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Linking genetic foundations of sleep disturbances to personality traits: a study of mid-life twins

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Summary

Risk of sleep disturbances depends on individuals' personality, and a large body of evidence indicates that individuals prone to neuroticism, impulsivity, and (low) extraversion are more likely to experience them. Origins of these associations are unclear, but common genetic background may play an important role. Participants included 405 twin pairs (mean age of 54 years; 59% female) from the National Survey of Midlife Development in the United States (MIDUS) who reported on their personality traits (broad and specific), as well as sleep disturbances (problems with falling asleep, staying asleep, waking early, and feeling unrested). Uni- and bivariate biometric decompositions evaluated contributions of genetic and environmental factors to associations between personality and poor sleep, as well as unique contributions from individual traits. Neuroticism, extraversion, conscientiousness, and aggressiveness were the strongest phenotypic predictors of poor sleep. Genetic sources of covariance were about twice as large as non-shared environmental sources, and only shared genetic background accounted for links between aggressiveness and poor sleep. Neuroticism and extraversion accounted for most of the genetic overlap between personality and sleep disturbances. The findings shed light on developmental antecedents of ties between personality and poor sleep, suggesting a larger role of common genetic background than idiosyncratic life experiences. The results also suggest that emotion-related traits play the most important role for poor sleep, compared to other personality traits, and may partially account for genetic associations with other traits.

KEYWORDS
aggression, 'Big Five', insomnia, positive affect, twin

1 | INTRODUCTION

Healthy sleep should be high in quality and free of disturbances—namely perceived as smooth, uninterrupted, and restorative (Buysse, 2014). Subjective sleep disturbances are consequential as they are a robust indicator of psychopathology, a common symptom of mental disorders, and a marker of physical health (Buysse, 2014; Krystal & Edinger, 2008). As a result, it is important to understand individual risk factors for poor sleep, as well as probe underlying aetiological connections.

While poor sleep is associated with a variety of sociodemographic differences, a key risk factor for poor sleep involves individuals' personality traits—individual differences in persistent patterns of thinking, feeling, and acting (McAdams & Pals, 2006). Evidence suggests sleep disturbances are fairly stable over time, which also implies that stable features of individuals, like personality traits, could be important concomitant factors (Stephan et al., 2018). Critically, understanding personality diatheses for poor sleep can aid in identification of individuals at most risk, recognition of person-based confounding factors that shape interpretations of consequences arising from poor sleep, and enriching the understanding of how insomnia develops.

Research on personality traits has converged on an empirically supported structure of individual differences that reflects combinations across five broad trait domains ('Big Five'). These interrelated domains are typically labelled *extraversion* (being outgoing/energetic versus solitary/reserved), *neuroticism* (being distressed/unstable versus resilient/assured), *agreeableness* (being friendly/compassionate versus critical/hostile), *conscientiousness* (being responsible/organised versus impulsive/careless), and *openness* (being curious/inventive versus traditional/uninventive; John et al., 2008). This structure of the five core traits is genetically based, persistent across the life-course, common to normal and abnormal personality expression, and relatively universal across cultures (DeYoung, 2010; Jang et al., 1998; Markon, 2009; McCrae & Costa, 1997).

These individual differences in personality are also arranged hierarchically, such that few broad trait domains (the Big Five) subsume a larger number of more specific and inter-linked trait facets, as well as underlie even broader meta-traits (DeYoung, 2010; Jang et al., 1998; Markon, 2009). Because the structure of personality is complex and intertwined, the finer-grained features sometimes lay at the borders of the Big Five domains. For example, trait hostility is considered a facet of neuroticism (given anger co-occurs with other negative emotions), yet it also relates closely to low agreeableness (DeYoung, 2010). As a result, considering more specific features of personality may be informative, as these fine-grained facets could be linked to sleep in opposing ways within a domain, masking connections at a more general level of personality description.

2 | PERSONALITY AND SLEEP DISTURBANCES

The most robust personality predictor of poor sleep and insomnia complaints is trait *neuroticism*. Neuroticism consistently foreshadows poor sleep quality, delayed, and interrupted sleep, and does so over and above demographic factors (Cellini et al., 2017; Duggan et al., 2014; Stephan et al., 2018). These 'neurotic' personality features also serve as risk factors for future insomnia and are concomitant with elevated anxiety and depression.

Also, low extraversion (particularly low positive affectivity) and low conscientiousness (high impulsivity) have been linked to reports of poorer sleep (Cellini et al., 2017; Duggan et al., 2014; Križan & Hisler, 2019). However, these ties are weaker and less consistent across studies than those of neuroticism, while agreeableness and openness show weak or inconsistent relations (Cellini et al., 2017; Duggan et al., 2014; Kim et al., 2015; Križan & Hisler, 2019). In this vein, a recent large-scale investigation of >22,000 individuals revealed neuroticism as the strongest and most robust predictor of poor sleep, followed by (low) conscientiousness and (low) extraversion, and negligible associations with agreeableness and openness (Stephan et al., 2018).

An important set of traits consistently linked to poor sleep are hostility and aggressiveness, which traverse both the agreeableness and neuroticism domains, and are thus not precisely assessed by the broader trait domains (i.e., Big Five measures). In this vein, although low agreeableness in general shows only weak or null links with poor sleep (Kim et al., 2015; Križan & Hisler, 2019; Sutin et al., 2020), hostility and aggressiveness have been repeatedly implicated in sleep disturbances (Hisler & Krizan, 2017; Križan & Hisler, 2019; Van Veen et al., 2021). In brief, personality traits predict sleep disturbances with similar power as important sociodemographic differences (e.g., age and sex; Križan & Hisler, 2019), as well as show incremental predictive utility above those other risk factors.

3 | STUDY PURPOSE

Despite strong associations between personality and poor sleep, the reasons for these links are not fully understood. While much work has focused on proximal mechanisms (e.g., pre-sleep rumination, Slavish et al., 2019; Lancee et al., 2017), a critical developmental source of individual differences in both personality and sleep problems that deserves consideration is genetic background. Both insomnia complaints and personality traits show substantial heritability and have been linked to single-nucleotide polymorphisms (SNPs). Behaviour genetic studies of poor sleep and insomnia complaints indicate that genetic background accounts for about 30%–40% of between-subject variance (Kocevska et al., 2021; Madrid-Valero et al., 2021). Moreover, genome-wide association studies (GWAS) also implicate hundreds of common polymorphisms as critical for insomnia complaints, with a recent meta-analysis estimating that these SNPs account for 2.6% of phenotypic variance (Jansen et al., 2019), although other studies of both insomnia disorder and sleep complaints have provided higher estimates (Lane et al., 2017; Song et al., 2020).

