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Associations of Personality Trait Level and Change With Mortality Risk in 11 Longitudinal Studies

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unsuccessful adaptation to life circumstances, which in turn influences mortality risk, or shared risk factors may impact personality trait change and mortality risk. In the latter case, personality trait change may serve as a "psychosocial vital sign" pointing toward increased risk. In 11 samples of middle-aged and older adults (combined N = 32,348), we used multilevel growth curve models to estimate personality trait level and personality trait change across three to 11 measurement occasions spanning 6–43 years. Next, we used Cox proportional hazards models to test whether personality trait level and personality trait change were associated with mortality risk. Higher conscientiousness (hazard ratio [HR] = 0.83), extraversion (HR = 0.93), and agreeableness (HR = 0.88) were associated with longer survival while higher neuroticism was associated with shorter survival (HR = 1.22). In contrast to personality trait level, we found limited evidence for associations between personality trait change and mortality risk. We discuss conceptual and methodological implications of the present findings that may guide future research on associations between personality trait change, health, and mortality.

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The research questions, hypotheses, and analytic approach were preregistered on the Open Science Framework at https://osf.io/kf2hn. Statistical analysis code can also be found on the Open Science Framework at

PERSONALITY TRAJECTORIES AND MORTALITY

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Big Five personality traits consistently predict mortality risk (e.g., Costa et al., 2014; Friedman et al., 1995; Graham et al., 2017; Jokela et al., 2013). Conscientiousness, agreeableness, extraversion, and low neuroticism are theorized to promote positive health behaviors, adaptive social functioning, and positive stress profiles that benefit health and ultimately support longevity. Theory and meta-analytic evidence suggest that these personality traits change across the lifespan (Ardelt, 2000; Bleidorn et al., 2022; Graham et al., 2020; Roberts & DelVecchio, 2000; Roberts & Mroczek, 2008; Roberts et al., 2006), yet little is known about how personality trait change is associated with longevity. In 11 independent samples of middleaged and older adults, the present research tested the hypothesis that personality trait change in midlife and old age is associated with mortality risk, above and beyond personality trait level. In the following sections, we describe theoretical models and empirical evidence for why individuals differ from one another in the direction or degree of personality trait change and why this might matter for health and longevity.

Individual Differences in Personality Trait Change

Mean-level personality trait change is greatest in adolescence and young adulthood with a peak around age 20 (Bleidorn et al., 2022; Roberts & Davis, 2016; Roberts & DelVecchio, 2000;

https://osf.io/kwcd7/. Four of the studies used in the present research have publicly available data; the remaining seven studies require an application process for data access. Details about data access are described in Supplemental Table S6. Emily C. Willroth was affiliated with Northwestern University when the majority of this research was conducted and with Washington University in St. Louis during the final stages of the writing and revision process. Emily C. Willroth presented findings from the current research at the Society for Personality and Social Psychology 2023 Annual Conference in Atlanta, GA. The views expressed in this article are those of the authors and do not necessarily represent the views of the National Institutes of Health, the U.S. Department of Veterans Affairs, or other support institutions.

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Roberts & Mroczek, 2008), but accumulating evidence suggests personality trait change continues into middle adulthood and may even accelerate in older adulthood (Bleidorn et al., 2022; Specht et al., 2011). In fact, cumulative mean-level change across the adult lifespan exceeds one full standard deviation for many Big Five traits (Bleidorn et al., 2022). On average, personality trait change trends in the direction of greater psychological maturity across most of the lifespan (Bleidorn et al., 2013; Graham et al., 2020; Roberts & Mroczek, 2008; Roberts & Nickel, 2021; Roberts & Wood, 2006; Specht et al., 2011, 2014), with increases in agreeableness in young adulthood, increases in conscientiousness through young adulthood and midlife, and decreases in neuroticism across the entire adult lifespan (Bleidorn et al., 2022). In older adulthood, meta-analytic evidence suggests that neuroticism continues to decrease, but trajectories of the other traits reverse, resulting in late-life decreases in all Big Five traits (Bleidorn et al., 2022). Importantly, however, not everyone follows these normative developmental trends. Instead, individuals' personality trait change trajectories vary around these population-level trends with some individuals changing more than others and some not changing at all for one or more traits or across all traits (e.g., Mroczek & Spiro, 2003; Schwaba & Bleidorn, 2018).

Theoretical models that describe population-level patterns of personality trait change provide useful foundations for considering drivers of individual differences in personality trait change. For

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example, neo-socioanalytic theory (Roberts & Nickel, 2021) posits that the expectations that accompany social roles impact personality development and that investment in new social roles drives personality trait change in the direction of increases in socially desirable traits (i.e., maturation; Bleidorn et al., 2013). As such, individual differences in personality trait change may reflect differences in the degree to which individuals take on and invest in new social roles, how successfully they adapt to those new social roles, and how they respond to the loss of social roles. Greater investment in or adaptation to new social roles should lead to greater-than-average personality maturation, whereas less investment in or difficulties adapting to new social roles should lead to less-than-average maturation or even personality trait change in the opposite direction of maturation. In midlife, some people invest in and adapt to increasing occupational and caregiving roles while others take on fewer responsibilities or struggle to adapt to the increasing demands that accompany their new roles. In older adulthood, this may manifest as some people adapting better to retirement and the loss of primary caregiver roles than others. Moreover, the Triggering situations, Expectancies, States/State expressions, and Reactions framework posits that personality trait change results from repeated short-term situational processes, which are likely to differ between individuals (Wrzus & Roberts, 2017).

We can also consider theories of late-life development, which posit that older adults select into and out of particular goals, experiences, and behaviors as they age (Heckhausen & Schulz, 1995). In turn, individual differences in these selection strategies may underlie individual differences in personality trait change. Socioemotional selectivity theory (Carstensen, 1993, 2006, 2021; Carstensen et al., 1999) and the theory of strength and vulnerability integration (Charles & Luong, 2013) suggest that as people age, they are more likely to prioritize positive emotional experiences and employ emotion regulation strategies that limit negative emotion experiences. Thus, as people age, they may be more likely to pursue positive emotional experiences that lead to decreases in neuroticism. Moreover, the selection, optimization, and compensation theory posits that older adults experience an increasing ratio of losses to gains as their physical, social, and cognitive functions decline (Baltes, 1997; Baltes & Baltes, 1990; Baltes et al., 2006; Freund & Baltes, 1998, 2002). To optimize development in response to these losses, older adults may select out of conscientious behaviors associated with occupational goal pursuit, may engage in less extraverted and agreeable behaviors associated with large social networks, and may become less open to novel intellectual and cultural experiences (Kandler et al., 2015). Together, these theories can explain normative declines in all Big Five traits in older adulthood (Bleidorn et al., 2022). At the same time, individual differences in selection strategies may underlie individual differences in personality trait change. For example, older adults who have the physical, social, and cognitive resources necessary to select into emotionally meaningful and enjoyable experiences may experience greater-than-average decreases in neuroticism, whereas older adults who experience difficulties coping with late-life challenges may increase in neuroticism.

