Dysfunctional Beliefs about Sleep and Insomnia Symptoms in Early Adulthood: A Twin and Sibling Study

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AMG – Alice Gregory is an advisor for a project sponsored by Johnson's Baby. She has written a book Nodding Off (Bloomsbury Sigma, 2018) and has a contract for a second book Sleepy Pebble (Nobrow). 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.

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All three authors (Melanie N. Schneider, Yulia Kovas and Alice M. Gregory) have contributed to this research paper by providing critical feedback shaping the analysis and the manuscript. M.N.S. helped design the study, ran the analyses and wrote the paper with advice from the other authors. A.M.G designed the study, gave advice on the analyses and the interpretation of the results and helped shape the manuscript. Y.K. gave advise on the analyses and the interpretation of the results and helped shape the manuscript.

Summary

This study examines the associations between dysfunctional beliefs about sleep (DBAS), its subtypes and insomnia symptoms and estimates the relative contribution of genetic and environmental influences on these variables and the associations between them. The data came from G1219, a twin/sibling study which comprises 862 individuals (aged 22-32 years, 34% male). The Insomnia Symptoms Questionnaire was used to measure insomnia symptoms and a 10-item version of the Dysfunctional Beliefs and Attitudes about Sleep Scale was used to assess DBAS. A higher DBAS score was associated with more insomnia symptoms. Overall DBAS showed mainly non-shared environmental influence (86%). The genetic correlation between DBAS and insomnia symptoms was large but not significant, the shared environmental correlation was very small, negative and not significant, while a moderate, significant overlap in the non-shared environmental influences was evident (non-shared environmental correlation = .32). For the association between the subscales of DBAS and insomnia symptoms no significant overlap for genetic (weak to strong associations) or shared environmental factors (very weak negative to strong associations) were indicated. Most of the non-shared environmental influences on the four variables were significantly, moderately correlated (non-shared environmental correlation = .24-.46). These findings help to deepen our understanding of cognitive theories of insomnia by dissecting one of its crucial elements and illuminating the factors involved in its association with insomnia symptoms.

Keywords: insomnia; DBAS; genetic influence; environmental influence; sleep; twins

Introduction

The concept of dysfunctional beliefs about sleep

Dysfunctional beliefs about sleep play a crucial role in cognitive theories of insomnia (Espie, Inglis, Harvey, & Tessier, 2000; Harvey, 2002; Morin, Blais, & Savard, 2002; Ong, Ulmer, & Manber, 2012). For example, the microanalytic model considers how insomnia is maintained as a vicious cycle of sleep disruption, feeding into arousal, dysfunctional beliefs about sleep and maladaptive behaviours – all of which again contribute to a sleep disturbance (Morin, 1993; Ellis, Gehrman, Espie, Riemann, & Perlis, 2012). Dysfunctional beliefs about sleep can be described as intrusive thoughts, excessive expectations or mistaken beliefs about sleep. For example, it would be dysfunctional to believe that we need 8 hours of sleep *every* night to be able to function during the day (Harvey, 2002; Morin, Blais, & Savard, 2002).

The association between dysfunctional beliefs about sleep and insomnia

Previous research has confirmed that dysfunctional beliefs about sleep and insomnia symptoms are associated. For example, when considering factors involved in developing and maintaining insomnia in a clinical sample and a healthy control group, insomnia was best predicted by dysfunctional beliefs about sleep and sleep quality (Palagini et al., 2015). In a long-term, follow up study (over 6 years) dysfunctional beliefs about sleep (as well as stress-related sleep vulnerability) were found to be strong predictors of insomnia (Yang, Hung, & Lee, 2014).

The crucial role that dysfunctional beliefs about sleep play in insomnia is highlighted by research that considers the treatment of insomnia. A meta-analysis of randomised controlled studies has shown that cognitive-behavioural therapy is effective in treating insomnia (Okajima, Komada, & Inoue, 2011). One of the central aims of CBT-I (cognitive-behavioural therapy for insomnia) is to correct dysfunctional beliefs about sleep (Sivertsen, Vedaa, & Nordgreen, 2013). In a recent study, using a randomised controlled trial, the authors compared cognitive therapy, behaviour therapy and cognitive-behavioural therapy as

treatment for insomnia (Eidelman et al., 2016). Regardless of which therapy method was applied, it was found that the greater the change in dysfunctional beliefs about sleep during treatment, the greater the improvement in insomnia symptoms (Eidelman et al., 2016). Even though research has shown that dysfunctional beliefs about sleep and insomnia are linked, the mechanisms underlying this association need to be further established.