Similarly, personality traits also show substantial heritability. Behaviour genetic studies estimate about 40%–50% of personality differences can be attributed to genetic background, with estimates similar across the trait domains, as well as trait hierarchy (Vukasović & Bratko, 2015). GWAS have also searched for common SNPs that may index personality differences, with most work focused on neuroticism, given its strong associations with health (Nagel et al., 2018).

Given both sleep disturbances and personality traits show substantial heritability, as well as strong phenotypic associations, it is critical to estimate (i) how much do sleep disturbances and personality traits share common genetic (versus environmental) sources of variation, and (ii) how much are these influences shared across traits or *specific* to particular traits. To this end, we employed a quantitative genetic approach to identifying sources of phenotypic associations between sleep disturbances and personality traits in a sample of mid-life twins. Quantitative genetic models draw on differential genetic similarity between family members in order to estimate how much variation in a particular phenotype (i.e., poor sleep) is due to genetic or environmental influences (Briley et al., 2018; Posthuma et al., 2003). As genetic variation temporally precedes phenotypic differences, such analyses can inform our understanding of the latent factors that contribute to underlying developmental process. Moreover, a link between poor sleep and personality traits through *environmental* factors (net of genetic similarities) constitutes strong evidence for some form of mutual causation across development (as in a co-twin control design), or at minimum presence of idiosyncratic life experience that impact the development of both personality and sleep (Briley et al., 2018).

To test how much genetic versus environmental factors contribute to links between sleep disturbances and personality, we drew on a sample of > 400 twin pairs from the National Survey of Midlife Development in the United States (MIDUS; Brim et al., 2004). Bivariate decompositions of variance and covariance between sleep disturbances and personality traits shed light on the relative importance of genetic background versus environment (Goal 1). Because self-reported personality traits also share variance with each other (e.g., due to common self-report method, or substantive association between the domains themselves), 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 et al., 2017). To the extent different traits show unique associations with sleep quality, concerns that common-method variance contributes to the findings would be minimised. Moreover, this approach enables estimation of how much of the overall genetic and non-shared environmental variance in poor sleep is accounted for by individual traits, speaking to the relative importance of these traits for aetiological factors of disrupted sleep (Goal 2).

To our knowledge, this is a first comprehensive analysis on the role of shared genetic (versus environmental) background in phenotypic associations between sleep disturbances and broad-spectrum personality traits, as prior analyses did not assess all the Big Five traits alongside distinct forms of sleep disturbance (Krizan et al., 2021). Because shared environmental effects appear negligible in shaping sleep disturbances 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; Briley & Tucker-Drob, 2012; Genderson et al., 2013), we tested the more parsimonious models where environment of origin (also known as shared environmental factors) is not a source of covariation between personality traits and subjective sleep quality in adulthood. Critically, as several GWAS analyses suggest that a genetic risk for elevated trait neuroticism predicts sleep difficulties, and that the two genetic profiles are strongly correlated (Jansen et al., 2019; Stephan et al., 2020), we anticipated significant genetic correlations between neuroticism and sleep disturbances. In brief, the present analysis provides novel evidence on genetic versus environmental bases of the ties between sleep disturbance and personality, while using validated measures across the broad spectrum of human personality differences including both general and specific traits.

4 | METHODS

4.1 | Participants

The sample includes adult twins who participated in the MIDUS (Brim et al., 2004). MIDUS investigates the role of behavioural, psychological, and social factors in understanding age-related differences in physical and mental health, and all research procedures have obtained ethics approval. All the data that can be accessed given appropriate permission are secured from the Inter-University Consortium for Political and Social Research (https://www.icpsr.umich.edu/).

The first wave of data collection took place in 1995–1996 (MIDUS 1), and a longitudinal follow-up on the original participants was conducted in in 2004–2006 (MIDUS 2). Relevant data were available and extracted from this second wave of collection only. Data were collected on 1914 twins in MIDUS 1 and 1484 twins in MIDUS 2. Because the present study is concerned with the genetic and environmental contributions to the phenotypic correlations among sleep and personality variables, we only use data from intact twin pairs who both completed both the sleep and personality assessments. Of those 1484 twins, 810 had sufficient data on the requisite measures for themselves and their co-twin. Thus, the final sample size was 405 twin pairs, consisting of 167 monozygotic (MZ), 143 same-sex dizygotic (DZ), and 95 opposite-sex DZ pairs.

The age of participants spanned 34–82 years (mean [SD] = 54.28 [11.24] years; median [first quartile–third quartile] = 53 [45–62] years; skew = 0.39; 59% female). In all, 95% of the participants identified their main racial origins as White, 2% as African American, 1% as Native American, >1% as Native Hawaiian or Pacific Islander, >1% as Asian, and 1% as other or unsure (complete demographic information can be found in Brim et al., 2004).

4.2 | Measures

4.2.1 | Sleep disturbances

Sleep disturbances were measured by averaging four items asking how often the participant experiences each of the following: 'Have trouble falling asleep', 'Wake up during the night and have difficulty going back to sleep', 'Wake up too early in the morning and be unable to get back to sleep', and 'Feel unrested during the day, no matter how many hours of sleep you had'. Items were rated on a 5-point scale (5 = almost always, 4 = often, 3 = sometimes, 2 = rarely, 1 = never). Answers to these four questions were averaged to estimate overall sleep disturbance ($\alpha = 0.79$, $\omega_T = 0.85$). Within MIDUS, these four items have been widely used and validated as an index of poor sleep and cover all major forms of sleep disturbances that parallel core symptoms of insomnia (e.g., Ravyts et al., 2019; Stephan et al., 2017).

