

Relationship between Insomnia and Quality of Life: Mediating Effects of Psychological and Somatic Symptomatology

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Abstract

Objectives: We aimed to explore the potential mediating effects of neuroticism, depressive and anxiety symptoms, and somatic symptoms in the relationship between insomnia and health-related quality of life (HRQoL). **Methods:** This was a cross-sectional family study, which recruited a total of 297 adolescents (eighty insomniacs as determined by clinical interview) and 318 parents (93 insomniacs). HRQoL was measured by short form-36 (SF-36) health survey. A series of questionnaires were employed to measure insomnia severity, neuroticism personality, and depressive, anxiety and somatic symptoms. **Results:** Participants with insomnia had lower HRQoL than those without insomnia (71.9 vs. 77.1, $P < 0.001$). Insomnia severity (as measured by Insomnia Severity Index) was significantly associated with HRQoL (correlation coefficient = -0.451 , $P < 0.001$). A total of 53% of the variance of SF-36 could be explained by the mediation model, which showed that a large proportion of the variances in the association between insomnia severity and HRQoL was mediated by depressive symptoms, somatic symptoms, and neuroticism personality trait. **Conclusions:** The close associations between insomnia severity and impaired HRQoL are largely mediated by psychological symptomatology and personality dimension. Further prospective study is warranted to investigate the long-term impact of insomnia symptoms on HRQoL and the roles of mood and somatic symptoms.

Keywords: Insomnia, mediating effect, psychological symptomatology, quality of life, somatic symptomatology

INTRODUCTION

Insomnia is the most common sleep disorder in the general population with a series of deleterious consequences on mental and physical health. Health-related quality of life (HRQoL), an individual's perceived physical and mental health, is considered as an important indicator in the assessment of disease burden and functional impairment as well as the evaluation of the efficacy of an intervention.^[1] It has been shown that insomnia exerts negative impacts on various domains of HRQoL in different populations and that the improvement of insomnia by intervention improves HRQoL accordingly.^[2]

On the other hand, insomnia is also closely associated with a constellation of mental and somatic problems as well as personality dimension (including depression,^[3] anxiety,^[4] somatic symptoms,^[5] and neuroticism^[6]), which also have significant impacts on HRQoL and functioning.^[7-9] Although previous studies have demonstrated close associations between insomnia and impaired HRQoL,^[2] the roles of mood and

somatic symptoms on the relationship between insomnia and impaired HRQoL have been rarely investigated in the existing literature. A previous study has shown that insomnia is associated with both increased psychological distress and poor HRQoL in the univariate analyses, but insomnia exerts its impact on only one aspect (vitality subscale) but not the other components of the HRQoL measure (short form-12 [SF-12] health survey) in the full adjusted model.^[10] Although this study implies that the relationship between insomnia and HRQoL might be confounded by other psychological factors, it fails to demonstrate how these psychological factors and which factors mediate the relationship.^[10] In this regard, further investigation on the roles of mood and somatic symptoms in

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the close relationship between insomnia and HRQoL should be warranted.

In the current study, we aimed to determine to what extent the psychological factors, including depressive, anxiety and somatic symptoms, and neuroticism, could account for the association between insomnia severity and impaired HRQoL.

