Journal of Research in Personality 81 (2019) 11–24

Contents lists available at ScienceDirect

Journal of Research in Personality

journal homepage: www.elsevier.com/locate/jrp

Full Length Article

Sleep: A pathway linking personality to mortality risk

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ARTICLE INFO

Article history:
Received 21 August 2018
Revised 4 April 2019
Accepted 25 April 2019
Available online 26 April 2019

Keywords:
Personality
Big Five
Mortality
Sleep
Health

ABSTRACT

Personality and sleep predict longevity; however, no investigation has tested whether sleep mediates this association. Thus, we tested this effect across a 20-year follow-up (N = 3759) in the Midlife Development in the United States cohort (baseline M_{age} = 47.15) using proportional hazards in a structural equation modeling framework. Lower conscientiousness predicted increased death risk via the direct, indirect, and total effect of quadratic sleep duration. Although there were no other direct personality-mortality effects, higher neuroticism and agreeableness and lower conscientiousness predicted increased death risk via the joint indirect effects of quadratic sleep duration and higher daytime dysfunction. Lower extraversion predicted increased mortality risk via the indirect effect of daytime dysfunction. Our findings have implications for personality-based health interventions.

1. Introduction

The Big Five personality characteristics predict all-cause mortality risk (Jokela et al., 2013). With ample support for this finding now established (e.g., Friedman et al., 1993; Turiano, Chapman, Gruenewald, & Mroczek, 2015), there is growing interest in the mechanisms underlying this association. One potential mechanism, sleep, has been associated with both personality (Duggan, Friedman, McDevitt, & Mednick, 2014; Gray & Watson, 2002) and longevity (Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002; Kronholm, Laatikainen, Peltonen, Sippola, & Partonen, 2011), yet no study has investigated whether sleep is a pathway linking personality to objective health outcomes. Thus, using data from the Midlife Development in the United States (MIDUS) study, we examined whether sleep duration and quality mediate the personality-mortality association across a 20 year follow-up period (see Fig. 1)[1].

Personality refers to characteristic patterns of cognition, affect, behavior, and motivation present across many contexts. The Big Five conceptualizes global personality structure as consisting of five hierarchical dimensions: conscientiousness, agreeableness, neuroticism, openness, and extraversion (Costa & McCrae, 1992; Goldberg, 1992). Among these traits, higher conscientiousness (i.e., the tendency to be self-disciplined, organized, and industrious) consistently predicts increased longevity across cultures (Iwasa et al., 2008), age groups (children: Friedman et al., 1993; middle-aged adults: Turiano et al., 2015; older adults: Costa, Weiss, Duberstein, Friedman, & Siegler, 2014), and follow-up periods (Friedman et al., 1993; Martin, Friedman, & Schwartz, 2007), as confirmed by meta-analyses (Jokela et al., 2013; Kern & Friedman, 2008). However, the studies of neuroticism (i.e., the tendency to be emotionally reactive and to experience depression, anxiety, and anger) and mortality risk have been less consistent, with some reporting no effect (Turiano et al., 2015) and others reporting inverse (Ploubidis & Grundy, 2009) or positive effects (Friedman et al., 1993; Martin et al., 2007). Some studies also suggest agreeableness (i.e., the tendency to be compassionate, compliant, and trusting), openness (i.e., the tendency to be intellectually curious, imaginative, and have liberal values), and extraversion (i.e., the tendency to be friendly and assertive, and to experience positive emotions) protect against death risk (agreeableness: Costa et al., 2014; openness: Ferguson & Bibby, 2012; extraversion: Iwasa et al., 2008). However, most studies report no effects of these traits on death risk (e.g., Turiano et al., 2015).

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[1] This study was not pre-registered. Statistical Analysis Software (SAS) code for our analyses are available upon request. We are not permitted to share our data because MIDUS study users must be registered through the Inter-University Consortium for Political and Social Research (IPCSR) at the University of Michigan. Data from MIDUS are publicly available at: http://midus.colectica.org.

https://doi.org/10.1016/j.jrp.2019.04.007
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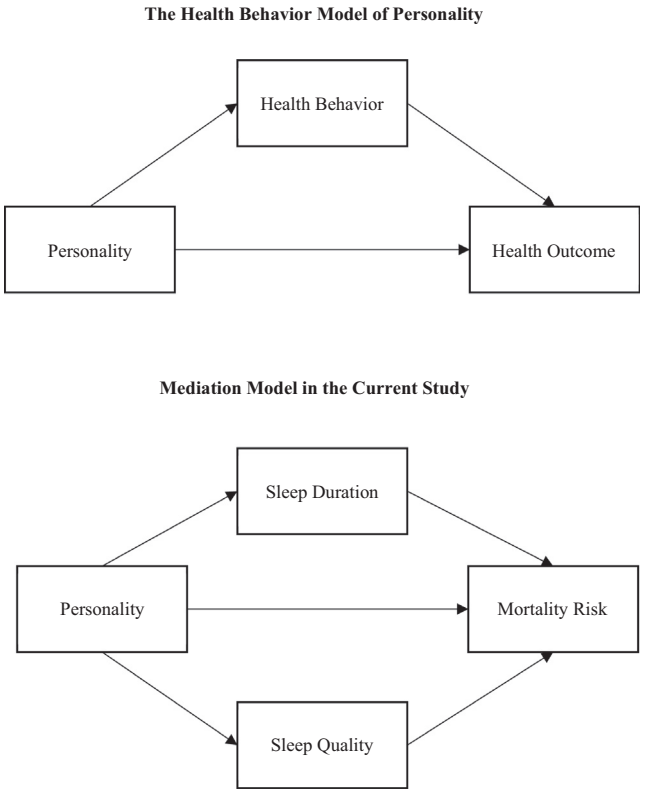


Fig. 1. In the *Health Behavior Model of Personality*, personality impacts health behaviors, which, in turn, influences health outcomes. In the *Mediation Model of the Current Study*, we examined whether personality predicted death risk through sleep duration and quality.

Given these personality-mortality findings, there is a body of literature examining whether health behaviors underlie this effect. These studies were guided by the Health Behavior Model (HBM) of personality (Smith, 2006; see Fig. 1), which postulates that aspects of personality lead one to engage in behaviors (e.g., smoking, alcohol use) that impact health over time. Earlier HBM studies were limited because traditional mediation tests allow for either discrete or continuous outcome variables but not ones such as mortality risk that are both continuous (i.e., survival time) and discrete (i.e., dead vs. alive). Thus, these studies inferred mediation (vs. formally testing it) by entering health behaviors as covariates into models testing the personality-mortality association, with these adjustments often attenuating the personality-mortality effect.

However, recent advances have extended the use of proportional hazards modeling in structural equation modeling (*SEM*) frameworks, allowing for mediational tests with outcomes that are both continuous and discrete (Asparouhov & Masyn, 2006; Muthén & Muthén, 1998). Importantly, this technique allows estimation of the statistical significance of indirect effects—a requirement for confirming mediation. A recent study using this technique with MIDUS cohort data found that alcohol use, smoking, and waist circumference explain 42% of the variance in the conscientiousness-mortality association (Turiano et al., 2015). Although prior HBM studies suggest that health behaviors, such as alcohol use, smoking, and physical activity, underlie the personality-mortality association (e.g., Hill, Turiano, Hurd, Mrcozek, & Roberts, 2013; Graham et al., 2017; Turiano et al., 2015), these behaviors do not account for all the variance in this pathway. Thus, other mechanisms need to be explored.

One such behavioral mechanism, sleep, shows robust associations with health outcomes (e.g., obesity: Cappuccio et al., 2008). Moreover, sleep is more than just a health-promoting behavior, it is an important health outcome itself, helping to regulate and maintain bodily hemostasis (Kryger, Roth, & Dement, 2016). Nevertheless, sleep has been overlooked in the personality-health literature, a surprising oversight given that sleep is a universal health behavior (i.e., relative to other health behaviors such as substance use and smoking) and sleep problems are amenable to treatment (Espie, 2002). Accordingly, we examined whether sleep duration (i.e., number of minutes slept per night) and quality (i.e., ease of falling asleep, staying asleep, and feeling rested upon waking) account for variance in the personality-mortality pathway. We focused on these aspects of sleep because they are available in MIDUS and commonly investigated in sleep-health studies (Buysse, 2014).

Among the Big Five, higher neuroticism has been associated with shorter sleep duration (Hintsanen et al., 2014; Otonari et al., 2012; Vincent, Cox, & Clara, 2009), with one investigation also linking it to both short and long sleep duration (i.e., the quadratic effect of sleep duration; Allen, Magee, & Vella, 2016). Higher neuroticism is also related to poorer sleep quality across diverse samples (college students: Duggan, Reynolds, Kern, & Friedman, 2014; Gray & Watson, 2002; community adults: Hintsanen et al., 2014; cross-culturally: Kim et al., 2015). Further, lower conscientiousness is typically associated with poorer sleep quality (e.g., Gray & Watson, 2002; Williams & Moroz, 2009; but see also Cellini, Duggan, & Sarlo, 2017) and shorter sleep duration (Randler, 2008; but see also Gray & Watson, 2002).

Other traits have shown less robust effects with sleep. Though most studies find no effect of extraversion on sleep quality and duration (Duggan et al., 2014; Gray & Watson, 2002), there is emerging evidence that higher extraversion is related to normal sleep duration (Otonari et al., 2012) and better sleep quality, both cross-sectionally (Allen et al., 2016; Randler, Schredl, & Göritz, 2017) and prospectively (Stephan, Sutin, Bayard, Krizan, & Terracciano, 2018). Although agreeableness has been positively associated with sleep duration (Randler, 2008), it has not been associated with sleep quality (Gray & Watson, 2002; Stephan, Sutin, Luchetti, Bosselut, & Terracciano, 2018). With the exception of one study linking lower openness to better sleep quality (Allen et al., 2016), this trait has not been associated with sleep quality or duration (Duggan et al., 2014; Gray & Watson, 2002).