Aspects of dysfunctional beliefs about sleep

To enable a detailed insight into the concept of dysfunctional beliefs about sleep, in addition to focusing on a global score for dysfunctional beliefs, the current study will focus on three established subscales (DBAS I: *beliefs about immediate consequences;* DBAS II: *beliefs about long-term consequences* and DBAS III: *beliefs about control*) (Espie et al., 2000; Morin et al., 1993). The use of the subscales has previously been found to be a fruitful line of enquiry. For example, one study (focusing only on the first two subscales) compared 'good sleepers', 'normal sleepers' and 'poor sleepers' with participants meeting the diagnostic criteria for insomnia in terms of different aspects of dysfunctional beliefs about sleep. It was found that all four groups differed significantly in their *beliefs about long-term consequences* (Jansson-Fröjmark, Lundquist, Lundquist, & Linton, 2008). The third subscale (*beliefs about the need for control over insomnia)* and the overall score were not included in this previous study as a shorted version of the DBAS-10 questionnaire was used (Jansson-Fröjmark et al., 2008).

Since there is limited research on the different aspects of dysfunctional beliefs about sleep, it is important to further examine their association with insomnia symptoms. This knowledge could potentially be used to improve treatment for insomnia in the future – such as indicating whether certain aspects of dysfunctional beliefs about sleep might require particular attention within the clinical setting.

Heritability of dysfunctional beliefs about sleep

In spite of the extensive research interest in dysfunctional beliefs about sleep, no research has explored genetic and environmental influences on individual differences for this variable. Dysfunctional beliefs about sleep play a crucial role in current theories and the treatment of insomnia (see, for example, Eidelman et al., 2016; Harvey, 2002; Ong et al., 2012) and the link between dysfunctional beliefs about sleep and insomnia symptoms is well established (see, for example, Morin et al., 2002; Palagini et al., 2015). Nonetheless, the role that genetic and environmental influences play in this association has yet to be explored. We expect the subscales to differ in their association with insomnia symptoms and, similarly, it is possible that there are differences in the aetiology of the overlap between the variables.

Aims of the current study

In summary, dysfunctional beliefs about sleep are an important element in theories of insomnia. They have been found to be associated with insomnia and are targeted in treatment. However, we need to know more about the aetiology of dysfunctional beliefs (and its subscales) and associations with insomnia symptoms, as no previous study has considered this research area from a behavioural genetics perspective. If a high genetic overlap between dysfunctional beliefs about sleep and insomnia symptoms is found, this may indicate that this symptom cluster may also be part of the same genetic cluster (as for sleep problems and depression disorders, as indicated in previous research findings for example) (Middeldorp, Cath, Van Dyck, & Boomsma, 2005). This may further suggest, that those experiencing dysfunctional beliefs about sleep may also be at increased genetic vulnerability for insomnia symptoms. If a high overlap for environmental influences is found instead, then this would encourage us to further investigate which environmental factors may be affecting these variables. Such information helps us get to the root of insomnia.

The aims of this study were to:

1) Examine the magnitude of the association between overall dysfunctional beliefs about

sleep, different aspects of dysfunctional beliefs about sleep and insomnia symptoms.

2) Estimate the relative contribution of genetic and environmental influences on a) overall dysfunctional beliefs about sleep, b) *beliefs about immediate consequences,* c) *beliefs about long-term consequences*, d) *beliefs about control* and e) insomnia symptoms.
3) Explore the relative contribution of genetic and environmental influences on the association between dysfunctional beliefs about sleep (as well as its subscales) and insomnia symptoms.

Method

Sample

Data from Wave 5 of the G1219 longitudinal twin/sibling study was the focus of this study as this is the only wave at which dysfunctional beliefs about sleep have been measured. Wave 5 included 862 individuals in total. After excluding 17 outliers, data from 223 monozygotic (MZ) twins, 404 dizygotic (DZ) twins and 218 siblings remained for the analyses (Denis et al., 2015). The participants were aged between 22 and 32 years (mean age 25 years) and 34% of them were male (Denis et al., 2015).

