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Why is personality tied to sleep quality? A biometric analysis of twins

Zlatan Krizan [a,*], Garrett Hisler [a], Robert F. Krueger [b], Matt McGue [b]

[a] Iowa State University, United States
[b] University of Minnesota, United States

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ABSTRACT

Despite consistent links between personality traits and poor sleep, little is known about genetic and environmental influences that may produce them. This study examined how much genetic background and environmental experiences contributed to phenotypic linkages between personality and subjective sleep quality. Seven hundred and thirty-four twin pairs from the Minnesota Study of Twin Aging and Development rated their sleep quality and provided personality reports. Bi-variate analyses revealed that genetic factors accounted for the majority of observed associations between subjective sleep quality and traits, but also that non-shared environmental experience played a role that varied across traits. The findings strongly implicate genotype in tying subjective sleep quality to personality variation, alongside non-shared environmental influences, and suggest indicate influences unique to individual traits.

1. Introduction

Sleep is one of the three pillars of health, alongside diet and exercise, and for good reasons. Poor sleep is an indicator of mental health difficulties, physical illnesses, serious psychiatric disorders, and an earlier death (Fernandez-Mendoza & Vgontzas, 2013; Harvey, 2008; Parthasarathy et al., 2015). People who get better sleep are also happier with their lives and perform better at work (Barnes, 2012; Ong, Kim, Young, & Steptoe, 2017). While governed by biological mechanisms (e.g., homeostatic sleep pressure, circadian rhythm), sleep also reflects environmental context (e.g., environmental noise, neighborhood) and individual differences (e.g., chronotype, personality; Bonnet & Arand, 2010; DeSantis et al., 2013; Randler, Schredl, & Göritz, 2017). In this vein, personality traits consistently predict how well people sleep, although the reasons for this association are unclear (Stephan, Sutin, Bayard, Križan, & Terracciano, 2018). To address sources responsible for linking personality to sleep, we employed behavioral-genetic methods with data from a large sample of aging twins to evaluate how genetic and environmental influences contributed to phenotypic ties between personality traits and subjective sleep quality.

1.1. Subjective sleep quality vs. sleep disturbance

How long people sleep is critical, but how well they sleep is no less important. Subjective sleep *quality* typically refers to overall perceptions of one's sleep as easily initiated, un-interrupted, and restorative, and is a critical component of sleep health (Buysse, 2014). When poor sleep chronically interferes with daily functioning (despite adequate sleep opportunity) it constitutes insomnia, a common syndrome that affects up to one third of the population and is associated with various psychiatric and physical health problems (Harvey, 2008; Mai & Buysse, 2008; Taylor et al., 2007).

As would be expected, complaints of poor subjective sleep quality are often associated with objective indicators of delayed, interrupted, or unrestorative sleep. For example, adults diagnosed with insomnia take longer to fall asleep and spend more time awake during the night (Kay, Buysse, Germain, Hall, & Monk, 2015; Keklund & Åkerstedt, 1997; Kurina et al., 2015; Libman et al., 2016). Moreover, individuals who report poor sleep may exhibit lower proportion of slow-wave ("deep") sleep, that is less electroencephalographic delta-wave activity associated with more restorative stages of sleep (Keklund & Åkerstedt, 1997; Krystal, Edinger, Wohlgemuth, & Marsh, 2002; Riedel & Lichstein, 1998). However, sleep complaints can also involve exaggerated or even confabulated perceptions of poor sleep, including overestimates of sleep latency, wakefulness in bed, or underestimates of sleep duration (Baker, Maloney, & Driver, 1999; Kay et al., 2015). As a result, reports of disrupted, poor-quality sleep are distinguished as a unique aspect of sleep in their own right, as they do not always reflect objective disruptions in sleep, yet nevertheless predict consequential outcomes in their own right even after accounting for other sleep characteristics (e.g., physical health, Buysse, 2014; Lichstein, 2017).

* Corresponding author at: Iowa State University, Ames, IA 50010, United States.
E-mail address: zkrizan@iastate.edu (Z. Krizan).

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1.2. Personality traits and subjective sleep quality

Subjective sleep quality shows large nightly fluctuations (e.g., due to environment, stress, or illness), but is also a relatively stable feature of individuals. For example, ratings of sleep quality changed little across two years in one study of healthy seniors (Hoch, Reynolds, Kupfer, & Berman, 1988), while another study using the Pittsburgh Sleep Quality Index (which queries sleep over the past month) found a stability correlation of 0.68 across one year (Knutson, Rathouz, Yan, Liu, & Lauderdale, 2006). Because of implications for health, it is important to identify individuals who chronically report poor sleep. To this end, personality traits have long been implicated in subjective sleep quality. We first review basic associations between personality traits and subjective sleep quality and then consider processes that may produce them.

1.2.1. Neuroticism

Traits from the neuroticism domain are consistently associated with insomnia complaints. Various measures of negative emotions including trait anxiety, depression symptoms, and emotional lability all consistently predict insomnia complaints or some form of poor sleep quality (Alvaro, Roberts, & Harris, 2013; Fuller, Waters, Binks, & Anderson, 1997; Koffel & Watson, 2009). More broadly, research focusing on normal personality traits and the general population has similarly implicated neuroticism (alongside all of its facets) in poor sleep, regardless of demographic status (Cellini, Duggan, & Sarlo, 2017; Dekker, Blanken, & Van Someren, 2017; Duggan, Friedman, McDevitt, & Mednick, 2014; Gray & Watson, 2002; Herlache, Lang, & Krizan, 2018; Hintsanen et al., 2014; Kim et al., 2015; Križan & Hisler, 2019; Soehner, Kennedy, & Monk, 2007; Weeks, Hayley, & Stough, 2019; Williams & Moroz, 2009). Associations with neuroticism are large and robust, typically falling in 0.30–0.40 range and extending across ages and cultures (Cellini et al., 2017; Hintsanen et al., 2014; Stephan, Sutin, Canada, & Terracciano, 2017). Even if over-reports of aversive experiences among neurotic individuals may inflate these associations (Suls & Howren, 2012; Watson & Pennebaker, 1989), neuroticism predicts actigraphically-recorded delayed sleep onset and wakefulness during the night (Križan & Hisler, 2019; Sutin et al., 2020).