Given these personality-sleep findings, it is important to explore whether sleep duration and quality mediate the personality-mortality effect because both sleep components have been associated with longevity (Duggan et al., 2014; Kojima et al., 2000). Specifically, sleep duration generally predicts increased death risk in a U-shaped manner (i.e., getting either too much or insufficient sleep increases the hazard of dying), with this effect replicating across diverse samples (childhood: Duggan et al., 2014; adulthood: Kripke et al., 2002; Kronholm et al., 2011; cross-culturally: Kojima et al., 2000), consistent with meta-analyses (Cappuccio, D'Elia, Strazzullo, & Miller, 2010; Da Silva et al., 2016). Short sleep duration is thought to be related to mortality risk due to its association with chronic health conditions (e.g., cardiovascular disease: Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; obesity: Cappuccio et al., 2008), whereas investigations into the mechanisms connecting long sleep durations to increased mortality are still unclear (Knutson & Turek, 2006). Though our understanding of the mechanisms linking long sleep duration and health are limited, they may include undiagnosed and unmeasured chronic disease, sleep fragmentation, immune function, depression, and/or fatigue (Grandner & Drummond, 2007; Grandner, Patel, Gehrman, Perlis, & Pack, 2010).

Like sleep duration, poorer sleep quality is associated with reduced life expectancy across diverse samples (cross-culturally: Kojima et al., 2000; but see also Chen, Su, & Chou, 2013). Poorer sleep quality is associated with increased morbidity such as cardio-

vascular disease (Hoevenaar-Blom, Spijkerman, Kromhout, van den Berg, & Verschuren, 2011), metabolic syndrome (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007), depression (Hale et al., 2013), and psychological well-being (Pilcher, Ginter, & Sadowsky, 1997). All of these factors partially contribute to why poorer sleep quality positively predicts death risk.

In sum, the purpose of this study was to examine how personality contributes to life expectancy by uniting segmented areas of research linking personality to sleep (Duggan et al., 2014; Gray & Watson, 2002); personality to mortality risk (Friedman et al., 1993; Turiano et al., 2015); and sleep to mortality risk (Duggan et al., 2014; Kojima et al., 2000), testing the contribution of nighttime health behaviors using the HBM. Specifically, the current study was guided by four research questions: First, how does personality predict mortality risk? We expected lower conscientiousness and higher neuroticism to be associated with increased mortality risk, based on previous studies (e.g., Friedman et al., 1993; Turiano et al., 2015).

Second, does sleep duration predict mortality risk in a linear and/or curvilinear manner? Given that sleep duration has been associated with increased mortality risk in both a linear (Chen et al., 2013; Kripke, 2002) and curvilinear (Duggan et al., 2014) manner, we first tested the linear and quadratic effects of this sleep component on mortality risk to determine whether and how sleep duration is related to death risk, before testing whether it significantly mediates the personality-mortality effect. We predicted that sleep duration would be associated with mortality risk in a U-shaped manner, consistent with previous findings (Cappuccio et al., 2010).

Third, which aspects of sleep quality are associated with mortality risk? Before testing whether sleep quality accounts for variance in the personality-mortality effect, we also explored whether and how sleep quality relates to death risk. Our sleep quality questionnaire included four items, three measuring sleep problems (e.g., trouble falling asleep) and one measuring daytime dysfunction due to sleep problems. First, we tested the direct effect of the full measure on death risk. Consistent with other investigations of sleep health, we also broke our measure into sleep problems and daytime dysfunction, examining the effect of these two components separately and together because sleep quality is a heterogeneous construct. Sleep maintenance complaints (e.g., trouble falling asleep, staying asleep, waking too early) are symptoms of insomnia. Feeling unrested may be an outcome of those processes, short sleep duration, or other processes, such as depression or narcolepsy (Buysee, Reynolds, Monk, Berman, & Kupfer, 1989; Buysse, 2014). Our tests of sleep quality were exploratory: we made no predictions about which aspects of sleep quality were more strongly related to mortality risk, with the exception that we expected the full sleep quality measure to be related to increased mortality risk.

Fourth, do sleep duration and quality mediate the personality-mortality association? We expected that sleep components would mediate the neuroticism-mortality and conscientiousness-mortality associations via the calculated significance of indirect effects. However, we made no predictions for the mediating effects of sleep components on the link between other personality traits and mortality, given the inconsistent findings for agreeableness, openness, and extraversion with sleep duration (Gray & Watson, 2002; Randler, 2008), sleep quality (Duggan et al., 2014; Gray & Watson, 2002), and mortality risk (Friedman et al., 1993; Turiano et al., 2015).

2. Method

2.1. Sample

Data were from the National Survey of Midlife Development in the United States (MIDUS), a longitudinal multidisciplinary study of psychosocial development and health in a national sample of adults (for review, see Brim, Ryff, & Kessler, 2004). MIDUS includes three waves of data: MIDUS 1, collected in 1995–1996; MIDUS 2, collected in 2004–2006; and MIDUS 3, collected in 2013–2016. We used data from MIDUS 1 and MIDUS 2, enabling us to utilize the largest number of mortality deaths, as well as the temporal ordering needed to establish mediation.

The MIDUS 1 sample included 7108 non-institutionalized English-speaking adults in the coterminous United States, aged 25 to 74. The sample was recruited using random digit dialing techniques, oversampling for men and older individuals to ensure adequate representation of these populations as they tend to be less likely to participate in research studies (e.g., Murthy, Krumholz, & Gross, 2004). Participants completed a telephone interview and a self-administered questionnaire (SAQ). MIDUS 1 participants were invited to participate in MIDUS 2. Of the 7108 participants, 4963 (75% adjusted for mortality) completed a phone interview at MIDUS 2. Of those 4,963 participants who completed the phone interview, 4032 (81%) completed an SAQ. Of these 4032 participants, 3759 (roughly 50% of the original full 7108 sample at MIDUS 1) provided complete MIDUS 1 and MIDUS 2 data used in our study and were included in our sample (see Appendix A for an attrition and mortality diagram). Note that we did not conduct a power analysis to determine our sample size prior to analyses because we utilized archival data and our sample size was finite. However, post hoc power analyses (for review see Hoenig & Heisey, 2001) suggest that effects equaling 0.11 in magnitude can be detected in the current sample, when power is 0.80 and alpha is 0.05. We handled missing data using listwise deletion as full information maximum likelihood (FIML) approaches are not available when using proportional hazards modeling in an SEM framework.

Compared to participants included in our sample (n = 3759), participants who dropped out or had missing data (n = 3349) were more likely to be male ($\chi^2 = 41.49$, $p < .001$); an ethnic/racial minority ($\chi^2 = 52.65$, $p < .001$); single ($\chi^2 = 83.53$, $p < .001$); less educated ($t = 13.15$, $p < .001$); lower on conscientiousness ($t = 6.94$, $p < .001$); higher on agreeableness ($t = -2.46$, $p < .05$); poorer sleep quality ($t = 3.91$, $p < .001$); had more sleep problems ($t = -3.36$, $p < .001$); higher daytime dysfunction ($t = -3.89$, $p < .001$); and were less likely to be retired ($\chi^2 = 18.68$, $p < .001$). There were no significant differences in age, neuroticism, openness, extraversion, and sleep duration.

2.2. Measures

Covariates. We adjusted our models for the following covariates measured at MIDUS 1: gender (0 = *female*; 1 = *male*), race (0 = *White;* 1 = *minority*), age, relationship status (0 = *married or living with a partner*, 1 = *not married or living with a partner*), and level of education (1 = *no school/some grade school*; 12 = *professional degree*). We also adjusted for retirement status (0 = *not retired*, 1 = *retired*), measured at MIDUS 2 (i.e., the same wave that sleep was measured), because of findings linking retirement to sleep disruption (Myllyntausta et al., 2017; Vahtera et al., 2009).

Personality. Personality was assessed at MIDUS 1, with the Midlife Development Personality Inventory (*MIDI*; Lachman & Weaver, 1997), which includes 25 adjectives measuring the Big Five personality dimensions. Participants rated how much each adjective described them, using a scale ranging from 1 (*not at all*) to 4 (*a lot*). The adjectives include: friendly, lively, active, talkative (extraversion; $\alpha = .78$); moody, worrying, nervous, calm (reverse coded; neuroticism; $\alpha = .74$); creative, imaginative, intelligent, curious, broad-minded, sophisticated, adventurous (openness; $\alpha = .77$); organized, responsible, hardworking, careless (reverse coded; conscientiousness; $\alpha = .59$); and helpful, warm, caring, soft-

hearted, sympathetic (agreeableness; α = .82). The *MIDI* (Lachman & Weaver, 1997) correlates strongly with NEO personality measures and has good construct validity (Lachman & Weaver, 1997; Mroczek & Kolarz, 1998). Scores for each trait were calculated by averaging the item responses. Higher scores represent higher standing on that trait.

Sleep Duration. Sleep duration was assessed at MIDUS 2 using self-reported weekday sleep duration (i.e., "How much sleep do you usually get at night [or in your main sleep period] on weekdays or workdays? Hours? Minutes?") and weekend sleep duration (i.e., "How much sleep do you get at night [or in your main sleep period] on weekends or your non-workdays? Hours? Minutes?"). Responses to the number of hours slept were recoded into minutes, then added to the number of minutes slept, yielding scores for weekend and weekday sleep duration in minutes. Following the method of Kong et al. (2011), these scores were averaged to yield a score for total sleep duration, using the formula: ([(5 × weekday duration) + (2 × weekend duration)]/7). Higher scores represent longer sleep duration.

