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RESEARCH ARTICLE

Sex differences in sleep quality and psychological distress: Insights from a middle-aged twin sample from Spain

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Summary

There is a moderate association between poor sleep and psychological distress. There are marked sex differences in the prevalence of both variables, with females outnumbering males. However, the origin of these sex differences remains unclear. The objectives of this study were to: (1) study genetic and environmental influences on the relationship between poor sleep quality and psychological distress; and (2) test possible sex differences in this relationship. The sample comprised 3544 participants from the Murcia Twin Registry. Univariate and multivariate twin models were fitted to estimate the magnitude of genetic and environmental influences on both individual variance and covariance between poor sleep quality and psychological distress. Sleep quality and psychological distress were measured using the Pittsburgh Sleep Quality Index and the EuroQol five-dimensions questionnaire, respectively. The results reveal a strong genetic association between poor sleep quality and psychological distress, which accounts for 44% (95%CI: 27%–61%) of the association between these two variables. Substantial genetic (rA = 0.50; 95%CI: 0.32, 0.67) and non-shared environmental (rE = 0.41; 95%CI: 0.30, 0.52) correlations were also found, indicating a moderate overlap between genetic (and non-shared environmental) factors influencing both phenotypes. Equating sexes in sex-limitation models did not result in significant decreases in model fit. Despite the remarkable sex differences in the prevalence of both poor sleep quality and psychological distress, there were no sex differences in the genetic and environmental influences on these variables. This suggests that genetic factors play a similar role for men and women in explaining individual differences in both phenotypes and their relationship.

KEYWORDS
psychological distress, sleep quality, twins

1 | INTRODUCTION

The relationship between sleep problems and mood disorders is especially important given the strong co-occurrence between these difficulties (Ohayon & Roth, 2003). Indeed, sleep problems are an essential part of the diagnosis of both depression and generalised anxiety disorder since symptoms related to sleep, such as insomnia or hypersomnia, are a criterion for the diagnosis of these disorders

(American Psychiatric Association, 2013). Many studies have found that depression and anxiety are associated with insomnia (Jansson-Fröjmark & Lindblom, 2008) and poor sleep quality (Meijer et al., 2010).

Age and sex are relevant to both symptoms of sleep and mood difficulties. Sleep complaints tend to increase from adolescence to adulthood; and it is common for adults to report poor sleep quality (Madrid-Valero et al., 2017). Regarding sex, women, compared with men, usually report greater sleep problems, such as insomnia and poorer sleep quality (Madrid-Valero et al., 2017; Zhang & Wing, 2006). A similar picture for anxiety and mood disorders can be found where women, compared with men, usually report a higher prevalence of these disorders (Albert, 2015; McLean et al., 2011). Even the clinical characteristics and treatment outcomes differ between men and women (Kang et al., 2020). It has also been hypothesised that the increased levels of depression/anxiety in women compared with men could also explain the sex differences in terms of insomnia symptoms (Voderholzer et al., 2003).

1.1 | Genetic and environmental influences on the association between poor sleep quality and psychological distress

Despite the frequency of poor sleep quality and psychological distress, the nature of the co-occurrence between these variables is still largely unknown. It is well established that sleep quality and anxiety/depression are moderately influenced by genetic factors. Previous twin studies in adult samples have shown that about 30%–40% of the variance in depression and anxiety is explained by genetic factors (Gasperi et al., 2017; Hettema et al., 2001). Similarly, two recent meta-analyses showed that on average 31%–44% of the variance in sleep quality is explained by genetic factors (Kocevska et al., 2021; Madrid-Valero et al., 2020).

Studies that have investigated the co-occurrence of problems in both realms have focussed on the relationship between sleep disturbances and anxiety/depression. For example, a substantial genetic overlap (genetic correlations >0.7) has been reported between insomnia and depression/anxiety (Gehrman et al., 2011; Gregory et al., 2016). Only a handful of studies have investigated the relationship between poor sleep quality and depression and anxiety focussing on non-clinical adult samples. Gregory et al. (2011) for example, found a substantial genetic overlap in adolescents/young adults (1556 individuals; mode age = 20 years [18–27 years]; rG >0.5), while no sex differences in this association were detected. Similar results regarding genetic overlap were found in a study using a sample of young/middle-aged adults (200 twin pairs; mean age = 29 years [18–65 years]; rG = 0.61), but this study was unable to compare the results across sexes due to their same-sex sample (Gasperi et al., 2017). However, as the authors stated in their manuscript, further analyses could yield potentially interesting insights into the role of sex in the link between sleep quality and psychological distress (Gasperi et al., 2017).