METHODS

Study procedure and participant selection

The current study was part of a longitudinal epidemiologic study on sleep problems among Hong Kong Chinese school children and their parents, which was started in 2003–2004 (baseline).^[11] The current report was based on the follow-up study (wave 2), which was conducted with a two-phase design to investigate the familial aggregation of insomnia and other mental distresses during 2008–2010.^[12,13] Phase 1 of the study involved a questionnaire assessment of sleep problems among each member of the families, which was initially recruited through local primary schools. Phase 2 was an in-depth family study that consisted of face-to-face structured diagnostic interviews to ascertain both insomnia disorder and mental disorders, and a series of psychometric assessments on sleep quality, mood and somatic symptoms, quality of life, and personality trait.^[14] The protocol of this study was approved by the Institutional Ethics Review Committee. All participants under 18-year-old gave their written assents and parental consents. All the participants aged 18 or above gave their written consents by themselves. Details of the participant recruitment have been described elsewhere.^[15] In brief, all adolescents with insomniac complaints (classified as “high risk” participants) including difficulty in initiating sleep (DIS), difficulty in maintaining sleep (DMS), and/or early morning awakening (EMA) for at least three times a week and/or usual sleep onset latency ≥ 30 min in the past 12 months as reported in the Phase 1 questionnaire study were invited to attend the Phase 2 in-depth study. Meanwhile, a group of adolescents without any insomnia complaint (classified as “low risk” participants) were also invited for further clinical interview in the Phase 2 study. In addition, biological parents and siblings aged over 6-year-old of the index adolescent participants were also invited to attend the Phase 2 study.

The Diagnostic and Statistical Manual of Mental Disorders, the fourth edition text revision criteria for insomnia disorder were employed to determine the diagnosis of insomnia.^[16] These criteria included (1) a predominant complaint of DIS, DMS, or EMA for at least 1 month; (2) causing significant distress or impairment in social, occupational (or academic in adolescents), and/or other important areas of functioning. In light of recent evidence suggesting that nonrestorative sleep (NRS), another subtype of insomnia,^[16] may have overlapping associations with other sleep disorders,^[17,18] and could have a longitudinal course independent of other subtypes of insomnia,^[12] we believed that NRS might be

better considered as a distinct sleep problem, and hence did not include it in the current study.^[19]

A total of 285 adolescents were contacted, and 236 of them finally participated with a response rate of 82.8%. Forty-one out of 95 adolescents with high-risk of insomnia disorder and 34 out of 141 adolescents with low-risk of insomnia disorder according to the questionnaire were finally diagnosed as insomnia disorder. In addition, a total of 224 mothers, 196 fathers, and 142 full siblings were recruited. As some key measures, such as personality measure, were added after having recruited some families, the current study only focused on those individuals with valid data on all measures. The current study included 297 adolescents (78.6%) and 318 middle-aged parents (75.7%) who completed the assessment of HRQoL. No age or sex difference was found between those participants who completed the HRQoL assessment and those who did not complete the HRQoL assessment.

Measure of health-related quality of life

SF-36 health survey was employed to measure the quality of life. SF-36 is a well-established generic instrument for measuring the HRQoL.^[20] The Chinese (Hong Kong) version of SF-36 health survey was found to be a reliable and valid measure of the quality of life in Chinese.^[21] A total of eight domains are measured by the SF-36, which include physical functioning, role limitations due to physical problems, bodily pain, general perception of health, vitality, social functioning, role limitations due to emotional problems, and mental health. The scores in the SF-36 range from 0 to 100, and higher scores indicate better HRQoL. The physical domains, including physical functioning, role limitations due to physical problems, physical problems, bodily pain, and self-rated health, constitute a Physical Component Scale (PCS). The mental domains, including vitality, social functioning, role limitations due to emotional problems, and mental health, constitute a mental component scale (MCS).

Measures of insomnia severity

The severity of insomnia symptoms was assessed by Insomnia Severity Index (ISI), which is a 7-item questionnaire assessing the subtype, severity, and impact of sleep difficulties in the past 2-week. ISI has shown to have satisfactory psychometric properties.^[22] Higher ISI scores indicate more severe insomnia symptoms.

Measures of somatic and pain symptoms

Somatic symptoms were measured by the 28-item Somatic Symptom Inventory (SSI-28), in which each somatic symptom was rated on a Likert scale from 1 (not at all) to 5 (a great deal), according to how much it bothered the participants over the past week.^[23]

Measures of depressive and anxiety symptoms

Depressive and anxiety symptoms were assessed by the Chinese version of Hospital Anxiety and Depression Scale (HADS), which has been validated locally in both adolescents and adults.^[24,25] The HADS is a 14-item self-report

questionnaire with two 7-item subscales for the measurement of anxiety and depressive symptoms. Each item is rated on a 4-point Likert scale from 0 to 3. The anxiety and depression subscores are summed up separately in the current study to estimate the severity of depressive symptoms and anxiety symptoms, respectively.