Sleep Quality. Sleep quality (α = .80) was assessed at MIDUS 2 using a four-item self-report measuring trouble falling asleep, staying asleep, waking too early, and feeling unrested during the day. Participants rated how often they experienced these problems using a scale ranging from 1 (*never*) to 4 (*almost always/four or more times per month*). For ease of interpretation, we reverse-scored item responses before averaging them. Thus, higher scores represented more optimal sleep quality. We also separated this measure into two constructs (i.e., sleep problems: items 1–3; α = .80; daytime dysfutncion: item 4) and tested effects using these components. For ease of interpretation, we did not reverse-score sleep problems and daytime dysfunction. Thus, higher scores on these scales indicated more sleep problems/daytime dysfunction.

Vital Status. There were 1,299 deaths in the entire MIDUS cohort by the censor date (October 15, 2015). Mortality data were obtained using several methods. First, 569 deaths were confirmed from National Death Index (NDI) reports obtained from 2006 to 2009. Second, 153 deaths were confirmed during the closeout phases of MIDUS 2 and 483 deaths confirmed during the closeout phases of MIDUS 3. Third, 94 deaths were confirmed as normal longitudinal sample maintenance was conducted. Only the month and year of death were recorded for the purposes of confidentiality. The 15th day of each month was assigned as the day of death for all decedents.

We utilized mortality data based on 3759 participants (403 or 10.72% deceased; mean survival time = 14.70 years; *SD* = 2.84; range: 8.98–20.15) who provided data on the independent variables. We should note that we did not use mortality data on all the aforementioned 1299 decedents because we were unable to use data from individuals who died before the sleep mediators were assessed at MIDUS 2. Survival time for deceased participants was the interval between when the SAQ data were received by the study team at MIDUS 1 and the date of their death. For participants who are still alive (censored observations), the survival time was the interval between MIDUS 1 and the censored date (October 15, 2015).

2.3. Data analysis

To test the direct effects of personality, sleep duration and sleep quality on mortality risk, we conducted a series of proportional hazards models (i.e., Cox models). We used proportional hazards modeling because this technique accounts for continuous survival times, varying ages at entry in the study, and discrete outcomes (i.e., dead vs. alive; Cox, 1992). All predictor variables were converted into standard deviation units for ease of interpretation. Thus, the Cox models presented herein yield an estimate of how much of a standard deviation increase or decrease in a predictor variable predicts the likelihood of dying over a certain time interval. In addition, we set our alpha level to 0.05.

We tested proportional hazards assumptions to ensure that the effects of each predictor were proportional over time (i.e., the strength and statistical significance of the effect was the same across all time points during the 20-year mortality follow-up). First, we mean centered survival time and each continuous predictor, created an interaction term between survival time and each covariate, and included it in the Cox model. We also investigated martingale residuals which provides a test of proportionality for each variable based on the empirical score process. There were no violations of proportionality.

Using a series of basic Cox models, we first tested the personality-mortality effect without formal tests of mediation so we could identify basic associations among personality and mortality risk. Model 1 included the Big Five traits unadjusted for demographic covariates (all Big Five traits were included in these models but Supplemental Appendix D contains results with each trait tested in its own model). In Model 2, we adjusted for MIDUS 1 covariates (i.e., age, gender, race, education, relationship status) to determine how adjustment for these confounds would affect the baseline personality-mortality associations. In a second step, we tested the linear and curvilinear effect of sleep duration[2]. Model 1 included personality traits, MIDUS 1 covariates, and the linear effect of sleep duration. Models 2 and 3 included the quadratic and cubic effect of sleep duration, respectively. In Model 4, we adjusted for retirement status, measured at MIDUS 2.

In a third step, we tested the effect of our full sleep quality measure and then separated sleep quality into separate components (i.e., sleep problems and daytime dysfunction), testing the effect of each component separately and together. We did this to identify the precise aspects of sleep quality that were driving sleep-mortality effects. Model 1 included personality traits, MIDUS 1 covariates, and the full sleep quality measure. Model 2 included the 3-item sleep problem measure. Model 3 included daytime dysfunction. Model 4 included sleep problems and daytime dysfunction. Finally, in Model 5, we adjusted for retirement status.

To formally test mediation, we utilized proportional hazards modeling in an SEM framework to estimate the direct and indirect effects on survival time (Asparoouhov et al., 2006). The maximum likelihood robust estimator (MLR) and Monte Carlo integration enables the program to calculate indirect effects similar to the Sobel method. A *product-of-coefficients* approach computes the ratio of the path from the predictor to the mediator and the path from the mediator to the outcome to its standard error. This technique provides standard errors, confidence intervals, and significance tests. The significance tests enabled us to determine whether there was mediation via the significance of an indirect effect. We calculated the indirect effect through any significant sleep predictors of mortality risk and a joint indirect effect through these sleep predictors.

3. Results

Table 1 provides descriptive statistics for our sample, stratified by survival status. Relative to those who survived, deceased individuals were more likely to be older ($t = -23.54$, $p < .001$); male ($\chi^2 = 14.50$, $p < .001$); less educated ($t = 5.72$, $p < .001$); single ($\chi^2 = 16.53$, $p < .001$); retired ($\chi^2 = 331.46$, $p < .001$) and scored

[2] Using the same procedure, we tested zero-order sleep duration (i.e., linear, quadratic, and cubic effects) and sleep quality effects, adjusted and unadjusted for demographics at MIDUS 1 and retirement at MIDUS 2. Across unadjusted and adjusted models, quadratic duration and higher daytime dysfunction predicted increased mortality even when personality traits were not included in the models.

Table 1
Descriptive statistics.

	Deceased (N = 403)	Alive (N = 3356)	Full Sample (N = 3759)	
Variables	Mean (*SD*) or %	Mean (*SD*) or %	Mean (*SD*) or %	Range
Age	59.93 (10.17)	45.62 (11.68)	47.15 (12.35)	20–75
Education	6.50 (2.48)	7.24 (2.45)	7.16 (2.46)	1–12
Gender				
Male	53.85%	43.86%	44.93%	
Female	46.15%	56.14%	55.07%	
Race				
White	95.78%	93.92%	94.12%	
Other	4.22%	6.08%	5.88%	
Relationship Status				
Partnered	67.74%	76.91%	75.92%	
Not partnered	32.26%	23.09%	24.08%	
Retirement Status				
Retired	67.25%	23.90%	28.54%	
Not Retired	32.75%	76.10%	71.46%	
Conscientiousness	3.40 (0.45)	3.46 (0.43)	3.45 (0.43)	1–4
Agreeableness	3.50 (0.48)	3.48 (0.49)	3.48 (0.49)	1–4
Neuroticism	2.16 (0.65)	2.23 (0.66)	2.22 (0.66)	1–4
Openness	3.01 (0.52)	3.01 (0.51)	3.01 (0.51)	1–4
Extraversion	3.20 (0.57)	3.19 (0.55)	3.19 (0.55)	1–4
Duration (minutes)	434.44 (85.16)	430.02 (63.07)	430.49 (65.79)	150–600
Sleep Quality	3.42 (1.01)	3.50 (0.86)	3.49 (0.88)	1–5
Daytime Dysfunction	2.58 (1.29)	2.54 (1.11)	2.54 (1.13)	1–5
Sleep Problems	2.59 (1.05)	2.48 (0.93)	2.49 (0.94)	1–5

lower on conscientiousness ($t = 2.44$, $p < .05$) and had more sleep problems ($t = -2.11$, $p < .05$). There were no significant differences in race, agreeableness, neuroticism, openness, extraversion, sleep duration, sleep quality, or daytime dysfunction.

Because weekday and weekend sleep duration were not normally distributed, we winsorized these variables by replacing six outlier data points with the 99th percentile value (600 minutes) before averaging weekday and weekend duration together to yield a score for total sleep duration. This normalized the distribution for these variables. Additionally, we excluded data from 63 participants in our analyses of sleep duration because they reported implausible weekday or weekend durations (i.e., seven minutes per night).

There were modest positive correlations between our sleep mediators (see Appendix B for bivariate correlations). Specifically, sleep duration was related to the full sleep quality measure ($r = 0.35$; $p < .001$) as well as each component of sleep quality (daytime dysfunction: $r = -0.21$; $p < .001$; sleep problems: $r = -0.35$; $p < .001$).

Table 2 depicts the baseline personality-mortality effect.[3] Model 1 indicated that lower conscientiousness and neuroticism directly predicted increased death risk. Model 2 indicated that older age, female gender, lower education and being single were associated with an increased hazard of dying. After adjustment for these confounds, the effect for lower conscientiousness remained significant (albeit reduced in magnitude by 26.6%; $HR_{baseline} = 0.852$; $HR_{adjusted} = 0.892$); however, the effect for neuroticism was attenuated to non-significance. Supplementary analyses suggest that adjusting for either age or education resulted in higher openness being associated with increased mortality risk. See Appendix D for personality-mortality associations when each personality trait is analyzed without adjustment for the other Big Five traits.

Table 3 depicts tests of sleep duration on mortality risk. Consistent with our prior models, Model 1 revealed that older age, female gender, lower education, not living with a partner, lower conscientiousness and higher openness predicted an increased hazard of dying. But the linear effect of sleep duration was not predictive of death risk. However, as seen in Model 2, the quadratic effect was significant: each standard deviation increase or decrease in sleep duration (approximately 65 minutes), relative to the mean (approximately seven hours in this sample), was associated with a 10% increased risk of dying over 20 years. Moreover, after adjusting for quadratic sleep duration, the effect of conscientiousness on mortality risk was no longer statistically significant (13.9 % drop in hazard ratio), but the effect of openness remained without any change to the hazard ratio. Model 3 indicated the cubic effect of sleep duration was nonsignificant. The quadratic effect of sleep duration was the only duration variable that remained significant. Thus, we tested this effect after adjusting for retirement in Model 5, with lower conscientiousness, higher openness, and being retired predicting increased death risk. Note the 95% confidence interval surrounding conscientiousness bordered the cutoff for significance versus nonsignificance, regardless of which sleep variables were included in the model. Fig. 2 depicts the effect of quadratic sleep duration: the effect is significant at two standard deviations below and above the mean, consistent with prior work. Lastly, we estimated how much the personality-mortality effect

Table 2
Personality predicting mortality.