4.2.2 | Midlife Development Inventory (MIDI)

The broader Big Five personality traits were measured using the MIDI (Lachman & Weaver, 1997). Participants were asked to indicate 'how well each of the following [adjectives] describes you'. Five adjectives were used to measure 'Extraversion' ($\alpha = 0.76$, $\omega_T = 0.80$), 'Agreeableness' ($\alpha = 0.82$, $\omega_T = 0.85$), and 'Conscientiousness' ($\alpha = 0.63$, $\omega_T = 0.70$). Four adjectives were used to measure 'Neuroticism' ($\alpha = 0.74$, $\omega_T = 0.83$), and seven adjectives were used to measure 'Openness' ($\alpha = 0.76$, $\omega_T = 0.85$). Items were rated on a 4-point scale (4 = a lot, 3 = some, 2 = a little, 1 = not at all). Items were reverse coded when necessary, so that higher average scores reflected higher levels of the trait. This particular measure has good psychometric properties and has been successfully used to index Big Five traits and their associations with health in ageing populations (Stephan et al., 2017).

4.2.3 | Multidimensional Personality Questionnaire (MPQ)

To assess more specific personality features, traits were also assessed using a shortened version of the MPQ (Tellegen & Waller, 2008). Participants were asked to rate how well they are described by each of 35 statements selected to assess 10 lower-order traits originally developed to represent three broader classes ('positive emotionality', 'negative emotionality', and 'constraint'). Items were rated on a 4-point scale (1 = true of you, 2 = somewhat true, 3 = somewhat false, or 4 = false). Items were reverse coded when necessary, so that higher values reflected higher standing on the trait.

The specific traits reflecting positive emotionality are 'well-being', 'social potency', 'social closeness', and 'achievement'. Well-being was assessed by three items ($\alpha = 0.75$). Social potency ($\alpha = 0.68$, $\omega_T = 0.80$), social closeness ($\alpha = 0.67$, $\omega_T = 0.73$), and achievement ($\alpha = 0.65$, $\omega_T = 0.68$) were assessed by four items. In terms of the Big Five, these traits are conceptually linked with core components of extraversion (Church, 1994).

The specific traits reflecting negative emotionality are 'stress reactivity', 'aggression', and 'alienation'. Stress reactivity ($\alpha = 0.76$) and alienation ($\alpha = 0.66$) were assessed by three items. Aggression was assessed by four items ($\alpha = 0.67$, $\omega_T = 0.84$). Note these traits are conceptually linked with neuroticism and low agreeableness. Specifically, stress reactivity is roughly equivalent to neuroticism and aggression is a strong marker of the negative pole of agreeableness (Church, 1994).

The specific traits reflecting behavioural constraint are 'control', 'traditionalism', and 'harm avoidance'. Control ($\alpha = 0.60$) and traditionalism ($\alpha = 0.59$) were assessed by three items. Harm avoidance was assessed by four items ($\alpha = 0.57$, $\omega_T = 0.60$). Constraint is conceptually linked with conscientiousness and low openness. Specifically, control is strongly associated with conscientiousness and traditionalism is moderately negatively associated with aspects of openness (Church, 1994). While reliability estimates for several MPQ scales did not reach ideal thresholds, they are acceptable for brief measures of broad constructs; high internal consistency may indicate that the brief measure fails to adequately cover the full breadth of the construct (Boyle, 1991). Critically, measuring these more specific traits provided an opportunity for conceptual replications of association with theoretically similar Big Five domains (e.g., MPQ well-being as a component of MIDI extraversion), as well as exploration of association at a more granular level with traits that lie at the border of broader trait domains (e.g., MPQ aggression). While simpler higher-order personality trait structures (e.g., meta-traits) were evaluated, given the substantive limitations of the recovered factors and the goal of this analysis to address traits indexing Big Five domains specifically, the analysis focuses on individual traits.

4.3 | Data preparation

Prior to conducting analyses, data were inspected for meeting the statistical assumption of normality. Visual inspection of the scale histograms and density plots suggested that the distributions of subjective sleep quality, stress reactivity, aggression, and alienation were all positively skewed, while agreeableness, extraversion, and conscientiousness were negatively skewed. Similar to previous personality-genetics research, skewed variables were transformed with a rank-based transformation. For positively skewed variables, a Blom transformation was applied. For negatively skewed variables, a cube-root transformation was applied (cf. Wright et al., 2017). Because age and gender differences were outside the scope of the present study, per standard practice, all variables were then regressed on sex, the linear and quadratic effects of age, and the age–sex interaction (McGue & Bouchard, 1984). The unstandardised residuals of these regressions were then used in subsequent analyses.

4.4 | Biometric decompositions

First, we estimated univariate ACE, ADE, and AE models to decompose variance in personality traits and subjective sleep quality into additive genetic (A), non-additive genetic (D), common environmental (C), and non-shared environmental components (E). Personality traits and sleep quality often show little to no common environmental or non-additive genetic variance components when examining broad, average estimates of variance (Genderson et al., 2013; Wright et al., 2017). As a result, we used the Akaike information criterion (AIC) to compare each ACE, ADE, and simpler AE models for each trait, and the model with the lowest AIC (and highest parsimony) was determined to best fit the observed data.

After modelling the appropriate univariate decompositions, we then estimated bivariate models, specifically 'Cholesky decompositions', of the covariance between sleep and personality traits to examine genetic and environmental contributions to their co-occurrence. Uni- and bivariate models were estimated in OpenMx (Boker et al., 2022), modifying sample scripts by Dr Hermine Maes (https://hermine-maes.squarespace.com/).

4.5 | Relative-weights analysis

Because personality traits share substantial genetic variance, it is difficult to make inferences on unique contributions of individual personality traits on sleep using traditional multiple regression techniques. 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). We ran these analyses to evaluate unique contributions of individual personality traits to genotypic ties with sleep disturbances. To do so, we first derived a genetic correlation matrix and a (unique) environmental correlation matrix from the bivariate models. Then, we simulated datasets with the study sample size and these correlation matrices as parameters. We used these simulated datasets to conduct relative-weights analyses to model the total and unique association of personality traits with sleep disturbances separately for genetic and non-shared environmental components (Tonidandel & LeBreton, 2011). This analysis allowed us to estimate the *total* genetic and environmental overlaps between all personality traits and sleep disturbances, as well as the *unique* overlaps of these components for each personality trait with disrupted sleep. We ran these analyses for both the overlap of the Big Five traits with poor sleep, as well as the more specific MPQ traits. This new set of orthogonal variables were then used as predictors of sleep disturbances. Estimates obtained from this analysis were transformed back to their original metric for interpretation (as in Wright et al., 2017).

