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Perceived Partner Responsiveness, Daily Negative Affect Reactivity, and All-Cause Mortality: A 20-Year Longitudinal Study

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Abstract

Objective: This study tested longitudinal associations between absolute levels of perceived partner responsiveness (PPR; how much people feel their romantic partners understand, care for, and appreciate them), daily negative affect (NA) reactivity and positive affect (PA) reactivity, and all-cause mortality in a sample of 1,208 adults over three waves of data collection spanning 20 years. We also tested whether longitudinal *changes* in PPR predicted mortality via affect reactivity.

Methods: Data were taken from the National Survey of Midlife Development in the United States (MIDUS). PPR was assessed at Waves 1 and 2, affect reactivity to stressors was assessed by daily diary reports at Wave 2, and mortality status was obtained at Wave 3.

Results: Mediation analyses revealed absolute levels of PPR at Wave 1 predicted Wave 3 mortality via Wave 2 affective reactivity in the predicted direction, but this did not remain robust when statistically accounting for covariates (e.g., marital risk, neuroticism), $\beta = .004$, 95% CI: [-.03, .04]. However, Wave 1-2 PPR *change* predicted NA (but not PA) reactivity to daily stressors at Wave 2, which then predicted mortality risk a decade later (Wave 3); these results held when adjusting for relevant demographic, health, and psychosocial covariates, $\beta = -.04$, 95% CI: [-.09, -.002].

Conclusions: These findings are among the first to provide direct evidence of psychological mechanisms underlying the links between intimate relationships and mortality and have implications for research aiming to develop interventions that increase or maintain responsiveness in relationships over time.

Keywords: partner responsiveness, affect reactivity, mortality, longitudinal, relationships, MIDUS

PPR = perceived partner responsiveness; **NA** = negative affect; **PA** = positive affect; **MIDUS** = Midlife Development in the United States; **M** = mean; **SD** = standard deviation; **SE** = standard error; **CI** = confidence interval; **OR** = odds ratio

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Two decades of research suggest that social relationships promote physical and psychological health and well-being. Positive social relationships are associated with lower susceptibility to illnesses ranging from the common cold to cancer (1-2), and a meta-analysis of 148 studies demonstrated that individuals with more supportive relationships have a 50% lower risk of death (3). Over and above simply being socially integrated, the *quality* of individuals' relationships are especially meaningful predictors of long-term health (4-6).

Several theoretical models propose that elements of relationship quality are linked to long-term health via mediating psychological mechanisms (1, 5, 7-9). When considering how relationships predict health, the strength and strain model of marital quality and health (7, 9) suggests that positive aspects of relationships (strengths) buffer against deleterious health outcomes, whereas negative aspects of relationships (strains) exacerbate deleterious health outcomes. Relational strengths and strains are linked to well-known psychological (e.g., cognitive or affective) and biological (e.g., endocrine or immune) pathways which, in turn, predict health. One important relational strength, postulated from the model, is *perceived partner responsiveness* (PPR), the extent to which individuals believe that their romantic partners care about, understand, and validate their thoughts and feelings (10-11). PPR is a core tenet of several influential relationship theories and is thought to be essential to attachment and healthy social functioning (9, 12-13). A basic function of PPR is to down-regulate negativity and bolster feelings of security (9, 14), which satisfies fundamental belongingness and bonding needs (15).

Recent studies provide strong evidence for associations between greater PPR and favorable health and well-being outcomes in adulthood. For example, PPR is linked to improved pain regulation (16-17), higher eudaimonic well-being (i.e., well-being associated with achieving one's

potential and finding meaning in life, 18), better subjective sleep quality and objective sleep efficiency (19), and steeper diurnal cortisol slopes—which are linked with better physical health outcomes (20)—over10 years (21). Of particular importance to the present study, Selcuk and Ong (22) found that PPR interacts with social support receipt to predict longevity; specifically, receiving high social support predicted greater risk of mortality a decade later when PPR was low, whereas receiving high social support was unrelated to mortality risk when PPR was high. This suggests that even typically helpful and supportive behaviors can be harmful for health if perceived as unresponsive, and that high PPR can buffer mortality risk. In the current research, we extend this finding in three main ways.

First, we investigated how mortality might be predicted by PPR alone, rather than the combination of PPR and social support receipt. Selcuk and Ong's (22) primary research question centered on understanding why received support is not always beneficial for health; in this study, our primary research question centered on understanding the mechanisms through which PPR and PPR change are linked—directly or indirectly—to mortality over a longer period (20 years vs. 10 years). Second, we tested, for the first time, whether longitudinal *changes* in PPR are linked to mortality. Given the inherently dynamic nature of relationships, predictors of relationship quality should not be static over time. Research suggests that PPR can change longitudinally; for instance, individuals in roommate dyads can create recursive "cycles of responsiveness" via compassionate goals (23-24). Other studies suggest that appreciation and responsiveness can be transferred between romantic partners via relevant behavioral displays (e.g., expressions of gratitude, 25). It may be, then, that longitudinal *increases* in PPR are associated with *better* health and well-being. Alternately, links between PPR change and health may emerge such that longitudinal *decreases* in PPR are associated with *worse* health and well-being. Most long-term relationships are characterized by declines in satisfaction and intimacy over time (26-27); perhaps for some individuals PPR may similarly decline.

Although changes in positive aspects of relationships are critically important for relationship quality and success (10, 26, 28), and potentially for health and well-being, no studies to our knowledge have tested potential longitudinal changes in PPR.

Finally, we identified and tested a theoretically plausible and potentially important pathway (i.e., affect reactivity to daily stressors) through which PPR might be indirectly linked to mortality. The effectiveness of PPR for promoting good personal outcomes depends partially on its capacity to soothe negativity and sustain positivity (9, 14). In studies of health and well-being, greater PPR robustly predicts lower negativity (e.g., reduced anxiety, anger, or depression), which, in turn, predicts better outcomes (18-19, 21). Links between responsiveness and health via higher positivity, however, appear to be more tenuous. For instance, Slatcher et al. (21) found that greater absolute levels of PPR predicted diurnal cortisol slopes 10 years later via reductions in general NA, but not increases in general PA. Believing a partner to be responsive thus seems to help individuals regulate NA longitudinally.

Affect regulation may be especially important when things go wrong in life (29). Individuals encounter stressors every day and may experience distinct affective reactions to those stressors (i.e., an increase in NA, termed *NA reactivity*, or a decrease in PA, termed *PA reactivity*). Over and above general levels of NA or PA, NA and PA reactivity to daily stressors can take a long-term toll on health (30). Daily stressors trigger immediate and distinct affective reactions that have downstream effects on health and well-being, just as major life stressors do. However, daily stressors are unique because they are much more common than major life stressors and can create psychological and physiological burdens when occurring frequently over time (31). Greater NA and PA reactivity to stressors are linked to chronic health problems (32), higher risk of mental disorders (33), elevated inflammation (34), poorer sleep (35), and higher mortality risk (36-38).