Predictors	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]
Conscientiousness	0.85 [0.77, 0.94]**	0.89 [0.80, 0.99]*
Agreeableness	1.10 [0.97, 1.24]	0.98 [0.86, 1.11]
Neuroticism	0.88 [0.79, 0.97]*	1.04 [0.93, 1.15]
Openness	0.99 [0.88, 1.11]	1.16 [1.02, 1.31]*
Extraversion	0.99 [0.87, 1.12]	0.92 [0.81, 1.05]
Age		3.40 [3.02, 3.83]***
Race		0.77 [0.47, 1.26]
Gender		1.61 [1.31, 1.99]***
Education		0.79 [0.71, 0.88]***
Married/partnered		1.70 [1.37, 2.11]***
AIC	6585.29	6030.15
SBC	6605.28	6070.14

Note. AIC = Akaike Information Criterion. SBC = Schwarz Bayesian (Information) Criterion. CI = Confidence Interval. *$p < .05$. **$p < .01$. ***$p < .001$.

[3] We tested all effects, modeling mortality using both survival time and attained age as the time metric and found the same results. We also tested the effect of sleep discrepancy (i.e., the difference between weekday and weekend sleep durations), but found this variable did not predict mortality. Results are available from the author upon request.

Table 3
Sleep duration predicting mortality.

Predictors	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]	Model 4 Hazard Ratio [95% CI]
Age	3.40 [3.01, 3.83]***	3.28 [2.90, 3.69]***	3.28 [2.91, 3.69]***	2.95 [2.55, 3.41]***
Race	0.78 [0.48, 1.27]	0.73 [0.45, 1.20]	0.74 [0.45, 1.20]	0.75 [0.46, 1.22]
Gender	1.61 [1.31, 1.99]***	1.59 [1.29, 1.96]***	1.59 [1.29, 1.96]***	1.56 [1.26, 1.93]***
Education	0.79 [0.71, 0.88]***	0.80 [0.72, 0.89]***	0.80 [0.72, 0.88]***	0.80 [0.72, 0.89]***
Married/Partnered	1.70 [1.37, 2.11]***	1.62 [1.30, 2.01]***	1.63 [1.31, 2.03]***	1.63 [1.32, 2.03]***
Conscientiousness	0.89 [0.80, 0.99]*	0.91 [0.82, 1.01]	0.91 [0.82, 1.00]	0.90 [0.81, 1.00]*
Agreeableness	0.98 [0.86, 1.12]	0.98 [0.86, 1.11]	0.98 [0.86, 1.11]	0.97 [0.85, 1.10]
Neuroticism	1.04 [0.94, 1.16]	1.02 [0.92, 1.14]	1.02 [0.92, 1.14]	1.02 [0.91, 1.13]
Openness	1.16 [1.03, 1.31]*	1.16 [1.03, 1.31]*	1.16 [1.03, 1.31]*	1.16 [1.03, 1.32]*
Extraversion	0.92 [0.80, 1.05]	0.92 [0.81, 1.05]	0.92 [0.81, 1.05]	0.93 [0.81, 1.06]
Linear Duration	1.04 [0.95, 1.14]	1.10 [1.01, 1.19]*	1.13 [0.99, 1.29]	1.10 [1.01, 1.19]*
Quadratic Duration		1.13 [1.08, 1.18]***	1.12 [1.07, 1.18]***	1.13 [1.08, 1.17]***
Cubic Duration			0.99 [0.97, 1.01]	
Retirement Status				1.39 [1.07, 1.79]*
AIC	6031.41	6005.79	6007.47	6001.31
SBC	6075.40	6053.78	6059.46	6053.29

Note. AIC = Akaike Information Criterion. SBC = Schwarz Bayesian Criterion. CI = Confidence Interval. *$p < .05$. **$p < .01$. ***$p < .001$.

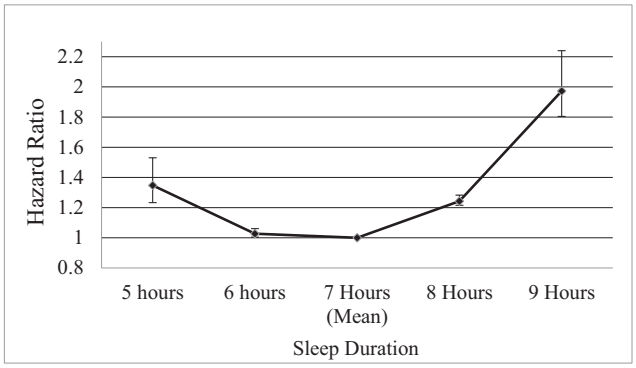


Fig. 2. The curvilinear effect of sleep duration on mortality risk. The data points represent the hazard ratio associated with raw hours of sleep duration that span roughly two standard deviations above and below the mean, with the error bars representing the 95% confidence interval.

changed after adjusting for linear and quadratic sleep duration, comparing the results from Tables 2 and 3, respectively. Adjusting for quadratic duration attenuated the effect of conscientiousness by 8.32% ($HR_{baseline} = 0.892$; $HR_{adjusted} = 0.901$), while the effect of openness increased by 3.74% ($HR_{baseline} = 1.156$; $HR_{adjusted} = 1.162$). Such changes in hazard ratios could be indicative of mediation but are not necessary criteria for the calculation of indirect effects (Preacher & Hayes, 2008).

Table 4 depicts the effect of sleep quality. Testing the full measure in Model 1 indicated that older age, female gender, lower education, not living with a partner, lower conscientiousness, and higher openness were associated with increased death risk. Breaking our measure into separate components (i.e., sleep problems, daytime dysfunction) and testing each separately (Models 2 and 3) and together (Model 4), revealed that daytime dysfunction, but not sleep problems, positively predicted death risk. Thus, it appeared that daytime dysfunction was driving the effect for the full sleep quality measure in Model 1. Accordingly, we only tested the daytime dysfunction-mortality effect after adjusting for retirement in Model 5, with the results for daytime dysfunction remaining the same and being retired predicting increased death risk. Comparing the baseline personality-mortality effects displayed in Table 2 with those adjusting for sleep quality in Table 4, we found the conscientiousness effect was attenuated 5.5% ($HR_{baseline} = 0.892$; $HR_{adjusted} = 0.898$), while the openness effect increased by 6.4% ($HR_{baseline} = 1.156$; $HR_{adjusted} = 1.166$).

Next, we estimated the direct, indirect, and total effect of all five personality traits on mortality risk via sleep components using proportional hazards in an SEM framework, adjusting for age, gender, race, education, living with a partner, and retirement status. We also adjusted for linear sleep duration in models testing mediation by quadratic sleep duration.[4]

First, we tested mediation via quadratic sleep duration. As seen in Fig. 3, lower conscientiousness was associated with increased death risk via the direct, indirect (path a*f), and total effect of quadratic sleep duration. Specifically, lower conscientiousness was associated with getting fewer hours of sleep, which predicted an increased risk of death. Additionally, there was an indirect effect (path b*f) from agreeableness to mortality via sleep duration such that higher agreeableness was associated with getting fewer hours of sleep, which predicted an increased risk of death. Lastly, there was also an indirect effect (path c*f) for neuroticism such that higher neuroticism was associated increased death risk via short or long sleep duration.

Next, we tested mediation via daytime dysfunction, as shown in Fig. 5. Higher agreeableness (path b*f), neuroticism (path c*f), lower conscientiousness (path a*f), and extraversion (path e*f) predicted increased death risk via the indirect effect of greater daytime dysfunction.

Finally, we tested mediation via the joint effect of quadratic duration and daytime dysfunction. As seen in Figure 6, higher neuroticism (sum of paths [c1*f1], [c2*f2]) and agreeableness (sum of paths [b1*f1], [b2*f2]) and lower conscientiousness (sum of paths [a1*f1], [a2*f2]) predicted increased death risk via the joint indirect effect of both sleep components. We should note that we only reported the significant effects here in text and refer readers to the Figs. 4–6 for a complete summary of the direct, indirect, and total effects from these models. See Appendix D for direct, indirect, and total effects when each personality trait is analyzed without adjustment for the other Big Five traits.

4. Discussion

We extended the personality-mortality literature by examining a novel behavioral mediator: sleep. First, we replicated prior

[4] Note, in all models, only higher openness directly predicted an increased hazard of dying, even after adjusting for the mediating effect of sleep components. See also Appendix C for additional analyses adjusting for self-rated health and waist circumference.

Table 4
Sleep quality predicting mortality.