Dysfunctional beliefs about sleep

In the present study the DBAS-10 was utilised. It is a shortened version of the dysfunctional beliefs and attitudes about sleep scale (DBAS; Morin et al., 1993), which comprises 10 items and three subscales (Espie et al., 2000). Each item was coded from 1 ('strongly disagree') to 10 ('strongly agree'), based on the participants' responses. For the subscale *beliefs about immediate consequences* (DBAS factor I), items 1 to 5 were added, giving a theoretical range from 5 to 50. For *beliefs about long-term consequences* (DBAS factor II), items 6 to 8 were added, resulting in a theoretical range from 3 to 30. For *beliefs about control* (DBAS factor III), items 9 and 10 were added, therefore the theoretical range

was from 2 to 20. DBAS I (*beliefs about immediate consequences*) includes the item "I need 8 hours of sleep to feel refreshed and function well during the day." DBAS II (*beliefs about long-term consequences*) includes the item "I am concerned that chronic insomnia may have serious consequences on my physical health.". DBAS III (*beliefs about control*) includes the item "When I have trouble falling asleep or getting back to sleep after night-time awakening, I should stay in bed and try harder." (Espie et al., 2000; Morin et al., 1993). The total scale score is the sum of all responses, with higher scores indicating more dysfunctional beliefs about sleep. In the current sample, the Cronbach's alpha for the overall DBAS was .78, DBAS factor I was .78, and DBAS factor II was .69. In line with the approach taken by Espie et al., 2000, the Cronbach's alpha for the DBAS factor III was not calculated for the current sample, because it only consisted of two items.

Insomnia symptoms

A 6-item version of the Insomnia Symptoms Questionnaire was utilised to measure insomnia symptoms (ISQ, Okun et al., 2009). The first five items are identical to the first five items in the published version of the ISQ. The sixth question is a single item that captures the distress/impairment criterion of insomnia diagnosis and was used in place of the eight individual distress/impairment items in the original ISQ. Each item of the ISQ was coded 0 - 4 based on frequency response (never/ don't know = 0; rarely = 1; sometimes = 2; frequently = 3; always = 4). The total scale score was calculated by summing the responses and ranges from 0 to 24, with higher scores meaning more severe insomnia symptoms. Cronbach's alpha for the ISQ in the current sample was .87 (as reported in previous studies - Gregory et al., 2016; Schneider et al., 2017).

Analyses

Data preparation

For all variables, outliers more than +/- 3 standard deviations away from the mean

were excluded from the sample (in total 17 cases) – as has been done in previous studies (see, for example, Gregory et al., 2016). None of the variables required transformation (skewness ranged from -.02, std. error = .08 for the overall DBAS to .97, std. error = .08 for DBAS factor II; kurtosis ranged from -.70, std. error = .17 for DBAS factor III to .33, std. error = .17 for DBAS factor III). All variables were age and sex regressed which is standard in genetic model fitting (Gregory et al., 2011, Bolhuis et al., 2014). For the phenotypic analyses, data from one randomly selected twin/sibling from each pair was used, to control for the non-independence of observations. For Wave 5 of the data collection, ethical approval was gained from Goldsmiths College, University of London. Written consent was given by all participants.

Twin and sibling analyses

The twin method is based on the assumption that dizygotic twins share on average 50% of their segregating genes, while monozygotic twins share 100% of their genes (Plomin, DeFries, Knopik, & Neiderhiser, 2013). Assuming that the environment is equally similar for both types of twins, this allows us to calculate the relative contribution of genetic (A, additive genetic), shared environmental (C) and non-shared environmental (E) influences (Neale & Cardon, 2013). Additive genetic influences are the genetic effects that 'add-up' to influence a phenotype. Shared environmental influences are those environmental factors which make members of the same family more similar to one another, in contrast to non-shared environmental influences which are the environmental influences that make members of the same family different. Note that E also includes measurement error (Knopik, Neiderhiser, DeFries & Plomin, 2016).