Of importance to the present work, recent research suggests that greater PPR predicts lower NA reactivity, which then predicts higher eudaimonic well-being a decade later (18). Notably, this research tested PPR and affect reactivity at the same time-point, leaving open the question as to whether PPR is prospectively linked to affect reactivity later in life, and whether NA reactivity underlies the links between PPR and physical health.

In the current study, we tested prospective associations of (a) absolute levels of PPR and (b) longitudinal changes in PPR with all-cause mortality, adjusting for relevant demographic, health, and psychosocial covariates. We also examined affect reactivity to daily stressors as a potential indirect pathway linking responsiveness to mortality. We investigated these associations in three waves of data separated by 10 years each, and tested the following hypotheses:

Hypothesis 1. *Greater absolute levels of PPR at Wave 1 would indirectly predict lower all-cause mortality at Wave 3 (20-year follow-up) via lower NA reactivity to daily stressors at Wave 2 (10-year follow-up).*

Hypothesis 2. *Beyond absolute levels of PPR at Wave 1, longitudinal changes in PPR from Wave 1-2 would indirectly predict all-cause mortality at Wave 3 via NA reactivity to daily stressors at Wave 2.*

We did not expect PPR to *directly* predict all-cause mortality, as prior research has not found evidence for direct links between responsiveness and longevity, and theoretical models of social relationships and health posit that the links between elements of relationship quality and health outcomes occur via psychological or physiological pathways (1-2, 5, 8-9, 39-40). Instead, we hypothesized that PPR would predict mortality *indirectly* via its links to affect reactivity to daily stressors (41).

We centered our hypotheses on NA reactivity because theory argues that PPR functions to diminish negativity in a health context (9, 11), in addition to robust evidence suggesting links between PPR, NA, and health outcomes (18-19, 21). However, we also tested PA reactivity given that prior research has found associations of PA reactivity with health over and above NA reactivity

(34, 37) and given that there is evidence for links between PA and health more generally (42). Predictions regarding PA reactivity were exploratory considering recent studies suggesting that the links between PPR and health are sometimes not explained by PA (21).

Method

Participants and Procedure

Data were taken from Waves 1-3 of the National Survey of Midlife Development in the United States (MIDUS), one of the largest studies on health in adulthood available in the US. Wave 1 of MIDUS comprises 7,108 individuals from four samples (3,487 individuals in the main national sample recruited via random-digit dialing, 757 individuals recruited via oversampling in metropolitan areas, 951 siblings of a randomly selected group of national sample members, and 1914 twins). Data were collected via phone interviews and self-administered questionnaires in 1995-1996 (Wave 1), 2004-2006 (Wave 2), and 2013-2014 (Wave 3). Participants were selected for the present analyses if they had complete data for Wave 1 PPR, Wave 2 PPR, Wave 2 daily NA and PA reactivity, and Wave 3 mortality. The final sample in this study comprised 1,208 individuals (52.6% female, 95.2% White). At Wave 1, participants were 25-74 years of age (M = 47.41, SD = 11.86). Approximately 4.4% of our sample did not complete high school, 49.5% graduated high school but did not complete college/university, 28.9% completed college/university, and 17% pursued a postgraduate degree (0.2% did not indicate their education level). Annual income ranged from $0 to $300,000 ($M$ = $85,871.13, SD = $60,332.66). In our sample, most participants (97.7%) were married or cohabiting with a partner.

Primary Measures

Wave 1-2 PPR. We used the same measure of PPR as previous investigations using the MIDUS data (18-19, 21-22). At Waves 1 and 2, participants answered three questions ("How much does your spouse or partner really care about you?" "How much does he or she understand the way

you feel about things?" and "How much does he or she appreciate you?"), which match the three components of PPR identified in previous literature (10-11). Individuals rated their responses on a scale of 1 (*a lot*) to 4 (*not at all*). PPR scores at each wave were created by reverse-scoring the three items and averaging across them, such that higher scores indicated greater PPR; $\alpha = .82$ (Wave 1) and $\alpha = .84$ (Wave 2). Wave 1 and 2 PPR were correlated, $r = .51$, $p < .001$, suggesting that PPR across time is related, but variable. We then calculated a residualized change score to represent the change in Wave 1-2 PPR over the decade by regressing Wave 2 PPR on Wave 1 PPR.

Wave 2 daily NA and PA reactivity. Each day for eight days, participants completed the Daily Inventory of Stressful Events (43), where they indicated whether they had experienced several common daily stressors that day, such as interpersonal conflict, a problem at work/home, or perceived discrimination (0 = *no*, 1 = *yes*). Additionally, participants indicated how often they had experienced 14 NA states (e.g., nervous, worthless, hopeless, irritable, frustrated, afraid) and 13 PA states (e.g., cheerful, proud, satisfied, calm and peaceful, confident, enthusiastic) each day on a scale of 0 (*none of the time*) to 4 (*all of the time*). Daily NA and PA reactivity, represented by two separate within-person slopes, were calculated using a two-level model where Level 1 modeled NA and PA as a function of stress exposure, with the intercepts representing NA and PA experienced on non-stressor days and the slopes representing the change in NA and PA from a non-stressor day to a stressor day. The Level 2 models estimated sample averages of the intercepts and slopes while adjusting for between-person stress exposure in average NA and PA. Thus, the within-person daily NA and PA reactivity scores consider between-person differences in both stress exposure and NA and PA, to measure how much reactivity individuals experience given the amount of stress they are exposed to and their typical levels of NA and PA (18, 33, 37).

Wave 3 all-cause mortality. Names of individuals who could not be contacted for a follow-up survey at Wave 3 were submitted to the National Death Index through October 2015 to ascertain

if participants were deceased (0 = *no*, 1 = *yes*). In our sample of 1,208 individuals, 100 (8.3%) were identified as deceased at Wave 3. The number of deaths differs somewhat from those reported in previous investigations of relational variables and mortality in MIDUS (22) because our study used a different sample of participants and examined Wave 3 (vs. Wave 2) mortality.

