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Changes in daily stress reactivity and changes in physical health across 18 years of adulthood

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Abstract

Background: Stress plays a pivotal role in physical health. Although many studies have linked stress reactivity (daily within-person associations between stress exposure and negative affect) to physical health outcomes, we know surprisingly little about how *changes* in stress reactivity are related to *changes* in physical health.

Purpose: The current study examines how change in stress reactivity over 18 years is related to changes in functional health and chronic health conditions.

Methods: Three measurement bursts from the National Study of Daily Experiences ($N = 2880$; 55% female) each included daily measures of stressor exposure and negative affect across 8 consecutive days, yielding 33 944 days of data across 18 years of adulthood. At each wave, participants reported their functional health limitations (ie, basic activities of daily living [ADL] and instrumental activities of daily living [IADL]) and chronic health conditions. Multilevel structural equation models simultaneously modeled stress reactivity at Level 1, longitudinal changes in stress reactivity at Level 2, and the association between changes in stress reactivity and changes in functional limitations and chronic conditions at Level 3.

Results: Higher levels of stress reactivity at baseline were associated with more functional health limitations 18 years later (ADLs: *Est.* = 0.90, $P = .001$; IADLs: *Est.* = 1.78, $P < .001$). Furthermore, individuals who increased more in their stress reactivity across the 18-year period also showed greater increases in their functional health limitations (ADLs: *Est.* = 4.02, $P = .017$; IADLs: *Est.* = 5.74, $P < .001$) and chronic conditions (*Est.* = 11.17, $P = .008$).

Conclusions: These findings highlight the strong connection between health and stress in daily life, and how they travel together across adulthood.

Lay Summary

Emotional reactivity to minor daily stressors has been shown to play a role in physical health, where individuals who are more reactive tend to also have poorer health. However, emotional reactivity to stress also changes across the lifespan with some people becoming more reactive over time and others becoming less reactive or remaining stable. The current study examines how change in stress reactivity over 18 years of adulthood is related to changes in functional health and chronic health conditions. Using data from the National Study of Daily Experiences, emotional responses to daily stressors were measured in 2880 individuals, capturing 33 944 days of data across 18 years of adulthood. Findings from this study indicate that individuals who were initially more reactive to daily stressors had greater functional health limitations 18 years later. Importantly, individuals who became more emotionally reactive to daily stressors across the 18-year period also experienced greater increases in their functional health limitations and number of chronic health conditions compared to individuals who were stable or became less emotionally reactive to daily stressors. These findings highlight the strong connection between health and stress in daily life, and how they travel together across adulthood.

Key words: multilevel SEM; stressor reactivity; functional health; chronic conditions; daily diary.

Introduction

Scientists have long recognized the tie between stress and physical well-being. Daily within-person associations between stress exposure and NA (henceforth referred to as stress reactivity) are related to several adverse health outcomes, including inflammation[1] and chronic conditions.[2] These findings typically rely on a measure of stress reactivity at one time point predicting physical outcomes years later. Thus, stress reactivity has been conceptualized as a dynamic process at micro timescales (ie, in daily life), and modeled as a static individual difference at macro timescales (ie, across years and decades). Yet, stress reactivity changes across

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adulthood,[3] as does physical health (eg, [4,5]). The current study uses an innovative method to examine how changes in stress reactivity are related to changes in 2 aspects of physical health: the presence of disease/illness states (assessed by number of chronic illness conditions) and functional health (assessed by a measure of limitations in ADL) across 18 years among a large national sample of middle-aged and older adults.

Stress reactivity as a daily risk factor for worse physical health

Ample research demonstrates that on days when an individual is exposed to a stressor, they report higher levels of negative affect (NA) than on stressor-free days.[6] The degree of stress reactivity differs across individuals, with some individuals more reactive than others. This reactivity, in turn, is related to worse health outcomes, including increased risk of morbidity,[2] mortality,[7] higher levels of inflammation,[1] poorer sleep efficiency,[8] and more affective disorders.[9,10]

Physiological arousal is one hypothesized pathway linking stress reactivity to poorer physical health. Accumulation of physiological changes as a result of stress reactivity is posited to lead to biological wear and tear and ultimately increased risk of illness as people grow older.[11] Consistent with this hypothesis, Piazza and colleagues[2] found that greater stress reactivity is associated with increased risk of reporting a chronic health condition nearly 10 years later using the first 2 waves of the National Study of Daily Experiences (NSDE). Absent from this and most other studies examining stress and health in daily life, however, is insight into how dynamic changes in the daily experience map onto health in the long-term.

Examining dynamic daily associations is important, as health and well-being are not static. Instead, they fluctuate daily in response to various stressors, emotions, social interactions, and physical activities. By examining individuals as dynamic processes, it is possible to capture this real-life variability, providing a more accurate understanding of how everyday experiences impact overall health. In addition, daily events, even minor ones, can accumulate over time to influence long-term health outcomes. Viewing individuals as dynamic processes helps in understanding how these small, everyday experiences contribute to larger health patterns, both positively and negatively.[12]

Changes in stress, emotion, and health over time

Stress reactivity captured at one point in time is an important predictor of both concurrent and future health and well-being. Yet, individuals do not necessarily remain stable in stress reactivity across time.[3,13,14] Research examining changes over time (as opposed to age differences) has relied predominantly on the NSDE study. Among NSDE participants, for example, average levels of NA, stressor exposure, and stress reactivity decrease over time.[3,15] While longitudinal declines in stressor exposure are consistent across young, middle, and older adulthood, the overall decrease in stress reactivity across the 18 years is moderated by age: younger adults decrease the most as they age, middle-aged adults are more stable across time, and older adults slightly increase over time. One study examining middle-aged and older adults (mean age 60 at baseline) and operationalizing stress slightly differently had participants record their NA and their overall perceived stress every night across 56 days every 2 years across 10 years. They found that NA had a stronger relationship with perceived stress in midlife, but this coupling (or reactivity) decreased with older age and over time.[16]

Three other non-NSDE based studies examined longitudinal change in affective reactivity to daily stressors among older adults, finding age-related increases over time.[14,17,18] In one study, participants (mean age of 80 years at baseline) in the Cognition, Health, and Aging Project (CHAP) came to a lab and reported their daily emotions and daily stressors for 6 days across 5 measurement bursts measured every 6 months over 2 years. Results indicate that average levels of stress reactivity increase across measurement bursts over 2 years.[14] Similarly, in the Daily Stress Interview (DAISI) study, participants reported increases in affective reactivity to daily stressors across 2 measurement bursts, 6 years apart.[17] Finally, in another study, adults ranging from 50 to 70 years-old reported their social conflicts and emotions hourly across 6 days, and then repeated the protocol for 3 days 6 years later. Participants reported increases in affect reactivity to momentary social conflict over time.[18]