1.2.2. Extraversion and conscientiousness

Sleep quality is also linked to other broad trait domains, but less strongly, broadly, and consistently than in the case of neuroticism. First, more extraverted individuals report better sleep, but these associations seem largely driven by underlying positive affectivity and enthusiasm, rather than other aspects of extraversion such as dominance or sensation seeking (Gray & Watson, 2002; Hintsanen et al., 2014; Kim et al., 2015; Križan & Hisler, 2019). For example, Gray and Watson (2002) reported a correlation of −0.13 between sleep quality and overall extraversion in a sample of students, but a correlation of −0.29 when linking sleep quality to positive affectivity more specifically. In a similar vein, more conscientious individuals report better sleep, with correlations typically in the 0.10–0.20 range (Dekker et al., 2017; Duggan et al., 2014; Gray & Watson, 2002; Križan & Hisler, 2019). However, these associations again vary depend on the facet; better sleep is mostly found among those high on self-control and industriousness, rather than those high on deliberation or orderliness (Kim et al., 2015; Križan & Hisler, 2019).

1.2.3. Hostility and aggressiveness

The final set of traits consistently linked to poor sleep involves hostility and aggressiveness, which are associated with both the agreeableness and neuroticism domains and are thus not precisely assessed by briefer Big Five instruments (e.g., the BFI). In this vein, although low agreeableness in general shows very weak (negative) links to sleep quality (Gray & Watson, 2002; Kim et al., 2015; Križan & Hisler, 2019; Sutin et al., 2020), hostility and aggressiveness have been repeatedly implicated in sleep disturbances (likely due to partial overlap with neuroticism). Individual differences in hostility encompass affective tendencies toward anger (e.g., trait anger), tendencies toward distrust and suspiciousness of others (e.g., hostile attributions), and behavioral tendencies toward aggression (e.g., physical aggressiveness, Martin et al., 2000). All of these features have been linked to sleep problems; children with angry temperaments or externalizing behavior have difficulties falling and staying asleep (Chervin, Dillon, Archbold, & Ruzicka, 2003; Reid, Hong, & Wade, 2009), while adults prone to anger, distrust, or aggression report more sleep interruptions and sleep of lower quality (Caska et al., 2009; Granö, Vahtera, Virtanen, Keltikangas-Järvinen, & Kivimäki, 2008; Hisler & Krizan, 2017; Križan & Hisler, 2019; Ottoni, Lorenzi, & Lara, 2011).

Finally, note that the associations of personality traits with subjective sleep quality reviewed so far extend to associations of these traits with measures of sleep continuity, such as interruptions during the night or unwanted wakefulness. Križan and Hisler (2019) reported a personality profile correlation of 0.77 between subjective sleep quality and (actigraphically-asessed) sleep continuity, confirming that a similar constellation of personality features describes subjective sleep quality as well as behavioral sleep continuity (i.e., falling asleep and staying asleep).

1.3. Why are personality traits tied to subjective sleep quality? A behavior-genetic approach

When understanding the etiology of these robust associations between personality traits and sleep quality, three general classes of explanations are possible, namely (1) causation by personality, (2) reverse-causation by sleep quality, and (2) association due to common-causes (Pearl, 2000; Wright, 1921). Due to relative stability of personality traits and obstacles to experimentation, the ultimate goal should be understanding the causal structure of influences that produce the links between sleep and personality, influences which may vary based on the trait domain (Briley, Livengood, & Derringer, 2018). As understanding the etiology of associations between personality traits and subjective sleep quality is challenging, it is critical to employ designs that provide evidence favoring one class of explanations over others. To this end, we employed a quantitative-genetics approach toward identifying sources of phenotypic associations between subjective sleep quality and personality traits in adulthood (Briley et al., 2018; McGue, Osler, & Christensen, 2010; Rohrer, 2018). Quantitative genetics draws on differential genetic similarity between family members in order to estimate how much diversity in a particular psychological phenotype (i.e., subjective sleep quality) is due to genetic or environmental influences (Fisher, 1918; Posthuma et al., 2003). As genetic variation precedes phenotypic differences, such analyses can inform our understanding of the underlying developmental process.

In order to estimate genetic influences, classic twin models compare patterns of phenotypic covariation across monozygotic and dyzogotic twins in order to yield "ACE" estimates of additive genetic effects (A, genetic sources of similarity), shared-environmental effects (C, environmental sources of similarity typically attributed to rearing environments), and non-shared environmental effects (E, sources of dissimilarity alongside measurement error). We applied this biometric approach to the bi-variate cases of individual personality traits and subjective sleep quality, where the *associations* (i.e., covariances) between these pairs of variables were similarly decomposed into gene-based,

rearing-environment, and person-specific influences (Evans, Gillespie, & Martin, 2002; Loehlin, 1996).

How does this design inform our understanding of the etiology of sleep-to-personality associations? Because shared environmental effects appear negligible in shaping subjective sleep quality and personality traits in adults (i.e., coming from the same household net of genetic similarity does not make sleep quality or personality of two adult siblings more similar, Genderson et al., 2013; Plomin & Deary, 2015), we hypothesized that sharing the environment of origin will not be a source of connection between personality traits and subjective sleep quality in adulthood. Rather, we hypothesized that both (1) genetic influences, and (2) non-shared environmental factors (i.e., person-specific experiences) will associate personality trait levels with quality of individuals' sleep.

Note that associating non-shared environmental sources of variance for personality and subjective sleep quality phenotypes can address important questions about causation. Following the logic of co-twin control designs, a tie between subjective sleep quality and personality traits through unique environmental influences (i.e., correlated E components) constitutes strong evidence for some form of mutual causation, or at minimum presence of idiosyncratic life experience that directly impact the development of both (Briley et al., 2018; McGue et al., 2010). Recording correlations between non-shared environmental variation in sleep quality and personality traits would thus suggest that idiosyncratic life experiences shaping one extend to the other regardless of inheritance, strengthening the case for environmentally-mediated causal linkage.

1.4. Are there common genetic and environmental influences on sleep and personality?

To evaluate to what extent genetic vs. environmental factors contribute to links between sleep quality and personality, we drew on a large population-based sample of twins from the Minnesota Twin Study of Adult Development and Aging (MTSADA, Finkel & McGue, 1993). Among an extensive assessment battery, study participants also completed a measure of subjective sleep quality and a systematic personality assessment on the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). Guided by findings and theory on personality traits and sleep reviewed earlier, we focused on the following traits, namely Neuroticism (MPQ Stress Reaction), Positive affectivity (MPQ Well Being), Planfulness (MPQ Control), Achievement-Striving (MPQ Achievement), Hostility (MPQ Alienation) and Aggressiveness (MPQ Aggression, see Church, 1994).