Covariates

Demographic covariates. Demographic variables included Wave 1 sex (0 = *female*, 1 = *male*; 52.6% female), age, ethnicity (0 = *White*, 1 = *non-White*; 95.2% White), education (0 = *high school or less*, 1 = *some college or more*; 45.9% some college or more), annual income, and whether participants remained in the same relationship from Wave 1-2 (0 = *no*, 1 = *yes*). To estimate whether participants were in the same relationship, we used the criteria outlined in previous MIDUS research (18); according to our best estimate, among the participants who were married or cohabiting at both waves, the majority (96.0%) remained in the same relationship from Wave 1-2.

Physical and mental health covariates. Physical and mental health variables were included as covariates to account for any potential confounds of the links between PPR, daily NA and PA reactivity, and mortality. Physical health variables were assessed at Wave 1 and included a one-item measure of participants' perceptions of their health rated on a scale of 1 (*poor*) to 5 (*excellent*), self-reported cardiovascular conditions (0 = *no*, 1 = *yes*), cancer diagnoses (0 = *no*, 1 = *yes*), and the sum of remaining chronic physical health conditions (Range = 0-17; M = 2.05, SD = 2.15). Participants also indicated with one item how often they had trouble falling/staying asleep on a scale of 1 (*almost every day*) to 6 (*not at all*). Mental health was assessed at Wave 1 with the depression scale of the Composite International Diagnostic Interview-Short Form (44); participants were given a score from 0 (*lowest depression*) to 7 (*highest depression*).

Relational covariates. Negative and positive relational variables were included as covariates to rule out the possibility that other aspects of relationships could explain the associations between

PPR, daily NA and PA reactivity, and mortality. Marital risk at Wave 1 was assessed with five items (45): One item measured how often participants felt their relationship was in trouble over the past year, rated on a scale of 1 (*never*) to 5 (*all the time*); one item measured participants' beliefs about the likelihood that they and their partner would separate at some point, rated on a scale of 1 (*very likely*) to 4 (*not at all likely*) and reverse-scored; and three items measured how much the participant and their romantic partner argue about money, household tasks, and leisure time activities, rated on a scale of 1 (*a lot*) to 4 (*not at all*) and reverse-scored. Marital risk scores were created by averaging across the five items, such that higher scores indicated greater marital risk ($\alpha = .77$).

Social support provision and receipt were assessed at Wave 1 with two one-item measures (45). Specifically, participants were asked to report the approximate number of hours per month they spend (a) providing or (b) receiving emotional support (e.g., comforting, listening to problems, giving advice) to or from their partner, respectively. In our sample, participants reported providing 0-189 hours of support per month ($M = 27.19$, $SD = 36.15$), and reported receiving 0-173 hours of support per month ($M = 23.08$, $SD = 32.15$). These items were free response, and thus some answers were unfeasible (e.g., participants reporting that they provided support 24 hours per day). Outliers on these variables were winsorized to ±2.5 SD of the mean (21, 46).

Personality covariates. Trait agreeableness and neuroticism at Wave 1 were included as covariates to address the possibility that the associations between PPR and PPR change, daily NA and PA reactivity, and mortality could be accounted for by dispositional personality characteristics (e.g., highly neurotic individuals reporting low levels of PPR and/or high NA reactivity). Agreeableness was assessed with five items (helpful, warm, caring, softhearted, and sympathetic) and neuroticism was assessed with four items (moody, worrying, nervous, and calm [reverse-scored]). All personality items were rated on scales of 1 (*a lot*) to 4 (*not at all*). Agreeableness and neuroticism

scores were created by averaging across the items of the relevant subscales such that higher scores indicated greater agreeableness (α = .79) and neuroticism (α = .76).

Data Analytic Strategy

To address potential analytic problems related to missing data we performed multiple imputation of missing values (2.6%) using the expectation maximization algorithm, which provides unbiased parameter estimates and improves statistical power of analyses (47-48). The expectation maximization algorithm does not allow value replacement for dichotomous data, so we used mode replacement to replace missing values for dichotomous variables (1.3%). We first tested associations among study variables using bivariate correlation analyses (see Table 1). We then tested the links between Wave 1 absolute levels of PPR, Wave 1-2 PPR change, Wave 2 daily NA and PA reactivity, and Wave 3 all-cause mortality using hierarchical regression. Lastly, we tested if Wave 1 PPR and Wave 1-2 PPR change predicted Wave 3 mortality via Wave 2 daily NA and PA reactivity using the PROCESS macro for SPSS (49). Bias-corrected confidence intervals for the indirect association were estimated based on 5,000 bootstrap samples. We tested four models per analysis: Model 1 included demographic covariates, Model 2 added physical and mental health covariates, Model 3 added relational covariates, and Model 4 added personality covariates. To facilitate interpretation and to provide estimates of effect size, all continuous variables were standardized.

Results

Primary Analyses

Table 2 displays the hierarchical logistic regression analyses predicting Wave 3 all-cause mortality. Neither Wave 1 absolute levels of PPR nor Wave 1-2 PPR change *directly* predicted mortality in any model. Wave 2 daily NA reactivity, however, significantly predicted mortality in all models such that greater daily NA reactivity was linked with a higher likelihood of death 10 years

later. Wave 2 daily PA reactivity did not predict mortality in any model, suggesting that the link between affect reactivity and mortality in our sample was tied primarily to NA.

Figure 1 displays mediation analysis results for Wave 1 PPR, and Figure 2 displays results for Wave 1-2 PPR change. Mediation models with PA reactivity were not significant (see the bottom halves of Figure 1 and Figure 2). Models 1 and 2 revealed indirect links between Wave 1 absolute levels of PPR and mortality via NA reactivity in the predicted direction, but these links were eliminated when relational (Model 3) and personality (Model 4) covariates were added (see the top half of Figure 1).

Analyses revealed a significant indirect association between Wave 1-2 PPR change and Wave 3 all-cause mortality through Wave 2 daily NA reactivity, which remained significant adjusting for all covariates (see the top half of Figure 2). Therefore, changes in PPR over a 10-year period (Wave 1-2) predicted NA reactivity at Wave 2, which, in turn, was associated with mortality risk 10 years later (Wave 3).

Auxiliary Analyses

In primary analyses, we tested Wave 1 covariates. However, we also tested models including Wave 2 covariates as more chronologically direct predictors of Wave 3 mortality. In physical and mental health covariate models, Wave 2 poorer perceived health predicted higher Wave 3 mortality (p = .008), and Wave 2 cardiovascular conditions marginally predicted mortality (p = .083). In relational covariate models, Wave 2 higher marital risk predicted higher Wave 3 mortality (p = .026). Notably, the associations between PPR change, NA reactivity, and mortality remained robust in Wave 2 covariate models.