Sibling pairs were included to increase power in the sample. Comparing MZ twin correlations with DZ/sibling correlations (within traits – e.g. just for insomnia symptoms) gives us a rough idea of the extent to which genes influence a trait. Considering the MZ and DZ/ sibling cross-twin-cross-trait correlations (e.g. the correlation between insomnia

symptom in twin 1 and dysfunctional beliefs about sleep in twin 2) gives us an idea of possible genetic overlap between traits (for a more detailed explanation, see Plomin et al., 2013 and Rijsdijk & Sham, 2002).

A univariate analysis was run for each variable (using OpenMX version 1, R version 3.0.3; Boker et al., 2011), applying maximum-likelihood model fitting analysis to estimate the relative contribution of A, C and E (Neale & Cardon, 2013).

The program R (with a package for genetic model fitting, called OpenMX) was utilised for the twin analyses, using maximum likelihood estimation to compare model fits (Boker et al. 2011).

As we obtained fewer participants than originally expected, novel power calculations were run after having collected all data/ run analyses in order to consider how to best interpret the estimates provided by the twin models. The results indicated that we had limited power (under .8 in all analyses) to detect significant heritability estimates of the magnitudes reported in the univariate analyses. Therefore, we decided to present the full (ACE) models to provide maximum information as power was limited due to our relatively small sample size. However, by comparing the ACE to the E model it was possible to consider if the variables showed familial influence. Familial influence (influence coming from A or C) is indicated if the E model fits significantly worse than the ACE (Schneider et al., 2018). We decided to take this approach instead of presenting, for example, the CE model or the AE model, as we had limited power to determine with certainty if the familial influence was coming from A or C. Being aware of our limited power is also the reason why we do not focus purely on the significance levels reported but also focus on the magnitude of effects when discussing our results (Schneider et al., 2018).

A bivariate analysis was run to examine the relationship between the genetic factors that affect overall dysfunctional beliefs about sleep and insomnia symptoms, as well as the relationship between environmental factors influencing these variables. A multivariate

correlated factors solution was used to explore this association in more detail by examining the genetic and environmental relationship between the three subscales of dysfunctional beliefs about sleep and insomnia symptoms. The correlated factors solution is based on the assumption that each trait has unique genetic, shared environmental, and non-shared environmental influences, and that these variable-specific influences may be correlated with the genetic, shared environmental, and non-shared environmental influences of other variables in the model (Loehlin, 1996; Schneider et al., 2018). Full models estimate additive genetic correlations (rA), shared environmental correlations (rC), and non-shared environmental correlations (rE).

Sensitivity analyses were performed whereby all analyses were re-run on raw data (without deleting outliers or regressing out age and sex), in order to examine whether or not different results were obtained. The sensitivity analyses showed similar results, except for *beliefs about immediate consequences*, which is discussed in the limitation section). The results of the sensitivity analyses are presented as an **Online Supplement**.

Results

Descriptive statistics

Descriptive statistics for each variable are summarised in **Table 1**. For overall dysfunctional beliefs about sleep, significant sex differences were found ($t(850) = -4.04$, $p < .01$, $d = .29$), with males reporting fewer dysfunctional beliefs about sleep than females. There was also a significant difference between males and females in terms of the *beliefs about immediate consequences* (DBAS factor I; $t(850) = -6.20$, $p < .01$, $d = .45$), with males showing on average lower scores than females. For insomnia symptoms, males also reported fewer insomnia symptoms than females ($t(625) = -3.28$, $p = .01$, $d = .25$).

Phenotypic analysis

The phenotypic correlations are displayed in **Table 2.** Higher overall dysfunctional beliefs about sleep were associated with more insomnia symptoms ($r = .37$, $p < .01$). Higher scores in *beliefs about immediate consequences* (DBAS factor I), *beliefs about long-term consequences* (DBAS factor II) and *beliefs about control* (DBAS factor III) were also associated with more insomnia symptoms (DBAS factor I: $r = .18$, $p < .01$; DBAS factor II: $r = .44$, $p < .01$; DBAS factor III: $r = .34$, $p < .01$).

MZ, DZ and sibling correlations

The MZ, DZ and sibling within-trait and cross-trait-cross-twin correlations for all variables are presented in **Table 3**. As the MZ correlations are substantially less than 1 for all of the traits, the importance of non-shared environmental influence is highlighted.