1.4.1. Neuroticism

What are the expected sources of personality-to-sleep-quality links? First, neuroticism and sleep quality both exhibit substantial genetic influences, so a shared genotype is a potential common-cause of these associations. Recent findings from genome-wide association studies have implicated numerous specific genes in both neuroticism and sleep disruptions (Hammerschlag et al., 2017; Nagel et al., 2018). Although these analyses so far do not point to shared specific genes, they do suggest that the genetic profile (i.e., polygenic risk scores) associated with insomnia predicts phenotypic differences in neuroticism (Hammerschlag et al., 2017). Similarly, an analysis of 1412 twin-children pairs found that observer-rated symptoms of insomnia, depression, and anxiety overlapped exclusively due to shared genetic effects (Gehrman et al., 2011), while a study by Heath, Eaves, Kirk, and Martin (1998) implicated genetic influences on high neuroticism and low extraversion in poor sleep quality. Plausible shared bio-chemical mechanisms that could be involved include stress reactivity, regulation of arousal, as well as circadian rhythmicity, although a phenotypic mediation is also possible (e.g., Jones et al., 2019; Mõttus, Realo, Vainik, Allik, & Esko, 2017; Turkheimer, Pettersson, & Horn, 2014). For example, if neuroticism undermines sleep quality over time, genetic risk-factors for neuroticism would be associated with poor sleep even without a direct genetic effect (Stephan et al., 2017).

With that said, it is also possible that idiosyncratic life experiences which shape neuroticism would ultimately impact sleep, such as undergoing divorce, losing employment, or dealing with chronic illness (Mroczek & Spiro, 2003; Stephan et al., 2017; Troxel, 2010). In sum, we hypothesized that neuroticism will exhibit strong phenotypic ties to poor sleep quality, that shared genetic influences will account for much of their associations, but that at least some overlap will be due to idiosyncratic life experiences tying the two together.

1.4.2. Positive affectivity

Second, when it comes to differences in extraversion, we focused on ties between sleep quality and positive affectivity (assessed by the Well Being scale in the MPQ), given its differential importance relative to other aspects of extraversion (Gray & Watson, 2002; Križan & Hisler, 2019; Ong et al., 2017). Positive affect may affect sleep quality directly by promoting healthy behavioral practices supportive of quality sleep (Steptoe, O'Donnell, Marmot, & Wardle, 2008), or indirectly by facilitating adaptive, problem-focused coping with stressors (Fredman, Gordon, Heeren, & Stouver, 2014). Alternatively, poor sleep could undermine chronic levels of positive affect by dampening vigor and enthusiasm (Armon, Melamed, & Vinokur, 2014; Koffel & Watson, 2009). Although extraversion may be associated with sleep-relevant genetic polymorphisms (Jiménez, Pereira-Morales, & Forero, 2017), there is currently little evidence on genetic links between extraversion facets and sleep quality. As a result, we hypothesized that positive affectivity will exhibit moderate phenotypic ties to sleep quality, with a smaller contribution of shared genetic influences (relative to neuroticism), and some contribution of shared unique life experiences. Although conscientiousness is important for sleep quality, only its planfulness and achievement-striving features were available in these data, and these scales were not very indicative of sleep quality in prior analyses (Križan & Hisler, 2019). Although we anticipated weak phenotypic ties of these traits with sleep quality, we nevertheless evaluated their genetic and environmental sources, anticipating some contributions from both.

1.4.3. Hostility and aggressiveness

Third and final, we anticipated moderate phenotypic links tying sleep quality to hostile and aggressive tendencies (Granö et al., 2008; Hisler & Krizan, 2017; Kamphuis, Dijk, Spreen, & Lancel, 2014). Due to the overlap of hostility and anger with neuroticism (Church, 1994; DeYoung, 2015), we anticipated substantial genetic contributions to their links with sleep quality. Because sleep, anger, and aggression have also been implicated in stressful life events (Kimonis, Centifanti, Allen, & Frick, 2014; Sun et al., 2016), we also expected (unique) environmental influences to contribute to links between sleep and hostility.

1.4.4. Contributions of specific personality traits to ties with sleep quality

Because self-reported personality traits also share method-based variance with each other (e.g., due to underlying meta-traits or responding styles), we also estimated the *unique* contribution of each personality trait to shared genetic and environmental variance of sleep quality (using Relative Weights Analysis, Johnson, 2000; Wright, Pahlen, & Krueger, 2017). To the extent different traits show unique associations with sleep quality across these

components, concerns that method-specific influences are driving the findings would be minimized. Moreover, this approach enabled us to estimate how much of the overall genetic and non-shared environmental variance in subjective sleep quality was accounted for by individual traits.

2. Method

2.1. Participants

Participants came from the Minnesota Twin Study of Adult Development and Aging (MTSADA) which was conducted between 1984 and 1994 (Finkel & McGue, 1993, 2007). MTSADA is a population-based sample which identified same sex twin pairs from the state of Minnesota birth records. Using public records (phone books, drivers' registrations, marriage licenses), 47% of identified twins from birth records were located and recruited by mail; 67% of those invited to the study agreed to participate. Only twins reared together in the same house were included in MTSADA and in total 797 twin pairs aged 27–95 were recruited. These individuals completed a range of measures assessing cognition, physical ability, physiology, life-style, and psychosocial issues. Because the current study is interested in the genetic and environmental contributions to the phenotypic correlations among sleep and personality variables, we only use data from participants who completed both sleep and personality assessments. This yielded a final sample size of 734 twin pairs, which consisted of 390 monozygotic and 344 dizygotic same sex twin pairs that were reared together in the same household (M_{age} = 58.71, range = 23–92; 58% female). Because of confidentiality assurances to participants and rules governing the use of this database we did not have access to other demographic information (e.g., race), and the data will not be made public. The hypotheses and analyses were not pre-registered.

2.1.1. Statistical power

As the goal of this analysis was to estimate associations at the genetic (rather than phyenotypic) level, we evaluated the power to identify correlations within a classic twin design. Based upon estimates provided by Verhulst (2017), assuming no common environmental contributions to personality and strong heritabilities (0.50), this sample should afford roughly 80% statistical power to detect a modest 0.20 genetic correlation.