Predictors	Model 1 Hazard Ratio [95% CI]	Model 2 Hazard Ratio [95% CI]	Model 3 Hazard Ratio [95% CI]	Model 4 Hazard Ratio [95% CI]	Model 5 Hazard Ratio [95% CI]
Age	3.38 [3.00, 3.81]***	3.38 [3.00, 3.82]***	3.48 [3.08, 3.92]***	3.51 [3.11, 3.97]***	3.09 [2.66, 3.58]***
Race	0.77 [0.47, 1.25]	0.77 [0.47, 1.26]	0.77 [0.47, 1.25]	0.77 [0.47, 1.25]	0.78 [0.48, 1.28]
Gender	1.65 [1.33, 2.03]***	1.63 [1.32, 2.01]***	1.64 [1.33, 2.03]***	1.63 [1.32, 2.01]***	1.62 [1.31, 2.00]***
Education	0.79 [0.71, 0.88]***	0.79 [0.71, 0.88]***	0.80 [0.72, 0.89]***	0.80 [0.72, 0.89]***	0.80 [0.72, 0.89]***
Relationship	1.69 [1.36, 2.10]***	1.70 [1.37, 2.11]***	1.66 [1.34, 2.06]***	1.65 [1.33, 2.05]***	1.68 [1.35, 2.08]***
Conscientiousness	0.90 [0.81, 0.99]*	0.89 [0.81, 0.99]*	0.90 [0.82, 1.00]	0.90 [0.82, 1.00]	0.90 [0.81, 1.00]
Agreeableness	0.98 [0.86, 1.11]	0.98 [0.86, 1.11]	0.96 [0.84, 1.09]	0.96 [0.84, 1.09]	0.95 [0.84, 1.08]
Neuroticism	1.01 [0.91, 1.13]	1.03 [0.92, 1.15]	0.99 [0.89, 1.10]	1.00 [0.89, 1.11]	0.98 [0.88, 1.10]
Openness	1.16 [1.02, 1.31]*	1.16 [1.02, 1.31]*	1.17 [1.03, 1.32]*	1.17 [1.03, 1.32]*	1.17 [1.03, 1.32]*
Extraversion	0.93 [0.81, 1.06]	0.92 [0.81, 1.06]	0.94 [0.83, 1.08]	0.94 [0.83, 1.08]	0.95 [0.83, 1.09]
Sleep Quality	0.91 [0.83, 1.01]				
Sleep Problems		1.04 [0.94, 1.15]		0.94 [0.84, 1.05]	
Daytime Dysfunction			1.22 [1.10, 1.34]***	1.25 [1.12, 1.40]***	1.22 [1.10, 1.35]***
Retirement Status					1.42 [1.10, 1.85]**
AIC	6028.83	6031.53	6018.03	6018.90	6012.66
SBC	6072.82	6075.52	6062.02	6066.88	6060.64

Note. AIC = Akaike Information Criterion. SBC = Schwarz Bayesian Criterion. CI = Confidence Interval. *$p < .05$. **$p < .01$. ***$p < .001$.

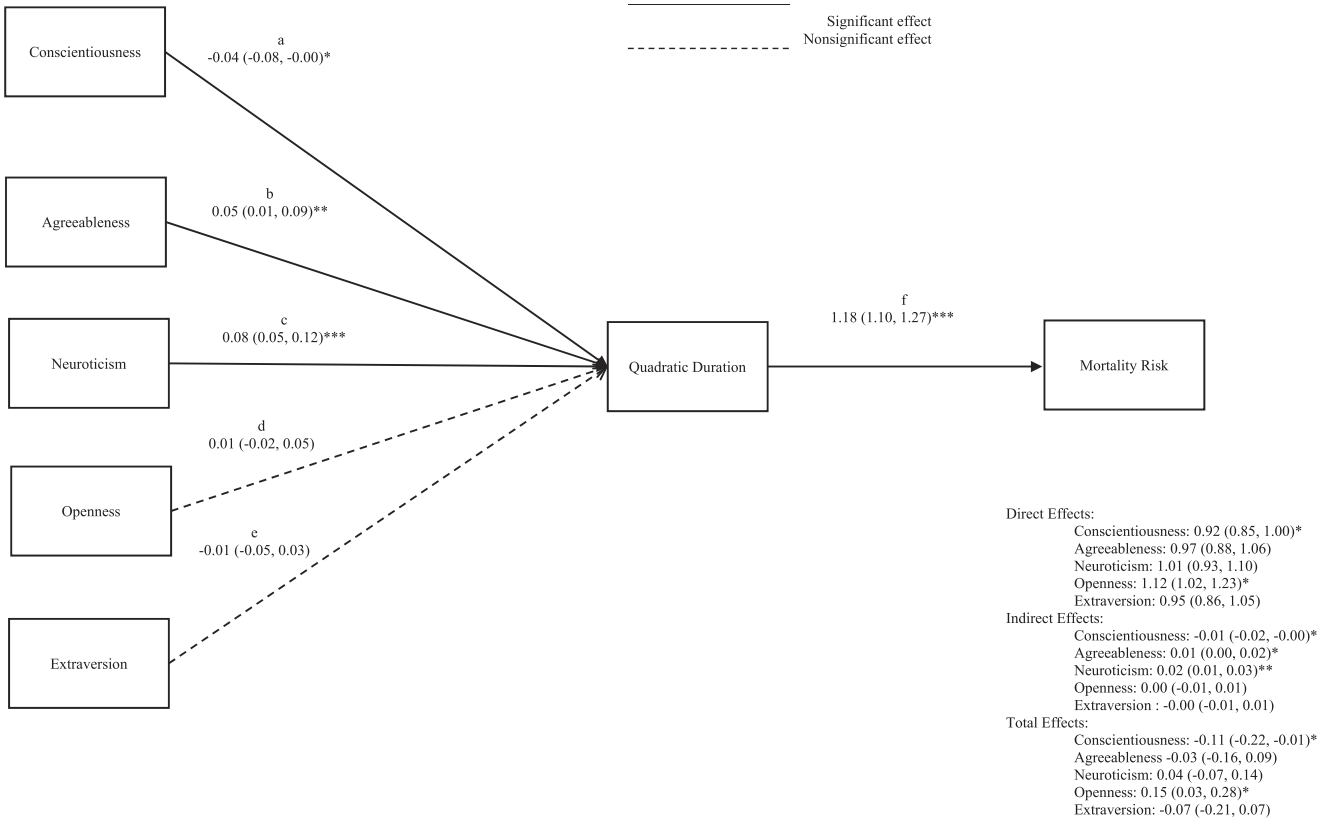


Fig. 3. Fully adjusted path model controlling for age, race, relationship status, education, linear sleep duration, and retirement status.* $p < .05$. ** $p < .01$. *** $p < .001$.

research linking lower conscientiousness and higher neuroticism to increased mortality risk (Jokela et al., 2013; Mroczek & Spiro, 2007). However, contrary to our expectations and previous findings (Friedman et al., 1993; Turiano et al., 2015), higher openness emerged as a predictor of increased death risk after adjusting for covariates. Second, we replicated findings linking short and long sleep duration (Cappuccio et al., 2010) and higher daytime dysfunction (Rockwood, Davis, Merry, MacKnight, & McDowell, 2001) to increased mortality risk. However, the full sleep quality index was not related to death risk. Finally, we found support for sleep as a mechanism of the personality-mortality association. Specifically, lower conscientiousness was associated with increased death risk indirectly via both sleep components. Higher agreeableness and neuroticism also predicted increased mortality risk via the indirect effect of both sleep components. Lastly, lower extraversion was related to increased mortality risk via the indirect effect of higher daytime dysfunction.

Consistent with prior meta-analytic work on sleep duration and mortality (Cappuccio et al., 2010), we are the first to use the MIDUS cohort to document that both short and long sleep durations were associated with increased 10-year all-cause mortality risk. Short sleep duration is associated with risk factors, such as inflammation

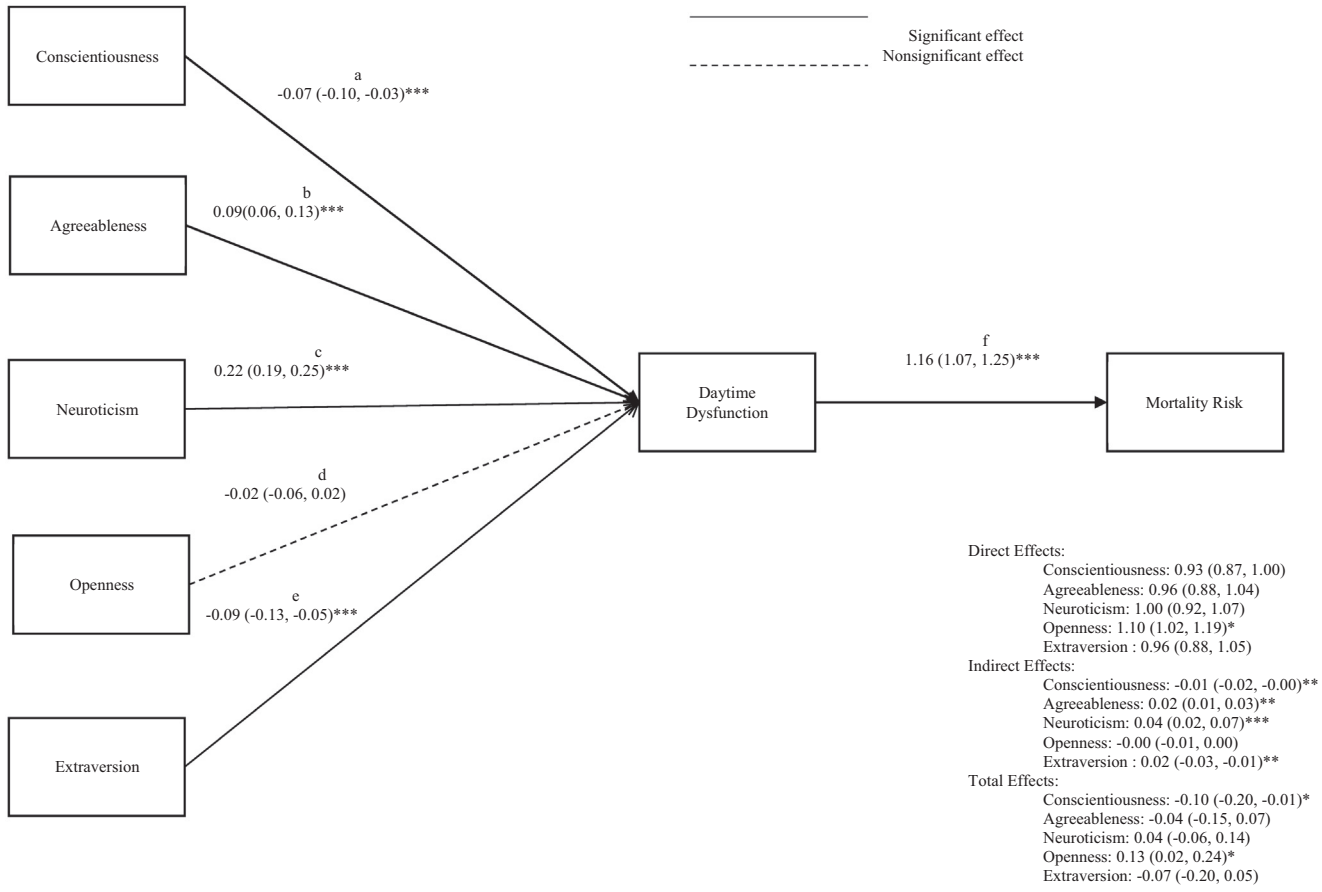


Fig. 4. Fully adjusted path model controlling for age, race, relationship status, education, and retirement status.* $p < .05$. ** $p < .01$. *** $p < .001$.