Personality Trait Change, Health, and Mortality

In this section, we describe multiple pathways by which individual differences in personality trait change may be linked to mortality risk and then turn to empirical evidence for personality trait change as a predictor of health and mortality. First, personality trait change may be linked to mortality risk through the same factors that link personality trait level with mortality risk, such as health behaviors (e.g., Mroczek et al., 2009; Turiano et al., 2015), social processes (e.g., Holland & Roisman, 2008), and stress experiences (e.g., Bogg & Roberts, 2013; Hampson, 2019). For example, conscientiousness has been associated with more positive health behaviors, less stressor exposure (Lee-Baggeley et al., 2005; Vollrath, 2000), and greater ability to cope with stressors when they do occur (Connor-Smith & Flachsbart, 2007; Penley & Tomaka, 2002). Longitudinal evidence suggests that people who increase in conscientiousness experience less stress over time, and these increases in conscientiousness and decreases in stress preceded changes in self-perceived physical health (Luo & Roberts, 2015). By contrast, neuroticism has been associated with greater stressor exposure and greater stress vulnerability (Schneider, 2004; Wang et al., 2018), suggesting that changes in neuroticism may alter stress experience across the lifespan. It has also been posited that increases in socially desirable traits may be associated with less stress from negative social interactions (Hampson, 2019). This may be especially true for extraversion and agreeableness, given that these traits are inherently social or interpersonal in nature. In sum, personality trait change may causally influence health and mortality risk by shifting traits in the direction of protection (i.e., protective health behaviors, more positive social relations, and better stress experience) or vulnerability (i.e., detrimental health behaviors, less positive social relations, and worse stress experience).

Personality trait change may also be a marker of successful or unsuccessful adaptation to life circumstances, which in turn may influence health and mortality risk. Successful adaptation to challenges in middle and older adulthood may confer health benefits and may be reflected in maturational patterns of personality trait change including increases in conscientiousness, extraversion, and agreeableness and decreases in neuroticism. Conversely, difficulties coping with challenges in middle and older adulthood may harm health and may manifest as increases in neuroticism and decreases in socially desirable traits. Finally, personality trait change may be associated with mortality risk due to shared risk factors. For example, the loss of a spouse has been associated with increases in both neuroticism (Mroczek & Spiro, 2003) and mortality risk (Boyle et al., 2011). Perhaps the most obvious shared risk factor for personality trait change and mortality risk is the onset of disease. A pooled analysis of four large samples found that the onset of a chronic disease preceded decreases in conscientiousness, extraversion, and openness and increases in neuroticism (Jokela et al., 2014). It has also been posited that increased vigilance to health threats in late life, which may reflect individual differences in actual health threats, may lead to increases in neuroticism (Kandler et al., 2015).

Taken together, personality trait change may causally impact mortality risk, or it may be the result of events or experiences that occurred earlier in the causal chain. These two explanations are complementary rather than competing. It is possible that personality trait change causally impacts mortality for some people. For others, an earlier confounding event such as the onset of chronic illness may cause personality trait change and increased mortality risk. Importantly, even if personality trait change is not causally related to mortality risk, it may serve as a "psychosocial vital sign" pointing toward increased risk (Matthews et al., 2016). The processes linking personality trait change to mortality may include mechanisms that are both shared and unique across the Big Five traits. Increases in

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conscientiousness in middle and late life are likely associated with increases in protective health behaviors (e.g., Takahashi et al., 2013), reductions in risky health behaviors, better management of health conditions, reductions in stressor exposure, and improved coping with midlife and late-life stressors, all of which may contribute to longevity. Increases in agreeableness and extraversion may support the maintenance and formation of protective social bonds throughout middle and older adulthood, which in turn promote longevity. Decreases in neuroticism may support longevity by reducing the likelihood of engaging in harmful health behaviors and reducing stressor exposure and reactivity. Conversely, increases in neuroticism may give rise to maladaptive coping strategies (e.g., substance use, rumination) that are harmful for health or may reflect increased vigilance in response to health challenges that precede or contribute to mortality. When considering processes that may be shared across several traits, successful adaptation to the challenges in middle and older adulthood may amplify normative maturational patterns of increases in conscientiousness, agreeableness, and extraversion and decreases in neuroticism.

Consistent with these possibilities, personality trait change has been associated with a variety of health outcomes, including selfrated health (Letzring et al., 2014; Magee et al., 2013; Turiano et al., 2012), health status (Wright & Jackson 2024a), physical fitness (Mõttus et al., 2012), and metabolic syndrome (Human et al., 2013; Magee et al., 2013). Likewise, research suggests a relationship between personality trait stability, in contrast to personality trait change or variability, and maintaining higher levels of cognitive function (Graham & Lachman, 2012; Klimstra et al., 2013; Terracciano et al, 2017). In addition, conscientiousness levels assessed within the same individuals in both childhood and adulthood have been shown to independently predict mortality (Martin et al., 2007), suggesting that nonstatic components of personality traits may be important for mortality.

Two studies have directly tested associations between personality trait change and mortality risk (Mroczek & Spiro, 2007; Sharp et al., 2019). Mroczek and Spiro (2007) found that men with high and increasing neuroticism had higher mortality risk than men without this combination; no such effect was observed for extraversion, and repeated assessments of the other three Big Five traits were not available. Results held when statistically adjusting for self-rated health, a measure of serious health conditions, and depressive symptoms. Sharp et al. (2019) found that decreases in openness in late life, but not neuroticism or extraversion, were associated with higher mortality risk, including after statistical adjustment for sociodemographic characteristics, physical illness, depressive symptoms, and cognitive ability. This finding was interpreted as reflecting a change in goal orientation in response to a shortened time horizon at the end of life, consistent with selection, optimization, and compensation theory (Baltes, 1997; Baltes & Baltes, 1990; Baltes et al., 2006; Freund & Baltes, 1998, 2002). These two studies provide initial evidence that personality trait change may be associated with mortality risk. Both studies were conducted within single samples that were limited in terms of gender (Mroczek & Spiro, 2007) or age (Sharp et al., 2019), and neither study examined all Big Five traits. Evidence for changes in specific traits predicting mortality risk were mixed across the two studies. The present research will address these limitations by examining associations between change in all Big Five traits and

mortality risk across 11 independent samples, providing strong tests of replicability and generalizability.