2.2. Measures

2.2.1. (Poor) subjective sleep quality

Sleep quality were measured by averaging two items asking "Was your sleep restless?" and "How often did worrying keep you awake?" over the past week. Difficulty falling asleep and fitful sleep are key signs of poor sleep (American Psychiatric Association, 2013; Irwin, 2015). Response options included: (1) "Rarely or none of the time (<1 day)", (2) "Some or little of the time (1–2 days), (3) "Occasionally or a moderate amount of the time (3–4 days), and (4) "Most or all of the time (5–7 days)." Answers to these two questions were averaged to estimate self-reported sleep quality (r = 0.47, α = 0.61). Note that while these items do not cover the entire construct of subjective sleep quality, such items tend to be highly intercorrelated with other indicators of subjective sleep quality and are common signs of poor sleep (Burgard & Ailshire, 2009; Harvey, 2003; Kalmbach et al., 2020; Kaplan, Hardas, Redline, & Zeitzer, 2017; Keklund & Åkerstedt, 1997). For example, Koffel and Watson (2010) found that reports of fragmented sleep' and 'anxiety at night' were among the strongest indicator of broader sleep disturbances. Moreover, utilizing brief assessment of subjective sleep quality indicates phenotypic links to personality and heritability estimates similar in magnitude to those found when longer measures are used (Lind & Gehrman, 2016; Stephan et al., 2017). Finally, to reduce the undue influence of content overlap between the measures, we also estimated models only using the 'restless' item as an indicator of sleep disturbances. Note that women reported more disturbance on both of these items, as would be expected based on prior findings (r[restless] = 0.09, p = .001; r[worrying] = 0.09, p = .002). There were no clear associations with age (r[restless] = −0.02, p = .37; r[worrying] = −0.08, p = .003), although this analyses was limited by the fact that most of the sample was older than 60.

2.2.2. Personality traits

Personality traits were measured with the 300-item version of the Multidimensional Personality Questionnaire (Tellegen & Waller, 2008). Participants were instructed to indicate true if the item described them and to indicate false if it did not. All items were coded so that zero indicated false and one indicated true, with higher values reflecting a higher standing on the trait. Although all 14 traits measured by this questionnaire are available in the data, our hypotheses focused on the following six traits given prior theory and findings: neuroticism, positive affectivity, planfulness, achievement-striving, hostility, and aggressiveness. We focus on these more specific aspects (rather than broad domains) given they were most specifically implicated in sleep functioning. All scale scores reflect simple aggregates.

2.2.3. Stress reaction (neuroticism)

Individuals high in stress reaction tend to more readily feel negative emotions such as nervousness, worry, and emotional vulnerability. This trait was assessed with the average of 26-items (α = 0.91), such as "I often find myself worrying about something", "My feelings are hurt rather easily", and "I am easily "rattled at critical moments." This scale correlates very strongly with other measures of neuroticism, exhibiting much lower correlations with other traits (Church, 1994; Križan & Hisler, 2019).

2.2.4. Wellbeing (positive affectivity)

Individuals high on this dimension have a disposition towards feeling positive emotions and being gregarious. A composite well-being score was created from the average score across the 24 Well-being items (e.g., "I am naturally cheerful", "My future looks very bright to me", "I have several pastimes or hobbies that are great fun"; α = 0.88). This scale correlates the most strongly with positive affect and gregariousness features of extraversion (Church, 1994).

2.2.5. Control (planfulness)

People high on this scale often plan ahead and tend to be careful and reflective. To measure this trait, participant responses to 24-items, such as "I almost never do anything reckless", "When faced with a decision I usually take time to consider and weigh all aspects", and "I like to stop and think things over before I do them", were averaged (α = 0.76). This scale correlates most strongly with planfulness aspects of conscientiousness (Church, 1993).

2.2.6. Achievement (achievement-striving)

People oriented toward achievement are driven to succeed and exhibit tendencies to work hard. An achievement score was created from the average score across the 21 items (e.g., "I enjoy putting in long hours", "I often go on working on a problem long after others would have given up", "I like to try difficult things"; α = 0.82). This scale correlates most strongly with conscientiousness and the activity facet of extraversion (Church, 1994).

2.2.7. Alienation (hostility)

Alienated individuals have the propensity to feel mistreated, used, or threatened by others. A hostility score was created through the mean of 20-items, (e.g., "some people go out of their way to keep me from getting ahead", "Many people try to push me around", "People often try to take advantage of me"; $\alpha = 0.83$). This scale correlates the most heavily with other measures of distrust from the agreeableness domain, but also trait anger (Church, 1994; Tellegen, 1995/2003).

2.2.8. Aggression (aggressiveness)

Twenty items measuring the tendency to engage in purposefully harming others and to enjoy watching other people experience harm were averaged to create an overall trait aggression score ($\alpha = 0.74$). Items measuring aggression included "When I get angry I am often ready to hit someone", "When someone hurts me, I try to retaliate (get even)", and "I enjoy violent movies." This scale correlates most heavily with general disagreeableness, but also trait anger (Church, 1994; Tellegen, 1995/2003).

2.3. Data preparation

Prior to conducting any analyses, data were inspected for meeting the statistical assumption of normality. It was expected that high levels of aggression or very poor sleep quality would be infrequently endorsed in this normative population and lead to skewed distributions. Visual inspection of the scale histograms suggested that subjective sleep quality, Stress reaction, Aggression, and Alienation all had a negative skew, while Well-being had a positive skew. Formal tests of normality via the Kolmogorov-Smirnov test confirmed the presence of skew. Following prior personality-genetics research, skewed variables were transformed with a rank-based transformation (i.e., Rankit; Bliss, 1967; Wright et al., 2017). After these variables were transformed, all variables were regressed on the linear and quadratic effects of age as well as sex and the age-sex interaction, because cohort and gender differences were outside the scope of the current study (McGue & Bouchard, 1984). The unstandardized residuals of these regressions were then used in the following analyses.

2.4. Data analytic plan

Univariate ACE models were first conducted to decompose variance in personality traits and subjective sleep quality into genetic (A), common environmental (C), and non-shared environmental components (E). Because personality traits and sleep quality often show little to no common environmental variance components when examining broad, average estimates of variance, we used the Akaike Information Criteria (AIC) to compare each ACE model to its respective simpler AE model (Butkovic, Vukasovic, & Bratko, 2014; Genderson et al., 2013; Wright et al., 2017). We also inspected the possibility of alternate models such as ADE (which estimates non-additive genetic effects wherein the correlation between monozygotic twins is more than twice the size of the correlation in dizygotic twins) and more parsimonious CE models**.** The univariate model with the lowest AIC was determined to best represent the observed data. After specifying the appropriate univariate model, we then estimated a series of bivariate models to examine the correlations between genetic and non-shared environmental components of sleep and personality traits. The programming syntax in Mplus 7 and all model results can be found at Open Science Framework: https://osf.io/64z8x/?view_only=316ba5f9378a45f392535f63c8b541a4. https://osf.io/64z8x/.