5 | RESULTS

5.1 | Preliminary analyses

Descriptive statistics and phenotypic correlations of sleep disturbances, the Big Five, and the MPQ traits are presented in Table 1. Neuroticism ($r = 0.31$), extraversion ($r = -0.19$), openness ($r = -0.17$), well-being ($r = -0.24$), stress reactivity ($r = 0.33$), and alienation ($r = -0.17$) all had moderate correlations with subjective sleep disturbances. Conscientiousness ($r = -0.11$), social potency ($r = -0.10$), aggression ($r = -0.12$), control ($r = 0.09$), and achievement ($r = -0.15$) had more modest but statistically significant ($p < 0.05$) correlations with sleep disturbances. Agreeableness, social closeness, traditionalism, and harm avoidance were not significantly correlated with sleep disturbances (all $p > 0.05$). Additional descriptive statistics and correlations for the individual indicators of sleep disturbances are presented in Supplemental Table S1.

Given the broad age range in the sample, we examined if sleep disturbances varied with age. The composite sleep disturbances score was not significantly correlated with age ($r = 0.002$), nor with any of its four items ($|r| < 0.03$, $p > 0.05$). Further, the composite sleep disturbances the mean did not vary across stratified subsamples of the older ($n = 403$; age ≥ 54 years; mean [SD] 2.53 [0.89]) and younger ($n = 407$; age < 54 years; mean [SD] 2.56 [0.83]) participants, nor did any of the means of the four items (mean differences <0.3 across subsamples). Correlations between sleep disturbances (both the composite score and individual items) and the personality traits in the older and younger subsamples are presented in Supplemental Tables S2 and S3. The associations were largely consistent across the two age groups (i.e., $\Delta r < 0.10$) with the exception of neuroticism ($r = 0.38$ in the younger subsample; $r = 0.26$ in the older subsample), aggression ($r = 0.17$ and 0.03), and alienation ($r = 0.29$ and 0.07) each of which had a stronger association with sleep disturbances in the younger compared to the older subsample.

5.2 | Univariate estimates

The fit statistics and results for the univariate models that estimate genetic and environmental contributions to variance are displayed in Table 2. The ACE, ADE, and AE models were estimated for each measure. The reduced AE model fit the data best for sleep disturbances and for 13 of the 15 personality traits that were examined. In each of these cases, the greatest proportion of variance was accounted for by unique environmental effects (E), with the remaining variance accounted for additive genetic effects (A). The full ACE model fit the data best for the trait of traditionalism. The ADE model fit the data slightly better than the ACE model for the trait of neuroticism. Additional univariate estimates for the sleep disturbances indicators are presented in Supplemental Table S4, as well as estimates for the sleep disturbances composite score across the older and younger subsamples. Broad-sense heritability estimates were similar across the items ($h^2 = 0.27$–0.35). Noting that stratifying the sample in this manner causes the biometric analyses to be underpowered (i.e., with 208 twin pairs per subsample), the older and younger subsamples also had similar heritability estimates ($h^2 = 0.25$ and 0.27, respectively). Due to low power (exacerbated further in bivariate models) and the observed similarity across age groups, subsequent analyses were not stratified by age.

5.3 | Bivariate analyses

Next, bivariate Cholesky decompositions of the covariances between each of the traits and sleep disturbances were executed. Similarly, for each pair of variables, three models were estimated: an ACE model, an ADE model, and a reduced AE model. In all cases the reduced AE model had the best fit to the data, as indicated by lower information criteria (AIC and Bayesian information criterion), so only results from the AE models are reported. These models tested whether genetic and non-shared environmental components of personality traits covaried with their respective components of sleep disturbances. As such, genetic and non-shared environmental correlations between sleep disturbances and both the Big Five and the MPQ traits are

TABLE 1 Descriptive statistics and correlations among sleep disturbances, 'Big Five', and Multidimensional Personality Questionnaire traits

Variable	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Sleep disturbances	2.54 (0.86)		0.31*	−0.04	−0.19*	−0.17*	−0.11*	−0.24*	−0.10*	−0.15*	−0.09	0.33*	0.12*	0.17*	−0.09*	0.00	−0.02
2. Neuroticism	2.05 (0.61)	0.31*		−0.11*	−0.17*	−0.22*	−0.18*	−0.35*	−0.17*	−0.23*	−0.16*	0.66*	0.33*	0.35*	0.00	0.00	0.02
3. Agreeableness	3.49 (0.50)	0.01	−0.11*		0.44*	0.33*	0.28*	0.24*	0.10*	0.20*	0.35*	−0.15*	−0.24*	−0.02	0.23*	0.11*	0.11*
4. Extraversion	3.11 (0.56)	−0.17*	−0.19*	0.45*		0.49*	0.28*	0.52*	0.43*	0.34*	0.41*	−0.23*	−0.11*	−0.16*	0.08	−0.02	−0.08
5. Openness	2.83 (0.53)	−0.17*	−0.23*	0.31*	0.49*		0.39*	0.49*	0.43*	0.47*	0.12*	−0.22*	−0.14*	−0.19*	0.12*	−0.22*	−0.22*
6. Conscientiousness	3.43 (0.42)	−0.09*	−0.17*	0.29*	0.28*	0.39*		0.21*	0.17*	0.37*	0.06	−0.25*	−0.26*	−0.26*	0.34*	0.01	0.05
7. Well-being	8.98 (1.77)	−0.23*	−0.37*	0.25*	0.52*	0.49*	0.20*		0.37*	0.46*	0.25*	−0.31*	−0.12*	−0.17*	0.10*	−0.05	−0.14*
8. Social potency	9.99 (2.27)	−0.12*	−0.15*	0.05	0.39*	0.42*	0.16*	0.35*		0.39*	0.18*	−0.15*	−0.03	−0.13*	−0.03	−0.09*	−0.15*
9. Achievement	12.11 (2.20)	−0.15*	−0.22*	0.17*	0.32*	0.47*	0.36*	0.45*	0.40*		0.12*	−0.16*	−0.14*	−0.08	0.19*	−0.03	−0.15*
10. Social closeness	12.09 (2.45)	−0.06	−0.15*	0.38*	0.42*	0.11*	0.07	0.25*	0.15*	0.11*		−0.21*	−0.14*	−0.16*	0.10*	0.13*	0.09*
11. Stress reactivity	6.10 (2.24)	0.32*	0.66*	−0.17*	−0.25*	−0.22*	−0.24*	−0.32*	−0.14*	−0.15*	−0.22*		0.42*	0.46*	−0.03	0.05	−0.09
12. Aggression	5.31 (1.74)	0.10*	0.33*	−0.28*	−0.13*	−0.14*	−0.25*	−0.13*	0.00	−0.11*	−0.16*	0.43*		0.41*	−0.06	−0.02	−0.12*
13. Alienation	5.17 (1.90)	0.18*	0.36*	−0.03	−0.17*	−0.18*	−0.25*	−0.18*	−0.12*	−0.06	−0.17*	0.47*	0.43*		0.00	0.11*	−0.01
14. Control	9.79 (1.46)	−0.09*	−0.02	0.23*	0.08	0.12*	0.34*	0.11*	−0.03	0.19*	0.10*	−0.03	−0.05	0.00		0.14*	0.16*
15. Traditionalism	8.56 (2.21)	0.02	−0.02	0.15*	0.00	−0.21*	0.01	−0.03	−0.11*	−0.03	0.15*	0.03	−0.05	0.08	0.14*		0.21*
16. Harm avoidance	12.19 (2.77)	0.01	0.01	0.18*	−0.04	−0.21*	0.06	−0.12*	−0.18*	−0.17*	0.13*	−0.11*	−0.15*	−0.04	0.16*	0.23*	