Twin/sibling analysis

Univariate analyses were run on all variables; the fit statistics, the results of the full ACE models and the estimates of A, C and E with 95% confidence intervals are shown in **Table 4**. Overall dysfunctional beliefs about sleep, *beliefs about long-term consequences* (DBAS factor II) and *beliefs about control* (DBAS factor III) showed small genetic influences which were non-significant (overall DBAS: A = .09, 95% CI = 0 - .31; DBAS factor II: A = 0, 95% CI = 0 - .32; DBAS factor III: A = .17, 95% CI = 0 - .32), shared environmental influences were also small or 0 and non-significant (overall DBAS: C = .05, 95% CI = 0 - .22; DBAS factor II: C = .13, 95% CI = 0 - .24; DBAS factor III: C = 0, 95% CI = 0 - .21). Non-shared environmental influences were large and significant (overall DBAS: E = .86, 95% CI = .69 - .99; DBAS factor II: E = .87, 95% CI = .68 - .99; DBAS factor III: E = .83, 95% CI = .68 - .99). For the *beliefs about immediate consequences* (DBAS factor I) small and significant genetic influence was indicated (A = .19; 95% CI = .01 - .38), no shared environmental influence was evident (C = 0, 95% CI = 0 - .22) and non-shared environment was large and significant (E = .81, 95% CI = .65 -.98). When the ACE model and the E model

were compared for these four variables, the fit did not decline significantly in any of the cases (overall DBAS: $\chi^2 = 6953.24$, df = 839, $p = .12$, AIC = 5275.92; DBAS factor I: $\chi^2 = 6106.98$, df = 839, $p = .07$, AIC = 4428.98; DBAS factor II: $\chi^2 = 5314.09$, df = 832, $p = .11$, AIC = 3650.09; DBAS factor III: $\chi^2 = 4882.15$, df = 839, $p = .11$, AIC = 3204.15), indicating no familial influence, confirming the aforementioned results. For insomnia symptoms a moderate (but non-significant) genetic influence was indicated (A = .36; 95% CI = 0 - .53), only a very small shared environmental influence was evident (C = .03, 95% CI = 0 - .32) and non-shared environment was large and significant (E = .61, 95% CI = .47 -.80). Non-shared environment appeared to be most important and familiality was found, as indicated by a decline in fit for the E model ($\chi^2 = 5135.58$, df = 837, $p < .01$, AIC = 3461.58).

Fit statistics for the bivariate and the multivariate analyses are presented in **Table 5**. The results of the bivariate analyses including overall dysfunctional beliefs about sleep and insomnia symptoms are shown in **Figure 1**. The genetic and shared environmental correlation between dysfunctional beliefs about sleep and insomnia symptoms were not significant (*r*A = .74, 95% CI = -1 - 1; *r*C = -.17, 95% CI = -1 - 1), and there was a moderate yet significant overlap in the non-shared environmental influences (*r*E = .32, 95% CI = .17 - .47) for the two traits.

The results of the multivariate analyses including *beliefs about immediate consequences* (DBAS factor I), *beliefs about long-term consequences* (DBAS factor II), *beliefs about control* (DBAS factor III) and insomnia symptoms are displayed in **Figure 2**. No significant overlap for genetic or shared environmental factors was indicated, see **Figure 2a and 2b**. As **Figure 2c** shows, the non-shared environmental influence between the subscales of the DBAS and insomnia symptoms were all significant and, moderately correlated (*r*E ranging from .24 to .46), except for *beliefs about immediate consequences* (DBAS factor I) and insomnia symptoms, for which the non-shared environmental correlation did not reach significance in this model (*r*E = .17, 95% CI = 0 - .33).

Discussion

Associations between variables

Dysfunctional beliefs about sleep and its subscales were all associated with insomnia symptoms. However, the association between the subscale *beliefs about immediate consequences* and insomnia symptoms was only weak, but significant. If these results are widely replicated, this might help to enhance treatment methods for insomnia in the future. Our findings are consistent with the idea that focusing on *beliefs about long-term consequences* and *beliefs about control* of the patient may be particularly effective for treating insomnia symptoms, due to the stronger associations of these two subscales (DBAS factor II and DBAS factor III) with insomnia symptoms as compared to *beliefs about immediate consequences*. Nonetheless, our results did not provide information about the direction of effects between variables, so future work would need to establish directly whether treatment focusing on this subscale is indeed useful in reducing symptoms of insomnia. Overall these associations are consistent with current theories which consider dysfunctional beliefs about sleep to be a central element in the development and maintenance of insomnia. The findings could contribute towards future extensions of the current models by adding detail about the specific types of dysfunctional beliefs about sleep likely to be most strongly associated with insomnia (see, for example, Harvey, 2002; Ong et al., 2012).