These results illustrate the relevant role of genetic factors in the relationship between sleep quality and depression/anxiety in adolescents and young adults as well as the scarcity of data from other groups with specific characteristics related to both outcomes (e.g. older age groups or different geographical area/cultural background). Consequently, little is known about the role of sex in the association between sleep quality and depression/anxiety in middle to old age adults, despite both phenotypes being substantially influenced by sex and the specific characteristics of these age groups regarding biological conditions (i.e. age-associated hormonal or neurobiological changes). Given that heritability is a population statistic, meaning that estimates can vary depending upon the specific population being considered, it is also important to study this association in a wide range of geographical locations for which estimates could vary.

Moreover, environmental factors relevant to sleep, anxiety, and depression can also differ between males and females according to age and are not the same for younger and older adults. For example, there can be sex and age differences in terms of care-giving responsibilities, shift-work, and comorbid diseases. Furthermore, sleep quality seems to be influenced by factors from the social environment which show ample cultural variation, such as the practice of daytime naps or siestas, commercial opening hours, late bedtimes, and prime time television hours (Sayón-Orea et al., 2013). However, samples used in previous studies in this area are limited in this respect.

Therefore, this study takes advantage of a Spanish population-based twin sample (composed of both same-sex and opposite-sex twin pairs). The objectives were to: (1) study genetic and environmental influences on the relationship between poor sleep quality and psychological distress in a large population-based sample of adult Spanish twins; and (2) test possible sex differences in this relationship.

2 | METHODS

2.1 | Participants

The sample comprised 3544 participants from a population-based registry in the region of Murcia, SE of Spain (Murcia Twin Registry, MTR). A full description of the MTR recruitment and data collection procedures is provided elsewhere (Ordoñana et al., 2019). This sample has been shown adequately to represent the Spanish adult population (Ordoñana et al., 2018). The sample was 59.9% female ($N = 2123$ participants). Zygosity distribution was as follows: 36.9% MZ ($N = 1308$ participants), 34.2% DZ same sex (DZ-SS; $N = 1211$ participants) and 28.9% DZ opposite sex (DZ-OS; $N = 1025$ participants). The mean age of the sample was 53.7 years (SD = 7.4). Data come from two different cohorts defined by year of birth. The first cohort was born between 1940 and 1966 and was interviewed in 2009/2010 when aged 53.8 years on average (SD = 7.4; range 43–71 years). The second cohort was born between 1967 and 1977 and was interviewed in 2018 when they were aged 46.3 years on average (SD = 3.3; range 43–51 years).

The MTR protocols and instruments, as well as the data collection procedures and the analysis derivatives thereof, have been approved by the Research Ethics Committee of the University of Murcia and

meet the legal requirements of confidentiality and protection of personal data. The participants provided written informed consent when interviewed in person or oral consent when a telephone interview was used.

2.2 | Measures

Sleep quality was measured using the widely used Pittsburgh Sleep Quality Index questionnaire (PSQI) (Buysse et al., 1989). The PSQI assesses sleep quality referencing the previous month. It comprises seven subscales: (1) subjective sleep quality, (2) sleep latency, (3) sleep duration, (4) habitual sleep efficiency, (5) sleep disturbances, (6) use of sleeping medication, and (7) daytime dysfunction. These seven subscales build the global scores which range from 0 to 21 where a higher score represents poorer sleep quality. In the current sample, Cronbach's alpha for the global score was 0.72.