Increases in stress reactivity among older adults appear to be at odds with improvements in affective well-being that researchers often observe among older adults (eg, [19]). According to socioemotional selectivity theory, older age is related to increases in social goals that derive emotional meaning and emotional well-being in life.[20] These motivations do not drive emotion regulation processes, per se, but they often result in greater attention to and preferences for positive (over negative) aspects of life, which benefits well-being (see [21]). Findings indicate that average levels of momentary affect improve over a 10-year period among a sample of adults ranging from teens to octogenarians,[19] and that older adults are less variable in daily reports of NA than are younger adults.[22,23] With age, social relationships remain one domain that is relatively well-preserved.[24,25] One study, for example, found that perceived control over daily interpersonal stressors across 10 years stayed stable, but control over non-interpersonal stressors declined.[26]

Yet, sometimes people find themselves in situations that are not consistent with their socioemotional goals. Strength and Vulnerability Integration (SAVI) predicts that when people are faced with an acute stressor, they are stripped from many age-related strengths that underly increased well-being with age (not encountering, avoiding, or directing attention away from negative aspects of life[27]). In stressful situations where people must down-regulate negative emotions, researchers often find few or no differences in emotion regulation abilities (eg, [28]). Furthermore, greater stress reactivity may place greater physiological stress on older adults given common age-associated biological decline and increased prevalence of functional and chronic health problems. Consistent with SAVI, Rush and colleagues[13] found that individuals who display greater increases in stress reactivity over a 9-year period report lower levels of psychological well-being and life satisfaction compared to individuals whose stress reactivity was more stable across time. Characterizing individuals by these dynamic daily associations can further contribute to our understanding of daily physical and emotional processes across the adult lifespan.

Present study

The present study uses data from the NSDE, a sub-study of the Midlife in the United States (MIDUS) project that embeds

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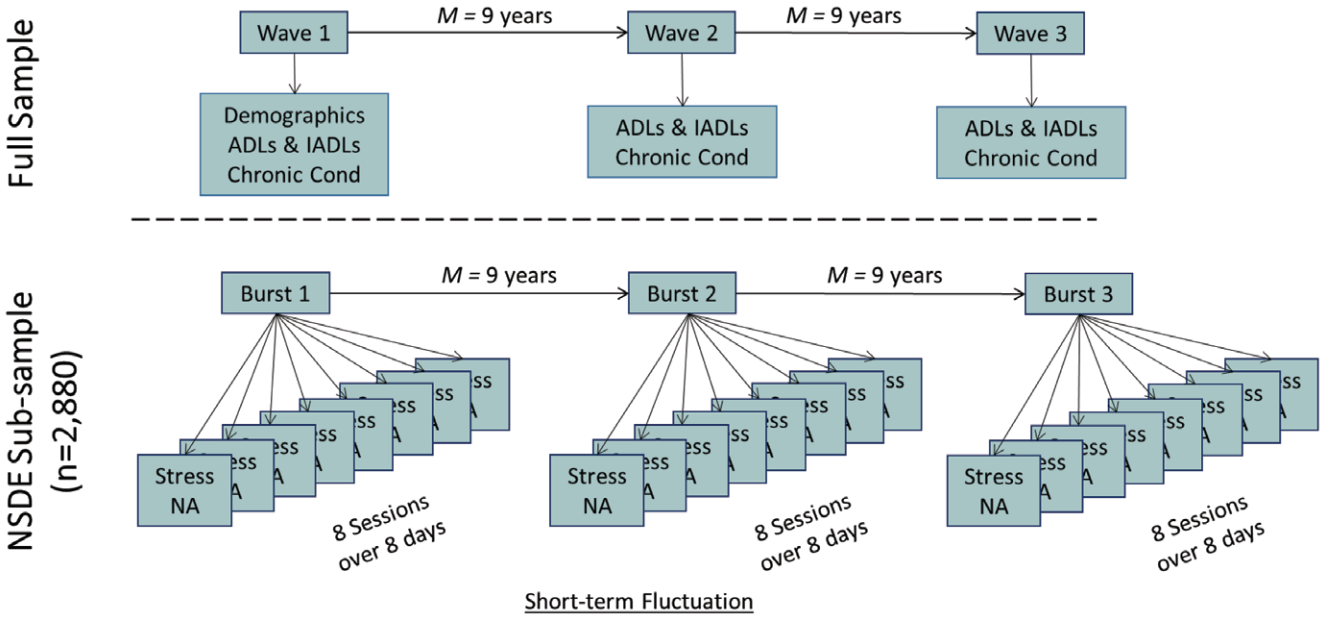

Figure 1. Midlife in the United States (MIDUS) study design. All participants completed Wave 1. A sub-sample completed the National Study of Daily Experiences (NSDE) daily assessments (2880 participants completed at least one of Burst 1, 2, or 3). Abbreviations: ADL, activities of daily living; Chronic cond, number of chronic conditions; IADL, instrumental activities of daily living; NA, negative affect.

intensive measurement burst data within a longitudinal panel design. This rich longitudinal measurement burst data allows us to examine how short-term (ie, daily) stress reactivity changes over longer intervals of time (ie, 18 years), and how this change relates to both overall levels of functional health and chronic conditions at 18-year follow-up, and to changes in health across 18 years. Compared to cross-sectional and single measurement burst designs, the present study's longitudinal examination across 3 bursts facilitates a more robust method for investigating links between daily stress reactivity and health by modeling developmental processes across the adult lifespan. We predict that individuals who increase in stress reactivity report greater declines in functional health and increases in chronic conditions than do those who are stable or who are becoming less reactive to stressors over time.

Furthermore, research to date has primarily used a 2-step approach to examine individual differences and patterns of change among short-term within-person associations, where within-person stress reactivity slopes are first extracted from a multilevel model then subsequently included as an individual difference variable in follow-up models. The present study uses an innovative approach that extends prior work[13] by simultaneously modeling individual differences in levels of stress reactivity; how stress reactivity changes over time; and individual differences in the rates of change in health outcomes, modeled as random slopes within a joint modeling multilevel structural equation modeling (MSEM) framework. Modeling these effects simultaneously within a single statistical model allows for a more computationally efficient modeling of the variability within and across levels of analysis (see [29–31]). The MSEM approach permits a sophisticated linking of processes operating on differing timescales, where short-term daily processes that unfold over time can be simultaneously predictive of slower developing changes in functional and physical health.

Methods

Participants and procedure

Participants were from the MIDUS project (https://www.icpsr.umich.edu/web/ICPSR/series/203), a publicly available data set consisting of multiple sub-projects using a large representative sample of American adults (aged 24–74 at baseline). The MIDUS project incorporates a large-scale longitudinal panel design, where participants completed 3 waves of a comprehensive survey on aspects of their health and well-being at approximately 9-year intervals. A random subset of these participants were invited to participate in the NSDE. Individuals who agreed to participate responded to end-of-day telephone interviews for 8 consecutive days that assessed daily levels of stress and affect (see [32,33] for a detailed description of data collection). Participants were predominantly White (84.5%, 11.3% Black, 1.3% Native American, 0.3% Asian, and 2.6% Other Race), Female (55%), and had some education beyond high school (67%).

