older, 76.5 versus 72.3 years, $t(179) = 3.68, p < .001$, and had worse self-reported current health, 2.4 versus 1.2, $t(179) = 3.72, p < .001$, but were not significantly different in past-year self-reported health, health relative to age peers, life satisfaction, marital status, percent working, percent retired, smoking status, or drinking status (results not shown).

**Procedure**

Instructions indicating when to complete the diary (approximately half hour before going to bed) and when to return the surveys (when all eight were completed), a preaddressed postage-paid envelope, and a form to be completed for payment were sent to each participant. For eight consecutive evenings, participants completed short semistructured questionnaires about their daily experiences (e.g., stressors, physical symptoms, positive and negative affect, memory failures, pain, and social support). At the conclusion of the 8-day period, participants returned the completed diaries; if they completed five or more of the 8 study days, they received $15; if they completed 4 or fewer days, they received $10.

**Measures**

**Daily stressors.**—Daily stressors were assessed through the Daily Inventory of Stressful Events (Almeida, Stawski, & Cichy, 2011; Almeida, Wethington, & Kessler, 2002), an instrument that assesses negative daily stressors. Participants answered questions regarding arguments, potential arguments, stressors that occurred at work/volunteer settings and home, network stressors (stressors that occurred to a network of friends and family), health-related events, and other stressors (stressors that may not have fit into the other categories) each day. For each person, for each day, we computed the sum of all stressors reported ($M = 0.85, SD = 1.10$, range $= 0–7$). The vast majority of stressor days were single-stressor days; very few days contained more than one stressor. Thus, we utilized a dichotomous daily stressor variable that simply indicated whether the participant reported at least one stressor on that day or not.

**Daily affect.**—The Positive and Negative Affect Schedule (PANAS: Watson, Clark, & Tellegen, 1988) was used to measure daily affect. The PANAS is a brief measure that consists of two 10-item mood scales. The scales contain words that describe different feelings and emotions (e.g., upset, enthusiastic), and participants were asked to indicate to what extent they felt each of the emotions on that day. Response options were 1 (very slightly or not at all) to 5 (extremely). These were summed across the 10 items to yield scores ranging from 10 to 50 for NA and PA. The PANAS scales have acceptably high internal consistency reliabilities, with alphas ranging from .84 to .90 (Watson et al., 1988; Watson & Walker, 1996).

**Daily physical symptoms.**—Daily physical symptoms were measured using a shortened version of Larsen and Kasimatis’s (1991) physical symptom checklist. The 16-item scale assessed symptoms such as aches/pain (headaches, backaches, and muscle soreness), gastrointestinal symptoms (poor appetite, nausea/upset stomach, constipation/diarrhea), chest pain or dizziness (symptoms often associated with cardiovascular functioning), and upper respiratory infection symptoms (sore throat, runny nose, congestion). Two additional items (cold/flu symptoms and joint pain) were also included in the checklist. Each day the respondents indicated whether they experienced each symptom over the past 24 hr.

**Bodily pain.**—Respondents also answered a single-item question about how much pain they experienced in the past 24 hr on a scale of 1 (no pain) to 100 (a lot of pain). We included it as covariate because recent work has indicated daily pain is an important correlate of daily affect (Almeida et al., 2011; Zautra et al., 2005).

**Neuroticism and extraversion.**—Neuroticism and extraversion are well-known predictors of PA and NA, and neuroticism is a well-established predictor of mortality as well. Thus, we controlled for both traits using measures obtained.
from a separate mail survey conducted in 2001. We used the Eysenck Personality Questionnaire-Q (EPI-Q) (Floderus, 1974), a short version of the Eysenck Personality Inventory (Eysenck & Eysenck, 1968). Both neuroticism and extraversion are assessed by 9 dichotomous items that yield scores ranging from 0 (low) to 9 (high). The EPI-Q has been used in Swedish twin studies (Floderus-Myrked, Pedersen, & Rasmussen, 1980), and in prior studies of the NAS, it has demonstrated good construct validity (Levenson, Aldwin, Bossé & Spiro, 1988; Mroczek & Spiro, 2003; Mroczek, Spiro, Aldwin, Ozer, & Bossé, 1993).