To evaluate *unique* contributions of personality traits to ties with subjective sleep quality, we derived two correlation matrices from these covariances: one for genetic components and another for non-shared environmental components. Using these correlation matrices we then conducted relative weights analyses to model the total and unique association of personality traits with sleep quality separately for genetic and non-shared environmental components (Tonidandel & LeBreton, 2011). In other words, this analysis allowed us to estimate the total overlap between all personality traits and subjective sleep quality as well as the unique overlap of each personality trait with subjective sleep quality. Relative weights analysis was used because personality traits share substantial genetic variance (Krueger, 2000). This collinearity makes traditional multiple regression approaches inappropriate for evaluating unique relations of each personality trait with sleep (though the results of a traditional regression analysis are provided in Appendices A and B). To circumvent collinearity, relative weights analysis applies a transformation to each predictor variable to create a new set of predictors that are orthogonal to each other, but maximally related to their respective original variable (Johnson, 2000). This new set of orthogonal predictors is then used to predict the outcome variable. Estimates obtained in this analysis were transformed back to their original metric for interpretation.

3. Results

Descriptive statistics and correlations of all study variables are presented in Table 1. All traits except achievement correlated with subjective sleep quality. Stress reaction, alienation, and wellbeing (r's = |0.19| to |0.39|) had notably large correlations with subjective sleep quality, while the rest of the personality traits had more modest associations with sleep (r's = |0.12| to |0.02|).

3.1. Univariate estimates

Univariate estimates of genetic, shared-environmental, and non-shared environmental variance are presented in Table 2. Subjective sleep quality and all personality traits only had significant genetic and non-shared environmental variance components. Correspondingly, the AE model fit was superior to the ACE model for these variables (all model AICs were slightly lower) so the AE model was used in subsequent bivariate analyses. ADE models did not provide meaningfully better fit for any variable and are therefore not discussed further.

3.2. Bivariate analyses

To test if genetic and non-shared environmental components of personality traits co-varied with their respective components in subjective sleep quality, a series of bivariate AE models were estimated for each personality trait pairing. To facilitate interpretation and comparison of covariance estimates, covariances were transformed into correlations.

All personality traits except achievement shared genetic variance with subjective sleep quality (all p's < 0.01; see Table 3). Consistent with close ties between subjective sleep quality and emotional functioning, traits most closely associated with emotional dispositions had the largest genetic correlations with sleep quality (stress reaction $r = 0.83$, 95% CI = 0.81 to 0.85; aggression $r = 0.50$, 95% CI = 0.44 to 0.55; wellbeing $r = 0.52$, 95% CI = 0.47 to 0.57, see Figs. 1 and 2). Smaller patterns of relations were apparent with traits more closely related to distrust and self-control (alienation $r = 0.38$, 95% CI = 0.73 to 0.44; control $r = -0.14$, 95% CI = −0.07 to -0.14).

In contrast to the genetic components, there were fewer and smaller correlations between non-shared environment influences and subjective sleep quality (see Table 4). Specifically, stress reaction ($r = 0.21$, 95% CI = 0.14 to 0.28), alienation ($r = 0.13$, 95%

Table 1
Descriptive statistics and correlations among study variables.

Variable	M	SD	1	2	3	4	5	6	7
1. Sleep quality	2.50	0.68	*0.61*	−0.39*	−0.19*	−0.12*	0.06*	0.02	0.23*
2. Stress reaction	1.35	0.26	−0.42*	*0.91*	0.47*	0.33*	−0.17*	−0.06*	−0.47*
3. Alienation	1.10	0.15	−0.19*	0.45*	*0.83*	0.35*	−0.14*	0.05*	−0.25*
4. Aggression	1.14	0.14	−0.11*	0.28*	0.34*	*0.74*	−0.26*	−0.07*	−0.18*
5. Control	1.70	0.17	0.06*	−0.15*	−0.13*	−0.25*	*0.76*	0.17*	0.00
6. Achievement	1.57	0.21	0.02	−0.07*	0.06*	−0.03	0.17*	*0.82*	0.25*
7. Wellbeing	1.82	0.19	0.26*	−0.48*	−0.25*	−0.20*	0.01	0.20*	*0.88*

* p < .05. Means, standard deviations, and correlations below the diagonal are reported prior to transformation and regression procedures. Correlations above the diagonal are reported *after* transformation and regression procedures. Reliabilities are reported in italics on the diagonal.

Table 2
Univariate estimates from ACE, ADE and best fitting model for each variable.

Variable	MZ pairs	DZ pairs	MZ ICC	DZ ICC	a^2 [95% CI]	d^2 [95% CI]	c^2 [95% CI]	e^2 [95% CI]	AIC
Sleep quality	324	273	0.54	0.14	0.11 [−0.11 to 0.32]	–	0.02 [-0.16 to 0.20]	0.55 [0.48 to 0.63]	3216.20
					13. [0.07 to 0.20]	0.00 [0.00 to 0.00]	–	0.55 [0.78 to 0.62]	3216.25
					0.13 [0.07 to 0.20]	**–**	**–**	**0.55 [0.48 to 0.62]**	**3214.25**
Stress reaction	299	273	0.64	0.24	0.41 [0.31 to 0.48]	–	0.00 [0.00 to 0.00]	0.52 [0.44 to 0.60]	3496.83
					0.09 [−0.36 to 0.53]	0.34 [−0.12 to 0.79]	–	0.50 [0.42 to 0.58]	3494.66
					0.41 [0.31 to 0.50]	**–**	**–**	**0.52 [0.44 to 0.58]**	**3494.83**
Alienation	299	273	0.51	0.36	0.23 [0.01 to 0.46]	–	0.05 [−0.13 to 0.23]	0.50 [0.42 to 0.58]	3296.89
					0.29 [0.21 to 0.37]	0.00 [0.00 to 0.00]	–	0.49 [0.42 to 0.56]	3297.16
					0.29 [0.21 to 0.36]	**–**	**–**	**0.49 [42 to 0.55]**	**3295.16**
Aggression	299	273	0.43	0.22	0.23 [0.14 to 0.31]	–	0.00 [0.00 to 0.00]	0.59 [0.51 to 0.67]	3378.95
					0.17 [−0.23 to 0.57]	0.06 [−0.36 to 0.48]	–	0.58 [0.50 to 0.67]	3378.88
					0.23 [0.14 to 0.31]	**–**	**–**	**0.59 [0.51 to 0.67]**	**3376.95**
Control	299	273	0.33	0.19	0.01 [0.01 to 0.01]	–	0.00 [0.00 to 0.00]	0.02 [0.02 to 0.02]	−987.80
					0.00 [0.00 to 0.00]	0.01 [0.01 to 0.01]	–	0.02 [0.02 to 0.02]	−991.23
					0.01 [0.01 to 0.01]	**–**	**–**	**0.02 [0.02 to 0.02]**	**−989.80**
Achievement	299	273	0.63	0.31	0.02 [0.02 to 0.02]	–	0.00 [0.00 to 0.00]	0.03 [0.02 to 0.03]	−424.15
					0.01 [−0.01 to 0.03]	0.01 [−0.01 to 0.03]	–	0.02 [0.02 to 0.02]	−424.76
					0.02 [0.02 to 0.02]	**–**	**–**	**0.03 [0.02 to 0.03]**	**−426.15**
Wellbeing	299	273	0.51	0.12	0.27 [0.18 to 0.36]	–	0.00 [0.00 to 0.00]	0.60 [0.52 to 0.69]	3456.29
					0.00 [0.00 to 0.00]	0.30 [0.20 to 0.39]	–	0.57 [0.49 to 0.66]	3453.30
					0.27 [0.18 to 0.36]	**–**	**–**	**0.60 [0.52 to 0.69]**	**3454.29**