The Present Research

The present research addressed limitations of the sparse prior research on personality trait change and mortality risk by examining change in all Big Five traits as predictors of mortality risk across 11 samples that are diverse in terms of gender, age, and country of origin. Based on prior research examining associations between personality trait level and mortality risk (e.g., Graham et al., 2017), we preregistered the following predictions: increases in conscientiousness, agreeableness, and extraversion and decreases in neuroticism would be associated with greater longevity; openness change would not be associated with longevity. At the time of preregistration, we were not aware of Sharp et al.'s (2019) finding of late-life increases in openness preceding mortality. However, it is not clear whether that finding would be expected to extend to changes in openness throughout midlife and older adulthood, or if it is specific to the end of life. In preregistered exploratory analyses, we also tested interactions between personality trait level and personality trait change predicting mortality risk. Although we did not make specific predictions concerning these interactions, there is some evidence from prior research that the combination of level and change may be associated with risk, such that the combination of high and increasing neuroticism is particularly harmful (Mroczek & Spiro, 2007).

Method

Ethics Statement

The Northwestern University Institutional Review Board (Protocol No. STU00207203-CR0003) approved the present research. After the lead author switched institutions, the Washington University in St. Louis Institutional Review Board approved the continuation of this research (Protocol No. 202207090).

Transparency and Openness

The research questions, hypotheses, and analytic approach were preregistered on the Open Science Framework (https://osf.io/kf2hn). Although we did our best to follow the preregistered plan as closely as possible, some deviations were necessary due to limitations of the data, and other deviations were suggested during the peer review process. Table 1 describes deviations from our preregistered plan, including a description of each deviation, the type of deviation, the reason for the deviation, and the timing of the deviation (see Willroth & Atherton, 2024, for a discussion of preregistration deviation reporting).

We used R versions 4.0. through 4.4. for all analyses. Statistical analysis code can also be found on the Open Science Framework (https://osf.io/kwcd7/). The present research involved secondary analysis of 11 existing studies. Sample size was predetermined based on the number of participants in each sample that met the inclusion criteria, which were preregistered and reported in the article. The measures used in the present research were drawn from these larger studies and were also preregistered. Four of the studies used in the present research have publicly available data; the remaining seven studies require an application process for data access. Details about data access are described in Supplemental Table S7.

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 Table 1

 Preregistration Deviations Table

No.	Detail type	Original wording	Deviation description	Reader impact
Devii 1	Deviations 1 Type: Sample Reason: New knowledge Timing: After data access	"We will conduct a coordinated analysis of 15 datasets."	We dropped three samples due to very low mortality rates (<5%) and one sample that had fewer than three measurement occasions of personality, resulting in the inclusion of 11	Including these data sets would result in lower data quality and underpowered findings. Moreover, even without these studies, we still report findings across 11 large data sets. Thus, the
0	Type: Analysis Reason: Plan not possible Timing: After results known	"In exploratory analyses, we will model quadratic change in the growth curve models for every study with four or more personality measurement occasions. Then, we will predict mortality from quadratic personality change in Cox regression models."	sumptes. The quadratic models were often too complex for the available data, resulting in model convergence problems and relatively uninterpretable nonproportional hazards (e.g., interactions between quadratic personality trait change and time in study), which led us to drop	Impact on study concustons is immed- on onission of the quadratic models prevented us from drawing conclusions about potential associations between nonlinear personality trait change and mortality. However, including these results may have led to overinterpretation of complex underpowered interaction effects.
ξ	Type: Analysis Reason: Peer review Timing: After results known	"Time will be assessed as age in years, centered at 60."	tness exproratory analyses from our investigation. During the peer review process, a reviewer pointed out that time in study centered at each participant's study midpoint is a more appropriate time metric for the growth curve models because this approach simply models a person's change over time rather than producing a biased, age- dependent slope.	Trait level estimates can now be interpreted as trait level at each person's study midpoint. Trait change estimates can now be interpreted as change over the study period. Results were relatively similar across the two approaches; however, this change resulted in lower correlations between trait levels and trait change, fewer violations of the proportional hazards assumption, fewer implausible point estimates, and smaller standard errors.
No.	Detail type	Original wording	Unregistered step description	Reader impact
Unre 1	Unregistered steps 1 Type: Analysis Timing: After results known	NA	In our preregistration, we neglected to specify that we would not proceed with testing Cox proportional hazards models when individuals did not significantly differ from one another in personality trait change. This was an oversight in the initial preregistration.	It does not make conceptual sense to investigate associations between individual differences in personality trait change if no such individual differences are observed. However, the absence of statistically significant individual differences for some traits in some data sets is an interesting and important finding in itself that may impact
6	Type: Analysis Timing: After results known	NA	We added unplanned sensitivity analyses comparing the results of our primary analytic approach to results from joint growth-survival models.	the way that readers interpret the study findings. Because conclusions converged across primary and sensitivity analyses, the addition of these analyses should increase reader's confidence in the overall study conclusions.

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Study and Sample Characteristics

We selected studies with at least three measurement occasions of personality (which is necessary to model personality trait change with growth models) and with long enough follow-up periods to observe at least 5% mortality (which is necessary to be sufficiently powered to predict mortality risk in the survival models). We included 11 studies, the majority of which are a part of the Integrative Analysis of Longitudinal Studies on Aging and Dementia (IALSA) network (Hofer & Piccinin, 2010), including several studies with publicly available data independent of their IALSA affiliation. These studies involve population cohorts; thus, although the IALSA network is interested in dementia, the samples have typically low prevalence rates of dementia. Within each sample, participants met eligibility if they had personality trait data at the first measurement occasion and personality trait data at one or more additional measurement occasions (i.e., two or more total measurement occasions). Although growth curve modeling requires three or more measurement occasions at the study level, participants with fewer measurement occasions can be included. We chose to include participants with two or more measurement occasions to balance competing goals of maximizing the number of participants included in analyses while excluding participants with only one measurement occasions since no personality trait change could be observed for those participants.

Sample characteristics are shown in Table 2. Across samples, the mean age at analytic baseline (i.e., the first personality assessment) ranged from 47 years old to 83 years old. Thus, across samples, personality trait change was assessed in either midlife, older adulthood, or across midlife through older adulthood. Studies are presented in order from the youngest to oldest mean age at analytic baseline. The youngest sample in the present research is the Midlife in the United States (MIDUS) study, which is a longitudinal probability sample of U.S. adults. The Veterans Affairs Normative Aging Study (NAS) is a longitudinal study of men residing in the Boston area that was initiated by the U.S. Department of Veterans Affairs in 1963 (Bell et al., 1972). The Wisconsin Longitudinal Study (WLS) is a longitudinal cohort study of high school graduates,

Table 2

Study and Sample Characteristics

born primarily in 1939, as well as a randomly selected sibling from each member of the WLS cohort (Herd et al., 2014). The Swedish Adoption/Twin Study of Aging (SATSA) is a longitudinal study of twins drawn from the Swedish Twin Registry (Finkel & Pedersen, 2004; Pedersen et al., 1991). The Seattle Longitudinal Study (SLS) is a study of psychological development in adulthood that began in 1956 (Schaie et al., 2004). The Health and Retirement Study (HRS) is a longitudinal panel study that surveys a representative sample of U.S. adults (Sonnega et al., 2014). The Longitudinal Aging Study Amsterdam (LASA) is a longitudinal study of older adults in the Netherlands (Huisman et al., 2011). The Lothian Birth Cohort 1936 (LBC) is a subset of a Scottish birth cohort who took an intelligence test at age 11 in 1947 and then were recruited into a longitudinal study at age 70 (Deary et al., 2007, 2012; Taylor et al., 2018). The Einstein Aging Study (EAS) is a longitudinal study of older adults over age 70 (Katz et al., 2012). The Berlin Aging Study (BASE) is a study of older adults over age 70 who lived in former West Berlin, Germany (Baltes & Mayer, 2001). Finally, the oldest sample used in the present research, Origin of Variances in the Oldest-Old: Octogenarian Twins (OCTO-Twin), is a longitudinal study of twin pairs over age 80 (McClearn et al., 1997).