Factors influencing dysfunctional beliefs about sleep

When comparing the MZ correlations to the DZ and the sibling correlations, for overall DBAS and the subscales of the DBAS, they did not differ largely (95% confidence intervals overlapped), hinting at the possibility that there is little or no genetic influence on these variables. For insomnia symptoms, the difference between the MZ and DZ correlation was larger (although not significantly, as indicated by overlapping 95% confidence intervals).

The twin analyses revealed that overall dysfunctional beliefs about sleep showed neither a significant genetic influence (except for *beliefs about immediate consequences*) nor shared environmental influence but was mainly influenced by the non-shared environment (including error). The results held up in the sensitivity analysis for all variables except for *beliefs about immediate consequences* (see **Online Supplement**). When re-running the analysis on the raw data (outliers still included, age and sex not yet regressed out), the genetic influence was not significant (95% CI spanning zero) but when A and C were dropped at the same time, the fit got worse, suggesting familiality. This inconsistency should be interpreted with caution. Considering the results presented in the main body of the paper together with the results of the sensitivity analysis for the *beliefs about immediate consequences*, it seems likely that if familial influence had played a role, this was not a robust finding in our sample. No familial influence was indicated for overall dysfunctional beliefs about sleep, *beliefs about long-term consequences* and *beliefs about control.* However, our results need to be interpreted with caution as decades of twin research reveals that most traits have some genetic influence (Polderman et al., 2015) and our sample was very small. Even though results should be treated with caution, they do give us an initial insight into the roots of dysfunctional beliefs about sleep, showing that environmental factors that make family members dissimilar might be key in explaining why one person may be more prone than another to beliefs such as "I need 8 hours of sleep to feel refreshed and function well during the day" than the other.

The estimate for genetic influence on insomnia symptoms was .36 but did not reach significance (95% confidence interval = 0 - .53), which may again reflect the small sample size. The estimate itself was in line with previous findings as the heritability of insomnia-related measures in adults typically falls into a range between .25 and .45 (Gehrman et al., 2011; with some exceptions, e.g. Wing et al., 2012). Furthermore, familiality was evident for this variable (influence was shown to come from A and/or C).

Factors influencing the association between overall dysfunctional beliefs about sleep and insomnia symptoms

The results of the correlated factor solution showed that neither the correlations between the genetic influences (which were moderate to high) nor the correlations between the shared environmental influences (which varied greatly from high negative to high positive) were significant in the multivariate model. This fits with the finding of no genetic or shared environmental influence either in the univariate analyses for overall dysfunctional beliefs about sleep and its subscales (except for *beliefs about immediate consequences*) and for these separate variables in the multivariate. However, the non-significant findings for genetic and shared-environmental correlations may again be related to the small sample size. All non-shared environmental correlations were significant, except for *beliefs about immediate consequences* and insomnia symptoms which was also the association with the lowest correlation in the phenotypic analysis. These results held up in the sensitivity analysis (except for *beliefs about immediate consequences* which showed a significant non-shared correlation with insomnia symptoms here, see **Online Supplement**). It can therefore be concluded that for the association of the DBAS subscales and insomnia symptoms, the non-shared environmental influences overlapped to some extent. The findings add to previous literature showing that dysfunctional beliefs about sleep are a useful area to target in the treatment of insomnia. As outlined above, certain areas of dysfunctional beliefs may be particularly useful to address (*beliefs about long-term consequences* and *beliefs about control*) (see, for example, Okajima et al., 2011; Eidelman et al., 2016) – although, this needs to be directly tested in future research. The findings help us to better understand the mechanisms underlying the link between dysfunctional beliefs about sleep (and its subscales) and insomnia symptoms, indicating that non-shared environmental influences are key for explaining this well established association that has already been outlined in various theories of insomnia (see, for example, Harvey, 2002). In contrast, our findings do not suggest that dysfunctional beliefs about sleep and insomnia symptoms are part of the same genetic cluster

(as would be indicated if a high, significant genetic correlation was found). Instead, our results indicate that non-shared environmental influences may be key in explaining the association between dysfunctional beliefs about sleep and insomnia symptoms.