Note. Bolded line indicates best fitting model.

Table 3
Genetic correlations from bivariate models.

Variable	1	2	3	4	5	6
1. Sleep quality	–					
2. Stress reaction	−0.83*	–				
3. Alienation	−0.38*	0.55*	–			
4. Aggression	−0.50*	0.60*	0.40*	–		
5. Control	0.14*	−0.24*	−0.13*	−0.46*	–	
6. Achievement	0.06	−0.09*	0.04	−0.13*	0.28*	–
7. Wellbeing	−0.52*	−0.61*	−0.35*	−0.47*	−0.61*	0.27*

* p < .01.

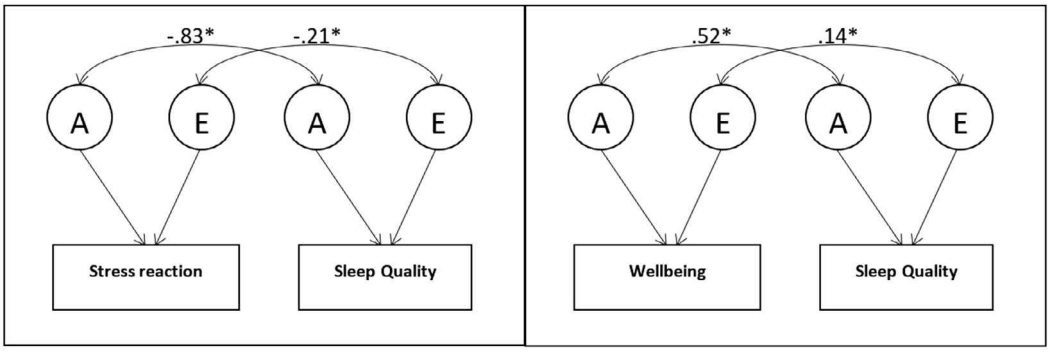

Fig. 1. Correlations between additive genetic (A) and non-shared environmental (E) components of associations between stress reaction (neuroticism) and sleep quality (left panel) and between wellbeing and sleep quality (right panel). *p < .01.

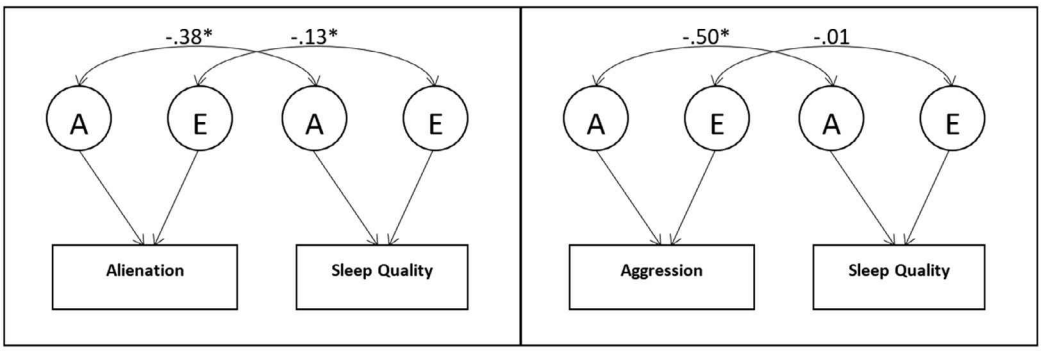

Fig. 2. Correlations between additive genetic (A) and non-shared environmental (E) components of associations between alienation and sleep quality (left panel) and between aggression and sleep quality (right panel). *p < .01.

Table 4
Non-shared environment correlations from bivariate models.

Variable	1	2	3	4	5	6
1. Sleep quality	–					
2. Stress reaction	−0.21*	–				
3. Alienation	−0.13*	0.42*	–			
4. Aggression	−0.01	0.19*	0.32*	–		
5. Control	0.04	0.12*	−0.15*	−0.17*	–	
6. Achievement	0.01	0.02	0.05	−0.05	0.09*	–
7. Wellbeing	0.14*	−0.39*	−0.20*	−0.06	0.24*	0.24*

* p < .01.

CI = 0.06 to 0.20), and wellbeing (r = −0.14, 95% CI = −0.07 to −0.21) had substantive non-shared environment correlations with subjective sleep quality (all p's < 0.01).

Across both genetic and non-shared environmental contributions, neuroticism emerged as the trait most closely tied to sleep quality. One reason for this may be that both stress reaction and sleep quality items refer to "worry." Associations between sleep quality and Stress Reaction could thus be inflated due to item content overlap, rather than due to the overlap of underlying constructs. To evaluate this possibility, we estimated the same bivariate AE model between stress reaction and sleep quality while omitting this item from sleep quality (only using the "restless" item). Critically, the correlations for both the genetic and non-shared environmental components between stress reaction and subjective sleep quality were virtually unchanged (only decreasing by 0.01 and 0.02, respectively). Thus, the covariances among stress reaction and subjective sleep quality seem minimally affected by this overlap in item content. Moreover, they suggest that measurement noise inherent in indicators with fewer items did not have a large impact on the results.

3.3. Relative weights analysis

We next sought to determine how much total genetic and non-shared environmental variance personality traits shared with sleep quality. Additionally, because personality traits share genetic and non-shared environmental variance with each other, we also examined the amount of genetic and non-shared environmental variance that each trait *uniquely* shared with subjective sleep quality. To do so, we used the constructed correlation matrices in Tables 3 and 4 to perform relative weights analysis separately for genetic and non-shared environmental components (Johnson, 2000).