Measures

Personality Traits

All samples included at least a subset of the Big Five traits (conscientiousness, neuroticism, extraversion, agreeableness, openness) assessed longitudinally at three or more measurement occasions using different measures of the Big Five. Table 3 shows the personality inventory, number of personality measurement occasions, and length of the personality assessment period for each study. Supplemental Table S1 displays additional information about the personality inventories used across studies, including the total number of items, the response scale, and example items. Figure 1 shows the time period in which personality trait change was assessed in each sample.

Sample	Analytic <i>N</i> (participant)	Analytic <i>N</i> (measurement occasion)	Country of data collection	Year of baseline assessment	% Female	M (SD) baseline age
MIDUS	4,092-4,093	10,766-10,805	United States	1994–1995	55	47.2 (12.4)
NAS	1,606	8,606-8,630	United States	1975	0	51.0 (8.8)
WLS	9,394-9,397	24,856-25,487	United States	1992/1993	54	53.0 (4.2)
SATSA	1,647-1,703	7,721-8,540	Sweden	1984	59	58.6 (13.8)
SLS	1,083	3,565	United States	2001	56	62.2 (14.4)
HRS	11,028-11,031	29,192-29,387	United States	2006/2008	60	67.3 (9.7)
LASA	1,623	4,477	The Netherlands	1992	52	68.1 (8.2)
LBC	787	2,663-2,673	United Kingdom	2004-2007	49	69.5 (0.8)
EAS	504-506	1,836-1,883	United States	2005-2016	61	79.0 (5.0)
BASE	210	554-677	Germany	1990-1993	50	79.8 (6.9)
OCTO	309	917–923	Sweden	1991	63	82.6 (2.2)

Note. Samples are shown in order from the youngest to oldest mean age at study baseline. Ranges for analytic N indicate ranges across the Big Five traits. The analytic N includes participants with personality trait data at analytic baseline and at least one additional measurement occasion; thus, it is smaller than the total sample N. MIDUS = Midlife in the United States Study; NAS = Normative Aging Study; WLS = Wisconsin Longitudinal Study; SATSA = Swedish Adoption/Twin Study of Aging; SLS = Seattle Longitudinal Study; HRS = Health and Retirement Study; LASA = Longitudinal Aging Study Amsterdam; LBC = Lothian Birth Cohort; EAS = Einstein Aging Study; BASE = Berlin Aging Study; OCTO = Origin of Variances in the Oldest-Old: Octogenarian Twins Study.

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 Table 3

 Personality and Mortality Assessment
 Characteristics and Descriptive Statistics

Sample	Personality inventory	Max. Person. MO	Max. Person. Follow-up (years)	Baseline conscien. (SD)	Baseline neurot. (SD)	Baseline extrav. (SD)	Baseline agree. (SD)	Baseline open. (SD)	Mortality follow-up (years)	Mortality rate (%)
MIDUS	Midlife Development Inventory (MIDI; Lachman & Weaver. 1997)	ε	18	8.15	4.09	7.32	8.26	6.7	24	14
NAS	Eysenck Personality Inventory Short Form (EPI-Q; Eysenck & Eysenck, 1968: Flodents 1974)	Π	43	NA	3.52	5.91	NA	NA	45	78
MLS	Big Five Inventory (John et al., 1991)	ю	19	7.69	4.42	5.64	7.47	5.25	19	20
SATSA	EPI (Eysenck, 1975; Neuroticism and extraversion) and NEO-Personality	6	30	NA	2.98	5.23	NA	5.99	35	70
	Inventory (Costa & McCrae, 1985; Openness)									
SLS	NEO-Personality Inventory–Revised (Costa & McCrae, 1992)	9	13	6.28	3.94	5.66	6.68	6.10	25	22
HRS	MIDI (Lachman & Weaver, 1997)	ю	8	7.94	3.49	7.38	8.45	6.51	10	16
LASA	Dutch Personality Questionnaire	ю	9	NA	1.99	NA	NA	NA	26	75
	(Luteijn, Starren, & van Dijk, 2000)									
LBC	International Personality Item Pool (IPIP50: Goldhers, 1992)	4	12	7.02	4.02	5.32	7.73	5.98	12	19
EAS	IPIP (Goldberg, 1992)	10	12	7.07	2.67	5.82	7.63	6.71	15	20
BASE	NEO-Five Factor Inventory (Costa & MoCros 1085)	5	13	NA	3.22	6.04	NA	5.43	26	100
OCTO	EPI-Q (Eysenck & Eysenck, 1968)	4	9	NA	2.51	5.60	NA	NA	16	100
<i>Note</i> . Sa Maximum in the Un Study; HF Variances	<i>Note.</i> Samples are shown in order from the youngest to oldest mean age at baseline. Personality traits were rescaled on a 0–10 scale across studies. Baseline <i>SD</i> = 1 for all traits across all studies. Max. = Maximum; Person. = Personality, MO = Measurement Occasion; conscien. = conscientiousness; neurot. = neuroticism; extrav. = extraversion; agree. = agreeableness; open. = openness; MIDUS = Midlife in the United States Study; NAS = Normative Aging Study; NA = not assessed; WLS = Wisconsin Longitudinal Study; SATSA = Swedish Adoption/Twin Study of Aging; SLS = Scattle Longitudinal Study; HRS = Health and Retirement Study; LASA = Longitudinal Aging Study Amsterdam; LBC = Lothian Birth Cohort; EAS = Einstein Aging Study; DASE = Berlin Aging Study; OCTO = Origin of Variances in the Oldest-Old: Octogenarian Twins Study.	t to oldest mean a t Occasion; cons f Study; NA = no = Longitudinal Ag dy.	tean age at baseline. Personality traits were rescaled on a 0–10 scale across studies. Baseline <i>SD</i> = 1 for all traits across all studies. Max. = conscien. = conscientiousness; neurot. = neuroticism; extrav. = extraversion; agree. = agreeableness; open. = openness; MIDUS = Midlife = not assessed; WLS = Wisconsin Longitudinal Study; SATSA = Swedish Adoption/Twin Study of Aging; SLS = Seattle Longitudinal al Aging Study; BASE = Berlin Aging Study; OCTO = Ongitu of al Aging Study; BASE = Berlin Aging Study; OCTO = Ongitu of	nality traits were runess; neurot. = neu Wisconsin Longitu n; LBC = Lothian	escaled on a 0–1 uroticism; extrav dinal Study; SA Birth Cohort; E	0 scale across st = extraversion; TSA = Swedish AS = Einstein A	udies. Baseline agree. = agree 1 Adoption/Twi Aging Study; B.	<i>SD</i> = 1 for al ablences, oper ablences; oper in Study of Ag ASE = Berlin	Il traits across all stuc = openness; MIDU ging; SLS = Seattle J Aging Study; OCTO	lies. Max. = S = Midlife Longitudinal = Origin of