Figure 1 depicts the NSDE data and study design. The NSDE data collection consisted of 3 bursts of daily assessments repeated at ~9-year intervals, providing longitudinal daily diary data across 18 years of adulthood (NSDE 1: ~1996, NSDE 2: ~2005, and NSDE 3: ~2017). Daily diary data were collected on a total of 33 944 days out of 37 576 possible days (completion rate = 90%). The present study included respondents who participated in any of the 3 NSDE bursts and at least one wave of the MIDUS surveys (N = 2880; # of daily assessments = 33 944; # of burst assessments = 4661). Descriptive statistics for all study variables are included in Table 1.

NSDE daily diary measures

Negative affect

Daily NA was assessed at each burst of the NSDE data collections. Across 8 consecutive days, participants were presented with a list of 6 negative emotions (*fidgety, nervous, worthless,*

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Table 1. Means and standard deviations of study variables.

	Wave 1/Burst 1 $N = 1499$			Wave 2/Burst 2 $N = 2022$			Wave 3/Burst 3 $N = 1176$		
Variable	*M*	*SD*	Range	*M*	*SD*	Range	*M*	*SD*	Range
Demographics									
Age	47.02	12.60	24–74	56.15	12.31	34–84	64.10	11.36	43–93
Female	0.55[a]	0.50	0–1	—	—	—	—	—	—
Education	0.67	0.47	0–1	—	—	—	—	—	—
Health									
ADL	1.13	0.44	1–4	1.26	0.59	1–4	1.37	0.70	1–4
IADL	1.52	0.73	1–4	1.77	0.87	1–4	1.99	0.94	1–4
Chronic cond	1.91	1.81	0–16	2.19	1.90	0–16	2.64	2.08	0–16
Burst-level variables									
Daily NA	0.19[b]	0.29	0–4	0.21[b]	0.28	0–4	0.17[b]	0.25	0–4
Daily stressor	0.40[c]	0.26	0–1	0.40[c]	0.27	0–1	0.39[c]	0.28	0–1

Abbreviations: ADL, activities of daily living; Chronic cond, number of chronic conditions; IADL, instrumental activities of daily living; NA, negative affect.
[a]Proportion of female participants.
[b]Aggregated across daily assessments.
[c]Proportion of stress days.

so sad that nothing could cheer you up, everything was an effort, and *hopeless*[34]) and asked to indicate how frequently they felt each emotion in the past 24 hours. Responses ranged from 0 (*none of the time*) to 4 (*all of the time*). Daily NA scores were computed by averaging across the items for each day. Multilevel omega was used to estimate within- and between-person reliability (see [35]). Within-person reliability estimates were .60, .58, and .54 and between-person reliability was .81, .82, and .82 for bursts 1, 2, and 3, respectively.

Daily stressors

Daily stressors were assessed using the Daily Inventory of Stressful Events.[36] The inventory consisted of 6 questions inquiring whether certain types of stressors had been experienced in the last 24 hours (eg, "In the past 24 hours, did you have an argument or disagreement with anyone?"). A dichotomous variable was used to characterize days as either stress days (at least 1 stressor was reported) or non-stress days (no stressor reported). A daily stressor was reported on ~40% of days during each of the 3 bursts.

MIDUS longitudinal panel measures

Functional health

At waves 1, 2, and 3 of the MIDUS survey, participants reported on their basic activities of daily living (ADLs) and instrumental activities of daily living (IADLs) to assess functional impairment.[37,38] Items in the ADL measure reflect an individual's ability to complete basic daily activities independently and include bathing or dressing oneself, walking one block, and climbing one flight of stairs. Items in the IADL measure reflect an individual's ability to engage in more complex daily activities that support independent living, including lifting or carrying groceries; climbing several flights of stairs; bending, kneeling, or stooping; walking more than a mile; walking several blocks; engaging in vigorous activity; and engaging in moderate activity. Participants indicated the extent to which their health limited these activities on a 4-point scale ranging from 1 (*not at all*) to 4 (*a lot*), with items averaged together such that higher scores indicated greater functional impairment (ADLs Cronbach's $\alpha = .73$, .81, and .85; IADLs Cronbach's $\alpha = .91$, .94, and .94 for waves 1, 2, and 3, respectively).

Chronic conditions

At waves 1, 2, and 3 participants reported their chronic health conditions from the past 12 months using a checklist of 30 possible health conditions (eg, asthma, diabetes, hypertension, and arthritis). We omitted "anxiety, depression, or some other emotional disorder," from analyses, due to its possible confounding role between NA and health conditions. Consistent with prior research, to avoid multiple reports of the same condition, and chronic conditions were coded into 16 groupings (see [2]). The number of chronic conditions was summed together to create a total score.

Covariates

Participant age at wave 1, sex, and education were included as covariates to adjust for sample heterogeneity. Age at wave 1 was centered at the grand mean in all statistical models. Sex and education were both coded as binary variables (sex: 0 = *male*, 1 = *female* and education: 0 = *high school degree or less*, 1 = *some college or more*).

Data analytic strategy

An MSEM framework that combines features of multilevel modeling and structural equation modeling was used to handle the hierarchically structured data from the measurement burst design and permit a multivariate examination of time-varying relationships between stress reactivity and physical health across timescales and levels of analysis.[39–41] An important feature of the MSEM approach is that random effects at each level can be modeled as either exogenous or endogenous variables at subsequent levels of analysis. That is, the latent random slopes (ie, the departure of individual stress reactivity scores from the overall group mean of stress reactivity) can be specified to represent individual differences in the within-person associations, and these individual differences can be included as predictors of concurrent or distal