Note: means, standard deviations (SDs), and correlations below the diagonal are reported prior to the transformation procedures. Correlations above the diagonal are reported after transformation and regression procedures.

*$p < 0.01$.

TABLE 2 Univariate parameter estimates and fit statistics for sleep disturbances, the 'Big Five', and Multidimensional Personality Questionnaire traits

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 disturbances	173	243	0.25	0.12	0.26 (0.00–0.39)	–	0.00 (0.00–0.00)	0.74 (0.62–0.89)	2797.2
					0.20 (0.00–0.39)	0.06 (0.00–0.41)	–	0.73 (0.60–0.88)	2797.2
					0.26 (0.13–0.39)	–	–	**0.74 (0.62–0.88)**	**2795.2**
Neuroticism	170	243	0.44	0.06	0.37 (0.19–0.49)	–	0.00 (0.00–0.00)	0.63 (0.53–0.75)	2763.3
					0.00 (0.00–0.00)	**0.40 (0.00–0.52)**	–	**0.59 (0.49–0.71)**	**2760.1**
					0.37 (0.25–0.49)	–	–	0.63 (0.53–0.75)	2761.3
Agreeableness	171	243	0.35	0.16	0.34 (0.02–0.47)	–	0.00 (0.00–0.00)	0.66 (0.55–0.80)	2774.3
					0.28 (0.00–0.47)	0.06 (0.00–0.47)	–	0.65 (0.54–0.79)	2774.3
					0.34 (0.21–0.47)	–	–	**0.66 (0.55–0.79)**	**2772.3**
Extraversion	171	243	0.36	0.17	0.36 (0.04–0.49)	–	0.00 (0.00–0.00)	0.64 (0.53–0.78)	2771.8
					0.30 (0.00–0.50)	0.07 (0.00–0.50)	–	0.63 (0.51–0.76)	2771.7
					0.36 (0.24–0.50)	–	–	**0.64 (0.53–0.77)**	**2769.8**
Openness	169	240	0.36	0.17	0.36 (0.03–0.49)	–	0.00 (0.00–0.00)	0.64 (0.53–0.78)	2751.0
					0.32 (0.00–0.49)	0.04 (0.00–0.49)	–	0.63 (0.52–0.76)	2750.9
					0.36 (0.24–0.49)	–	–	**0.64 (0.53–0.76)**	**2748.9**
Conscientiousness	171	243	0.30	0.22	0.21 (0.00–0.48)	–	0.12 (0.00–0.35)	0.67 (0.54–0.82)	2772.6
					0.36 (0.02–0.49)	0.00 (0.00–0.00)	–	0.64 (0.53–0.77)	2773.3
					0.36 (0.23–0.49)	–	–	**0.64 (0.53–0.77)**	**2771.3**
Well-being	172	244	0.29	0.11	0.27 (0.00–0.39)	–	0.00 (0.00–0.00)	0.73 (0.62–0.87)	2789.1
					0.19 (0.00–0.39)	0.08 (0.00–0.41)	–	0.72 (0.60–0.86)	2789.0
					0.27 (0.14–0.39)	**–**	**–**	**0.73 (0.62–0.87)**	**2787.1**
Social potency	173	243	0.41	0.18	0.42 (0.16–0.55)	–	0.00 (0.00–0.00)	0.58 (0.48–0.71)	2767.5
					0.27 (0.00–0.55)	0.17 (0.00–0.56)	–	0.56 (0.46–0.70)	2767.1
					0.42 (0.29–0.55)	–	–	**0.58 (0.48–0.70)**	**2765.5**
Achievement	172	243	0.34	0.11	0.32 (0.08–0.45)	–	0.00 (0.00–0.00)	0.68 (0.57–0.81)	2780.1
					0.09 (0.00–0.43)	0.26 (0.00–0.49)	–	0.65 (0.53–0.79)	2779.2
					0.32 (0.19–0.45)	–	–	**0.68 (0.57–0.81)**	**2778.1**
Social closeness	173	243	0.40	0.18	0.39 (0.09–0.51)	–	0.00 (0.00–0.00)	0.61 (0.51–0.74)	2768.9
					0.32 (0.00–0.51)	0.07 (0.00–0.51)	–	0.60 (0.49–0.73)	2768.9
					0.39 (0.27–0.52)	–	–	**0.61 (0.51–0.73)**	**2766.9**
Stress reactivity	172	243	0.33	0.12	0.30 (0.00–0.42)	–	0.00 (0.00–0.00)	0.70 (0.59–0.83)	2781.1
					0.20 (0.00–0.42)	0.11 (0.00–0.43)	–	0.69 (0.57–0.83)	2781.0
					0.30 (0.18–0.42)	–	–	**0.70 (0.59–0.83)**	**2779.1**
Aggression	172	244	0.39	0.13	0.38 (0.16–0.51)	–	0.00 (0.00–0.00)	0.62 (0.52–0.75)	2775.5
					0.10 (0.00–0.49)	0.31 (0.00–0.55)	–	0.59 (0.48–0.73)	2774.2
					0.38 (0.25–0.51)	**–**	**–**	**0.62 (0.52–0.75)**	**2773.5**
Alienation	172	243	0.46	0.18	0.46 (0.26–0.59)	–	0.00 (0.00–0.00)	0.54 (0.36–0.66)	2755.9
					0.20 (0.00–0.57)	0.29 (0.00–0.61)	–	0.51 (0.42–0.64)	2754.5
					0.46 (0.33–0.59)	**–**	**–**	**0.54 (0.45–0.66)**	**2753.8**
Control	173	243	0.29	0.15	0.18 (0.00–0.39)	–	0.08 (0.00–0.30)	0.74 (0.62–0.88)	2786.7
					0.27 (0.00–0.39)	0.00 (0.00–0.00)	–	0.72 (0.61–0.85)	2786.9
					0.27 (0.15–0.39)	–	–	**0.72 (0.61–0.85)**	**2784.9**
Traditionalism	170	242	0.42	0.28	**0.18 (0.00–0.49)**	**–**	**0.21 (0.00–0.43)**	**0.61 (0.50–0.73)**	**2741.2**
					0.42 (0.15–0.53)	0.00 (0.00–0.00)	–	0.57 (0.49–0.68)	2743.6
					0.42 (0.31–0.53)	–	–	0.57 (0.49–0.68)	2741.6