Moreover, because one of our key hypotheses involved longitudinal changes in a relational variable (i.e., PPR change), we ran additional models that included changes in marital risk, support provision, and support receipt from Wave 1-2. Inclusion of those variables did not alter our primary

results, and the other relational change variables did not predict mortality via affect reactivity to daily stressors.

Finally, we tested the interaction of PPR and sex in our models; analyses revealed no sex differences in associations between PPR, affect reactivity, and mortality.

Discussion

In a large sample of married and cohabiting adults in the US, we found robust prospective links between *changes* in PPR over a 10-year period, daily NA reactivity, and all-cause mortality, adjusting for demographic, physical and mental health, and psychosocial covariates known to be associated with relationships and health. We did not find strong evidence for links between Wave 1 absolute levels of PPR and Wave 3 mortality via Wave 2 NA reactivity (i.e., the predicted associations emerged in some, but not all, models). PPR was not indirectly associated with mortality via PA reactivity, a finding consistent with prior research demonstrating that responsiveness-health associations appear to be driven by NA rather than PA (21). To our knowledge, this study is the first to examine longitudinal alterations in PPR or changes in any marker of relationship quality predicting mortality. Our findings thus provide an important advance in understanding the links between relationship functioning and health by showing that changes in PPR over time predict longevity, even after adjusting for whether individuals remain in the same relationship.

Our findings are consistent with existing studies on the separate relations among PPR and health (21), PPR and affective reactivity (18) and affective reactivity and health (36-38). For example, our research extends a prior study that demonstrated links between absolute levels of PPR and NA reactivity assessed at the same time-point in MIDUS (18). By assessing NA reactivity at Wave 2 (vs. Wave 1), we show that these links are robust over time. Moreover, the current study is novel in that it links a critically-important relationship construct with a psychological mediator and mortality together in a single prospective investigation, a rare occurrence in longitudinal psychology studies

(50-51). Although previous research has found evidence that PA reactivity is sometimes linked to longevity (37), a similar pattern did not emerge in our sample. Prior studies, nonetheless, have not considered PPR when predicting mortality from affect reactivity to stressors. Our findings suggest that, when considering PPR—a construct theoretically and empirically tied to reducing NA in a health context (9)—the link between affect reactivity and health is driven by NA rather than PA.

Are the results of this study practically meaningful? The size of associations between PPR, affect reactivity, and mortality are small, but comparable to previously reported effects of relationship quality (3, 6, 22). Additionally, the effect sizes of other behaviors (e.g., exercise, fruit and vegetable consumption) and health are also small, as noted in a recent meta-analysis of marital quality and health (6). Thus, the associations found in this research are small but potentially mighty when put in context with other health behaviors.

The nature of our affect reactivity measure involved reactivity to inherently distressing events (e.g., conflict), which perhaps lends itself more strongly to increases in NA. PPR, however, matters not only when things go wrong, but also when things go right. General PA is associated with health and well-being (42), and capitalization of happy experiences is important for relationship functioning (52). Perhaps inherently positive daily events are also longitudinally associated with health. In this case, PPR as a relational strength may predict health, well-being, and longevity through different affective pathways (9).

The correlational nature of our study makes it impossible to be certain of the direction of PPR change in predicting affect reactivity and mortality risk. It is possible that increases in PPR are linked to lower NA reactivity and, in turn, lower mortality risk. Studies suggest that expressing gratitude (25) and holding compassionate goals (23-24) potentially foster responsiveness in relationships over time, and maintaining high levels of love predicts happier relationships (53). It is also possible that decreases in PPR are linked to higher NA reactivity and, in turn, higher mortality

risk. If PPR naturally declines longitudinally as satisfaction and intimacy do (26-27), then decreases in PPR might be particularly detrimental. An interesting question raised by these findings is precisely how PPR may be increased or maintained over time. Understanding the specific processes (e.g., partner behaviors, life events) that underlie changes in PPR could illuminate other psychological pathways to health and potentially inform clinical interventions. It is important not only to identify the types of relationship dynamics that influence responsiveness longitudinally, but also to consider the types of responsiveness interventions (e.g., person-level, couple-level) that may be effective.

Our findings must be considered in light of some limitations. These data are correlational, so we are unable to make definitive causal claims about the associations between PPR change, daily NA reactivity, and mortality. Nevertheless, prior literature and theoretical models of relational quality and health suggest that PPR predicts health and well-being through lower NA compared to the reverse (9, 18-19, 21). Moreover, in the present research we tested prospective associations between responsiveness and mortality and found effects even after statistically adjusting for relevant covariates, which bolsters our confidence in our conclusions. A second limitation is that the MIDUS sample is not racially diverse (95.2% White), limiting the generalizability of our findings. Replications of these findings in more heterogeneous samples across different cultures will provide the strongest case for the capacity of PPR to predict health.

Another limitation of this study is that the MIDUS sample comprises reasonably healthy individuals, so the total number of deaths across the 20-year span of the study was small (100 out of 1,208). This introduces some potential issues of statistical power, given that in logistic regression power is related not only to sample size, but also to variation in the outcome (i.e., the number of cases). Nevertheless, this is a potential limitation of any study of mortality involving a healthy sample, and to our knowledge there are no other existing datasets wherein the relational and affective processes we examined could be tested with the same depth. Interestingly, we might expect

perceived partner responsiveness to have similar links to mortality via affect reactivity even in a comparatively unhealthy sample, given that high-quality social relationships predict health in both healthy and unhealthy samples of participants (3, 54).

Taken together, these findings validate PPR as a meaningful predictor of longevity via psychological mechanisms (e.g., reducing NA) with implications for romantic relationship functioning that raise interesting questions about how partners help each other regulate negativity in the face of daily stressors. PPR is a tractable and potentially modifiable target for marital therapy that could lead to beneficial reductions in negative reactions to daily stressors as well as improvements in health.

References

1. Cohen S. Social relationships and health. Am Psychol 2004;59:676-84.
2. Uchino BN. Social support and health: A review of physiological processes potentially underlying links to disease outcomes. J Behav Med 2006;29:377-87.
3. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: A meta-analytic review. PLoS Med 2010;7:e1000316.
4. Burman B, Margolin G. Analysis of the association between marital relationships and health problems: An interactional perspective. Psychol Bull 1992;112:39-63.
5. Kiecolt-Glaser JK, Newton TL. Marriage and health: His and hers. Psychol Bull 2001;127:472-503.
6. Robles TF, Slatcher RB, Trombello JM, McGinn MM. Marital quality and health: A meta-analytic review. Psychol Bull 2014;140:140-87.
7. Slatcher RB. Marital functioning and physical health: Implications for social and personality psychology. Soc Personal Psychol Compass 2010;4:455-69.
8. Sbarra DA, Coan JA. Relationships and health: The critical role of affective science. Emot Rev in press.
9. Slatcher RB, Selcuk E. A social psychological perspective on the links between close relationships and health. Curr Dir Psychol Sci 2017;26:16-21.
10. Reis HT, Clark MS, Holmes JG. Perceived partner responsiveness as an organizing construct in the study of intimacy and closeness. In: Mashek D, Aron A, editors. The handbook of closeness and intimacy. Mahwah: Erlbaum; 2004. p. 201-25.
11. Reis HT. Perceived partner responsiveness as an organizing theme for the study of relationships and well-being. In: Campbell L, Loving TJ, editors. Interdisciplinary research on close

relationships: The case for integration. Washington DC: American Psychological Association; 2012. p. 27-52.