In total, personality traits shared 67% of genetic variance with subjective sleep quality (see Table 5), where 19% of the total variance in subjective sleep quality was attributable to genetic variance. Multiplying these two percentages suggests that genetic variance in personality traits accounted for approximately 13% of the total phenotypic variance in subjective sleep quality (cf. Heath et al., 1998). Inspecting each trait's uniquely shared genetic variance with subjective sleep quality showed that all traits had unique genetic covariance with subjective sleep quality. Notably, Stress Reaction accounted for most of the total shared genetic variance (42% out of the 67%). Alienation, aggression, and Wellbeing uniquely shared between 5 and 10% of genetic variance with subjective sleep quality, whereas control and achievement only shared 1%.

Table 5
Relative weights of personality trait genetic covariance predicting genetic variance in sleep quality.

	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Rescaled relative weight (% of overall model R^2 explained)
Stress reaction	**0.42***	0.39 to 0.46	63.0
Alienation	**0.05***	0.03 to 0.06	6.9
Aggression	**0.09***	0.07 to 0.11	13.8
Control	**0.01**	−0.00 to 0.01	0.92
Achievement	**0.01**	−0.00 to 0.02	0.95
Wellbeing	**0.10***	0.08 to 0.12	14.5
Total	**0.67**		

* p < .05.

In terms of non-shared environmental covariance, personality traits accounted for 5% of the total non-shared environmental variance in subjective sleep quality (see Table 6). Given that 81% of the total variance in quality was attributable to non-shared environmental influences, this indicates that idiosyncratic influences on personality traits accounted for approximately 4% of the total phenotypic variance. Only stress reaction (3%) and alienation (1%) uniquely accounted for non-shared environmental variance of sleep quality. Taken together, these findings implicate personality

Table 6
Relative weights of personality trait non-shared evnironmental covariance predicting non-shared evnironmental variance in sleep quality.

	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Rescaled relative weight (% of overall model R^2 explained)
Stress reaction	**0.03***	0.01 to 0.05	63.16
Alienation	**0.01***	0.00 to 0.03	23.07
Aggression	**0.00**	−0.01 to 0.00	0.87
Control	**0.00**	−0.00 to 0.01	0.56
Achievement	**0.00**	−0.01 to 0.00	0.55
Wellbeing	**0.01**	−0.00 to 0.02	11.79
Total	**0.05**		

* $p < .05$.

traits in subjective sleep quality both in term of common genetic influences as well as idiosyncratic influences of life experiences. With that said, genetic influences associated with personality contributed more to the phenotypic associations of traits with sleep quality than did non-shared environmental experiences across multiple traits.

4. Discussion

How well individuals sleep is key to their health, well-being, and success, yet some individuals are more vulnerable to poor-quality sleep. Personality traits emerged as robust predictors of sleep quality in prior research, with neuroticism, extraversion, conscientiousness, and hostility playing the most important roles. In order to examine the etiology of these associations, the current study utilized a quantitative-genetic approach to estimate their genetic and environmental sources. Beyond replicating past phenotypic associations, the findings also provided novel evidence implicating shared genetic influences as a major pathway tying personality traits to differences in subjective sleep quality (at least in terms of restless and worry-disrupted sleep). This was especially true for traits reflecting emotional functioning. Moreover, as evident in correlations between non-shared environmental influences, the findings suggest that unique life experiences shaping personality differences also shape differences in sleep, providing compelling evidence for a dynamic developmental relation between personality and sleep (net of genetic effects). Finally, the genetic contributions of personality to subjective sleep quality were distinct across different traits, implicating trait-specific mechanisms in these associations with sleep.

4.1. Genetic influences on phenotypic ties between personality and sleep quality

Across different personality traits, the data revealed large and systematic contributions of shared genetic influences on the associations between sleep and personality. These findings clearly point to genetic influences as a critical developmental pathway shaping personality and subjective sleep quality. Genetic correlations were especially prominent for traits capturing emotional functioning, namely neuroticism and positive affectivity, which also exhibited the strongest phenotypic associations. Neuroticism again stood out as the most important predictor of subjective sleep quality relative to other traits, both at the genetic and environmental level. Most of the overlap between personality traits and subjective sleep quality was due to genetic influences, however, highlighting the key role of genetic pathways in tying personality to sleep. This held true even in the analyses examining separate contributions of traits to subjective sleep quality, suggesting it was not only due to genetic influences on methodology (e.g., self-report styles). The findings also implicate distinct mechanisms tying personality to subjective sleep quality, as multiple traits showed unique genetic contributions to subjective sleep quality. Besides neuroticism, positive affectivity and aggressiveness stood out as the most important contributors to genetic ties between personality and sleep quality.

How does genetic background exert such an important influence on tying personality differences to how well one sleeps? The most direct genetic mechanisms is pleiotropy, where common genes exert simultaneous effects on personality and sleep. For example, it is possible that genes contributing to serotonin mechanisms in the brain impact both the personality phenotype (e.g., chronic emotion regulation) and subjective sleep quality (e.g., distress about sleep). However, recent large-scale genome-wide association studies struggled to identify a common gene or -nucleotide polymorphism shared across insomnia symptoms and neuroticism (Hammerschlag et al., 2017; Nagel et al., 2018). Given the polygenetic nature of both traits and sleep problems, the search for common genes may be difficult with extremely small contributions of any given genetic variant. A genetic profile indicative of insomnia and poor sleep quality is thus predictive of neuroticism, but shared genes have not yet been identified. Although the current findings could be taken as strong evidence for existence of pleiotropy, there are other pathways.

One key alternative is when genetically-influenced aspects of personality (e.g., emotional reactivity, enthusiasm) impact sleep via phenotype, rather than direct genetic influence. Individuals who are more stable and happier sleep better (Duggan et al., 2014; Križan & Hisler, 2019; Ong et al., 2017; Stephan et al., 2017). Given evidence that propensities toward stress, anxiety, anger, and lethargy directly disrupt sleep, such individuals are more likely to exhibit poor sleep quality. Ultimately, this combination of genetic influences specific to personality and subsequent behavioral-causal effects of personality traits on sleep could contribute to genetic correlations. This may be an underappreciated developmental pathway between genetic bases of personality and its ties to sleep quality in need of examination

Moreover, gene-environment interplay is likely to play an important role in shaping these estimates. For example, genotypic differences are likely to influence environments individuals experiences in their lives (via both passive and active mechanisms). For example, individuals who are prone to anger and aggression will ultimately inhabit more conflict-ridden and hostile environments (Anderson, Buckley, & Carnagey, 2008). Such gene-based environmental selection will contribute to genetic linkages between personality traits and subjective sleep quality even if pleiotropy is not involved. Although the current data cannot favor one of these interpretations over the others, they nevertheless highly the important role environments likely play as mediating genetic pathways that yield phenotypic associations between traits and sleep quality.