Statistical methods

Descriptive statistics are presented as percentages for discrete variables and as means (standard deviation) for continuous variables. To compare the independent differences in HRQoL between insomniac and noninsomniac participants, linear model in generalized estimating equations was employed to adjust for potential confounding effects of age and sex [Figure 1].

A bootstrapping technique developed by Preacher and Hayes was employed to examine the direct and indirect effects of insomnia severity/poor sleep quality (independent variable [IV]) on HRQoL outcomes (dependent variable [DV]) through neuroticism, depressive and anxiety symptoms, and somatic symptoms (mediators) after adjusting for age and sex (covariates).^[26] As suggested by the authors, a single mediation path for IV on DV is not likely to happen in medical and social sciences research;^[26] hence, several mediators (neuroticism, depressive and anxiety symptoms, and somatic symptoms) with close associations with both HRQoL outcomes and sleep measures were included in the model. A series of bootstrapped multiple mediation models were tested, which adopted ISI as IV, SF-36, SF-36 MCS, or SF-36 PCS respectively as DV, and HADS-anxiety, HADS-depression, SSI, and neuroticism as mediators. Bootstrapping provides the most powerful and reasonable method to obtain confidence interval (CI) for a specific indirect effect, even for variables with an abnormal distribution. We employed 20,000 bootstraps to generate all models. A bias-corrected and accelerated (BCa) bootstrapped 95% CI (BCa 95% CI) was also generated for each model to estimate the indirect effect of each path mediating the effect of IV on DV. Figure 2 shows the details about the paths for the total effects, indirect effects, and direct effects of IV on DV. Path c indicates the total effect of IV on DV. Path a (including a1, a2, a3, a4) indicates the effects of IV

on the mediators. Path b (including b1, b2, b3, b4) indicates the respective effect of different mediators on DV. The indirect effect ($a \times b$) of IV on DV through these paths was calculated as the sum of $a1 \times b1$, $a2 \times b2$, $a3 \times b3$, and $a4 \times b4$. Path c' is the direct effect of IV on DV, which is the difference between total effect and indirect effect.

$P < 0.05$ or 95% CI not including zero (for BCa 95% CI) was considered as a statistically significant level. SPSS 19.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for all tests.

RESULTS

Of the overall assessed participants with valid data on SF-36 ($n = 615$), 180 participants were classified as current insomniacs, and 454 of them were determined as noncurrent insomniac based on the clinical interview. Insomniacs are more likely to be females (60.6% vs. 49.8%, $P = 0.014$) when compared with noninsomniacs. There was no difference in age between insomniacs and noninsomniacs (31.7 ± 16.3 -year-old vs. 30.5 ± 16.4 -year-old, $P = 0.41$).

Associations of health-related quality of life with insomnia diagnosis and other psychometric measures

Figure 1 shows the differences in SF-36 total scores, SF-36 MCS, and SF-36 PCS between insomniacs and noninsomniacs after adjustment for age and sex. As compared to the noninsomniacs, insomniacs had lower scores in SF-36 total scores (71.9 vs. 77.1, $P < 0.05$), SF-36 MCS (70.6 vs. 75.5, $P < 0.05$), and SF-36 PCS (73.2 vs. 78.5, $P < 0.05$), which indicated that insomniacs had poorer HRQoL than noninsomniacs. Subgroup analyses also revealed that the impacts of insomnia disorder on SF-36 total scores were consistently found in adolescents and adults as well as both sexes (data not shown). Furthermore, correlation analyses revealed that SF-36 and its two major components were negatively associated with insomnia severity as measured by ISI (correlation coefficients = -0.348 – -0.454 ,