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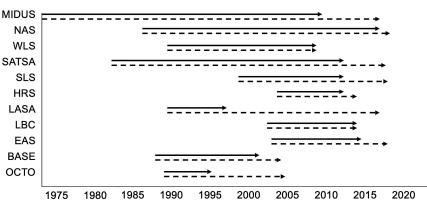


Figure 1 *Personality Trait Change and Mortality Follow-Up by Sample*

Note. The *x*-axis displays calendar year. The solid arrows depict the time period in which personality trait change was assessed in each sample. The dashed arrows depict the length of mortality follow-up in each sample. MIDUS = Midlife in the United States Study; NAS = Normative Aging Study; WLS = Wisconsin Longitudinal Study; SATSA = Swedish Adoption/Twin Study of Aging; SLS = Seattle Longitudinal Study; HRS = Health and Retirement Study; LASA = Longitudinal Aging Study; Amsterdam; LBC = Lothian Birth Cohort; EAS = Einstein Aging Study; BASE = Berlin Aging Study; OCTO = Origin of Variances in the Oldest-Old: Octogenarian Twins Study.

We transformed personality trait scores for comparability across studies by converting each trait to a 0-10 scale (that is, a percent of maximum possible score, scaled to 10 rather than 100 to aid in convergence; Cohen et al., 1999). We then calculated the baseline mean and standard deviation of the transformed scale, standardized the transformed scores based on these new baseline statistics, and finally, added back the baseline mean. The units are interpreted in standard deviation units, and the mean is on a 0-10 scale.

Mortality

All samples included mortality information (death status and death dates), obtained through a reliable source, such as national mortality databases (e.g., U.S. National Death Index, Swedish Death Index) or official death certificates. The precise date of death was masked in some studies to limit reidentification of participants; in these cases, year of death was used in survival analyses. Survival time was calculated in months since analytic baseline. If the month of death was not available, we subtracted the interview year from the death year and multiplied by 12 so that the resulting variable was in units of months. Surviving participants were right-censored at the final mortality follow-up date. Figure 1 shows the mortality follow-up period in each sample.

Covariates

Because months since study entry was used as the survival time metric in the Cox regression models, baseline age (centered at age 60) was included as a covariate to account for age differences in survival odds. In addition, we included self-reported gender (0 = men, 1 = women) and education as covariates to account for potential sociodemographic confounders of the association between personality trait change and mortality.

Baseline education may impact both personality trait change and mortality risk (i.e., a confounding relationship), but it may also be a mediator of the relationship between personality level and mortality risk. Specifically, higher levels of particular personality traits (e.g., higher conscientiousness) may lead to higher educational attainment, which in turn may influence mortality risk. Because it is inappropriate to adjust for a mediator (Rohrer, 2018), we have included results of models in which education was not included as a covariate in Supplemental Figures S1 and S2. The direction and statistical significance of all meta-analytic effects remained the same as in primary models.

Analytic Approach

We modeled our overall analytic approach based on Mroczek and Spiro (2007). First, personality trait level and change were estimated in unconditional growth models using the lme4 package in R (Bates et al., 2015). Second, the associations of personality trait level and personality trait change and mortality risk were estimated in Cox proportional hazards models, adjusting for sociodemographic covariates, using the survival package in R (Therneau, 2022). We used discrete time in study in units of decades centered at each participant's study midpoint as the time metric in the growth curve models estimating personality trait change (e.g., a value of 0.2 indicates 2 years after the participant's study midpoint). We chose to use decades as the unit of measurement to avoid extremely small random slope values and, in turn, extremely large hazard ratios for the personality change associations with mortality. For the Cox proportional hazards models predicting mortality risk, we used time in study in months as the time metric.

We selected this two-step approach rather than joint growth-survival models due to complexities associated with employing joint models in a coordinated data analysis framework, including long computation times and model convergence issues. However, joint growth-survival models afford some benefits over this two-step approach including accounting for error in the estimation of trait change. To balance the strengths of the joint growth-survival model with the challenges of implementing this type of model across a large number of samples and analysts, we conducted joint growth-survival models in half of the samples (MIDUS, WLS, LASA, and EAS) and then compared conclusions across the two modeling approaches. We chose these data sets because (a) the first author had access to the raw data for these data sets whereas coauthors conducted analyses for many of the other data sets; (b) these data sets had limited proportional hazards violations for personality trait level and personality trait change, enabling direct comparisons between the two modeling approaches; and (c) the joint growth-survival models converged with reasonable computation times (e.g., minutes to hours rather than days). We conducted the joint growth-survival models using the JMbayes2 package in R (Rizopoulos & Papageorgiou, 2024).

Estimating Personality Trait Change

To estimate linear change for each Big Five trait, we estimated up to five separate growth models within each study (one for each trait that was assessed). We modeled fixed and random intercepts and fixed and random linear slopes. Coefficients were estimated using restricted maximum likelihood. Random intercepts and random slopes (i.e., empirical Bayes estimates) were outputted and added to the data set for each participant and then entered into Cox proportional hazards models as predictors of mortality risk.

Predicting Mortality Risk From Personality Trait Level and Change

To predict mortality risk from personality trait level and change, we estimated up to five separate Cox proportional hazards models within each study (one for each trait that was assessed). Survival time (i.e., time in study in months) was the dependent variable in each model. Individual intercepts (i.e., personality trait level) and slopes (i.e., personality trait change) from the linear growth curve models (i.e., empirical Bayes estimates) were included as predictor variables. Baseline age, gender, and education were included as covariates. In a parallel set of exploratory analyses, we additionally added an interaction term between personality trait level and personality trait change.

For all Cox proportional hazards models, we tested the proportional hazards assumption by checking the scaled Schoenfeld residuals. When a given predictor violated the proportional hazards assumption, we added interactions between the violating predictor(s) and time in study. Specifically, we used the timeSplitter function from the Greg package in R (Gordon & Seifert, 2022) to split the follow-up period into 6-month increments. When interactions between personality trait level and time in study or personality trait change and time in study were included in the model, the meaning of the effects for personality trait level and personality trait change can be interpreted as the associated risk of mortality in the first 6 months of the follow-up period, and the interaction term can be interpreted as change in risk for each subsequent month of survival.