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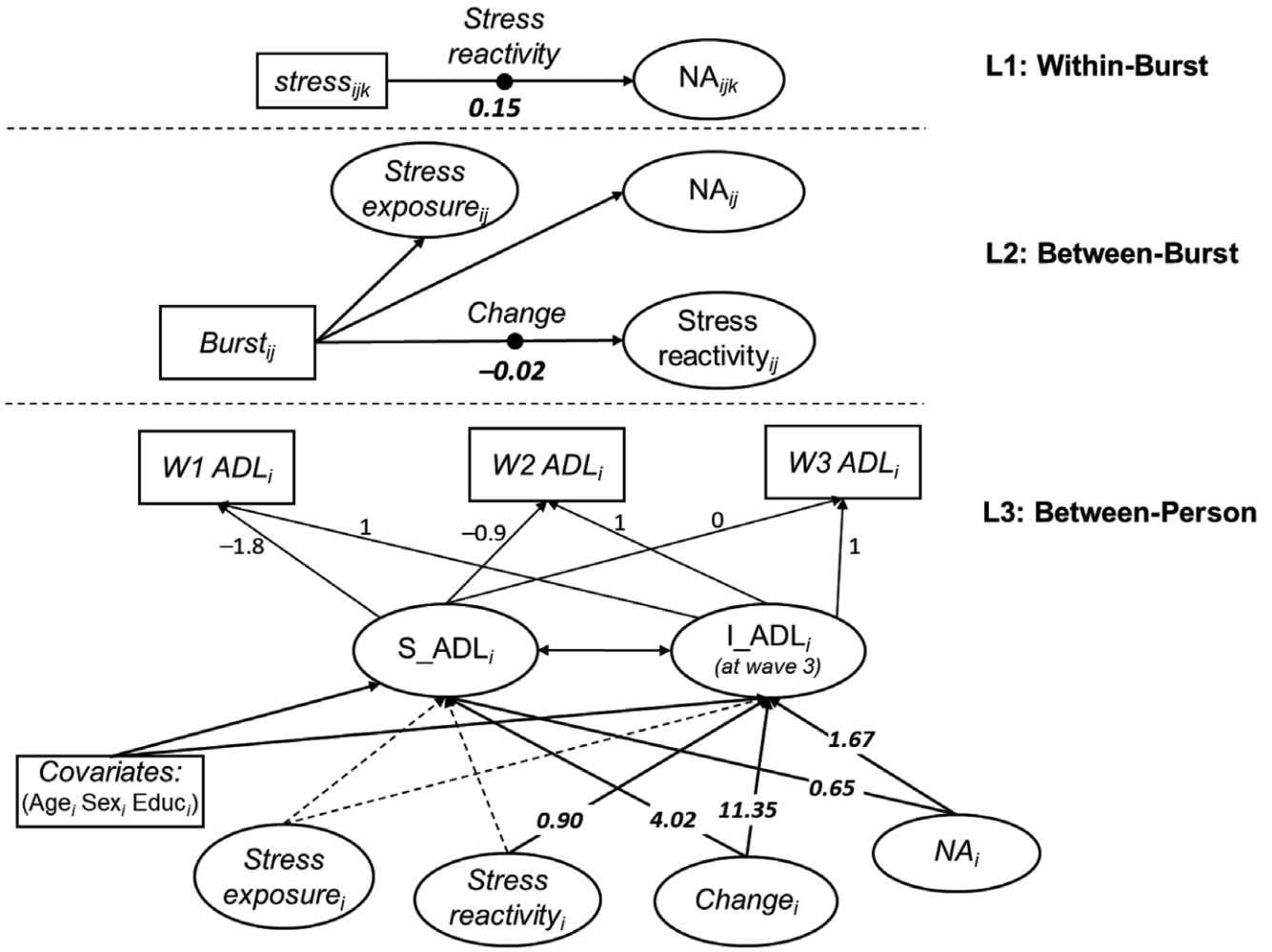

Figure 2. Estimated 3-level structural equation model predicting longitudinal changes in activities of daily living (ADLs). Daily assessments are nested within-bursts and bursts of measurements are nested within people. Ovals indicate variables were estimated within the model. Black dots indicate that pathway was modeled as a random slope. Note: Values are unstandardized coefficients. Dotted paths are non-significant, estimated values for these paths are omitted from display (*p*s > .05). All other paths (italicized values) are statistically significant, *p*s < .05. Abbreviations: Educ, education; I_ADL, ADL intercept centered at wave 3; NA, negative affect; stress, stress day; S_ADL, ADL slope.

outcomes (ie, functional health and chronic health conditions). Furthermore, the current measurement burst design that assesses individuals across multiple timescales can be modeled in a manner that permits random effects from lower levels of analysis to also be specified as random effects at subsequent levels, linking change processes across timescales.

The current research capitalizes on this flexibility by specifying daily within-burst associations between stress and NA as a random slope that may also change within individuals over longer intervals of time (ie, across bursts). The long-term change in the random short-term association can further be specified as a random slope, permitting individual differences in the magnitude of change in reactivity to daily stressors across 18 years of adulthood. Within the MSEM, a latent growth curve model was simultaneously estimated to capture individual levels and changes in health, and the extent that long-term changes in daily stress reactivity accounted for changes in health across 18 years of adulthood. Daily measurement occasions were nested within measurement bursts and measurement bursts were nested within people, resulting in 3-levels of analysis. Model specification for each level of analysis is described next and the full model is depicted in Figure 2.

Level 1 (within-burst)

At the within-burst level, daily stressor exposure$_{ijk}$ was included as a predictor of daily levels of NA$_{ijk}$. The subscript *ijk* in Figure 2 indicates that both stressor exposure and NA could vary across days (*k*), measurement bursts (*j*), and individuals (*i*). Stress reactivity was modeled as the daily within-person association between stressor exposure and NA. Because stressor exposure was a dichotomous variable, stress reactivity can be defined as the difference in NA on stressor days compared to non-stressor days. This daily within-person association between stressor exposure and NA (ie, stress reactivity) was modeled as a random slope and was permitted to vary across bursts and individuals. That is, the strength of the daily stressor-NA association could differ across bursts within an individual, as well as across individuals. A *t*–1 lagged NA autocorrelation term was included to model the serial autocorrelation between daily NA and previous day NA.

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Level 2 (between-bursts)

At the second level of analysis, the random stress reactivity$_{ij}$ slope was modeled as a latent endogenous variable that varies across bursts and individuals. Burst-level NA$_{ij}$ and stress exposure$_{ij}$ were also modeled as a latent endogenous variables that represent the mean NA and proportion of stress days, respectively, for person *i* during burst *j*. A Burst$_{ij}$ variable (where time was coded as change across decades, ie, *burst 1* = 0; *burst 2* = 0.9; *burst 3* = 1.8) was included as a predictor of burst-level NA$_{ij}$, stress exposure$_{ij}$, and stress reactivity$_{ij}$ to examine whether there was a within-person change across bursts in the level of NA, proportion of stress days reported, or the strength of the daily stressor-NA association, respectively. The long-term change in stress reactivity across bursts was modeled as a random slope, permitting individual differences in the magnitude of change in the daily within-person association of stress and NA across bursts. That is, modeling whether some individuals differed in the extent to which their stress reactivity changed across the 18-year period from burst 1 to burst 3 (cf. [13] for a more detailed description of model specification).

Level 3 (between-person)

Individual differences in *stress reactivity*$_i$ and the magnitude of long-term changes in stress reactivity (ie, *change*$_i$) were modeled as latent slopes, indicating that they are estimated from the model and reflect the strength of the daily stress reactivity association at burst 1 (ie, when burst = 0) and amount of change in stress reactivity, respectively, for individual *i*. NA$_i$ and stress exposure$_i$ were modeled as latent means that reflect average levels of NA and the proportion of stressor days, respectively, for individual *i* across days and bursts.

To model long-term changes in health, a latent growth curve model was specified at Level 3 (see Figure 2). The latent growth curve specified a latent intercept (eg, I_ADL$_i$) and slope (eg, S_ADL$_i$) for levels of health and changes in health across the 3 waves of data (18 years) by loading onto the observed health variables at each wave. The time metric was re-centered such that the 0 value represented health levels at wave 3. To accomplish this, the latent slope was specified such that wave 1 loaded with a value of −1.8, wave 2 with a value of −0.9, and wave 3 with a value of 0. Thus, the intercept could now be interpreted as health levels at wave 3 (ie, when time = 0). The slope estimate would still be interpreted as a linear change in health across 18 years from wave 1 to wave 3, where a 1-unit change in the time metric reflects a 10-year timespan.