Psychological distress was assessed by self-report, using the "depression or anxiety" domain of the Spanish version of the EQ5D (EuroQol five-dimensions questionnaire) (Rabin & de Charro, 2001; Ramos-Goñi et al., 2018). This domain of the EQ5D questionnaire offers a reasonable valid prediction of depression and anxiety disorders (König et al., 2010; Supina et al., 2007). Additionally, the EQ5D is substantially correlated with other measures of psychological distress, suggesting good convergent validity (Jutte et al., 2015). Participants from cohort 1 answered the EQ5D-3 L version of the questionnaire in which they had to choose among three different options selecting the one that best describes themselves at the present day ("I am not anxious or depressed"; "I am moderately anxious or depressed"; and "I am extremely anxious or depressed"). Participants from cohort 2 answered the EQ5D-5 L version, which has five response options (1: "I am not anxious or depressed"; 2: "I am slightly anxious or depressed"; 3: "I am moderately anxious or depressed": 4: "I am severely anxious or depressed" and 5: "I am extremely anxious or depressed"). Data from cohort 1 (3 L) and cohort 2 (5 L) were homogenised by translating data from the 5 L into the 3 L version, using a validated method as proposed by the questionnaire developers (https://euroqol.org/eq-5d-instruments/eq-5d-5l-about/valuation-standard-value-sets/crosswalk-index-value-calculator/) (Herdman et al., 2011). Once homogenised (three levels of psychological distress), given that the third category was very infrequent (2.8%), it was collapsed with the second one. Therefore, the variable was dichotomised as follows: (1) "I am not anxious or depressed" and (2) "I am moderately/extremely anxious or depressed". In other words, participants were dichotomised in those with symptoms of psychological distress and those with no symptoms.

Zygosity was established through either a DNA test ($N = 338$ twin pairs) or a 12-item questionnaire focussed on the degree of similarity and mistaken identity between twins. This questionnaire was accurate in over 96% of the cases.

2.3 | Statistical analysis

Making use of the difference in similarity between MZ twins (who share 100% of their DNA) and DZ twins (who share on average 50% of their segregating DNA) the variance of a trait can be broken down into genetic and environmental components (Knopik et al., 2017). Genetic influences can be further decomposed into additive genetic factors (A; the sum of allelic effects across all loci) and non-additive genetic effects (D; the effects of genetic dominance and, possibly, epistasis). The environmental component can also be decomposed into shared-environmental (C: environmental influences that make family members more alike, e.g. socioeconomic status, parenting style, childhood diet, or peer influences shared by both twins) and non-shared or individual factors (E: environmental influences that make family members less alike, e.g. accidents, differential parental treatment, differential prenatal exposure and measurement error) (Rijsdijk & Sham, 2002; Verweij et al., 2012).

The decision to fit an ACE or ADE model is usually made based on the pattern of correlations between MZ and DZ twins. An ACE model is selected when the DZ twin correlation is greater than half of the MZ twin correlation; an ADE model is selected if the DZ twin correlation is less than half of the MZ twin correlation (which suggests possible non-additive genetic effects) (Verweij et al., 2012).

ADE univariate models were fitted for sleep quality and psychological distress, as suggested by the pattern of correlations. As the pattern of correlations for sleep quality could also indicate an ACE model, this was also fitted to allow comparison and selection of the best fitting model. Sleep quality was treated as a continuous variable. However, psychological distress was analysed using a liability threshold model since this variable was dichotomous. In this model, an unobserved liability, normally distributed with a mean value of 0 and a variance of 1, underlying the measured categories of psychological distress is assumed (Rijsdijk & Sham, 2002). Nested models (i.e. AE and E) were also run to check if one (or two) components of the model

TABLE 1 Descriptive statistics

	Male	Female	MZ	DZ	Total sample
Total *N* (%)	1421 (40.1)	2123 (59.9)	1308 (36.9)	2236 (63.1)	3544 (100%)
Age, Mean (SD)	51.41 (7.1)	51.02 (7.3)	49.65 (6.5)	52.07 (7.5)	51.17 (7.2)
Poor sleep quality, Mean (SD)	4.56 (3.58)	5.71 (4.0)	5.30 (3.9)	5.21 (3.9)	5.24 (3.9)
Participants with psychological distress, *N* (%)	191 (13.5)	471 (24.2)	217 (17.7)	445 (20.9)	662 (19.7)

Note: There were significant differences between men and women ($p < 0.05$) for both poor sleep quality and psychological distress.

could be dropped without a significant decrease in model fit. The model and submodels goodness of fit were compared using the likelihood-ratio chi-square test and the Akaike's information criterion (AIC). Assumptions of twin models (homogeneity of means and variances across twin order and across zygosity) were checked in the saturated models and met the twin modelling assumptions.

Next, a mixed (means/threshold) multivariate model was also fitted. This model allows us to estimate the genetic and environmental influences on both individual variances and also on the sources of covariance. In other words, with this model we can estimate aetiological correlations (i.e. rA, rC/rD, and rE) which inform us to what extent the latent variables (A, C/D, and E) for poor sleep quality and psychological distress overlap. As discussed previously, both phenotypes (poor sleep quality and psychological distress), show marked sex disparities. Therefore, sex differences in variance decomposition were tested by fitting a sex-limitation model. Sex-limited expression may occur in two different ways. Scalar sex limitation is where effects of a factor could be larger for one sex compared with the other. Non-scalar sex limitation is where some factors may have an effect on one sex but not on the other (Neale et al., 2006).