12. Bowlby J. Attachment and loss. Vol. 3. New York: Basic Books; 1980.
13. Mikulincer M, Shaver PR. The role of attachment security in adolescent and adult close relationships. In: Simpson JA, Campbell L, editors. The Oxford handbook of close relationships. New York: Oxford University Press; 2013. p. 66-89.
14. Slatcher RB, Schoebi D. Protective processes underlying the links between marital quality and physical health. Curr Opin Psychol 2016;13:148-52.
15. Baumeister RF, Leary MR. The need to belong: Desire for interpersonal attachments as a fundamental human motivation. Psychol Bull 1995;117:497-529.
16. Master SL, Eisenberger NI, Taylor SE, Naliboff BD, Shirinyan D, Lieberman MD. A picture's worth: Partner photographs reduce experimentally induced pain. Psychol Sci 2009;20:1316-8.
17. Machin AJ, Dunbar RIM. The brain opioid theory of social attachment: A review of the evidence. Behaviour 2011;148:985-1025.
18. Selcuk E, Gunaydin G, Ong AD, Almeida DM. Does partner responsiveness predict hedonic and eudaimonic well-being? A 10-year longitudinal study. J Marriage Fam 2016;78:311-25.
19. Selcuk E, Stanton SCE, Slatcher RB, Ong AD. Perceived partner responsiveness predicts better sleep quality through lower anxiety. Soc Psychol Personal Sci 2017;8:83-92.
20. Adam EK, Quinn ME, Tavernier R, McQuillan MT, Dahlke KA, Gilbert KE. Diurnal cortisol slopes and mental and physical health outcomes: A systematic review and meta-analysis. Psychoneuroendocrinology 2017;83:25-41.
21. Slatcher RB, Selcuk E, Ong AD. Perceived partner responsiveness predicts diurnal cortisol profiles 10 years later. Psychol Sci 2015;26:972-82.

22. Selcuk E, Ong AD. Perceived partner responsiveness moderates the association between received emotional support and all-cause mortality. Health Psychol 2013;32:231-5.
23. Crocker J, Canevello A. Creating and undermining social support in communal relationships: The role of compassionate and self-image goals. J Pers Soc Psychol 2008;95:555-75.
24. Canevello A, Crocker J. Creating good relationships: Responsiveness, relationship quality, and interpersonal goals. J Pers Soc Psychol 2010;99:78-106.
25. Gordon AM, Impett EA, Kogan A, Oveis C, Keltner D. To have and to hold: Gratitude promotes relationship maintenance in intimate bonds. J Pers Soc Psychol 2012;103:257-74.
26. Baumeister RF, Bratslavsky E. Passion, intimacy, and time: Passionate love as a function of change in intimacy. Pers Soc Psychol Rev 1999;3:49-67.
27. Karney BR, Bradbury TN. Neuroticism, marital interaction, and the trajectory of marital satisfaction. J Pers Soc Psychol 1997;72:1075-92.
28. Laurenceau JP, Barrett LF, Pietromonaco PR. Intimacy as an interpersonal process: The importance of self-disclosure, partner disclosure, and perceived partner responsiveness in interpersonal exchanges. J Pers Soc Psychol 1998;74:1238-51.
29. Kiecolt-Glaser JK, McGuire L, Robles TF, Glaser R. Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. Annu Rev Psychol 2002;53:83-107.
30. Almeida DM. Resilience and vulnerability to daily stressors assessed via diary methods. Curr Dir Psychol Sci 2005;14:64-8.
31. Zautra AJ. Emotions, stress, and health. New York: Oxford University Press; 2003.
32. Piazza JR, Charles ST, Sliwinski MJ, Mogle J, Almeida DM. Affective reactivity to daily stressors and long-term risk of reporting a chronic physical health condition. Ann Behav Med 2013;45:110-20.

33. Charles ST, Piazza JR, Mogle J, Sliwinski MJ, Almeida DM. The wear and tear of daily stressors on mental health. Psychol Sci 2013;24:733-41.

34. Sin NL, Graham-Engeland JE, Ong AD, Almeida DM. Affective reactivity to daily stressors is associated with elevated inflammation. Health Psychol 2015;34:1154-65.

35. Ong AD, Exner-Cortens D, Riffin C, Steptoe A, Zautra A, Almeida D. Linking stable and dynamic features of positive affect to sleep. Ann Behav Med 2013;46:52-61.

36. Wilson RS, Bienias JL, Mendes de Leon CF, Evans DA, Bennett DA. Negative affect and mortality in older persons. Am J Epidemiol 2003;158:827-35.

37. Mroczek DK, Stawski RS, Turiano NA, Chan W, Almeida DM, Neupert SD, Spiro III A. Emotional reactivity and mortality: Longitudinal findings from the VA Normative Aging Study. J Gerontol Ser B 2015;70:398-406.

38. Chiang JJ, Turiano NA, Mroczek DK, Miller GE. Affective reactivity to daily stress and 20-year mortality risk in adults with chronic illness: Findings from the National Study of Daily Experiences. Health Psychol in press.

39. Miller G, Chen, E, Cole SW. Health psychology: Developing biologically plausible models linking the social world and physical health. Annu Rev Psychol 2009;60:501-24.

40. Pietromonaco PR, Powers SI. Attachment and health-related physiological stress processes. Curr Opin Psychol 2015;1:34-9.

41. Zhao X, Lynch Jr JG, Chen Q. Reconsidering Baron and Kenny: Myths and truths about mediation analysis. J Consum Res 2010;37:197-206.

42. Pressman SD, Cohen S. Does positive affect influence health? Psychol Bull 2005;131:925-71.

43. Almeida DM, Wethington E, Kessler, RC. The Daily Inventory of Stressful Events: An interview-based approach for measuring daily stressors. Assessment 2002;9:41-55.