(Continues)

TABLE 2 (Continued)

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
Harm avoidance	172	244	0.32	0.19	0.23 (0.00–0.44)	–	0.08 (0.00–0.32)	0.69 (0.57–0.83)	2779.7
					0.32 (0.00–0.45)	0.00 (0.00–0.00)	–	0.67 (0.57–0.80)	2780.0
					0.32 (0.20–0.45)	–	–	**0.67 (0.57–0.80)**	**2778.0**

Abbreviations: AIC, Akaike information criterion; CI, confidence interval; DZ, same-sex dizygotic twins; ICC, intraclass correlation coefficient; MZ, monozygotic twins.

TABLE 3 Phenotypic, genetic, and non-shared environmental correlations between personality traits and sleep disturbances from bivariate models.

Variable	Phenotypic, r	Genetic, r_g (95% CI)	Non-shared environmental, r_e (95% CI)
Big Five traits			
Neuroticism	0.31*	0.37* (0.09 to 0.63)	0.29* (0.18 to 0.40)
Agreeableness	−0.04	0.05 (−0.26 to 0.39)	−0.08 (−0.20 to 0.05)
Extraversion	−0.19*	−0.22 (−0.49 to 0.10)	−0.20* (−0.32 to −0.07)
Openness	−0.17*	−0.05 (−0.33 to 0.30)	−0.22* (−0.34 to −0.10)
Conscientiousness	−0.11*	−0.19 (−0.50 to 0.12)	−0.07 (−0.20 to 0.05)
MPQ traits			
Well-being	−0.24*	−0.40* (−0.71 to −0.06)	−0.20* (−0.32 to −0.08)
Social potency	−0.10*	0.10 (−0.17 to 0.48)	−0.22* (−0.33 to −0.09)
Achievement	−0.15*	−0.15 (−0.45 to 0.20)	−0.15* (−0.27 to −0.02)
Social closeness	−0.09	−0.46* (−0.79 to −0.19)	0.05 (−0.07 to 0.18)
Stress reactivity	0.33*	0.67* (0.40 to 0.97)	0.22* (0.10 to 0.33)
Aggression	0.12*	0.37* (0.08 to 0.67)	0.05 (−0.08 to 0.17)
Alienation	0.17*	0.43* (0.17 to 0.71)	0.09 (−0.03 to 0.22)
Control	−0.09*	−0.29 (−0.69 to 0.04)	0.01 (−0.10 to 0.13)
Traditionalism	0.00	−0.09 (−0.36 to 0.17)	0.03 (−0.09 to 0.16)
Harm avoidance	−0.02	−0.05 (−0.37 to 0.27)	−0.02 (−0.14 to 0.10)

Note: 95% confidence interval (CI) displayed in brackets. All values derive from AE models.
Abbreviations: MPQ, Multidimensional Personality Questionnaire.
*$p < 0.05$.

reported in Table 3. Additional tables displaying the genetic and non-shared environmental correlations between individual sleep disturbances indicators and the Big Five and MPQ traits are displayed in Supplemental Tables S5–S8. Crucially, these supplemental findings do not suggest that scientific conclusions depend on the specific form of sleep disturbance.

Significant genetic correlations with sleep disturbances were observed for neuroticism ($r_g = 0.37$), well-being ($r_g = -0.40$), social closeness ($r_g = -0.46$), stress reactivity ($r_g = 0.67$), aggression ($r_g = 0.37$), and alienation ($r_g = -0.43$). Moderate correlations that fell short of traditional significance thresholds were observed for extraversion ($r_g = -0.22$), conscientiousness ($r_g = -0.19$), and control ($r_g = -0.29$). Significant non-shared environmental correlations with sleep disturbances were observed for neuroticism ($r_e = 0.29$), extraversion ($r_e = -0.20$), openness ($r_e = -0.22$), well-being ($r_e = -0.20$), social potency ($r_e = -0.22$), achievement ($r_e = -0.15$), and stress reactivity ($r_g = 0.22$).

5.4 | Relative-weights analyses

Because personality traits share genetic and non-shared environmental variance with each other (Table 3), we next aimed to estimate the *unique* overlap between a given trait and sleep disturbances. To do so, we used the constructed genetic and non-shared environmental correlation matrices (e.g., matrix of genetic correlations between each of the Big Five traits with sleep disturbances and each other) to perform four separate relative-weights analyses (Johnson, 2000). First, we ran an analysis on the genetic overlap between the Big Five and sleep disturbances (Table 4). The second analysis corresponded to the non-shared environmental overlap between the Big Five and sleep disturbances (Table 5). Finally, we examined the genetic (Table 6) and non-shared environmental (Table 7) overlap between the MPQ traits and sleep disturbances.

Altogether, the Big Five traits shared 29% of genetic variance with sleep disturbances. Returning to univariate estimates, 26% of the

TABLE 4 Relative weights of Big Five trait genetic covariance predicting genetic variance in sleep disturbances.

Variable	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Rescaled relative weight (% of overall model R^2 explained)
Neuroticism	0.13*	0.08 to 0.19	44.0
Agreeableness	0.03*	0.01 to 0.06	10.4
Extraversion	0.10*	0.06 to 0.14	33.7
Openness	0.01	−0.01 to 0.01	3.1
Conscientiousness	0.03*	0.00 to 0.06	8.7
Total	0.29		

*$p < 0.05$.