Individual differences in (1) stress reactivity at burst 1, (2) the magnitude of changes in stress reactivity across 18 years, (3) mean levels of NA, and (4) mean levels of stress exposure were used to predict individual differences in changes in functional health (ADLs and IADLs) and chronic conditions across 18 years; as well as estimated levels of health at wave 3 (18-year follow-up). A set of observed covariates was included to adjust for the effects of wave 1 age (centered at the grand mean), sex, and education on wave 3-levels and changes in health. All effects were estimated simultaneously using Bayesian estimation, which makes use of all available data. Mplus version 8.10 software[42] was used to fit all models.

Results

An empty 3-level model revealed that, according to the intraclass correlation coefficients (ICCs), 46% of total variation in NA was within-burst, 18% was between-burst, and 36% was between-person. Table 2 presents the findings from the full MSEMs. Each of the health outcomes (ie, ADLs, IADL, and chronic conditions) was examined in separate models.

Daily within-person associations over time

The daily within-person associations over time (ie, the within-burst and between-burst effects, see Table 2) were consistent across all models regardless of the health outcome. As an example, the estimates from the model where ADLs is the health outcome are described in this paragraph (and presented in Figure 2). The significant association between stressor exposure and NA indicates that on days when individuals were exposed to a stressor, their NA was higher than on days when they did not report a stressor (ie, stress reactivity). This effect is shown by the significant baseline stress reactivity effect in burst 1 (*estimate* = 0.15, *pSD* = .007, $P < .001$, CrI$_{95}$ = 0.13 to 0.16). Furthermore, some individuals were more emotionally reactive to stressors than others, and for some bursts more than others, indicated by significant burst-specific and person-specific variations in the strength of the daily association between stress and NA (see between-burst and between-person random effects estimates of Stress reactivity from Table 2).

Individuals, on average, became less reactive to daily stressors over the 18-year period. From Table 2, the fixed effect of stress reactivity change across time was significant (*estimate* = −0.02, *pSD* = .006, $P < .001$, CrI$_{95}$ = −0.03 to −0.01), indicating that the strength of the daily association between stress and NA displayed a significant linear decline across the 3 measurement bursts. In addition, the between-person random effects estimate of stress reactivity change in Table 2 indicates significant individual differences in the degree of change in stress reactivity across bursts. As an illustrative example, Figure 3A displays the average (fixed) change in stress reactivity (solid black line), as well as individual deviations in the degree of stress reactivity change (colored dotted lines) for 5 individuals to highlight the multiple levels of random slopes, wherein there are individual deviations in stress reactivity during each burst of measurement (depicted in the balloons) as well as individual deviations in the degree of stress reactivity change across the three bursts of data spanning 18 years.

Long-term changes in stress reactivity and health

The latent growth curve specified at Level 3 revealed a significant linear increase in slope estimates across the 18-year period, indicating increases in functional limitations, ADLs (*estimate* = 0.20, *pSD* = .068, $P < .001$, CrI = 0.08 to 0.33) and IADLs (*estimate* = 0.39, *pSD* = .078, $P < .001$, CrI = 0.24 to 0.53), and in number of reported chronic health conditions (*estimate* = 0.83, *pSD* = .168, $P < .001$, CrI = 0.51 to 1.16). Figure 3B displays the average change trajectory (thick solid red line) in functional limitations, as well as individual trajectories of functional health limitations (thin black lines) across the three waves of data spanning 18 years.

Linking changes in stress reactivity and health across timescales

The primary effect of interest was whether long-term changes in daily stress reactivity (ie, stress reactivity change) accounted

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Table 2. Three-level structural equation modeling analyses of the effects of daily stress reactivity on health.