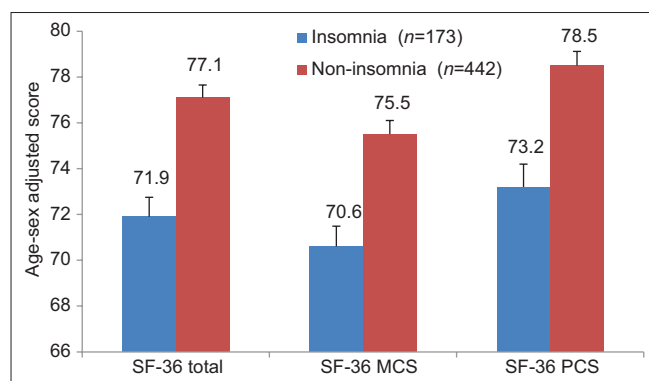


Figure 1: Differences in quality of life between participants with and without insomnia adjustment for age and sex.

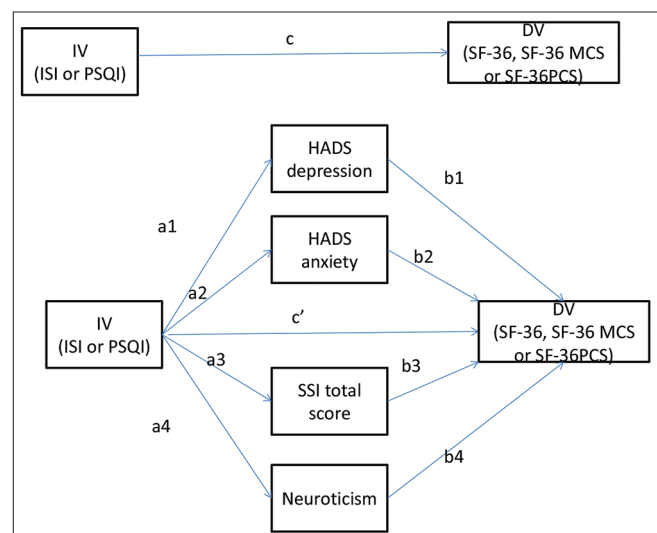


Figure 2: Graphical depiction of the mediation model.

$P < 0.001$) [Table 1]. In addition, HRQoL, as measured by SF-36, was variably associated with anxiety symptoms, depressive symptoms, somatic symptoms, and neuroticism personality (correlation coefficients = -0.374 to -0.594 , $P < 0.001$) [Table 1].

Mediation analyses

Table 2 shows the mediation analyses of the direct and indirect effects of ISI on SF-36 and its two major components through HADS anxiety score, HADS depression score, SSI, and neuroticism. A total of 53% of the variance of SF-36 could be explained by Model 1, which delineated the direct and indirect effects of ISI on SF-36. A large proportion of the total effect (path c) of ISI on SF-36 was accounted for by indirect effect (path a \times b) rather than direct effect (path c') (path coefficient = -0.75 ± 0.07 vs. -0.28 ± 0.08). The effect of ISI on SF-36 was significantly mediated by HADS depression (path coefficient = -0.18 ± 0.04 , $P < 0.05$), SSI total (path coefficient = -0.26 ± 0.05 , $P < 0.05$), and neuroticism (path coefficient = -0.26 ± 0.05 , $P < 0.05$) but not HADS anxiety (path coefficient = -0.05 ± 0.05 , $P > 0.05$), as indicated by the BCa bootstrapped 95% CI. For more details about the path coefficient in each path, please refer to Figure 3.

For the direct and indirect effects of ISI on SF-36 MCS (Model 2), a total of 51% of the variance of SF-36 MCS could be explained by this model. The path coefficient for the total effect of ISI on SF-36 MCS was -1.14 ± 0.10 ($P < 0.0001$), which was largely accounted for by the indirect effect (path coefficient = -0.77 ± 0.08 , $P < 0.05$). In this model, all four

variables significantly mediated the effects of ISI on SF-36 MCS. Among them, neuroticism was found to exert the largest effect (path coefficient = -0.29 ± 0.05 , $P < 0.05$), followed by HADS depression (path coefficient = -0.21 ± 0.05 , $P < 0.05$).