Random Effects Meta-Analysis

We used random-effects models (Borenstein et al., 2010) to calculate the overall weighted mean effect size, standard error, and 95% confidence intervals (CIs) across samples. To examine between-study heterogeneity in effect sizes, we report Cochrane's Q and I^2 (see Figures 2–4 and Supplemental Figures S1 and S2). I^2 indicates the proportion of between-study variance in effect sizes that is due to meaningful heterogeneity versus random error (Higgins & Green, 2008). However, I^2 is imprecise and can be biased in small meta-analyses like those reported in this article and, thus, should be interpreted with caution (von Hippel, 2015).

Results

Growth Curve Models

Results from the growth curve models are shown in Supplemental Table S2. The fixed intercept can be interpreted as mean level of a given trait at the study midpoint. The fixed slope can be interpreted as mean linear personality trait change per decade in units of standard deviations. Standard deviations of random effects reflect the model-estimated degree of individual differences in personality trait level and personality trait change, respectively. The correlation between the fixed and random effects describes the model-estimated relationship between personality trait level and personality trait change. To evaluate whether individuals significantly differed from one another in personality trait change, we used log likelihood ratio tests to compare the fit of growth curve models with and without random linear age slopes. Supplemental Table S3 shows the log likelihoods for the models with and without random linear age slopes and the p value corresponding to the log likelihood ratio test.

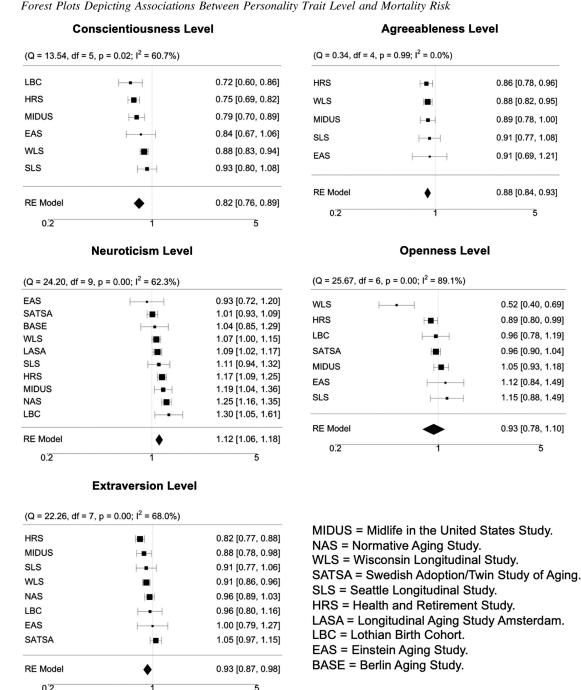
On average, conscientiousness decreased in all six samples in which it was assessed. The decrease was statistically significant in five samples (WLS, SLS, HRS, LBC, and EAS) and statistically nonsignificant in one sample (MIDUS). The effect size ranged from near 0 to 0.33 *SD*s of decline per decade. Individuals significantly differed from one another in conscientiousness change in all six samples in which it was assessed.

On average, neuroticism significantly decreased in five samples (MIDUS, NAS, WLS, SATSA, and HRS), significantly increased in two samples (EAS, BASE), and did not significantly change in four samples (SLS, LASA, LBC, OCTO). Within the samples in which neuroticism significantly decreased, the effect size ranged from 0.04 to 0.15 *SD*s of decline per decade. Within the samples in which neuroticism significantly increased, the effect size ranged from 0.22 to 0.41 *SDs* of increase per decade. Individuals significantly differed from one another in neuroticism in all samples except OCTO.

On average, extraversion significantly decreased in seven samples (MIDUS, WLS, SLS, HRS, EAS, BASE, OCTO), significantly increased in one sample (NAS), and did not significantly change in two samples (LBC, SATSA). Within the samples in which extraversion significantly decreased, the effect size ranged from 0.03 to 0.40 *SD*s of decline per decade. In the sample in which extraversion significantly increased, the effect size was 0.02 *SD*s of increase per decade. Individuals significantly differed from one another in extraversion in all samples in which it was assessed except BASE and OCTO.



Forest Plots Depicting Associations Between Personality Trait Level and Mortality	v Risk
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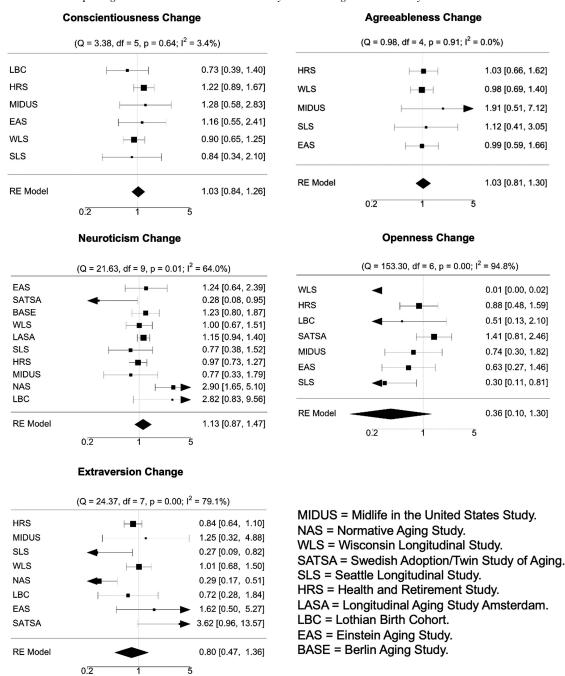


Note. For each trait and sample, the effects of personality trait level and personality trait change were estimated within the same model, and baseline age, gender, and education were included as covariates. The complete results including the effects of covariates are shown in Supplemental Table S4. RE Model = mean weighted effect size from random-effects meta-analysis.

On average, agreeableness significantly increased in two samples (WLS, SLS), significantly decreased in two samples (MIDUS, HRS), and did not significantly change in two samples (LBC, EAS). Within the samples in which agreeableness significantly increased, the effect size ranged from 0.04 to 0.19 SDs of increase per decade. In the samples in which in significantly

decreased, the effect size ranged from 0.06 to 0.08 SDs of decline per decade. Individuals significantly differed from one another in agreeableness in all samples in which it was assessed except LBC.

On average, openness decreased in all eight samples in which it was assessed. The decrease was statistically significant in seven



Note. For each trait and sample, the effects of personality trait level and personality trait change were estimated within the same model, and baseline age, gender, and education were included as covariates. The complete results including the effects of covariates are shown in Supplemental Table S4. RE Model = mean weighted effect size from random-effects meta-analysis.

samples (MIDUS, WLS, SATSA, SLS, HRS, EAS, BASE) and statistically nonsignificant in one sample (LBC). The effect size ranged from near 0 to 0.33 *SD*s of decline per decade. Individuals significantly differed from one another in neuroticism change in all samples in which it was assessed except BASE.