Analytic Strategy

Proportional hazards models were used to predict mortality status (Cox, 1972). For all predictors (except age) the odds ratios reflected odds of having died for a 1-unit increase, or difference, in that predictor. Consistent with previous daily stress research, emotional reactivity was defined as the within-person association, a slope or coefficient, between the occurrence of any daily stressors (often dichotomous, as here) and positive and negative affect, respectively. More precisely, this coefficient captures a shift in affect in response to a change in the dichotomous daily stressor variable (stressor day or not).

We attempted to combine both stages of the analysis (multilevel modeling [MLM] and survival analysis) in a single, joint model in Mplus (Muthén & Muthén, 1998–2010), using a multivariate MLM to estimate the time-varying stress–affect slopes (both stressor-related increases in NA and stressor-related decreases in PA) with intercepts and reactivity slopes directly predicting survival. Others have employed such joint estimation but usually with simpler models and larger N’s (e.g., Ghisletta, McArdle & Lindenberger, 2006; Guo & Carlin, 2004; Henderson, Diggle & Dobson, 2000; McArdle, Small, Bäckman & Fratiglioni, 2005). However, for our model, the joint estimation was unsuccessful because of convergence issues, possibly due to the complexity of our model. Thus, we applied a two-stage model where survival models were run separately from MLM. In the first stage we employed multilevel models to estimate reactivity coefficients:

Level 1: \[ A_{ij} = a_{0j} + a_j \text{ Stressor Day}_{ij} + e_{ij} \] (1)

Level 2: \[ a_{0j} = \beta_{00} + \beta_{01} \text{ Person–Mean Stress}_j + u_{0j} \] (2)

\[ a_j = \beta_{10} + u_j \] (3)

At Level 1, the value \( a_{0j} \) is an intercept and reflects the affect score (positive or negative) when the predictor, the dichotomous stressor variable, is zero. In other words, it is the amount of affect on a nonstressor day. The coefficient \( a_j \) is a slope parameter reflecting the association between (dichotomous) stressor day and affect. For some people, this slope will be larger and for others smaller, or even zero. It thus indexes emotional reactivity or the extent to which affect is responsive to stress (Larsen & Ketelaar, 1991). At Level 2, \( B_{00} \) and \( B_{01} \) represent the sample average level of affect and reactivity effect, respectively. \( B_{10} \) represents person-mean frequency of stressors across the study period and is included to account for individual difference in stressor frequency, and for grand-mean centering the Level-1 stressor effect (i.e., stress reactivity slope). Additionally, \( u_{0j} \) and \( u_j \) are variances reflecting individual differences or deviations from the sample average level of affect and reactivity estimates, respectively. These deviations can be estimated for each individual and used as predictors in further analyses (a similar approach was used by Mroczek & Spiro, 2007). It is important to note that we used grand-mean centering to create the emotional reactivity slopes (Hoffman & Stawski, 2009). As such, each person’s frequency of stressor days was also included (as a Level-2, or person-level, predictor) to account for individual differences in stressor frequency. The mean of these emotional reactivity slopes was 1.69 (SD = 1.67, range = −1.89 to 9.02) for NA reactivity, and −0.18 (SD = 1.15, range = −3.87 to 9.59) for PA reactivity.

For illustration, someone with a slope, or reactivity coefficient, of 1.69 (the sample mean) for NA reactivity had a 1.69 increase in PANAS daily NA on stressor days compared with nonstressor days. Noting the range, some people had increases that were much more than that, thus capturing the individual differences in NA emotional reactivity. However, it is interesting to note that some people had negative slopes, indicating decreases in NA on stressor days. An approximation of effect size was obtained by obtaining the ratio of within-person variance from the intercept-only (baseline) model to the within-person variance from the model that included stressors. This variance was 9.8 in the baseline model and declined to 9.1 when stressors were added, indicating 7% of the within-person variation in NA was explained by stressors.

For PA reactivity, a person with a slope at the sample mean (−0.18) had a decrease of 0.18 in PA on stressor days compared with nonstressor days. Yet, again there was a considerable range around that mean, thus capturing the wide individual differences in PA reactivity. Some people had much larger decreases, as much as a 3.87-point drop in PA on stressor days. Again, similar with NA reactivity, there were some individuals with positive slopes, thus denoting increases in PA on stressor days. However, there were few of these people, as indicated by the sample mean of −0.18. As mentioned earlier, we computed the ratio of within-person variances from the intercept-only to the stressors model. The variance was 19.5 in the baseline model and declined to 18.6, indicating 5% of the within-person variation in PA was explained by stressors.