	ADL		IADL		Chronic conditions	
Variable	Estimate (*pSD*)	95% CrI	Estimate (*pSD*)	95% CrI	Estimate (*pSD*)	95% CrI
Fixed effects						
Within-burst variables						
NA intercept	0.125 (.005)[c]	[0.116, 0.135]	0.125 (.005)[c]	[0.116, 0.135]	0.126 (.005)[c]	[0.117, 0.136]
Baseline stress reactivity	0.147 (.007)[c]	[0.134, 0.161]	0.148 (.007)[c]	[0.135, 0.161]	0.146 (.007)[c]	[0.132, 0.159]
Between-burst variables						
NA change	0.010 (.004)[b]	[0.002, 0.017]	0.009 (.004)[b]	[0.002, 0.016]	0.008 (.004)[a]	[0.001, 0.016]
Stress exposure change	−0.019 (.005)[c]	[−0.029, −0.010]	−0.019 (.005)[c]	[−0.028, −0.009]	−0.018 (.005)[c]	[−0.028, −0.009]
Stress reactivity change	−0.020 (.006)[c]	[−0.034, −0.008]	−0.022 (.006)[c]	[−0.034, −0.011]	−0.020 (.006)[c]	[−0.032, −0.008]
Between-person variables predicting health						
Intercept (wave 3 health)	1.489 (.137)[c]	[1.203, 1.768]	1.771 (.184)[c]	[1.384, 2.066]	2.010 (.339)[c]	[1.368, 2.701]
Intercept on stress reactivity	0.901 (.387)[b]	[0.268, 1.779]	1.780 (.587)[c]	[0.880, 3.179]	1.000 (.936)	[−0.726, 2.924]
Intercept on stress reactivity change	11.353 (3.203)[b]	[2.097, 15.378]	6.651 (4.569)	[−4.240, 12.323]	25.432 (8.222)[b]	[5.628, 37.834]
Intercept on NA	1.674 (.151)[c]	[1.387, 1.976]	1.679 (.184)[c]	[1.334, 2.055]	4.163 (.438)[c]	[3.304, 5.016]
Intercept on stress exposure	−0.158 (.113)	[−0.380, 0.062]	0.183 (.133)	[−0.073, 0.448]	0.876 (.301)[b]	[0.289, 1.467]
Intercept on age	0.015 (.001)[c]	[0.012, 0.017]	0.032 (.002)[c]	[0.029, 0.035]	0.053 (.004)[c]	[0.046, 0.060]
Intercept on sex	0.096 (.031)[b]	[0.033, 0.156]	0.258 (.039)[c]	[0.176, 0.334]	0.431 (.085)[c]	[0.265, 0.596]
Intercept on education	−0.219 (.034)[c]	[−0.285, −0.150]	−0.336 (.043)[c]	[−0.421, −0.251]	−0.209 (.095)[a]	[−0.397, −0.024]
Slope (change in health)	0.203 (.068)[c]	[0.075, 0.333]	0.393 (.078)[c]	[0.236, 0.534]	0.828 (.168)[c]	[0.508, 1.164]
Slope on stress reactivity	0.124 (.206)	[−0.248, 0.573]	0.326 (.254)	[−0.109, 0.868]	−0.533 (.572)	[−1.747, 0.478]
Slope on stress reactivity change	4.023 (1.836)[b]	[0.250, 6.927]	5.740 (1.687)[c]	[1.714, 8.228]	11.169 (3.686)[b]	[2.330, 16.938]
Slope on NA	0.653 (.088)[c]	[0.487, 0.831]	0.255 (.102)[b]	[0.055, 0.466]	0.565 (.267)[a]	[0.029, 1.095]
Slope on stress exposure	0.001 (.064)	[−0.131, 0.124]	0.072 (.071)	[−0.065, 0.210]	−0.338 (.178)	[−0.675, 0.018]
Slope on age	0.006 (.001)[c]	[0.005, 0.008]	0.009 (.001)[c]	[0.007, 0.011]	0.011 (.002)[c]	[0.007, 0.015]
Slope on sex	0.030 (.018)	[−0.005, 0.065]	0.042 (.022)	[−0.013, 0.081]	−0.010 (.049)	[−0.104, 0.087]
Slope on education	−0.090 (.020)[c]	[−0.128, −0.050]	−0.126 (.002)[c]	[−0.017, −0.079]	−0.062 (.055)	[−0.166, 0.045]
Random effects						
Within-burst NA	0.058 (.001)	[0.057, 0.060]	0.058 (.001)	[0.057, 0.059]	0.058 (.001)	[0.057, 0.060]
Between-burst						
NA intercept	0.007 (.001)	[0.006, 0.008]	0.007 (.001)	[0.005, 0.009]	0.007 (.001)	[0.005, 0.009]
Stress exposure	0.037 (.001)	[0.034, 0.039]	0.037 (.001)	[0.034, 0.039]	0.036 (.001)	[0.034, 0.039]
Stress reactivity	0.023 (.002)	[0.019, 0.027]	0.023 (.002)	[0.020, 0.028]	0.023 (.002)	[0.019, 0.027]
Between-person						
NA intercept	0.025 (.001)	[0.022, 0.027]	0.025 (.001)	[0.022, 0.027]	0.025 (.001)	[0.022, 0.027]
Stress exposure	0.037 (.002)	[0.033, 0.040]	0.037 (.002)	[0.033, 0.040]	0.037 (.002)	[0.033, 0.040]
Stress reactivity	0.014 (.002)	[0.010, 0.019]	0.013 (.002)	[0.009, 0.018]	0.014 (.002)	[0.010, 0.019]
Stress reactivity change	0.001 (.000)	[0.001, 0.001]	0.001 (.000)	[0.001, 0.001]	0.001 (.000)	[0.001, 0.001]
Residual variance						
Wave 1 health	0.089 (.011)	[0.066, 0.111]	0.153 (.023)	[0.113, 0.201]	1.208 (.102)	[1.008, 1.403]
Wave 2 health	0.184 (.008)	[0.169, 0.201]	0.285 (.013)	[0.261, 0.310]	1.278 (.062)	[1.164, 1.404]
Wave 3 health	0.184 (.018)	[0.148, 0.218]	0.189 (.029)	[0.136, 0.251]	1.358 (.127)	[1.118, 1.616]
Health intercept	0.142 (.058)	[0.059, 0.261]	0.454 (.045)	[0.361, 0.536]	1.570 (.369)	[0.854, 2.256]
Health slope	0.036 (.016)	[0.007, 0.064]	0.048 (.020)	[0.016, 0.089]	0.117 (.076)	[0.018, 0.297]

Abbreviations: ADL, activities of daily living; CrI, credibility interval; IADL, instrumental activities of daily living; NA, negative affect; *pSD*, posterior standard deviation.
Results are based on 33 944 daily assessments ($N = 2880$). Estimates of fixed effects are reported as unstandardized regression coefficients. Estimates of random effects are reported as variances.
[a] $P < .05$.
[b] $P < .01$.
[c] $P < .001$.

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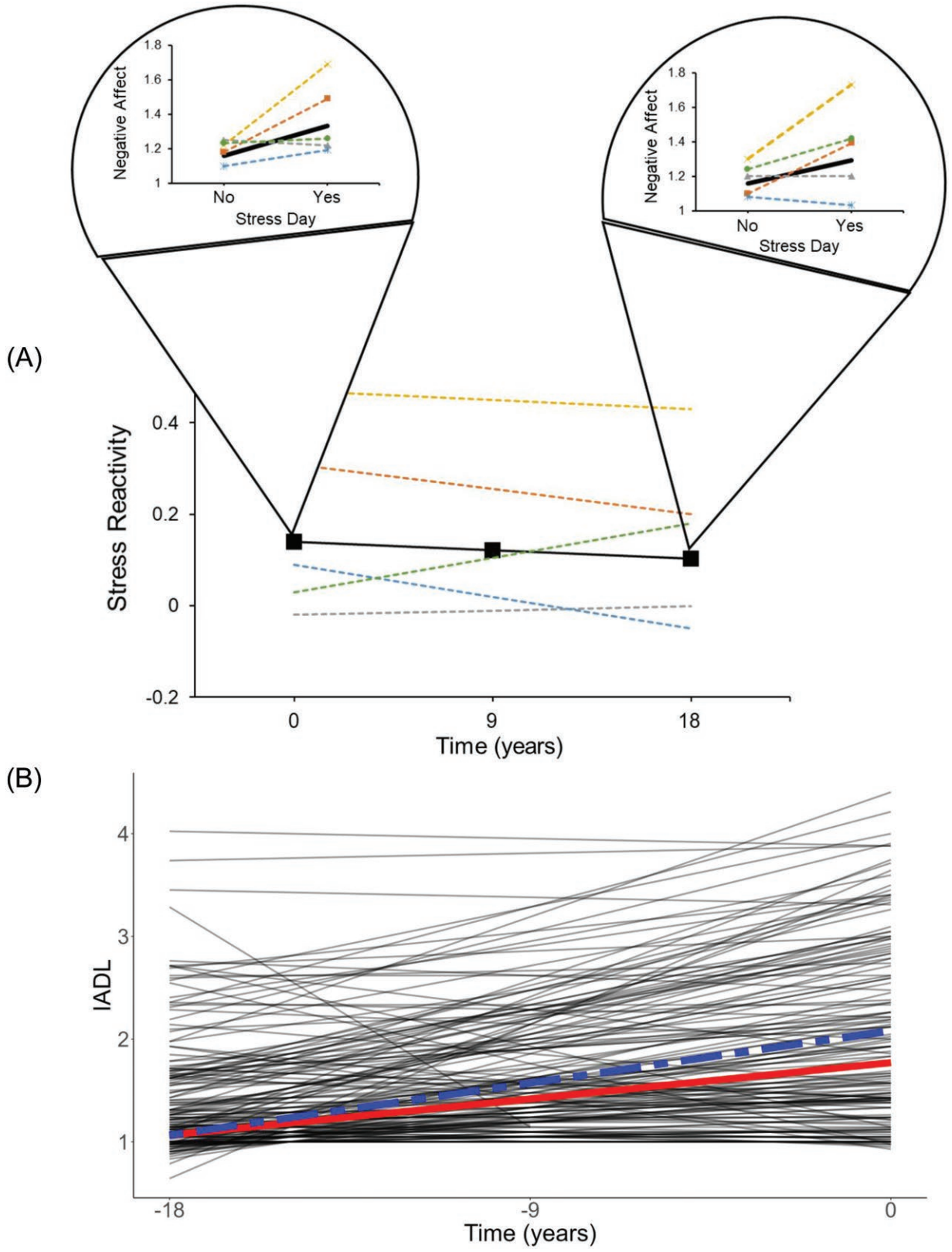