Model 3 in Table 2 summarizes the results for the direct and indirect effects of ISI on SF-36 PCS. The variance (38%) explained by this model was less than those explained by Models 1 and 3 (53% and 51%, respectively). The direct effect of ISI on SF-36 PCS was not statistically significant (path coefficient = -0.19 ± 0.11 , 0.0875), which indicated that the total effect of ISI on SF-36 PCS was mediated by other mediators. The indirect effect (path coefficient = -0.72 ± 0.08 , $P < 0.05$) of ISI on SF-36 PCS was mainly accounted for by SSI (path coefficient = -0.38 ± 0.07 , $P < 0.05$), followed by neuroticism (path coefficient = -0.22 ± 0.06 , $P < 0.05$), and HADS depression (path coefficient = -0.16 ± 0.06 , $P < 0.05$). However, HADS anxiety did not play a significant role in mediating the effect of ISI on SF-36 PCS.

DISCUSSION

In this community-based study, we confirmed that individuals with insomnia disorder have poorer HRQoL than those individuals without insomnia disorder. The HRQoL as measured by SF-36 was associated with insomnia severity, mood symptoms, neuroticism, and somatic symptoms. Mediation analyses further demonstrated that the close associations between insomnia severity and impaired HRQoL are both mediated directly through insomnia *per se* and more interestingly, indirectly through other psychological and somatic symptomatology. In particular, the indirect pathway via mood and somatic symptoms has accounted a more significant effect on the impaired HRQoL.

In keeping with the previous study,^[2] our study confirmed that we confirmed that individuals with insomnia disorder have poorer HRQoL than those individuals without insomnia disorder. In addition, this study further found that the impact of insomnia on HRQoL already occurs as early as adolescents. In a relatively older population, Dixon *et al.* have shown that the impact of insomnia on HRQoL was weaker in older patients, which may be mediated by the multiple medical comorbidity in aged group.^[27] However, in the current study with younger subjects, we did not found age differences in the impact of insomnia on HRQoL.

By taking mood and somatic symptoms into account in the mediation model, the results of the current study provides further understanding on the consequences of insomnia

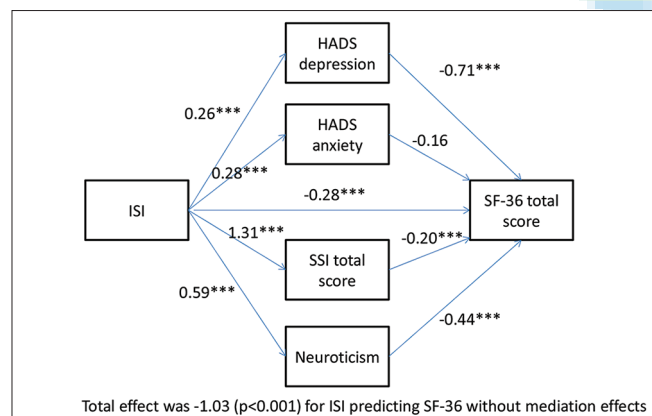


Figure 3: Graphical depiction of the mediation models for the relationships of Insomnia Severity Index and Short Form-36. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$; bootstrap 20000.

Table 1: Correlations between quality of life and other psychometric measures

	ISI	HADS anxiety	HADS depression	SSI total	Neuroticism
SF-36 total	-0.451***	-0.535***	-0.548***	-0.534***	-0.575***
SF-36 MCS	-0.454***	-0.580***	-0.570***	-0.460***	-0.594***
SF-36 PCS	-0.348***	-0.374***	-0.405***	-0.488***	-0.430***

*** $P < 0.001$. Partial correlations controlling for age and sex. ISI=Insomnia Severity Index, HADS=Hospital Anxiety Depression Scale, SSI=Somatic Symptoms Inventory, MCS=Mental Component Scale, PCS = Physical Component Scale, SF-36=Short Form-36