TABLE 5 Relative weights of Big Five non-shared environmental covariance predicting non-shared environmental variance in sleep disturbances.

Variable	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Re-scaled relative weight (% of overall model R^2 explained)
Neuroticism	0.07*	0.02 to 0.11	38.9
Agreeableness	0.01	−0.04 to 0.02	3.1
Extraversion	0.05*	0.01 to 0.09	29.5
Openness	0.05	−0.00 to 0.08	26.6
Conscientiousness	0.00	−0.04 to 0.01	1.9
Total	0.18		

*$p < 0.05$.

TABLE 6 Relative weights of Multidimensional Personality Questionnaire traits genetic covariance predicting non-shared environmental variance in sleep disturbances.

Variable	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Re-scaled relative weight (% of overall model R^2 explained)
Well-being	0.09*	0.06 to 0.12	13.1
Social potency	0.04*	0.02 to 0.06	5.7
Achievement	0.01	−0.00 to 0.01	1.2
Social closeness	0.09*	0.06 to 0.13	14.0
Stress reactivity	0.22*	0.19 to 0.26	33.5
Aggression	0.04*	0.03 to 0.06	6.6
Alienation	0.09*	0.06 to 0.11	12.6
Control	0.07*	0.05 to 0.12	11.9
Traditionalism	0.01*	0.00 to 0.02	1.6
Total	0.66		

*$p < 0.05$.

TABLE 7 Relative weights of Multidimensional Personality Questionnaire traits non-shared environmental covariance predicting non-shared environmental variance in sleep disturbances.

Variable	Raw relative weight (R^2)	Raw relative weight 95% confidence interval	Re-scaled relative weight (% of overall model R^2 explained)
Well-being	0.02*	0.00 to 0.06	19.5
Social potency	0.04*	0.01 to 0.08	35.8
Achievement	0.00	−0.01 to 0.02	4.1
Social closeness	0.01	−0.01 to 0.03	4.9
Stress reactivity	0.03*	0.01 to 0.07	30.2
Aggression	0.00	−0.01 to 0.01	1.6
Alienation	0.00	−0.01 to 0.01	1.2
Control	0.00	−0.01 to 0.02	2.2
Traditionalism	0.00	−0.02 to 0.01	0.4
Total	0.10		

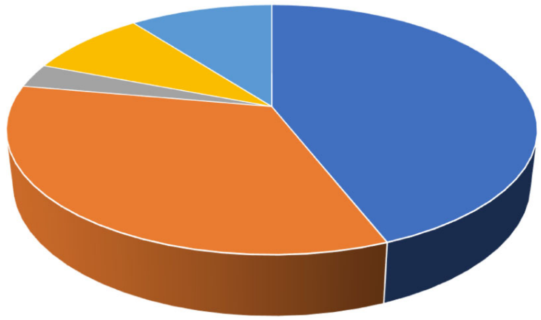
*$p < 0.05$.

total variance in sleep disturbances was attributable to genetic variance. Multiplying these two proportions suggests that genetic variance in the Big Five traits accounted for ∼8% of the total phenotypic variance in sleep disturbances. Neuroticism (13%) and extraversion (10%) accounted for 78% of the genetic overlap between the Big Five and sleep disturbances, with smaller but statistically significant contributions from conscientiousness (3%) and agreeableness (3%).

Similarly, the Big Five traits shared 18% of non-shared environmental variance with sleep disturbances. In all, 74% of the total variance in subjective sleep disturbances was attributable to non-shared environmental variance. Thus, non-shared environmental variance in the Big Five traits accounted for ∼13% (74% × 18%) of the total phenotypic variance in subjective sleep. Neuroticism (7%) and extraversion (5%) accounted for 68% of the genetic overlap between the Big Five and Sleep disturbances, while none of the other traits provided substantial contributions.

The MPQ traits shared 66% of genetic variance with sleep disturbances, in effect accounting for ∼17% (26% × 66%) of the total phenotypic variance in sleep disturbances. Each trait, except for achievement, provided a statistically significant contribution, with the most variance accounted for by stress reactivity (22%). The MPQ traits shared 10% of non-shared environmental variance with sleep disturbances, in effect accounting for ∼7% (74% × 10%) of the total phenotypic variance in sleep disturbances. Social potency (4%), stress reactivity (3%), and well-being (2%) provided statistically significant contributions.

In sum, these results suggest that personality and sleep disturbances share common genetic influences, as well as idiosyncratic unique environments that contribute to their co-variation. Genetic influences contributed more than non-shared influences across assessments of both specific and general personality traits. Traits associated with negative emotionality (neuroticism, stress reactivity, alienation, aggression) and positive emotionality (extraversion,



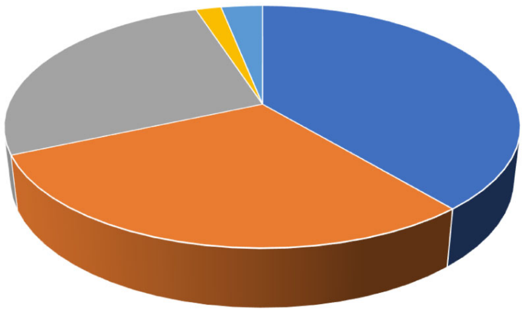


FIGURE 1 Relative contributions of personality traits to sleep disturbances in terms of (Top) genetic covariance and (Bottom) non-shared environmental covariance.

well-being, social closeness) had the largest and most unique contributions, both genetically and environmentally.

6 | DISCUSSION

This analysis aimed to address (i) how much do poor sleep and personality traits overlap due to common genetic (versus environmental) bases, and (ii) how much are these sources of sleep disturbances shared across the traits or specific to particular traits. The univariate results were fairly consistent with past findings regarding heritability of sleep complaints and personality traits, indicating moderate heritability of sleep disturbances (27%), as well as heritability for personality traits ranging from 30% to 40%. Similarly, best fitting models for both indicated only genetic and non-shared environmental influences were present, confirming a lack of shared environmental effects on insomnia complaints and personality, as observed in prior research with adults (Vukasović & Bratko, 2015).