(Patel et al., 2009), in conditions such as cardiovascular disease and cancer (Berg & Scherer, 2005; Buysse, 2014). However, long sleep duration is often considered a marker for deteriorating health (Cappuccio, Cooper, Delia, Strazzullo, & Miller, 2011).

Moreover, higher daytime dysfunction is associated with conditions that increase mortality risk, such as cardiovascular disease (Newman et al., 2000). Thus, we were not surprised that daytime dysfunction was also related to increased mortality, but we should note that our null effect for the full sleep quality measure diverges from some investigations (e.g., Kojima et al., 2000). Nevertheless, sleep problems (e.g., trouble falling asleep) and daytime impairment (e.g., feel unrested upon waking or nonrestorative sleep) appear to be different constructs, showing divergent patterns with health outcomes (e.g., sleep problems: cardiovascular disease; daytime impairment/nonrestorative sleep: sleep disorders, respiratory disease, cancer: Zhang et al. (2013)). Moreover, some evidence suggests that nonrestorative sleep or daytime impairment shows a stronger association with role impairment (i.e., one's ability to carry out work and daily activities; daytime sleepiness) than sleep problems (Roth et al., 2006). This may be why we uncovered divergent associations between sleep problems and daytime impairment with mortality risk in our sample, with greater daytime impairment being related to greater death risk but not sleep problems (nor the full sleep quality measure). However, we utilized a one-item measure of daytime dysfunction; thus, future studies should test the replicability of this effect with more comprehensive measures of daytime functioning.

Consistent with previous findings, lower conscientiousness was related to increased death risk (Jokela et al., 2013). However, contrary to our expectations and previous findings (e.g., Turiano et al., 2015; Turiano, Spiro, & Mroczek, 2012), openness showed a positive association with death risk, but only after adjusting for possible confounding demographic variables. The magnitude of these associations increased after adjustment for sleep. Thus, beyond sleep behavior, higher openness increases mortality risk through other avenues. For example, previous investigations of the MIDUS cohort linked higher openness, as well as increases in openness over time, to more smoking, alcohol, and illicit drug use (Turiano, Whiteman, Hampson, Roberts, & Mroczek, 2012). Alternatively, because prior personality-mortality investigations of the MIDUS cohort (Chapman, Fiscella, Kawachi, & Duberstein, 2010; Turiano et al., 2015) found no effects for openness, this trait may represent a late onset risk factor for poor health outcomes as the current study used an older sample than previously published reports. Additionally, self-ratings of evaluative traits, such as openness (John & Robins, 1993), tend to be less accurate than nonevaluative traits (e.g., extraversion, conscientiousness; Vazire, 2010), potentially contributing to inconsistencies in openness-health findings (Isreal et al., 2014). Thus, testing the replicability of our novel openness effect is important because it may represent a Type I error.

Other novel results from our study included our mediation effects. First, consistent with previous neuroticism-mortality findings (e.g., Jokela et al., 2013), there was no direct effect connecting this trait to death risk. Rather, consistent with previous personality-sleep findings (Allen et al., 2016; Duggan et al., 2014; Vincent et al., 2009), individuals higher on neuroticism reported greater daytime dysfunction and shorter and longer sleep durations, which increased the hazard of dying across 20 years. Prior studies indicate that individuals higher on neuroticism may expe-

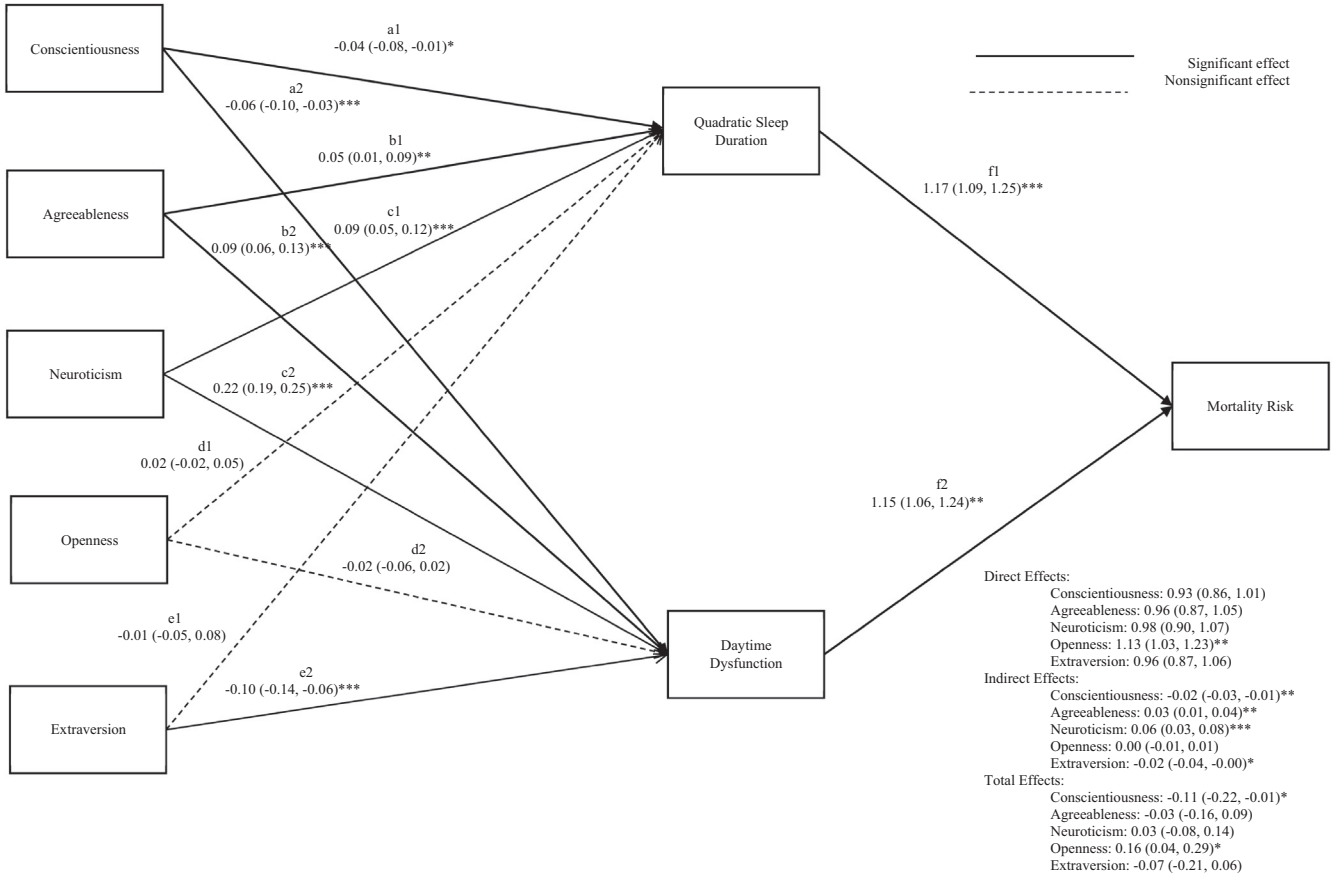


Fig. 5. Fully adjusted path model controlling for age, race, relationship status, education, linear sleep duration, and retirement status. * $p < .05$. ** $p < .01$. *** $p < .001$.

rience abnormal sleep duration and more sleep difficulties because they have difficulty regulating emotions, causing increased anxious arousal that interferes with falling asleep, staying asleep, and, ultimately, feeling rested during the day (Cellini et al., 2017; Slavish et al., 2018). Moreover, consistent with previous findings (Duggan et al., 2014), lower conscientiousness was associated with greater daytime dysfunction and short and long sleep duration, which increased the risk of dying. The poor self-regulation (e.g., inconsistent sleep schedules; increased alcohol use) characteristic of low conscientiousness is thought to disrupt sleep (Duggan et al., 2014), which may be why this aspect of personality was connected to mortality risk via sleep components in our sample.

However, based on our data, we are unable to discern why higher agreeableness would be connected to increased death risk via the indirect effect of both sleep components because the evidence for an agreeableness-sleep effect is mixed (Duggan et al., 2014). Similar to openness, agreeableness is a highly evaluative trait (John & Robins, 1993), making it more susceptible to inaccuracies in self-ratings and perhaps inconsistencies in findings (Vazire, 2010). Moreover, the evidence for an extraversion-health effect is also mixed (Duggan et al., 2014; Gray & Watson, 2002; but see also Stephan et al., 2018). Nevertheless, we found lower extraversion predicted death risk via the indirect effect of higher daytime dysfunction. Given a recent study linking higher extraversion to better sleep quality in middle-aged adults from the MIDUS sample (Stephan et al., 2018), there may be age differences in the magnitude of associations between extraversion and sleep quality (with associations stronger in older cohorts). Alternatively, the MIDUS extraversion personality questionnaire primarily includes content related to sociability and activity, but not assertiveness. Thus, perhaps differences in extraversion-sleep findings stem from measurement differences, with perhaps measures of extraversion including positive affect, sociability and activity showing stronger associations with sleep quality than inventories that also cover dominance. Thus, further research is needed to determine whether these effects generalize beyond our sample.