Twin analyses were performed using the package OpenMx in R. Age and sex were added (although sex was not added to the sex-limitation models) to the model as covariates, as is standard procedure in twin modelling. Cohort was also included as a covariate to account for possible cohort effects.

3 | RESULTS

Descriptive statistics are displayed in Table 1. Women showed higher levels of both poor sleep quality ($\overline{X}_{male} = 4.56$; $\overline{X}_{female} = 5.71$ $p < 0.05$) and psychological distress ($\%_{male} = 13.5$; $\%_{female} = 24.2$; $p < 0.05$).

3.1 | Univariate twin models

The results of univariate twin models for the total sample are presented in Table 2. Intrapair correlations were higher for MZ twins (rMZ = 0.31 and 0.54 for poor sleep quality and psychological distress respectively) than for DZ twins (rDZ = 0.16 and 0.21). This pattern of correlations indicates that genetic factors are playing a substantial role in both phenotypes. In both cases the non-additive-genetic component (D for psychological distress; C for sleep quality) could be dropped without significant deterioration of the model fit and, therefore, the nested AE submodels provided the best fits. Regarding poor sleep quality, 31% (95%CI: 23%–38%) of the variance was explained by genetic factors and 69% (95%CI: 62%–77%) by non-shared environmental factors. As for psychological distress 51% (95%CI: 38%–63%) of the variance was attributable to genetic factors and 49% (95%CI: 37%–63%) to non-shared environmental factors. Univariate sex-limitation models were also fitted. There were no significant sex differences, for poor sleep quality ($AIC_{ADE\ qualitative\ \&\ quantitative\ sex-differences} = 8499.78$; $AIC_{ADE\ quantitative\ sex-differences} = 8497.78$; $AIC_{ADE\ no\ sex-differences} = 8492.96$)

TABLE 2 Univariate results

	Model	Model for comparison	A (95% CI)	C (95% CI)	D (95% CI)	E (95% CI)	Df	-2LL	AIC	DiffLL	Diffdf	*p*	rMZ	rDZ
Poor sleep Quality													0.31 (0.23, 0.39)	0.16 (0.08, 0.23)
	ACE		0.32 (0.10, 0.54)	−0.01 (−0.18,0.15)		0.69 (0.62, 0.77)	3080	8479.03	2319.03					
	ADE		0.28 (−0.03, 0.59)		0.02 (−0.31, 0.37)	0.69 (0.62, 0.77)	3080	8479.03	2319.03					
	AE	**ADE**	**0.31 (0.23, 0.38)**		/	**0.69 (0.62, 0.77)**	**3081**	**8479.05**	**2317.05**	**0.02**	**1**	**0.88**		
	E	AE	/		/	1 (1,1)	3082	8540.57	2376.57	61.52	1	<0.001		
Psychological Distress													0.54 (0.39, 0.66)	0.21 (0.07, 0.34)
	ADE		0.24 (−0.33, 0.79)		0.30 (−0.30, 0.92)	0.46 (0.33, 0.61)	3337	3181.22	3195.22					
	AE	**ADE**	**0.51 (0.38, 0.63)**		/	**0.49 (0.37, 0.63)**	**3338**	**3182.19**	**3194.19**	**0.96**	**1**	**0.33**		
	E	AE	/		/	1 (1,1)	3339	3230.34	3240.34	48.15	1	<0.001		

Note: The bold text identifies the best fitting model.

Abbreviations: A, additive genetic influences; AIC, Akaike information criterion; C, common environmental influences; CI, confidence interval; D, nonadditive genetic influences; df, degrees of freedom; E, unique environmental influences; *p*, significance value of the likelihood ratio chi-square test.

or for psychological distress ($AIC_{ADE\ qualitative\ \&\ quantitative\ sex-differences} = 3200.25$; $AIC_{ADE\ quantitative\ sex-differences} = 3198.25$; $AIC_{ADE\ no\ sex-differences} = 3195.18$). For both variables the comparison between the ADE model allowing sex differences (quantitative) and the ADE model not allowing sex differences was non-significant ($p = 0.758$ and $p = 0.365$ for poor sleep quality and psychological distress, respectively) so the most parsimonious model was selected (no sex differences).