4.2. Life experiences are important for tying personality to sleep quality

Even if genetic background played a large role for linking sleep to personality, there was strong evidence that person-specific life-experiences contributed. Across neuroticism, hostility, and positive affect, such experiences underlied associations of traits with subjective sleep quality. Note these associations were likely deflated due to measurement error inherent in unexplained variance. Furthermore, because they emerged regardless of shared genetic background (and any household sources of similarity), they implicate dynamic causal processes between personality traits and sleep quality (Briley et al., 2018). At minimum, they suggest that

person-specific experiences tie personality dispositions to how well a person sleeps, and call for identifying experiences that drive these associations.

Adverse and traumatic events could play an important role in such a pathway. Although a large literature has identified the corrosive long-term impact of trauma on subjective sleep quality, the impact of such events on long-term personality change is only at a nascent stage of understanding (Bleidorn et al., 2018). However, some post-traumatic reactions can be chronic in a large number of individuals (e.g., chronic post-traumatic stress disorder, Simon, 1999), highlighting one example of how life events could serve as common causes of change in both sleep quality and personality. Alternatively, environmental factors that affect sleep quality (e.g., a noisy and dangerous sleep environment) could potentially affect personality over time (e.g., increased stress reactivity due to a threatening habitat).

Finally, environments may suppress or reveal genetic influences on sleep and personality. Multiple analyses have implicated stressful or hostile environments as amplifiers of genetic differences in hostility or antagonism (Krueger, South, Johnson, & Iacono, 2008). As a result, the relative genetic contributions to phenotypic ties between sleep and personality may be larger when environments support expressions of underlying genetic propensities. Although the current data does not directly speak to such effects, they remain a core direction for future research.

4.3. Limitations and future directions

These findings also have limitations. First, only a brief measure of subjective sleep quality was used in this sample. This measure did not inquire about whether sleep was restorative or whether it was difficult to fall back asleep once awake. Also, one of the items referred to "worry," which reduced its construct specificity although worry interfering with sleep initiation is an important indicator of insomnia and sleep disturbances (Harvey, 2003; Kalmbach et al., 2020). Nevertheless, the estimates did not substantively change when analyzing only the single item capturing restlessness during sleep. Prior analyses also suggest that behavioral-genetic estimates are not too impacted by the brevity of the measure (Lind & Gehrman, 2016; Stephan et al., 2017), but it is nevertheless likely that personality traits matter for some aspects of subjective sleep quality more than others. Because the current data only captured restless sleep and worry interfering with falling asleep, these findings do not speak to other specific aspects of subjective sleep quality or other sleep characteristics (e.g., sleep timing, sleep duration). Future studies will be needed to examine these possibilities, as well as examine if they extend to more diverse samples.

Second, while the MPQ provides a comprehensive assessment of personality (Waller, DeYoung, & Bouchard, 2016), it does not neatly dovetail the big five personality dimensions that serve as the organizing framework for most contemporary research on traits and sleep. This is both a limitation as well as an advantage. It is a limitation as the trait domain of conscientiousness that is important for subjective sleep quality is not adequately represented by the MPQ scales. While features of planfulness, organization, and persistence are captured in this study (by the Control and Achievement scales), the most important features of conscientiousness implicated in poor sleep quality, namely self-control and impulsivity are not. As a result, these findings do not provide limited answers on the genetic and environmental contributions to links between conscientiousness and subjective sleep quality. Use of the MPQ provides an advantage by allowing for a finer-grained analysis of personality dispositions that go beyond the classic Big-5 personality traits. Such finer-grained analyses can reveal associations not generalizable to the Big-5 (Mõttus, 2016). Thus, by focusing on traits typically subsumed by the Big-5 dimensions, using the MPQ scales allowed for a more nuanced analysis of different trait dispositions and subjective sleep quality.

Third, these findings do not speak directly to physiological or behavioral sleep parameters. Individual differences in subjective sleep quality are only somewhat reflective of actual differences in sleep behavior, so the present estimates are likely to be shaped by a variety of reporting biases. For example, genetic associations identified here could reflect genetic influences on complaining during personality assessment. However, as different traits exhibited distinct genetic (and non-shared environmental) associations with subjective sleep quality, such concerns are minimized (i.e., such biases should affect all valenced traits similarly). Finally, personality profiles associated with subjective sleep quality were strongly indicative of behavioral sleep continuity in prior research, suggesting that patterns documented here may translate to behavioral aspects of sleep health (Križan & Hisler, 2019; Sutin et al., 2020). Even if that were not the case, subjective sleep quality is important in its own right as an aspect of overall health and a core clinical complaint (Buysse, 2014).

In the end, the developmental processes tying sleep to personality are complex, likely to change over time, and likely to be sensitive to environmental context. Our findings are consistent with an important influence of genetic background, but also likely reflect phenotypically-mediated influences of genetic influences on one but not the other, as well as gene-environment correlations. The last possibility suggests that age may serve as a qualifying factor, as active gene-environment correlations are likely to play a stronger role later in life (Briley et al., 2018). The relative stability of personality may also afford it causal primary in developmental processes, but longitudinal designs that simultaneously assess change in both alongside genetic influences will be critical to marshalling evidence. Although the current sample did not afford adequate power to model age as a qualifier of bi-variate associations, future research should take lifespan processes into account. Also, the shared impact of non-systematic, individual-specific influences on both personality traits and sleep quality strongly points to life events or features of external environments that could bind one's personality to subjective sleep quality. Targeted measurement of personality, sleep, and major life events that are likely to play etiological roles in this association should be the primary focus for ongoing research.

5. Conclusions

As sleeping well does reflect one's character, this study thought to estimate genetic and environmental sources of ties between personality traits and subjective sleep quality. The findings confirmed the substantive role that differences in emotional functioning play in subjective sleep quality, and further revealed shared genetic influences as a critical factor. Moreover, they point to an important role of idiosyncratic life experiences in binding subjective sleep quality to personality, regardless of genetic background. Thus, they highlight social and life experiences as potentially important mediators of genetic influences, calling for understanding the influence of genes that extends beyond pleiotropy.

6. Contributions

Z. Krizan was responsible for all stages of research (excluding original study design). G. Hisler contributed to data preparation, analysis, and report-writing. R. F. Krueger contributed to data-analysis and report writing. M. C. McGue contributed to study conceptualization.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jrp.2020.104048.

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