Figure 3. (A) Change in within-person association between stress and NA (ie, stress reactivity) across 3 bursts (18 years). Black square (solid line) represents average within-person association between stress and NA and change in average within-person association across bursts (Δstress reactivity = −0.02 per 10 years, $P < .001$). Colored dotted lines represent individual participants with varying strengths of within-person association within and across bursts. Lines of the same color represent same individual across bursts. (B) Change in functional health limitations (IADL) across 3 waves (18 years). The thick solid (red) line represents average change in IADLs across waves (*slope* = 0.39 per 10 years, $P < .001$). The thick dashed (blue) line represents change in IADLs for individual who increased slightly in stress reactivity (Δstress reactivity = +0.01). The thin black lines represent individual trajectories.

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for changes in health across 18 years of adulthood (ie, Slope). Results from the MSEM confirmed this hypothesis for functional limitations and chronic conditions. Individuals who became more reactive to daily stressors across the 18-year period (relative to those who were stable or became less reactive) also increased more in their functional health limitations (both ADLs and IADLs) and number of reported chronic health conditions (see Figure 2 and Table 2; slope on stress reactivity change). For a 1-unit difference in stress reactivity change, individuals increased by 0.4023 and 0.5740 units more in limitations of ADLs and IADLs per year. Interpreting these values further, the average person decreased in stress reactivity by 0.02 units per decade (fixed effect of stress reactivity change = −0.02), and also the average person increased in their ADLs by 0.0203 units per year (ADL slope = 0.203 per decade). Though unlikely for there to be a 1-unit difference in stress reactivity change, the difference between the typical person and someone who showed a slight increase in their stress reactivity (eg, stress reactivity change slope = +0.01) would be estimated to increase in their ADL limitations by 0.032 units per year (0.03 × 0.4023 = 0.012 units more per year than the average ADL change of 0.0203), which is 37% faster than the average individual. Across the 18 years, an individual increasing in stress reactivity would be estimated to have increased in their ADL score by 0.576 units (on a 4-point scale) compared to the average increase of 0.365 units.

Similarly, an individual increasing in stress reactivity (stress reactivity change = +0.01) would be estimated to have increased in their IADL score by 1.017 units across the 18 years compared to an average increase of 0.707 units, reflecting a 30% greater increase (see Figure 3B). The average individual was estimated to increase by 1.490 chronic conditions across the 18 years, whereas an individual increasing in stress reactivity was estimated to increase by 2.094 chronic conditions, reflecting a 29% greater increase.

Stress reactivity levels at baseline did not uniquely account for changes in functional health limitations nor changes in chronic conditions (Table 2, slope on stress reactivity). Thus, once we account for how individuals change in their stress reactivity, those who were more reactive initially did not differ in their rates of change in health relative to those who were less reactive at baseline.

Accounting for wave 3 health.

Changes in stress reactivity also accounted for levels of health 18 years later at wave 3 (see Table 2; intercept on stress reactivity change). Individuals who became more reactive across the 18-year period had higher levels of basic functional limitations (ADLs) and more chronic health conditions at wave 3 relative to individuals who decreased in their reactivity. For a 1-unit increase in stress reactivity change, ADLs were 11.35 units higher at wave 3 and chronic conditions were 25.43 units higher. Therefore, an individual who had a stress reactivity change score of +0.01 would be expected to report their functional limitations 0.34 units higher at wave 3 (on a scale from 1 to 4) than an individual who changed the average amount in their stress reactivity (ie, their stress reactivity change score was −0.02; 11.35 × 0.03 = 0.34). This amounts to a 19% higher ADL value (1.829 vs. 1.489). Similarly, an individual slightly increasing in stress reactivity is estimated to report 2.77 chronic health conditions at wave 3 compared to an individual changing the typical amount in stress reactivity, who is estimated to report 2.01 chronic health conditions at wave 3 (a 27% difference).

Initial level of stress reactivity was also related to health at wave 3. Those who were more reactive to daily stressors at baseline had significantly more functional health limitations (both ADLs and IADLs) at wave 3, 18 years later (Table 2; intercept on stress reactivity). These results were present after adjusting for age, sex, education, and average levels of both NA and stress exposure. Furthermore, higher average level of NA was reliably related to greater increases and levels of functional limitations and chronic conditions, whereas higher levels of stress exposure were only related to greater levels of chronic conditions at wave 3. Figure 2 displays the unstandardized estimates from the 3-level MSEM predicting ADLs.

Discussion

Stress reactivity is often linked to poorer health outcomes (eg, [1,2]). Consistent with prior findings showing effects across almost 10 years,[2] levels of stress reactivity at baseline predicts worse functional health 18 years later. The current study further examined not only baseline levels, but whether *changes* in stress reactivity are related to poorer health. Results revealed that changes in stress reactivity are associated with changes in functional health and chronic illness across an 18-year period, indicating that people who become more emotionally reactive to daily stressors over time also decrease in their physical functioning and increase in their number of chronic illnesses. When examining the relative effects of these findings, the degree to which people change in reactivity over time is a greater predictor of their ADLs, IADLs, and chronic conditions than their level of reactivity at baseline.

Previous research on the link between stress reactivity and health over time was primarily based on 2 waves of data across a 9-year follow-up, with stress reactivity scores predicting health outcomes years later.[2,9] What is unclear from this initial work is: (1) the extent to which stress reactivity changes over time, and (2) whether these concomitant changes are related to health outcomes beyond baseline effects. The current study, which incorporated 2 additional waves of NSDE data spanning 18 years of follow-up, augments this research by illustrating that people often change in their levels of stress reactivity over time[3,14] and that this change in reactivity maps onto changes in health outcomes over time. Consistent with prior findings, our models show that stress reactivity declines across adulthood.[3] Results are also consistent with well-established findings that functional limitations and chronic illnesses increase with age.[5] These contrasting trajectories of declining stress reactivity paralleling increasing health problems appear to present a paradox, until we examine within-person patterns over time. People who increase in reactivity, or who decrease to a lesser extent than their peers, are also those who have increased functional limitations and chronic illnesses. In addition, changes in stress reactivity are more strongly related to changes in health than baseline levels of stress reactivity. One potential explanation for this finding is that the levels of stress reactivity from where a person begins is not as important for their health as how they change in reactivity over time.