Cox Proportional Hazards Models

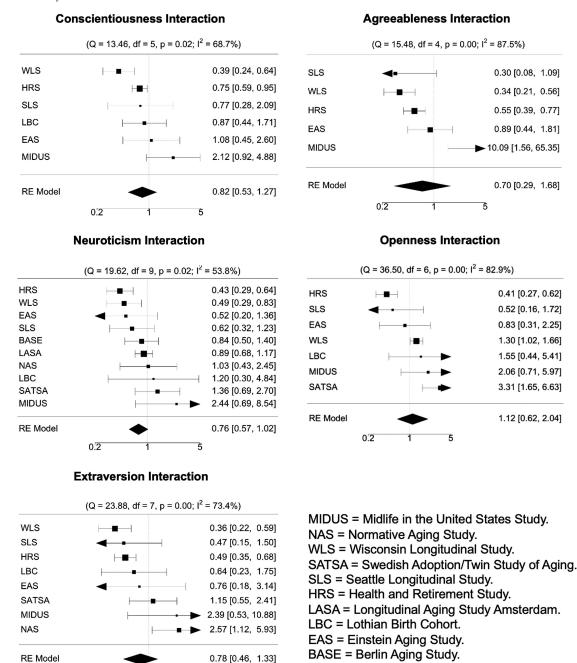
Because the central focus of the present study is associations between personality trait change and mortality, and associations between personality trait level and mortality have been reported elsewhere for most of these samples (Graham et al., 2020), we did

Figure 3

Forest Plots Depicting Associations Between Personality Trait Change and Mortality Risk

Figure 4

Forest Plots Depicting Interactions Between Personality Trait Level and Personality Trait Change Predicting Mortality Risk



Note. For each trait and sample, the simple effects of personality trait level and personality trait change were estimated within the same model, and baseline age, gender, and education were included as covariates. RE Model = mean weighted effect size from random-effects meta-analysis.

not proceed with testing Cox proportional hazards models for trait– sample pairings in which individuals did not significantly differ from another in personality trait change (i.e., neuroticism in OCTO, extraversion in BASE and OCTO, agreeableness in MIDUS and

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0.2

LBC, openness in BASE). This resulted in excluding OCTO from all Cox proportional hazards analyses.

Hazard ratios for personality trait level can be interpreted as the ratio of the hazard rate for two people who are 1 SD apart in a given

trait at their personal study midpoint, adjusting for linear personality trait change, baseline age, gender, and years of education. Hazard ratios for personality trait change can be interpreted as the ratio of the hazard rate for someone who increased by 1 *SD* in a given trait per decade compared to someone who did not change in that trait, adjusting for personality trait level, baseline age, gender, and education.

Personality Trait Level and Mortality Risk

Associations between personality trait level and mortality risk are shown in Figure 2 and Supplemental Table S4. For models in which personality trait level violated the proportional hazards assumption, the hazard ratio reflects the hazard in the first 6 months of the followup period (i.e., openness in WLS and SLS).

Higher conscientiousness level was associated with lower mortality risk in all six samples in which the effects of conscientiousness were assessed. The effect was statistically significant in four samples (MIDUS, WLS, HRS, LBC). The mean weighted effect size was 0.82 (95% CI [.76, .89]), indicating an 18% reduction in risk for individuals 1 *SD* above the mean of conscientiousness relative to individuals at the mean of conscientiousness.

Higher neuroticism level was associated with higher mortality risk in nine of the 10 samples in which the effects of neuroticism were assessed. The effect was statistically significant in six samples (MIDUS, NAS, WLS, HRS, LASA, LBC). The mean weighted effect size was 1.12 (95% CI [1.06, 1.18]), indicating a 12% increase in risk for individuals 1 *SD* above the mean of neuroticism relative to individuals at the mean of neuroticism.

Higher extraversion level was associated with lower mortality risk in six out of the eight samples in which the effects of extraversion were assessed. The association was statistically significant in three samples (MIDUS, WLS, HRS). The mean weighted effect size was 0.93 (95% CI [0.87, 0.98]), indicating a 7% reduction in risk for individuals 1 *SD* above the mean of extraversion relative to individuals at the mean of extraversion.

Higher agreeableness level was associated with lower mortality risk in all five samples in which the effects of agreeableness were assessed, and the effect was statistically significant in three samples (MIDUS, WLS, HRS). The meta-analytic effect size was 0.88 (95% CI [0.84, 0.93]), indicating an 11% reduction in risk for individuals 1 *SD* above the mean of extraversion relative to individuals at the mean of extraversion.

Higher openness level was significantly associated with lower mortality risk in two samples (WLS, HRS). In WLS, there was a statistically significant interaction between openness level and time in study, indicating that openness was associated with lower mortality risk early in the follow-up period but became associated with greater mortality risk as survival time increased. The association between openness level and mortality risk was nonsignificant with effects in both directions in the other five samples in which it was assessed. The mean weighted effect was statistically nonsignificant, HR = 0.93 (95% CI [0.78, 1.10]).

Personality Trait Change and Mortality Risk

Associations between personality trait change and mortality risk are shown in Figure 3 and Supplemental Table S4. For models in which personality trait change violated the proportional hazards assumption, the hazard ratio reflects the hazard at the beginning of the follow-up period (i.e., neuroticism and extraversion in SATSA and openness in WLS). For all Big Five traits, the mean weighted effect size was statistically nonsignificant, suggesting that personality trait change was not consistently associated with mortality risk. Below we describe the handful of statistically significant individual study results for trait change.

Increases in neuroticism were associated with higher mortality risk in two samples (NAS and SATSA). In NAS, the hazard ratio was 1.25, indicating a 25% increase in risk for an individual who increased 1 SD per decade in neuroticism compared to an individual who did not change in neuroticism. In SATSA, there was a statistically significant interaction between neuroticism change and time in study such that increases in neuroticism were associated with lower mortality risk, and this protective effect became more pronounced across the follow-up period. Increases in extraversion were associated with lower mortality risk in two studies (NAS and SLS). The hazard ratio ranged from 0.27 to 0.29, suggesting that an individual who increased 1 SD per decade in extraversion is at 71% to 73% lower risk compared to an individual who did not change in extraversion. Increases in openness were associated with lower mortality risk in two studies (WLS and SLS). In SLS, the hazard ratio was from 0.30, suggesting that an individual who increased 1 SD per decade in openness is at 70% lower risk compared to an individual who did not change in openness. In WLS, there was a statistically significant positive interaction between openness change and time such that increases in openness were associated with lower mortality risk early in the follow-up period but became associated with greater mortality risk as survival time increased.