44. Kessler RC, Andrews G, Mroczek D, Ustun B, Wittchen HU. The World Health Organization Composite International Diagnostic Interview short-form (CIDI-SF). Int J Methods Psychiatr Res 1998;7:171-85.
45. Rossi AS. Caring and doing for others: Social responsibility in the domains of family, work, and community. Chicago: University of Chicago Press; 2001.
46. Wilcox R. Trimming and winsorization. In: Armitage P, Colton T, editors. Encyclopedia of biostatistics. Vol. 6. Chichester: Wiley; 1998. p. 4588-90.
47. Enders CK. A primer on maximum likelihood algorithms available for use with missing data. Struct Equ Modeling 2001;8:128-41.
48. Scheffer, J. (2002). Dealing with missing data. Res Lett Inf Math Sci 2002;3:153-60.
49. Hayes AF. Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. New York: Guilford Press; 2013.
50. Kemeny ME. An interdisciplinary research model to investigate psychosocial cofactors in disease: Application to HIV-1 pathogenesis. Brain Behav Immun 2003;17:62-72.
51. Farrell AK, Imami L, Stanton SCE, Slatcher RB. Affective processes as mediators of links between close relationships and physical health. Soc Personal Psychol Compass under invited review.
52. Gable SL, Reis HT. Good news! Capitalizing on positive events in an interpersonal context. Adv Exp Soc Psychol 2010;42:195-257.
53. Acevedo BP, Aron A. Does a long-term relationship kill romantic love? Rev Gen Psychol 2009;13:59-65.
54. Idler EL, Boulifard DA, Contrada RJ. Mending broken hearts marriage and survival following cardiac surgery. J Health Soc Behav 2012;53:33-49.

Table 1
Correlations among Study Variables

	Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1	Wave 1 PPR	—	.04	-.12**	.01	.002	.14**	.09**	-.09**	.01	.01	.09**	-.002	.05+	-.11**	.15**	-.10**	-.60**	.10**	.18**	.02	.11**	-.17**
2	Wave 1-2 PPR Change		—	-.11**	-.06*	.03	.12**	.13**	-.07*	-.04	.02	.06+	.03	.01	-.02	.03	-.02	-.10**	-.03	.003	-.03	.05+	-.03
3	Wave 2 NA Reactivity			—	-.06*	.06*	-.05+	-.10**	.08**	-.13**	-.10**	-.13**	.01	.001	.14**	.16**	.16**	.19**	-.01	-.02	-.02	-.03	.29**
4	Wave 2 PA Reactivity				—	.01	.06+	.001	-.01	.03	-.03	-.06+	-.02	.02	.05	-.01	.001	.03	-.05	-.06+	.03	-.12**	-.01
5	Wave 3 Mortality[a]					—	.08**	.34**	-.01	-.05	-.08**	-.08**	.15**	.11**	.05+	-.05	-.01	-.07*	-.03	-.05+	.001	-.01	-.03
6	Sex[b]						—	.12**	.003	.07*	-.02	.03	.02	-.02	-.18**	.08**	-.12**	-.05+	-.08**	-.05+	-.04	-.26**	-.13**
7	Age							—	-.06*	-.06*	-.06*	-.07*	.17**	.19**	.06+	-.002	-.14**	-.26**	-.04	-.05+	.17**	.06*	-.22**
8	Ethnicity[c]								—	-.01	-.04	-.07*	-.04	-.01	.04	-.02	.03	.08**	.05	.04	-.04	-.03	-.03
9	Education[d]									—	.22**	.16**	-.05+	-.03	-.09**	.06*	-.06+	.01	-.07*	-.06+	-.03	-.03	-.13**
10	Annual Income										—	.21**	-.12**	-.03	-.05+	.05+	-.05	-.01	-.06*	-.07*	.07*	-.03	-.07*
11	Perceived Health											—	-.19**	-.09**	-.38**	.22**	-.16**	-.09**	-.08**	-.05+	.06*	.07*	-.17**
12	Cardiovascular[e]												—	.05	.04	-.08**	.03	-.01	.04	.04	.04	.02	.04
13	Cancer[e]													—	-.02	-.002	-.04	-.06*	.04	.01	.02	.08**	-.02
14	Other Conditions														—	-.34**	.23**	.13**	.02	.01	-.01	.03	.26**
15	Sleep Quality															—	-.20**	-.17**	-.04	-.02	.03	-.03	-.28**
16	Depressive Symptoms																—	.15**	.03	.02	.002	.01	.23**
17	Marital Risk																	—	-.01	-.08**	-.09**	-.13**	.25**
18	Support Provision																		—	.90**	-.01	.06*	.01
19	Support Receipt																			—	-.01	.06*	.03
20	Same Marriage[e]																				—	.03	-.04
21	Agreeableness																					—	-.07*
22	Neuroticism																						—

Note. N = 1,208. PPR = perceived partner responsiveness; NA = negative affect; PA = positive affect. Higher scores on continuous variables indicate greater standing on the variable (e.g., greater PPR). Continuous variables are standardized.
[a]0 = not deceased, 1 = deceased; [b]0 = female, 1 = male; [c]0 = White, 1 = non-White; [d]0 = high school or less, 1 = some college or more; [e]0 = no, 1 = yes
+$p < .10$, *$p < .05$, **$p < .01$