3.2 | Multivariate models

Models allowing for different types of sex differences were fitted (i.e. scalar and non-scalar sex limitation models). Nested models were also compared with full models and the best fit was provided by an AE model without sex differences (Table 3).

There was a significant phenotypic correlation between poor sleep quality and psychological distress (rPh = 0.44; 95%CI: 0.39, 0.48). The results did replicate and reinforce those observed in univariate analyses for both sleep quality (A = 31%; 95%CI: 23%–37%; E = 69%; 95%CI: 63%–77%) and psychological distress (A = 49%; 95%CI: 36%–62%; E = 51%; 95%CI: 38%–64%). In addition, both genetic (rA = 0.50; 95%CI: 0.32, 0.67) and non-shared environmental (rE = 0.41; 95%CI: 0.30, 0.52) correlations were found significant, suggesting a substantial genetic and environmental overlap between these two phenotypes (Figure 1). As for the bivariate heritability, it was found that 44% (95%CI: 27%–61%) of the phenotypic association between poor sleep quality and psychological distress was explained by genetic factors, with the rest attributable to non-shared environmental factors (56%, 95%CI: 39%–74%).

TABLE 3 Multivariate models fit

	Parameters	AIC
Saturated	38	19895.26
Non-scalar ADE	26	19895.46
Scalar ADE	19	19887.32
No sex differences ADE	13	19882.44
Non-scalar AE	20	19884.89
Scalar AE	15	19882.34
No sex differences AE	**11**	**19878.61**

Abbreviations: A, additive genetic influences; AIC, Akaike information criterion; D, nonadditive genetic influences; E, unique environmental influences.

4 | DISCUSSION

This study adds new information about the aetiology of the complex relationship between poor sleep quality and psychological distress. Sex-differences in variance decomposition were tested and, despite the remarkable sex gap in the prevalence of the symptoms of these phenotypes, we did not detect significant differences between men and women in genetic and environmental contributions to phenotypic variance. That is, we found no evidence of quantitative or qualitative genetic differences between men and women for these phenotypes or their association. Our results also highlight that the phenotypic association between poor sleep quality and psychological distress is almost equally explained by genetic and unique environmental influences. Furthermore, moderate genetic and environmental correlations were found, indicating a significant overlap between genetic (and non-shared environmental) factors influencing poor sleep quality and psychological distress.

4.1 | Genetic and environmental influences on poor sleep quality and psychological distress

For both phenotypes, a substantial proportion of the phenotypic variance was explained by genetic factors. Our results showed that for psychological distress, around half of the variance was explained by genetic factors. The heritability estimate in our sample is at the upper

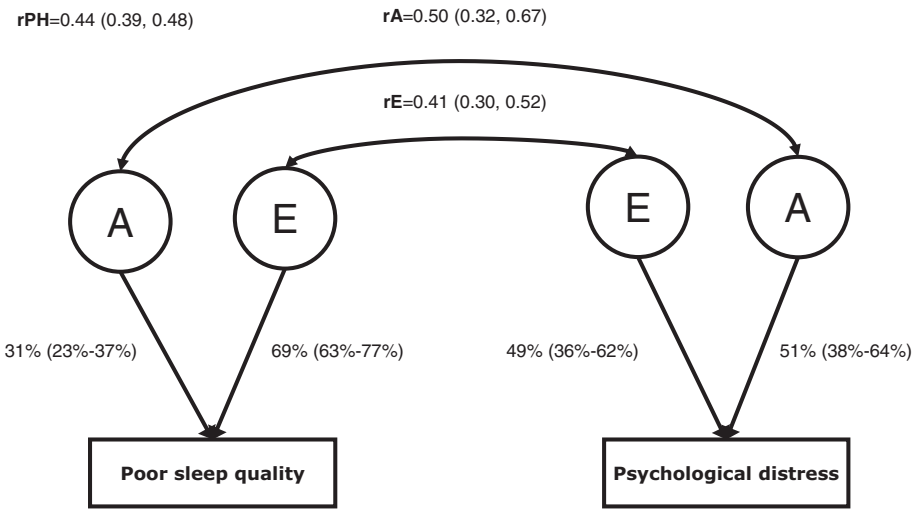

FIGURE 1 Bivariate model poor sleep quality and psychological distress. A, additive genetic influences; E, non-shared environmental influences. rA, additive genetic correlation; rE, non-shared environmental correlation; rPH, phenotypic correlation. rPH, rA, and rE were statistically significant ($p < 0.001$)

end of the range found in previous studies (Gasperi et al., 2017; Gregory et al., 2011; Hettema et al., 2001; Nivard et al., 2015; Sullivan et al., 2000). Regarding sleep quality, around one third of the variance was explained by genetic factors. This heritability estimate matches well with previous publications and the aforementioned meta-analyses (Kocevska et al., 2021; Madrid-Valero et al., 2020).