The findings above could be interpreted as indicating a causal link, where greater stress reactivity leads to worse health outcomes. This interpretation is consistent with theories positing that chronic, high levels of stress-related

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physiological arousal create wear and tear on the system, leading to poorer health, and with past studies showing that greater stress reactivity elevated the risk for subsequent physical health problems years later.[1,2,7] Unfortunately, the research design currently makes it impossible to confirm this possibility, because the data could also be interpreted such that declines in health led to increased stress reactivity. With additional measurement bursts, we will be able to test lagged associations to determine whether changes in stress reactivity precede or follow changes in physical health, or perhaps bidirectional associations often observed between physical and emotional experiences.[43]

Just as stress researchers offer predictions leading from greater stress reactivity to poorer health, health researchers theorize that poorer physical health leads to greater distress. People with physical disabilities report lower well-being than people without physical disabilities.[44,45] In a study examining the directional nature of this phenomenon, Lucas[46] examined happiness trajectories several years before and after the onset of physical limitations. People experienced moderate to large drops in reported happiness at the time of disability and remained at these lower levels in the ensuing years. Similarly, prior research has found that people report higher levels of depressive symptoms and loneliness for several years after experiencing a stroke.[47,48] Moreover, changes in emotional well-being following the onset of physical disability appear to be relatively long-lasting.[49–51] Consistent with this past research, it is possible that health declines may lead to enduring changes in daily emotional processes such as stress reactivity. It is also possible that these processes feed into one another, such that health declines lead to greater stress reactivity, which then leads to more health declines.

It remains an open question whether the temporal structure of the NSDE maps onto the timescale of any underlying causal processes. For example, prior research found that year-to-year changes in daily emotional well-being coincided with year-to-year changes in physical health, but changes in one did not predict subsequent changes in the other.[52] This suggests that if lagged relationships exist, they may occur on a more rapid timescale than can be captured with traditional widely spaced longitudinal and measurement burst designs. Perhaps that is why baseline reactivity predicts chronic health conditions at 10-year follow-up, but not 18 years later.[2] Importantly, regardless of the causal direction of the observed association, the current findings suggest that the daily emotional lives of people experiencing health declines are characterized by increases in stress reactivity. Given the widespread harmful effects of stress reactivity, this suggests that stress reactivity may be an important target of intervention[53] among people experiencing physical health declines.

The present findings need to be interpreted within the context of the study. This study used data from NSDE, the largest publicly accessible longitudinal daily diary study in existence. This study has provided much needed insight into how reactivity to daily stressors change over time, and how age-related changes compared to age differences in cross-sectional studies. These findings, however, pertain to the cohorts and population sampled for this study, and it remains untested whether a different study sampling different cohorts and assessing stress reactivity over time will yield the same results. Future studies examining different cohorts of aging adults will allow us to determine whether findings are specific to certain cohorts or generalize across time.

In addition, the present study operationalized stress reactivity as the difference in NA between stressor days and non-stressor days. We used this operationalization to assess how an identified event (as opposed to an emotional mood or appraisal) was related to emotional distress. However, future research should consider other operationalizations of stress reactivity to better understand temporal dynamics within and across stressor versus non-stressor days and potential carryover effects of stressors on NA (see[54]). Furthermore, future studies examining stress reactivity over time could provide information regarding whether these findings generalize to different types of stressors or different aspects of stress reactivity (eg, reactivity to stressor intensity). A potential limitation of the current operationalization includes the less than optimal within-person reliabilities of the NA scale. However, within-person reliability estimates are typically lower than between-person estimates[55] and the magnitude of the within-burst association between stress and NA was consistent with other studies that have used more reliable NA measures (eg, [6]).

The following constraints on generalizability should also be considered when interpreting the present findings. The current study included a national probability sample of US adults that was diverse with respect to age, gender, socioeconomic status, and geographical location within the United States. However, the sample was majority non-Hispanic white and does not represent the current racial and ethnic composition of the United States, nor did it include any individuals from outside of the United States. Theoretically, increases in stress reactivity should be associated with worse physical health regardless of race, ethnicity, culture, or geographic region. However, individuals from minoritized sociocultural groups experience unique stressors in the form of chronic and daily discrimination,[56] and thus it is important to consider the effects of those unique stress experiences on links between stress responses and health outcomes. For example, past work on a phenomenon called "skin-deep resilience" has found evidence that in some contexts, psychological resilience may come at a paradoxical cost to physical health among particular minoritized groups (eg, [57–59]). Moreover, the daily stress experiences of individuals living in low- and middle-income countries are likely to be quite different from the daily stress experiences of individuals living in the United States. Thus, future research should seek to conceptually replicate the current findings in more racially and geographically diverse samples.

The findings have implications for behavioral medicine. Understanding health through dynamic associations allows for the identification of personalized health patterns. This approach can inform tailored interventions that align with an individual's daily routines, behaviors, and stress responses, offering a more precise method to promote health and well-being.[60] Furthermore, a dynamic characterization of daily stress has broader implications for public health, as it allows policymakers to identify population-level trends in stress and outcomes. Such insights can guide the development of more effective, evidence-based public health strategies that account for the daily realities of diverse groups.[61]

Conclusion

This is the first study, to our knowledge, to extend findings from the literature focused on how higher levels of baseline

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stress reactivity is related to future health by examining whether changes in stress reactivity over time accounted for changes in health. Findings reveal that changes in stress reactivity are related to changes in health, specifically to changes in functional health and chronic illness. These findings highlight the strong connection between health and stress in daily life, and how they travel together across adulthood. Moreover, because change in reactivity is more predictive of some health outcomes than is baseline reactivity, there is hope that decreasing our reactivity could potentially result in improved health outcomes and vice versa.

Author contributions

Jonathan Rush (Conceptualization, Formal analysis, Methodology, Visualization, Writing—original draft), Susan T. Charles (Conceptualization, Writing—original draft, Writing—review & editing), Emily C. Willroth (Conceptualization, Writing—original draft, Writing—review & editing), Eric S. Cerino (Conceptualization, Formal analysis, Writing—review & editing), Jennifer R. Piazza (Conceptualization, Writing—review & editing), and David M. Almeida (Conceptualization, Funding acquisition, Resources, Writing—review & editing)

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Conflicts of interest

None declared.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee.

Transparency statements

This study's design and its analysis were not pre-registered on any official website, although plans for these specific hypotheses using these data were articulated in the NIH grant: U19-AG051426-07. Data are publicly available and can be found at the following website: https://www.icpsr.umich.edu/web/ICPSR/series/203. In addition, a MIDUS-Colectica Portal (https://midus.colectica.org) contains rich searchable metadata, links to helpful documentation, and the ability to download customized datasets. All analyses were completed using Mplus v8.10[42], and both scripts and output are available upon request from the corresponding author. These data have been publicly accessible for over 20 years, with numerous studies including measures of health, daily stress, and negative affect from both the MIDUS survey and the daily diary project (see the following website for a list of publications: www.midus.wisc.edu/findings/index.php). No study has examined predictive associations among longitudinal trends across the 3 waves of data presented here.

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