Table 2

Hierarchical Logistic Regression Models Predicting Wave 3 All-Cause Mortality

	Model 1 (Nagelkerke's R^2 = .30)			**Model 2** (Nagelkerke's R^2 = .32)			**Model 3** (Nagelkerke's R^2 = .33)			**Model 4** (Nagelkerke's R^2 = .33)		
Variable	Coeff(*SE*)	OR	95% CI	Coeff(*SE*)	OR	95% CI	Coeff(*SE*)	OR	95% CI	Coeff(*SE*)	OR	95% CI
Primary												
Wave 1 PPR	-.15(.13)	0.86	[0.67, 1.11]	-.13(.13)	0.88	[0.68, 1.14]	-.11(.16)	0.90	[0.65, 1.24]	-.10(.16)	0.91	[0.66, 1.25]
Wave 1-2 PPR Change	-.06(.15)	0.95	[0.70, 1.27]	-.06(.16)	0.94	[0.69, 1.28]	-.07(.16)	0.93	[0.68, 1.27]	-.07(.16)	0.94	[0.69, 1.28]
Wave 2 Daily NA Reactivity	.41(.12)***	1.50	[1.19, 1.90]	.35(.12)**	1.42	[1.12, 1.79]	.35(.12)**	1.42	[1.12, 1.81]	.35(.12)**	1.43	[1.12, 1.82]
Wave 2 Daily PA Reactivity	.02(.12)	1.02	[0.80, 1.29]	.003(.12)	1.00	[0.79, 1.28]	-.01(.12)	0.99	[0.78, 1.26]	-.02(.13)	0.98	[0.77, 1.25]
Demographics												
Gender[a]	.49(.24)*	1.63	[1.01, 2.62]	.57(.25)*	1.78	[1.09, 2.90]	.58(.25)*	1.79	[1.09, 2.94]	.54(.26)*	1.71	[1.02, 2.87]
Age	1.70(.17)***	5.48	[3.91, 7.70]	1.72(.19)***	5.60	[3.89, 8.06]	1.76(.20)***	5.78	[3.93, 8.51]	1.76(.20)***	5.81	[3.93, 8.58]
Ethnicity[b]	.35(.57)	1.41	[0.46, 4.35]	.32(.58)	1.37	[0.44, 4.27]	.30(.58)	1.35	[0.43, 4.21]	.30(.59)	1.35	[0.43, 4.27]
Education[c]	-.19(.25)	0.83	[0.51, 1.36]	-.16(.26)	0.85	[0.52, 1.41]	-.15(.26)	0.87	[0.52, 1.44]	-.15(.26)	0.86	[0.51, 1.43]
Annual Income	-.004(.15)	1.00	[0.75, 1.33]	.07(.15)	1.07	[0.79, 1.44]	.06(.15)	1.06	[0.79, 1.42]	.06(.15)	1.06	[0.78, 1.43]
Physical and Mental Health												
Perceived Health				-.08(.15)	0.93	[0.69, 1.23]	-.09(.15)	0.91	[0.68, 1.22]	-.08(.15)	0.92	[0.69, 1.24]
Cardiovascular Conditions[d]				.54(.31)+	1.71	[0.94, 3.13]	.50(.31)	1.64	[0.89, 3.04]	.50(.32)	1.66	[0.89, 3.07]
Cancer Diagnoses[d]				.36(.34)	1.43	[0.73, 2.81]	.29(.35)	1.33	[0.67, 2.64]	.30(.35)	1.35	[0.68, 2.69]
Other Chronic Conditions				.05(.15)	1.05	[0.79, 1.39]	.06(.15)	1.06	[0.80, 1.42]	.06(.15)	1.07	[0.79, 1.43]
Sleep Quality				-.08(.13)	0.92	[0.71, 1.19]	-.07(.13)	0.93	[0.72, 1.21]	-.08(.14)	0.93	[0.71, 1.21]
Depressive Symptoms				.26(.15)+	1.29	[0.97, 1.72]	.25(.15)+	1.29	[0.96, 1.72]	.25(.15)+	1.29	[0.96, 1.73]
Relational												
Marital Risk							-.001(.15)	1.00	[0.75, 1.33]	-.01(.15)	0.99	[0.75, 1.32]
Support Provision							.27(.22)	1.30	[0.84, 2.02]	.27(.23)	1.31	[0.84, 2.04]
Support Receipt							-.37(.25)	0.69	[0.43, 1.12]	-.38(.25)	0.69	[0.42, 1.12]
Same Marriage Wave 1-2[d]							-.59(.29)*	0.55	[0.31, 0.97]	-.58(.29)*	0.56	[0.32, 0.99]
Personality												
Agreeableness										-.08(.13)	0.93	[0.72, 1.19]
Neuroticism										-.01(.15)	1.00	[0.74, 1.34]

Note. N = 1,208. PPR = perceived partner responsiveness; *SE* = standard error; CI = confidence interval; OR = odds ratio. Model 1 = analysis with demographic covariates; Model 2 = analysis adding physical and mental health covariates; Model 3 = analysis adding relational covariates; Model 4 = analysis adding personality covariates. Higher scores on continuous variables indicate greater standing on the variable (e.g., greater PPR). 95% CI is for the OR. In these models, a CI that does not include 1 indicates a statistically meaningful association.

[a]0 = female, 1 = male; [b]0 = White, 1 = non-White; [c]0 = high school or less, 1 = some college or more; [d]0 = no, 1 = yes