In the second stage of modeling, these MLM-derived (person-level) slopes, indexing either NA or PA reactivity,
were then entered into a proportional hazards model (survival analysis Cox, 1972) to predict mortality risk. A formal expression of that model (with only two predictors) is

\[ h(t_i) = h_0(t) e^{\beta_{\text{age}} + \beta_{\text{emotional reactivity}}} \]  

(4)

where \( h(t) \) is an individual \( i \)'s risk of dying (or hazard: \( h \)) at time \( t \) (see Singer & Willet, 2003, p. 512), \( h_0(t) \) represents the general baseline hazard function, and the terms in the exponent, \( \beta_{\text{age}} \) and \( \beta_{\text{emotional reactivity}} \), are the effects of these variables on risk of dying.

This was a novel analysis in that a dynamic aspect of affect—emotional reactivity—was utilized as a predictor of an important health outcome, mortality. Given that daily stressor-related changes in PA are, on average, decreases in PA, we multiplied the PA reactivity slopes by \(-1\) for conceptual clarity. Thus, a hazard ratio greater than 1 would indicate increased mortality risk as a function of a decrease in PA (net of control variables).

**RESULTS**

Prior to conducting survival analyses to test the main hypotheses regarding the impact of emotional reactivity on mortality, we tested whether PA had a blunting role on the effect of stressor days on NA. In MLM, we used PA, the stressor day variable, and their interaction to predict NA. The interaction was not significant, \( t(1208) = 1.29, p < .20 \), indicating that PA may not necessarily blunt the effect of stress on NA.

For the primary analyses examining mortality, proportional hazards (survival) analyses were conducted in two steps. The first used the level of PA and NA (average daily level across the 8 days of the diary study), whereas the second used the two emotional reactivity indicators to predict mortality risk.

The proportional hazards model assumes that the effect of each predictor is the same at all points in time, and we tested nonproportionality in two ways. First, we created interaction terms between each predictor and the time metric—in this case survival time—and tested whether each predicted mortality risk. None of the interactions approached statistical significance, suggesting that the proportional hazard assumption was not violated. In a second step, we estimated the martingale residuals (Lin, Wei, & Ying, 1993) based on 10,000 random simulations that compare the observed residuals against the simulated residuals for each predictor. Observation of the plots for each predictor indicated that the observed residuals and simulated residuals did not deviate markedly. In addition, the Kolmogorov–Smirnov type test did not approach statistical significance for any of the predictors. Based on these findings, there is no evidence to suggest that the proportionality assumption was violated.

**Daily Affect Predicting Mortality**

In Table 1, Model 1 represents a baseline model of covariates (i.e., age, average daily bodily pain, average number of daily physical symptoms, extraversion, and neuroticism), and Model 2 adds the effects of mean PA and NA over the 8 days. In Model 1, only age was a statistically significant predictor of mortality risk although pain was borderline for significance. A 1-year increase in age was associated with a 1.109 (10.9%) increase in likelihood of mortality. Model 2 shows that neither daily average positive or negative affect was significantly related to mortality risk. We compared Models 1 and 2 using a Likelihood Ratio Test, which indicated that the models were not significantly different from one another, \( \chi^2(2) = 0.70 \), not significant.

**Emotional Reactivity Predicting Mortality**

The second part of our analysis considered whether the indicators of PA and NA emotional reactivity were predictive of mortality. We included an additional covariate in these models, daily stressor frequency, because there is great individual variation in stressor exposure, and this may influence the degree to which some people are emotionally reactive. Some people are exposed very frequently, others much less so. We also controlled for all the aforementioned covariates: age, average daily bodily pain, average daily number of physical symptoms, extraversion, and neuroticism. As mentioned earlier, we compared Models 1 and 2 in Table 2 using a Likelihood Ratio Test, which indicated they were significantly different from one another, \( \chi^2(2) = 39.31, p < .0001 \).