Our results confirm the significant association between poor sleep quality and psychological distress. The phenotypic correlation was almost equally explained by genetic and non-shared environmental factors; a similar result to that of Gasperi et al. (2017). A significant genetic correlation was also found, suggesting substantial genetic overlap (i.e. genes influencing poor sleep quality also influence psychological distress). Moderate genetic correlations have been reported previously, between poor sleep quality and depression (Gasperi et al., 2017; Gregory et al., 2011) and between poor sleep quality and anxiety (Gregory et al., 2011). The genetic correlations between these variables reported previously are slightly higher (although with overlapping CI) compared with our study which could be due to the different measures for psychological distress and also sample characteristics (e.g. age of the sample, geographical location). A substantial correlation for non-shared environmental factors was also found, which suggests that some of those influences which are idiosyncratic for each individual (e.g. stressful job demands, partner circumstances, or life events) could be linked to both poor sleep quality and psychological distress. However, both genetic and environmental correlations are notably lower than unity which demonstrates the preservation of considerable specificity for genetic (e.g. gene variants related to specific pathways) and non-shared environmental factors (e.g. room temperature) affecting either sleep or psychological distress. The observation of those significant correlations for both genes and non-shared environment implies that similar factors influence both phenotypes – suggesting the presence of genetic pleiotropy (i.e. the same genes influencing different traits) and non-shared environmental circumstances impacting on both phenotypes (e.g. stressful experiences). However, these data are not necessarily incompatible with a causal relationship – with disturbed sleep causing psychological distress or vice versa. Future research is needed to address different mechanisms which can explain these associations.

This study also started with the objective of testing sex differences on the association between poor sleep quality and psychological distress. Our result show that, despite differences in the prevalence of both phenotypes, the estimated genetic and environmental influences on these variables are not significantly different between men and women. The analysis of sex differences in the genetic architecture of sleep quality and psychological distress has been elusive and there are no definitive conclusions in this regard. Indeed, previous studies have reported inconsistent findings, including stronger genetic influences in males, females, and no detectable differences (Jansen et al., 2019; Kang et al., 2020). There are also reports from molecular genetic studies (i.e. GWAS or WES) of sex-specific genetic associations (Aragam et al., 2011; Kang et al., 2020; Sullivan et al., 2009). However, replication of these findings is still needed and it is not clear whether such variations would be sufficient to explain the entire scope of phenotypic differences between males and females.

Some explanations have been postulated to explain the higher prevalence in women for both poor sleep quality and psychological distress; they still remain speculative, though. Both, physiological and social dynamics have been alluded to in this regard. For example, a factor that has been extensively studied to explain these differences in middle-aged adults is menopausal status and its associated hormonal variation which could affect women's sleep quality and/or psychological distress through the destabilising effects of the cyclic oestradiol fluctuations or vasomotor symptoms. Yet, there is no complete consensus for this effect (Xu et al., 2011). In fact, using some data from the current sample, we previously investigated the role of menopausal status on sleep quality and observed that this effect was completely overshadowed by the effect of age (i.e. menopausal status was not a significant predictor of poor sleep quality once age was added into the model) (Madrid-Valero et al., 2017). More recently, it has been postulated that a higher genetic burden is needed for males compared with females to develop depression, which could contribute to the higher masculine resilience against depressive disorder (Kang et al., 2020). Additionally, women are more likely to suffer from some chronic health problems that could increase the risk of sleep difficulties and distress, such as osteoporosis, fibromyalgia, or back pain (Lallukka et al., 2012; Murtagh & Hubert, 2004). However, none of these explanations would satisfactorily account for the lack of sex differences in our heritability estimates. Clearly, more research is needed on these grounds.