Importantly, our mediation effects demonstrate that personality can be related to death risk (or health) via the indirect effect of sleep (or other health behaviors), even in the absence of a direct effect. Historically, mediation guidelines (Baron & Kenny, 1986) required a significant direct effect between the focal predictor(s) and outcome (in this study personality and mortality risk) for mediators to even be considered as explanatory variables of that direct effect. However, more recent empirical and simulation evidence suggests that such direct effects are not necessary for establishing mediation effects (Hayes, 2009; Zhao, Lynch, & Chen, 2010). In fact, when indirect effects are estimated, significant indirect effects emerge in the absence of significant direct effects roughly half the time (Rucker, Preacher, Tormala, & Petty, 2011).

Shrout and Bolger (2002) also discuss the importance of identifying proximal versus distal mediation processes because the direct effect is often underpowered in tests of mediation (Rucker et al., 2011; Shrout & Bolger, 2002). If personality were a proximal causal agent of mortality risk, we would expect any change in personality to predict changes in mortality risk shortly thereafter, and, thus, a significant direct effect. However, personality change occurs over many years (Roberts, Walton, & Viechtbauer, 2006), and any effects of personality on mortality risk need to accumulate over years. For example, lower levels of conscientiousness are associated with increased tobacco and alcohol use and a host of other

problematic behaviors (Bogg & Roberts, 2004). Nevertheless, engagement in these behaviors in the short term might not necessarily lead to an immediate increase in mortality risk as the damaging effects of these behaviors take years to influence health. Thus, because personality and mortality are more distally related, we would expect weaker evidence for the direct effect of personality on mortality. Rather, we would expect the personality-sleep association, as well as the sleep-mortality associations to be more strongly and proximally associated, making them easier to quantify statistically (Shrout & Bolger, 2002). In sum, these separate paths involved in the calculation of the indirect effect are more powerful than the simple direct effect of personality on mortality risk.

Overall, our results are consistent with findings linking personality to sleep (Duggan et al., 2014; Gray & Watson, 2002) and sleep to mortality risk (Duggan et al., 2014; Kripke et al., 2002) and include several strengths. First, we utilized a parsimonious modeling technique to formally test mediation using proportional hazards, giving us empirical evidence of a novel behavioral pathway connecting personality to longevity: sleep. Additionally, our mediation tests of quadratic sleep duration advanced the personality-health literature methodologically. Traditionally, researchers assume linear relations between independent, mediator, and dependent variables when testing mediation. However, many variables are related in a nonlinear manner, including sleep duration and mortality risk (Duggan et al., 2014). Thus, testing mediation of quadratic sleep duration was theoretically sound, allowing us to detect an effect that would otherwise be overlooked. These mediation tests also represented a more statistically robust approach than alternative methods, such as testing this effect in subgroups (i.e., short, average, long duration), which masks important individual differences and inflates the risk of obtaining spurious results (Hayes & Preacher, 2010). Testing quadratic sleep duration also allowed us to connect personality to sleep duration, an effect that rarely emerges in the personality-sleep literature (Gray & Watson, 2002), perhaps because curvilinear effects are often overlooked (for an exception of a study linking higher neuroticism to quadratic sleep duration see Allen et al., 2016).

Finally, our study remedies limitations from prior HBM findings because the behavioral mediators in these personality-mortality studies were measured concurrently with personality (e.g., Hill et al., 2013; Graham et al., 2017; Turiano et al., 2015). However, such cross-sectional mediation studies complicate the temporal ordering in the mediated chain of effects because no time has elapsed that would allow personality to causally predict the mediator (Preacher, 2015). Accordingly, we extended these studies by testing not only the contribution of novel behavioral mediators (sleep duration and quality), but utilizing a longitudinal mediational design that allowed personality to prospectively predict sleep, and sleep behavior to then prospectively predict mortality risk. Such temporal ordering is necessary in mediational analyses to get closer to specific causal agents.

Notably, our tests of the HBM also add to the literature aimed at developing personality-based health interventions by exploring pathways linking personality traits to premature death. Our results have implications for personality-based health interventions, offering insights into what treatments would be effective for whom. Although everyone would likely benefit from improving their sleep health (Buysse, 2014), our findings suggest that individuals scoring lower on conscientiousness and extraversion and higher on neuroticism and agreeableness may be especially at risk for poorer sleep outcomes. Accordingly, these individuals might benefit the most from treatments aimed at normalizing sleep duration and improving daytime functioning. Alternatively, changing these aspects of personality could improve sleep for these individuals, enhancing their health and longevity (Roberts et al., 2017). These findings also highlight the utility of using personality assessment in personalized medicine to identify those at risk for health problems, whether it be for sleep problems or poor health outcomes.

Despite these practical applications, our study included some limitations. First, our sample was predominately White, highly educated, and middle-aged, potentially limiting generalizability. Nevertheless, our investigation is the first to test whether sleep components explain the personality-mortality association in a large national sample. Moreover, we are not aware of any research suggesting that our findings would differ in more diverse samples (e.g., higher neuroticism predicts poorer sleep cross-culturally: Kim et al., 2015). Additionally, because sleep data were only available for the second and third waves of MIDUS, we did not have the three data points required to test bidirectional effects between personality and sleep. However, testing these effects remains important: neither personality traits nor sleep are static variables (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004; Roberts et al., 2006). Because sleep data were not collected at the first wave of MIDUS, we were also unable to test whether baseline sleep better accounted for differences in mortality risk beyond personality traits. Relatedly, we were unable to control for sleep disorders in our analyses as MIDUS does not include adequate data on whether participants were diagnosed with, or treated for, sleep disorders.

Additionally, because this study was an initial attempt to understand how sleep relates to the personality-mortality association, we refrained from using a more stringent alpha correction to avoid making a Type II error in this early-stage research. Because participants excluded from our sample due to missing data were more likely to be lower on conscientiousness and higher on agreeableness and daytime dysfunction, our indirect effects for conscientiousness and agreeableness may have been underestimated. Moreover, we utilized a shortened personality scale, possibly limiting our ability to detect effects because the Big Five personality dimensions were not comprehensively covered (e.g., the agreeableness scale measured prosocial aspect of this dimension, with no coverage of antagonism, hostility, etc.).

Lastly, it remains unclear whether participants reported their sleep durations based on the amount of time spent in bed vs. time sleeping, as this distinction was not made with the MIDUS sleep questionnaire. Future investigations should use behavioral measures such as actigraphy (Hall, 2010) to disentangle whether personality traits are linked with sleep perceptions as well as actual sleep behaviors, and also the extent to which time spent in bed relative to time spent asleep (i.e., low sleep efficiency) is a possible mechanism of personality-health associations. Though the literature on how personality differentially predicts subjective vs. objective sleep measures is sparse, a recent study with a MIDUS subsample linked lower neuroticism and higher conscientiousness to more sleep continuity (measured via actigraphy) and better subjective sleep quality. However, the other three personality dimensions were only predictive of subjective sleep quality, not objective sleep outcomes (Križan & Hisler, 2019).

4.1. Conclusions

In sum, we extended the personality-mortality literature by testing whether sleep accounted for this effect. In the process, we advanced the use of proportional hazards in an *SEM* framework within the personality-health literature. Our results also have important applications for personality-based health interventions, informing our understanding about who might be at risk for premature death and why. Overall, our findings suggest that short and long sleep duration and daytime dysfunction may be important pathways linking aspects of personality to reduced life expectancy.

Author contributions

Shantel Spears contributed to the study conception and design, analysis and interpretation of data, and drafting of the manuscript.

Hawley Montgomery-Downs contributed to the study design and the critical revision of the manuscript.

Shari Steinman contributed to the study design and the critical revision of the manuscript.

Katherine Duggan contributed to the critical revision of the manuscript.

Nicholas Turiano contributed to the study conception and design, interpretation of data, and the critical revision of the manuscript.

Declarations of interest

None.

Funding

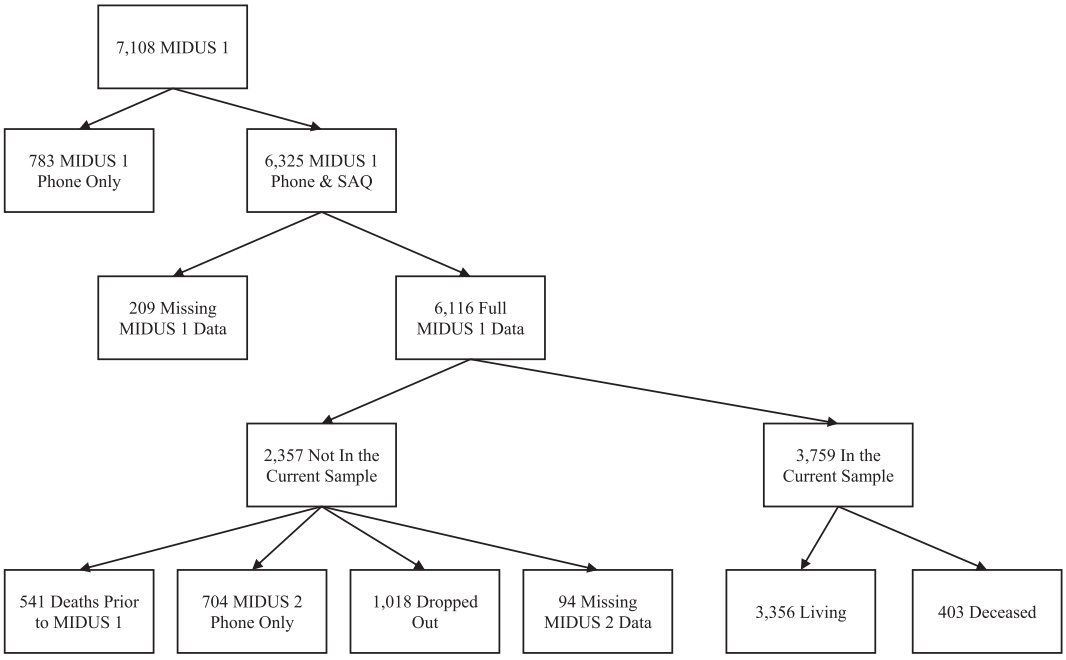
This research was supported by the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife Development. The MIDUS II research was supported by a grant from the National Institute on Aging (P01-AG020166) to conduct a longitudinal follow-up of the MIDUS I investigation. Research support for K. A. D. was provided by the National Heart, Lung, and Blood Institute at the National Institutes of Health (T32HL007560 and L30HL143741).