Subsequently, we examined whether emotional reactivity to daily stressors was predictive of mortality. As described earlier, emotional reactivity was defined as the magnitude of the within-person coupling of having experienced any stressors on a given day and levels of positive and negative affect, respectively, on that same day. Thus, our index of reactivity quantifies the magnitude of within-person change in daily PA and NA that is associated with the experience of any daily stressors. We ran models that both included and omitted estimates of mean level of PA and NA, in addition to emotional reactivity slopes. Results were the same

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Model 1</th>
<th>Model 2</th>
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<tbody>
<tr>
<td><strong>Predictor</strong></td>
<td><strong>HR (95% CI)</strong></td>
<td><strong>HR (95% CI)</strong></td>
</tr>
<tr>
<td>Age</td>
<td>1.11** (1.03–1.19)</td>
<td>1.10* (1.02–1.18)</td>
</tr>
<tr>
<td>Pain</td>
<td>1.02 (0.99–1.05)</td>
<td>1.02* (0.99–1.04)</td>
</tr>
<tr>
<td>Physical symptoms</td>
<td>1.13 (0.84–1.52)</td>
<td>1.16 (0.86–1.57)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>1.02 (0.83–1.24)</td>
<td>0.99 (0.80–1.22)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.19 (0.97–1.47)</td>
<td>1.15 (0.91–1.44)</td>
</tr>
<tr>
<td>Negative affect</td>
<td>1.06 (0.92–1.20)</td>
<td></td>
</tr>
<tr>
<td>Positive affect</td>
<td>1.00 (0.94–1.05)</td>
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Note. HR = hazards ratio.

*p < .06, *p < .05, **p < .01.
over both, and neither PA nor NA level was significantly predictive of mortality risk. Model 2 of Table 2 shows the results of this model. Only PA emotional reactivity (daily stressor-related decreases in PA) was associated with mortality. A 1-unit difference in stressor-related decreases in PA was associated with a 2.32 (132%) increase in mortality risk (Cohen’s $d = 0.47$). However, stressor-related increases in NA (emotional reactivity) were not predictive of mortality over the follow-up.

**Discussion**

Four different emotion variables—average daily negative and positive affect and NA and PA emotional reactivity—were tested as potential predictors of mortality, for more than 10 years of follow-up. We expected all four to be significant predictors because static indicators of affect (e.g., our two daily average variables) have been linked to health outcomes (e.g., Kok et al., 2013; Wilson et al., 2003) and because dynamic aspects of affect (e.g., our two emotional reactivity variables) should theoretically predict important physical health outcomes such as mortality. However, only PA emotional reactivity was predictive of mortality. Specifically, larger decreases in daily PA in response to daily stressors were associated with more than a doubling in mortality risk over almost a decade of follow-up.

Given that participants were generally healthy at the outset and given that we controlled for physical symptoms and pain we can rule out that poor physical health or impending death was the impetus for decreases in daily PA in response to negative stressors. Moreover, these daily estimates of affect precede (by years in many cases) the event of mortality. Indeed, this is the first study to our knowledge that has demonstrated a prospective effect of a positive affect–related emotional reactivity indicator on mortality.

Why would stressor-related decreases in PA elevate mortality risk but not stressor-related increases in NA? We speculated earlier that Zautra coworkers’s (2001) dynamic affect theory may provide an explanation. If PA mitigates NA during periods of stress, then those individuals who experience the greatest decreases in PA when stressors occur may have a lessened ability to counteract the negative psychological effects of stress. At least among older adults, the experience of increased NA in response to stress may not be as important as the potentially crucial countervailing force of PA. However, as noted earlier, we directly tested this in a preliminary analysis by entering PA, stressors, and their interaction to predict NA. The interaction was not significant, meaning that PA may not necessarily have a blunting effect. Stressor-related decreases in PA may be damaging to health for reasons having nothing to do with NA. It may be that maintaining PA in the face of stressors has a salutatory effect on health through other mechanisms, such as sustaining better health behaviors or seeking out social support. This finding suggests that maintaining daily PA even when stressed may be good in and of itself, irrespective of what it does to NA.

Finally, why did levels of NA and PA not predict mortality? Reactivity (PA or NA) is a process and taps into the extent to which emotions are subject to external events. This may be more detrimental for health, at least among older adults, than simple level of NA or PA. Dynamic indices that reflect the extent that a person is “pulled” emotionally by stressors may have a larger effect in older adulthood than level of an emotion. Future research needs to clarify this, however.