Alternatively, prevalence differences could be influenced by different specific age-related environmental factors, gender-related social conditions, or sex differences in gene-by-environment interactions. For example, men are more likely to report shift-work or the emergence of age-related diseases as reasons for disrupted sleep, whereas women are more likely to report partner snoring/noises or role as a care-giver (e.g. caring for children or older people) to explain their sleep difficulties (Madrid-Valero et al., 2018). As for psychological distress, women appear to be, in general, more vulnerable to negative socioeconomic factors. Also, failures in interpersonal relationships seemed to play a stronger aetiological role in major depression for women than for men, whereas men appear to display more sensitivity to external factors such as those related to career goals (Kendler & Gardner, 2014). All these variables could interact with genetic factors facilitating or preventing the development of depressive symptoms with a gender bias. Thus, Arnau-Soler et al. (2019) identified nominally significant positive GxE effects in women, but not in men, in a validation of the diathesis-stress theory of depression (albeit this effect could be due to the male lower sample size). This points towards possible sex-specific differences in the effect of genetic risk of depression in response to stressful life events. Other studies have reported gender diversity in the interaction of specific genetic variants with environmental stressors with mixed and non-conclusive results (Das, 2020; Kang et al., 2020; Li et al., 2013). Again, whether these differences are enough to produce the ample sex-related variation and why they are not apparent in our standardised estimates should be further analysed. All in all, these factors highlight the complex relationship that exists between poor sleep quality and psychological distress.

Even though some important research questions have yet to be elucidated, the reported results bear important implications in terms of health promotion and disease prevention and treatment. Thus, the bivariate heritability and the genetic and non-shared environmental correlations between poor sleep quality and psychological distress indicate the need for considering both difficulties together when designing health promotion plans and activities. Additionally, the fact that the phenotypic association between the mentioned variables is substantially explained by non-shared environmental factors point to the utility of exploring environmental sources of distress that could contribute to both outcomes. Moreover, treatment options should consider investigating deeply and tackling comorbidity as a basic strategy. In this regard, previous publications have shown the benefits of taking into account sleep quality for treating mood disorders (Gebara et al., 2018). Finally, in terms of research requirements, efforts should aim to identify genetic variants and environmental influences contributing to both conditions, as well as possible gene × environment interactions, using large enough samples and appropriate designs to elucidate the origin of sex differences both in poor sleep quality and psychological distress.

4.2 | Limitations

This study has some limitations that must be addressed. Sleep quality and psychological distress were self-reported. A widely used questionnaire was used to measure sleep quality (i.e. PSQI). However, our psychological distress measure was somewhat simplistic and a more detailed scale would have provided additional power to the analyses. Nonetheless, ultra-brief questionnaires (e.g. PHQ-2) have shown adequate performance compared with a standard diagnostic interview, as well as established depression scales (Löwe et al., 2005); and the EQ-5D in particular has been shown previously to be valid for measuring psychological distress (Jutte et al., 2015; König et al., 2010; Supina et al., 2007). Regardless, future work should use more in depth measures to assess sleep and psychological distress. Finally, the use of a representative non-clinical sample is an advantage and allows us to extrapolate these results to the general population. However, it might be possible that the aetiology of the relationship between poor sleep quality and psychological distress is different in clinical populations and this needs to be tested directly.

AUTHOR CONTRIBUTIONS

JJMV, JRO, and AMG conceptualised the original idea and constructed the methodology. JJMV, FGJ, and JRO participated in data collection. JJMV, RMK, and JRO performed statistical analyses. JJMV and JRO wrote the original manuscript in consultation with RMK, AMG, and FGJ. All authors have read and agreed to the final version of the manuscript.

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CONFLICT OF INTEREST

Alice Gregory is an advisor for a project initially sponsored by Johnson's Baby. She is a consultant for Perrigo (2021+). She receives royalties for two books *Nodding Off* (Bloomsbury Sigma, 2018) and *The Sleepy Pebble* (Flying Eye, 2019). She has another contract with Lawrence King Publishers (publication due 2022). She is a regular contributor to *BBC Focus* magazine and has contributed to other outlets (such as *The Conversation*, *The Guardian* and *Balance Magazine*). She occasionally receives sample products related to sleep (e.g. blue light blocking glasses) and has given a paid talk to a business. She is a specialist subject editor at *JCPP* (sleep) for which she receives a small honorarium. She has contributed a paid article to *Neurodiem*. The other authors have nothing to declare.

DATA AVAILABILITY STATEMENT

Data could be available under restrictions.

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