Interactions Between Personality Trait Level and Personality Trait Change

Results from the Cox proportional hazards models used to predict mortality risk from the interaction of personality trait level and personality trait change are shown in Figure 4 and Supplemental Table S5. Simple effects of personality trait level and personality trait change and baseline age, gender, and years of education were included as predictors in the model. For models in which personality trait level or change violated the proportional hazards assumption, the hazard ratio reflects the hazard at the beginning of the follow-up period (i.e., neuroticism and extraversion in SATSA and openness in WLS and SLS). For all Big Five traits, the mean weighted effect size for the interaction term was statistically nonsignificant, suggesting that personality trait level and personality trait change did not reliably interact to predict mortality risk.

Sensitivity Analyses

Supplemental Figures S1 and S2 show results of models without adjustment for education. The direction and statistical significance of all mean weighted effect sizes remained the same as in primary analyses. Supplemental Table S6 shows the results of the joint growth–survival models next to the results from the original two-step modeling approach. The two modeling approaches resulted in the same conclusion in all instances.

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Discussion

We examined associations of personality trait *level* and personality trait *change* with mortality risk across 11 independent samples. Higher conscientiousness, extraversion, and agreeableness and lower neuroticism were associated with longer survival. These associations are consistent with prior research. For example, Graham et al. (2017) used 15 studies that partially overlap with those used in the present study and found that higher conscientiousness, extraversion, and agreeableness and lower neuroticism was associated with longer survival. Compared to personality trait level, we found limited evidence for replicable associations between personality trait change and mortality risk. At least three potential explanations for these null results should be considered.

First, Big Five personality trait change may not be associated with mortality risk, above and beyond personality trait level. It may be the case that stable between-person differences in personality traits matter for health and mortality more than age-related changes in traits. For example, someone who is high in conscientiousness may engage in more health protective behaviors across their entire life, regardless of whether they become more or less conscientiousness as they age. Indeed, given that many health behaviors are habitual in nature, personality trait change may not necessarily result in health behavioral change. Consistent with this explanation, several studies have found that health behaviors are more strongly related to between-person individual differences in personality trait level and are less consistently related to within-person personality trait change (e.g., Jokela et al., 2018). Even if personality trait change leads to changes in health behaviors, social relationships, or stress processes, personality trait change occurring in midlife and older adulthood may not have enough time to impact mortality risk.

The second possibility is that the relationship between personality trait change and mortality is more complex than the simple linear and interactive relationships that we tested in the present investigation. Because the direction and degree of personality trait change differs across the lifespan (Bleidorn et al., 2022; Specht et al., 2014), nonlinear trait change may be more strongly associated with mortality than linear personality trait change. This may be especially true when considering personality trait change that spans multiple developmental periods. Additionally, there is some evidence that absolute personality trait change, regardless of direction, may be associated with health outcomes (Human et al., 2013). In addition to linear and nonlinear changes in mean levels of personality traits, other dynamic aspects of personality may be related to mortality. For example, previous research has observed associations of health behaviors and health outcomes with changes in personality profile consistency (e.g., Stephan et al., 2014) and with within-person variability in personality (Wright & Jackson, 2024b). These dynamic aspects of personality may be related to mortality even in the absence of associations between individual differences in mean-level personality trait change and mortality. Moreover, accounting for within-person variability in personality may enable more precise estimates of the true association between individual differences in mean-level personality trait change and outcomes such as mortality (see Wright & Jackson, 2024b, for further discussion of this point). The present study highlights the strong data requirements for testing the effects of linear personality trait change on mortality risk. Tests of more complex associations would likely have even stronger data requirements (e.g., more measurement occasions and possibly larger samples). Thus, future research should test these possibilities in more focused investigations involving datasets that meet these strong requirements.

A third possibility is that methodological and statistical limitations of this study obscured the association between personality trait change and mortality risk. Although the present research has several methodological strengths, including the use of 11 large samples (Ns = 210 to 11,031) with between three and 11 measurement occasions of personality across 6 to 43 years in each study, the complexity of the research question may necessitate even more stringent data requirements. In the next section, we describe methodological considerations for future research on personality trait change, health, and mortality.

Methodological Considerations for Research on Personality Trait Change and Mortality

First, future research should aim for more measurement occasions of personality, which would facilitate more reliable estimates of personality trait change and may be better able to statistically disentangle personality trait level from personality trait change. We included studies with as few as three measurement occasions and participants with as few as two measurement occasions. Second, when possible, future research should include long follow-up periods after the first several measurement occasions of personality to allow enough time to pass for personality trait change to impact risk and for enough mortality to occur after measurement of personality trait change. In some of the present studies, such as MIDUS and WLS, trait change was assessed across three measurement occasions that largely overlapped with the mortality follow-up period (see Figure 1). Thus, the majority of participants with three measurement occasions of personality survived until the end of the follow-up period, creating a confound between the reliability of the personality trait change estimates and the outcome of interest. In addition to including a large number of personality measurement occasions and a long mortality follow-up period, the spacing of these measurement occasions requires careful consideration. For example, multiple measurement occasions of personality spanning young adulthood to midlife followed by multiple decades of mortality follow-up in midlife and late life may be ideal from a modeling perspective but may be less theoretically meaningful if personality trait change in older adulthood is of interest. Researchers should consider these trade-offs when designing future studies of personality trait change and mortality.

Last, the current investigation focused on simple associations between personality trait level, personality trait change, and mortality. Numerous underlying causal processes could explain the presence or absence of such associations. For example, newer formulations of the health behavior model of personality (Friedman et al., 2014) emphasize dynamic associations among personality, physiology, behavior, social relations, and health across the lifespan and implications of these dynamic processes for longevity. This model suggests that personality and personality change are analogous to maintaining or altering the course of a ship, such that personality's impact is long-lasting but not necessarily permanent and may be influenced by prior levels or changes in any of the processes in the causal model. Moreover, some of the steps in the causal chain may occur over shorter or longer timescales than others. Thus, to appropriately model the dynamic relations among personality traits, longevity, and the many potential mediating factors, future research will need to use frequently repeated measures of personality and plausible mediators (e.g., behavior, physiology, social relations, health) over long periods of time followed by long follow-up periods to track mortality (i.e., measurement burst designs). This will allow for dynamic modeling of potentially reciprocal relations among personality, behavior, physiology, social relations, health, and ultimately their impact on longevity.

Concluding Comment

In a coordinated analysis of 11 independent samples, the present investigation provided a preregistered test of associations of personality trait level and personality trait change with mortality risk. We replicated previous findings concerning associations between personality trait level and mortality risk but found limited evidence for consistent associations between personality trait change and mortality risk. The extent to which personality trait change is associated with mortality risk is an important research question with the opportunity to inform intervention efforts. Although we did not observe replicable associations between personality trait change and mortality in the present study, naturally occurring personality trait change is relatively small in magnitude compared to the more substantial personality trait change that often occurs in response to interventions (Roberts et al., 2017). Thus, it remains an open question whether personality trait change in response to interventions may modulate mortality risk (Mroczek, 2014). Future research focusing on personality trait change, health, and mortality risk can benefit from the lessons we learned in the present investigation.

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