**Limitations**

Generalizability is limited by a number of factors. The sample on which the daily diary study was conducted (the VA NAS) is all male participants. This is in part because when the study was founded in the 1960s, the VA focused on veterans, who were largely men. The NAS was also established at a time where medical studies often excluded women. Although we have recruited NAS wives to be part of the study since 2001, we did not have mortality information on them. Additionally, the sample is less representative than we would wish in other ways. It is largely white, and does not include many Hispanics. Finally, although nearly 20% of the diary subsample were decedents at the present time, it is possible that our results may change as more participants die in the future. It will be important for us to revisit these data when more of the participants have died, to determine if effects become stronger or weaker.

**Conclusion**

Our findings show that dynamic processes like emotional reactivity that take place over “micro” periods such as days can predict global or “macro” outcomes such as mortality. Second, they highlight the potentially important role of dynamic PA in health, illness, and longevity. Future studies should continue to clarify the role of positive emotions, especially their dynamic features, on health outcomes. However, we must reiterate that in this study, nondynamic

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**Table 2. Proportional Hazards Regression Results for Exposure and Emotional Reactivity to Daily Stressors Predicting Mortality**

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<tr>
<td>Age</td>
<td>1.10* (1.02–1.19)</td>
<td>1.10** (1.03–1.20)</td>
</tr>
<tr>
<td>Pain</td>
<td>1.02* (1.00–1.04)</td>
<td>1.03 (0.99–1.05)</td>
</tr>
<tr>
<td>Physical Symptoms</td>
<td>1.12 (0.83–1.50)</td>
<td>1.09 (0.69–1.78)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>0.99 (0.80–1.21)</td>
<td>0.80 (0.60–1.12)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>1.15 (0.92–1.44)</td>
<td>0.96 (0.70–1.52)</td>
</tr>
<tr>
<td>Daily stress frequency</td>
<td>1.82 (0.49–6.81)</td>
<td>2.31 (0.02–6.00)</td>
</tr>
<tr>
<td>Negative affect</td>
<td>0.84 (0.27–2.60)</td>
<td></td>
</tr>
<tr>
<td>Positive affect</td>
<td>0.96 (0.89–1.02)</td>
<td></td>
</tr>
<tr>
<td>Emotional reactivity (NA)</td>
<td>1.88 (0.13–28.10)</td>
<td></td>
</tr>
<tr>
<td>Emotional reactivity (PA)</td>
<td>2.32** (1.32–4.00)</td>
<td></td>
</tr>
</tbody>
</table>

*Note. HR = hazards ratio.

$p = .06$, $^* p < .05$, $^{**} p < .01$. 

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...
PA (daily average PA) did not predict mortality. Given that other studies have found an effect of nondynamic PA on health outcomes, future work should attempt to reconcile these findings. For example, perhaps the fact that this study included only men may account for this discrepancy.

Our work also adds to the growing body of evidence, demonstrating that many psychological variables are, in and of themselves, key predictors of illness and mortality and are not just a by-product of deteriorating physical health. However, the present findings are also unique in that they show a psychological process—emotional reactivity—predicts mortality. Our initial step using multilevel models allowed us to capture an emotional process. By obtaining within-person associations that quantify in a single value an estimate of emotional reactivity, we were able to then use those values to predict an important health outcome, mortality.

This finding is theoretically important because it demonstrates the long-term threat posed by emotional reactivity. As described earlier, many theoretical models of stress and health argue that the greater such reactivity, the greater the physical health cost, perhaps due to excessive HPA axis activations (Cacioppo, 1998; Kiecolt-Glaser et al., 2002). In essence, emotional reactivity, whether PA or NA based, is an indicator of the fight-or-flight response (Gray, 1981, 1994), which itself is deeply intertwined with HPA axis functioning. Chronic decreases in PA may be accompanied by overactivation of the HPA axis, therefore creating eventual damage downstream via the by-products of HPA activation (e.g., cortisol, inflammatory cytokines, alpha amylase; Hellhammer, Wüst & Kudielka, 2009; Kiecolt-Glaser et al., 2002; McEwen, 1998). Perhaps the most likely pathway from stressor-related decreases in PA and mortality is through HPA functioning, but there may be other plausible pathways, both physiological and behavioral, for example, health behaviors.

Regardless of the explanatory pathway, this study shows that a dynamic emotional process, in contrast to a static emotion measurement, predicts mortality. It hints that a potentially fruitful but understudied aspect of emotional processing is important for health and longevity. How much one reacts emotionally to stressful events may be as important as how one generally feels in influencing physical health and longevity.

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**References**


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