+p < .10, *p < .05, **p < .01, ***p < .001

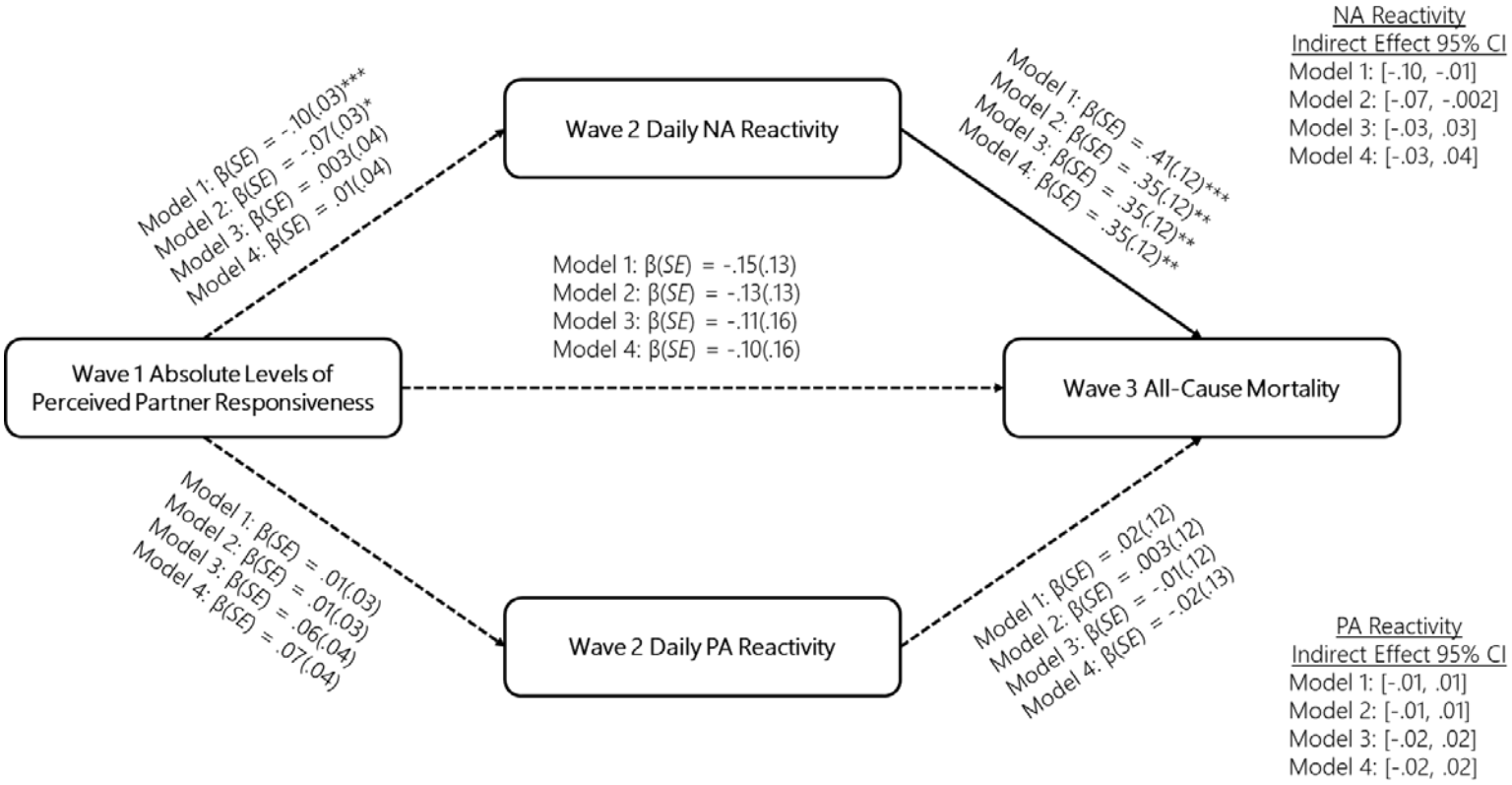

Figure 1. Direct and indirect associations between Wave 1 PPR, Wave 2 daily NA reactivity, Wave 2 daily PA reactivity, and Wave 3 all-cause mortality. *N* = 1,208. *SE* = standard error; CI = confidence interval. Model 1 = analysis with demographic covariates; Model 2 = analysis adding physical and mental health covariates; Model 3 = analysis adding relational covariates; Model 4 = analysis adding personality covariates. Higher scores on continuous variables indicate greater standing on the variable (e.g., greater PPR). Continuous variables are standardized. In these models, a CI that does not include 0 indicates a statistically meaningful association.

*$p < .05$, **$p < .01$, ***$p < .001$

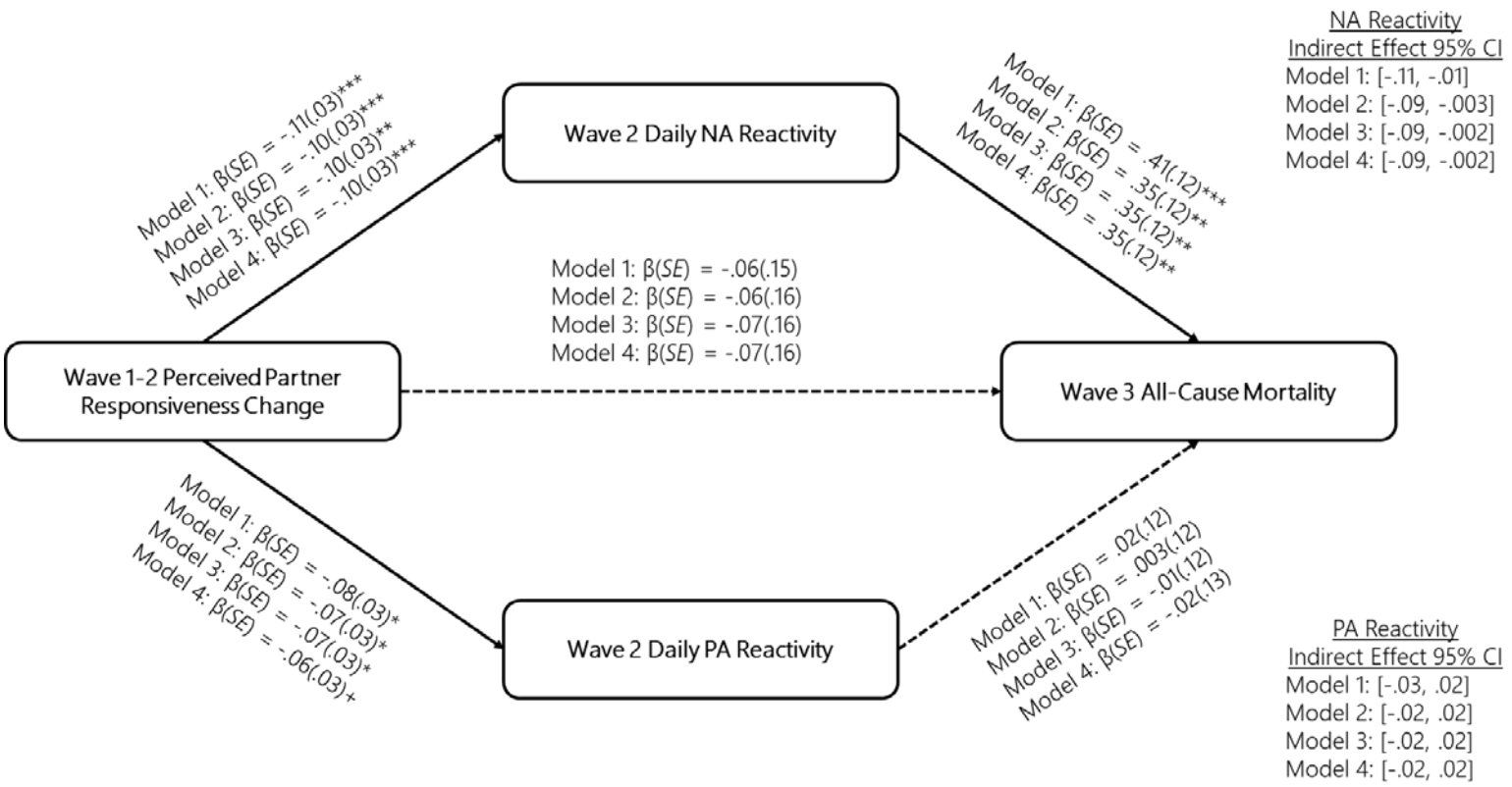

Figure 2. Direct and indirect associations between Wave 1-2 PPR change, Wave 2 daily NA reactivity, Wave 2 daily PA reactivity, and Wave 3 all-cause mortality. *N* = 1,208. *SE* = standard error; CI = confidence interval. Model 1 = analysis with demographic covariates; Model 2 = analysis adding physical and mental health covariates; Model 3 = analysis adding relational covariates; Model 4 = analysis adding personality covariates. Higher scores on continuous variables indicate greater standing on the variable (e.g., greater PPR). Continuous variables are standardized. In these models, a CI that does not include 0 indicates a statistically meaningful association.

$+p < .10$, $*p < .05$, $**p < .01$, $***p < .001$