Critically, the findings reveal robust genetic correlations between sleep disturbances and multiple traits at varying levels of generality. In accordance with studies suggesting similar genetic profiles of neuroticism and sleep disturbances based both on biometric and genome-wide analyses (Jansen et al., 2019; Stephan et al., 2020), the present results indicate neuroticism is the trait most strongly linked to sleep disturbances at both the phenotypic and genotypic level, confirmed by findings with several different trait measures indicative of neuroticism (e.g., stress reactivity, alienation). In fact, neuroticism accounted for the vast majority (78%) of genetic co-variance between personality differences and sleep disturbances (Figure 1). Put simply, genetic vulnerabilities for neuroticism are the most important personality factor for genetic vulnerabilities to sleep disturbance.

Nevertheless, the findings clearly implicate other traits as important risk or protective factors, at both the phenotypic and genotypic level. Phenotypic associations implicate low extraversion and related positive affect/well-being facets as a strong predictor of poor sleep, albeit somewhat weaker than neurotic traits. Furthermore, bivariate decompositions indicated moderate to strong genetic correlations between extraversion traits and poor sleep, which sometimes rivalled neuroticism in strength. As with phenotypes, genotypic associations indicated that positive emotions were the strongest extraversion-related indicator of (fewer) sleep disturbances. Thus, while neuroticism predicted more phenotypic variance in poor sleep, both neuroticism and extraversion seemed to contribute a similar amount of genetic variance, albeit in opposite directions.

The final set of traits implicated in poor sleep were conscientiousness, aggressiveness, and alienation (i.e., hostility). These associations were more modest at the phenotypic level (<0.20), which is consistent with past findings (Hisler & Krizan, 2017; Križan & Hisler, 2019). However, the genetic correlations were more substantial, indicating that genetic background played an especially important role in linking individual differences in self-regulation and poor impulse control to poor sleep.

Non-shared environmental factors also contributed sizably to ties between personality traits and poor sleep. For trait indicators of the neuroticism domain, unique environmental experiences accounted for some overlap, although the estimates were roughly half the size of genetic correlations for these traits. Thus, while genetic background may be relatively less important than environmental factors, overall, for sleep problems and traits (as evident in univariate estimates), genetic factors appear more important than unique environmental factors for tying poor sleep to personality makeup. In other words, genetic influences are a considerable (perhaps primary) source of the link between poor sleep and personality traits related to neuroticism and extraversion.

In contrast, idiosyncratic environments that differentiate identical twins did not seem to play a role in tying conscientiousness and aggressiveness to poor sleep; only genetic correlations were evident. This finding suggests that tendencies to control behaviour and to be socially restrained indicate poor sleep only due to common genetic influences, not shared environmental factors. This stands as an important contrast to the emotion-based traits of neuroticism and extraversion, for which idiosyncratic environmental linkages with poor sleep were substantial.

Finally, the results also reveal important information about unique contributions of individual traits. Specifically, the most robust contributions to genetic linkages between traits as a whole and poor sleep were found for neuroticism and extraversion, suggesting that genetic linkages with other traits may be partially attributed to overlap of those traits themselves with neuroticism and extraversion. While findings with specific traits (assessed via MPQ) implicated a larger number of contributing traits, here too stress reactivity (i.e., neuroticism) showed the largest contribution. These findings suggest that at both the phenotypic and genotypic level it is critical to consider pre-existing differences in extraversion and neuroticism that are confounded with other personality differences.

7 | LIMITATIONS AND FUTURE DIRECTIONS

While providing a novel glance into genetic and environmental factors responsible for ties of poor sleep to traits across the personality spectrum, the present findings also have limitations that are important to address. First, the findings are based on a sample of middle-aged, mostly White adults—to what extent these estimates generalise to younger or more diverse samples needs to be evaluated. Second, the present results do not specify the proximate, developmental mechanisms that undergird the genetic link between poor sleep and personality. On the one hand, distinct phenotypic effects of the same underlying genes (i.e., pleiotropy) can create genetic correlations between phenotypes, although genome wide analyses have not yielded common polymorphisms that influence both personality traits and sleep disturbances, notwithstanding correlated genetic profiles (Jansen et al., 2019; Stephan et al., 2020). On the other hand, these genetic correlations can also be *phenotypically mediated* (Turkheimer et al., 2014)—namely, genetic disposition to particular personality traits can lead individuals into environments that undermine sleep, through both selective and evocative processes, even in the absence of pleiotropy (Hindley et al., 2022). Prior evidence suggest that more neurotic individuals occupy roles and environments that induce more stress (e.g., strained relationships), which could then contribute to the development of sleep disturbances (Barclay et al., 2011; Luo et al., 2022). As a result, future work needs to disentangle distinct developmental pathways that link personality and sleep, ideally using a longitudinal study design that can account for gene–environment correlations.

Second, the sample was not sufficiently large to examine sociodemographic moderators of the genetic and environmental links between personality and sleep, which may qualify findings. For example, age may serve as a qualifying factor, as active gene–environment correlations may play a stronger role later in life, which would produce higher estimates of genetic contributions in older individuals (Briley et al., 2018). Future work also should test for gene–environment interactions that have been observed for personality, e.g., more stressful environments may change the relative contribution of personality traits to poor sleep (South et al., 2017).

8 | CONCLUSIONS

The present findings emphasise the importance of individual differences in personality for experiencing poor sleep—both phenotypic and genetic correlations were sizeable and transcended several trait domains, albeit with varying effect sizes. Emotion-related traits appeared most critical for poor sleep, with genetic contribution often double the size of environmental contributions. However, as emphasised, it is possible that common genetic influences are, at least in part, environmentally mediated. Regarding aggressiveness and conscientiousness, only genetic background appeared responsible for associations with poor sleep. The present results emphasise genetic ties between insomnia complaints and personality traits, further implicating insomnia as a non-specific sleep disorder that is tied to broader personality functioning, rather than a pathology that more closely tied to comorbid sleep disorders than to personality (Jansen et al., 2019). As a result, assessing and considering personality factors for modelling risk of poor sleep and insomnia has high potential relevance for future work.

AUTHOR CONTRIBUTIONS

Zlatan Krizan was primarily responsible for project conception, analysis plan, and report writing. Colin Freilich conducted the analyses and contributed to writing major sections of the report. Colin Freilich, Robert F. Krueger, and Frank D. Mann provided input on analysis, interpretation, and editing of the manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors report no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data underlying this article is part of the Midlife in the United States Study and is publicly available to researches given appropriate permissions are secured via a signed use agreement (https://www.icpsr.umich.edu/web/pages/NACDA/midus.html).

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