Future directions

The current findings indicate where to focus research efforts aimed at elucidating the aetiology of dysfunctional beliefs about sleep. This has the potential to increase insight into the development and maintenance of insomnia, as dysfunctional beliefs about sleep play a crucial role in the development of this disorder (Morin, 1993). Since non-shared environment was the key influence for dysfunctional beliefs about sleep and was important in explaining associations with insomnia symptoms, we suggest that future research should attempt to specify these influences. Research that attempts to specify environmental factors influencing dysfunctional beliefs is limited. However, previous literature flags up certain environmental factors which are associated with insomnia or sleep quality in general and which may be good candidates to consider, given the associations between insomnia/ sleep quality and dysfunctional beliefs about sleep (see, for example, Carney et al., 2010; Hiller, Johnston, Dohnt, Lovato, & Gradisar, 2015; Palagini et al., 2015; Yang et al., 2014).

For example, negative life events have been found to be related to insomnia and sleep quality (Barclay, Eley, Rijsdijk, & Gregory, 2011; Bernert, Merrill, Braithwaite, Van Orden, & Joiner, 2007; Vahtera et al., 2007) – and may be a good candidate to investigate in relation to dysfunctional beliefs about sleep.

An MZ differences design can be used to help specify non-shared environmental influences on variables. This is based on the idea that we know that MZ twins share 100% of their genes, as well as 100% of their shared environment (Plomin et al., 2013). Therefore, any discrepancy between MZ twins must come from non-shared environmental influence (or measurement error). This design allows for the identification of candidate non-shared environmental factors, un-confounded by genetic factors (Barclay et al., 2013; Vitaro,

Brendgen, & Arseneault, 2009). In the current sample we had limited power to use this approach but we suggest this as an avenue for further research in larger-scale studies assessing these variables.

Limitations

The twin design has some limitations, which are discussed elsewhere (Knopik et al., 2016). For example, whilst the results from twin studies are used to draw conclusions about individual differences in the general population, it is possible that twins may not be representative of the wider non-twin population (Knopik et al., 2016).

A further limitation relates to the sample size. This was a small sample for a twin study which meant that some of the confidence intervals were wide and slight inconsistencies occurred in the sensitivity analysis (see above). Contrary to our expectations the estimates of genetic influence did not reach significance in any of the univariate analyses, except for DBAS factor I (*Beliefs about immediate consequences*). The estimate for genetic influence on insomnia symptoms (A = .36) is in line with previous findings (see, for example, Gehrman et al., 2011), even though significance was not reached. No previous twin study exists to compare the estimates of genetic influence on overall DBAS and its subscales to. Therefore, the current results should be interpreted with caution and further work using larger samples would be of value.

Another limitation relates to the use of self-report measures which may have artificially inflated the associations. This was necessary given the scope of the study (i.e. assessing numerous variables in a sample of many hundreds of participants) and is also considered to be the optimal approach to assessing certain phenotypes (for example, insomnia symptoms) (Schneider et al., 2018). Nonetheless, future work should try to incorporate additional information (for example, symptoms rated by other reporters and objective measures of sleep).

Finally, it should be noted that we have utilized a shortened version of the ISQ in the current study. This version has been used in previous studies and has a Cronbach's alpha of was .87 (see Gregory et al., 2016; Schneider et al., 2018). Future research should look to further establish the reliability/validity of this particular version of the measure.

Conclusion

The current findings give us a novel insight into the concept of dysfunctional beliefs about sleep, its subscales and the association between dysfunctional beliefs about sleep and insomnia symptoms. This helps to deepen our understanding of the cognitive theories of insomnia by dissecting one of its crucial elements and illuminating the factors involved in its association with insomnia symptoms. A more detailed understanding of insomnia holds the promise of improving treatment in the future. These results now need to be validated in a larger sample. The results presented here raise new questions, such as which environmental factors are involved in the development of dysfunctional beliefs about sleep.

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