Role of Funder

The funders had no role in the study design, data analysis and interpretation, or manuscript preparation.

Appendix A

Attrition and mortality diagram.



Appendix B. Descriptive statistics for study variables.

	M (SD) or %	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age	47.15 (12.35)	–													
2. Education	7.16 (2.47)	−0.09***	–												
3. Sex (male)	45%	0.03	0.12***	–											
4. Race (minority)	6%	−0.05***	−0.01	−0.04**	–										
5. Partnered	24%	−0.01	0.04**	−0.10***	0.08***	–									
6. Retired	29%	0.66***	−0.07***	−0.05**	−0.04*	−0.01	–								
7. Agreeableness	3.48 (0.49)	0.09***	−0.09***	−0.27**	0.02	0.02	0.06***	–							
8. Extraversion	3.19 (0.55)	0.03*	−0.03	−0.07***	0.04*	−0.02	0.01	0.52***	–						
9. Neuroticism	2.22 (0.66)	−0.16***	−0.10***	−0.11***	−0.01	0.05**	−0.08***	−0.03	−0.15***	–					
10. Conscientiousness	3.45 (0.43)	0.03	0.10***	−0.12***	−0.01	−0.04**	0.02	0.28***	0.27***	−0.19***	–				
11. Openness	3.01 (0.51)	−0.01	0.21***	0.06***	0.05***	0.06***	−0.01	0.34***	0.50***	−0.17***	0.27***	–			
12. Duration [a]	430.49 (65.79)	0.03	0.06**	−0.03	−0.06***	−0.01	0.04*	−0.06***	−0.02	−0.06***	0.03	−0.03	–		
13. Sleep Quality [b]	3.49 (0.88)	0.01	0.09***	0.14***	−0.01	−0.03	−0.01	−0.05**	0.07***	−0.28***	0.09***	0.08***	0.35***	–	
14. Dysfunction [c]	2.54 (1.13)	−0.16***	−0.08***	−0.11***	−0.01	0.06***	−0.10***	0.02	−0.11***	0.28***	−0.11***	−0.10***	−0.21***	−0.71***	–
15. Sleep Problem [d]	2.49 (0.94)	0.05***	−0.08***	−0.14***	0.01	0.01	0.05***	0.05***	−0.04**	0.23***	−0.06***	−0.06***	−0.35***	−0.96***	0.49***

Note. *$p < .05$. **$p < .01$. ***$p < .001$. a = Duration in minutes. B = Full sleep quality measure. c = Daytime Dysfunction. d = Three-item sleep problem measure.

Appendix C

For transparency purposes and to demonstrate robustness of our effects, we also included results from models that were a part of our initial submission of this manuscript. We originally included self-rated physical health as a covariate at baseline to adjust for differences in health when personality was assessed. During the review process we agreed with reviewer comments that self-rated health was more in line with the mediational chains of the personality-health association and not a confound and thus we removed it from our models. When we did include self-rated health as a baseline covariate (Table 3), we find that self-rated health was strongly predictive of mortality risk (HR = 0.78; 95% CI = 0.70–0.87) and it reduced the conscientiousness direct effect on mortality to non-significance (HR = 0.92; 95% CI = 0.83–1.03). However, the openness direct effect on mortality remained unchanged.

We also had originally included waist circumference as a covariate when sleep variables were included in models in our original submission because this variable could act as a proxy for sleep apnea. However, the etiology of adiposity is inherently tied to personality processes so like self-rated health, we agreed with reviewers that this variable is more than likely on the causal pathway and not a confound. When we included self-rated health and waist circumference in models that included the sleep duration variables (Table 4), we found that SRH (HR = 0.83; 95% CI = 0.75–0.92) and waist circumference (HR = 1.17; 95% CI = 1.05–1.32) were strongly predictive of mortality risk. Self-rated health reduced the conscientiousness direct effect on mortality to non-significance (HR = 0.93; 95% CI = 0.83–1.04). However, the openness direct effect on mortality maintained significance and slightly increased in strength (HR = 1.20; 95% CI = 1.05–1.36). When self-rated health and waist circumference were included in models with sleep dysfunction (Table 4), self-rated health reduced the conscientiousness direct effect on mortality to non-significance (HR = 0.93; 95% CI = 0.83–1.03). However, the openness direct effect on mortality maintained significance and slightly increased in strength (HR = 1.20; 95% CI = 1.06–1.37).

Finally, we tested the robustness of the indirect effects by also including self-rated health and waist circumference as covariates in our mediation models. For sleep duration, the conscientiousness (IE = -0.003; 95% CI = -0.011-0.004) and agreeableness (IE = -0.007; 95% CI = -0.001-0.015) indirect effects were reduced to non-significance. For daytime dysfunction, only the conscientiousness indirect effect was reduced to non-significance (IE = -0.005; 95% CI = -0.012-0.002). In summary, these analyses suggest substantial variance that self-rated health accounts for in the personality-mortality association via sleep behavior.

Appendix D. Personality predicting mortality with each big five trait in separate models.

See Table D1 and D2.

Table D1
Personality predicting mortality unadjusted for other big five personality traits.

Predictors	No covariate Model Hazard Ratio [95% CI]	Covariate Model[$] Hazard Ratio [95% CI]
Conscientiousness	0.89 [0.81, 0.98]**	0.90 [0.82, 0.99]*
Agreeableness	1.04 [0.94, 1.15]	0.95 [0.86, 1.05]
Neuroticism	0.91 [0.82, 1.01]	1.06 [0.95, 1.17]
Openness	0.99 [0.90, 1.09]	1.05 [0.95, 1.16]
Extraversion	1.00 [0.91, 1.11]	0.94 [0.86, 1.04]

Note. CI = Confidence Interval. *$p < .05$. **$p < .01$. ***$p < .001$.[$] = model adjusted for age race, gender, education, and married/partnered. Each personality effect is unadjusted for the other Big Five traits.

Table D2
Personality Predicting Mortality Unadjusted for Other Big Five Personality Traits.

Predictors	Quadratic Duration Hazard Ratio [95% CI]			Dysfunction Hazard Ratio [95% CI]		
	Direct Effect	Indirect Effect	Total Effect	Direct Effect	Indirect Effect	Total Effect
Conscientiousness	0.93 [0.86, 1.00]*	−0.009 [−0.149, −0.001]***	−0.106 [−0.202, −0.010]***	0.94 [0.88, 1.01]	−0.021 [−0.033, −0.008]***	−0.107 [−0.192, −0.021]**
Agreeableness	0.96 [0.88, 1.04]	0.008 [−0.001, 0.016]	−0.049 [−0.152, 0.054]	0.95 [0.89, 1.02]	0.001 [−0.005, 0.008]	−0.064 [−0.154, 0.026]
Neuroticism	1.02 [0.94, 1.12]	0.020 [0.010, 0.030]***	0.050 [−0.050, 0.160]	1.01 [0.94, 1.09]	0.048 [0.022, 0.074]***	0.059 [−0.036, 0.153]
Openness	1.05 [0.96, 1.13]	0.001 [−0.006, 0.008]***	0.059 [−0.045, 0.164]	1.04 [0.97, 1.10]	−0.017 [−0.028, −0.006]**	0.034 [−0.059, 0.126]
Extraversion	0.96 [0.89, 1.04]	0.000 [−0.007, 0.006]	−0.048 [−0.152, 0.055]	0.97 [0.90, 1.04]	−0.020 [−0.032, −0.008]***	−0.065 [−0.156, 0.026]

Predictors	Duration & Dysfunction Hazard Ratio [95% CI]		
	Direct Effect	Indirect Effect	Total Effect
Conscientiousness	0.94 [0.87, 1.01]	−0.027 [−0.042, −0.012]***	−0.106 [−0.202, −0.010]*
Agreeableness	0.96 [0.89, 1.04]	0.008 [−0.002, 0.018]	−0.046 [−0.148, 0.056]
Neuroticism	0.99 [0.91, 1.08]	0.063 [0.034, 0.091]***	0.049 [−0.056, 0.155]
Openness	1.06 [0.98, 1.15]	−0.015 [−0.028, −0.001]***	0.062 [−0.042, 0.167]
Extraversion	0.98 [0.90, 1.06]	−0.020 [−0.035, −0.005]**	−0.050 [−0.153, 0.054]

Note. CI = Confidence Interval. *$p < .05$. **$p < .01$. ***$p < .001$. Model adjusted for age race, gender, education, married/partnered, linear sleep duration, and retirement status. Each personality effect is unadjusted for the other Big Five traits.

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