Table 2: Bias corrected and accelerated bootstrapped mediation of the effects of Insomnia Severity Index on quality of life through neuroticism, anxiety, depression, and somatic symptoms

Model	ISI			
	Path coefficient±SE	BCa 95% CI**		P
		Lower	Upper	
Model 1: Adjusted R²=0.53				
SF-36				
Total effect (c)	-1.03±0.09	-	-	<0.001
Direct effect (c')	-0.28±0.08	-	-	0.0008
Indirect effect (a × b)	-0.75±0.07	-0.90	-0.60	-
HADS depression	-0.18±0.04	-0.28	-0.11	-
HADS anxiety	-0.05±0.05	-0.13	0.04	-
SSI total	-0.26±0.05	-0.37	-0.16	-
Neuroticism	-0.26±0.05	-0.36	-0.17	-
Model 2: Adjusted R²=0.51				
Mental health component				
Total effect (c)	-1.14±0.10	-	-	<0.001
Direct effect (c')	-0.37±0.09	-	-	0.0001
Indirect effect (a × b)	-0.77±0.08	-0.93	-0.61	-
HADS depression	-0.21±0.05	-0.31	-0.12	-
HADS anxiety	-0.13±0.05	-0.23	-0.05	-
SSI total	-0.14±0.06	-0.25	-0.03	-
Neuroticism	-0.29±0.05	-0.40	-0.20	-
Model 3: Adjusted R²=0.38				
Physical health component				
Total effect (c)	-0.92±0.11	-	-	<0.001
Direct effect (c')	-0.19±0.11	-	-	0.0875
Indirect effect (a × b)	-0.72±0.08	-0.90	-0.57	-
HADS depression	-0.16±0.06	-0.28	-0.06	-
HADS anxiety	0.04±0.06	-0.07	0.16	-
SSI total	-0.38±0.07	-0.53	-0.26	-
Neuroticism	-0.22±0.06	-0.35	-0.13	-

All models adjusted for age and sex; ** $P<0.05$ for 95% CI does not include zero. ISI=Insomnia Severity Index, HADS=Hospital Anxiety Depression Scale, SSI=Somatic Symptoms Inventory, CI=Confidence interval, -=Not applicable, SE=Standard error, BCa=Bias-corrected and accelerated

symptoms and the potential roles of mood and somatic symptoms in the relationship between insomnia and quality of life. Insomnia has been shown to be closely associated with a number of mood and somatic symptoms.^[5] We have previously reported that chronic insomnia is associated with an increased risk of future chronic medical conditions and psychiatric disorders in both children and adults.^[13,28] Previous studies have also suggested that there is likely a bidirectional association between insomnia and depression^[29] as well as between insomnia and somatic symptoms.^[30] Our models suggested that insomnia severity is not only related to mental health component of HRQoL but also physical health component of HRQoL, despite some degree of attenuation. This finding suggested that insomnia affects both mental and physical health.^[28] In the recent decade, increasing studies have suggested that insomnia is a risk factor of both mental disorders and a number of chronic medical conditions, including

stroke,^[31] type 2 diabetes,^[32] and cardiovascular diseases.^[33] In this regard, effective management of insomnia may be potentially beneficial to individual's overall health condition, including both physical and mental aspects.

Furthermore, we found that neuroticism, a common personality construct that underlies both insomnia and depression,^[6] also partially mediates the impact of insomnia symptoms on HRQoL. It has been shown that neuroticism is associated with poor HRQoL in the general population^[34] and patients with chronic medical conditions, such as Parkinson's diseases^[35] and chronic heart failure.^[36] The reasons for the mediation effect of neuroticism in the association between insomnia symptoms and HRQoL remain unclear. It is unlikely the mediation effect of neuroticism is confounded by mood and somatic symptoms, which were included in the models.

Future direction

A series of cross-sectional studies have confirmed that insomnia is associated with poor quality of life. However, the long-term impact of insomnia on HRQoL is still largely unknown. In this regard, future longitudinal study is warranted to delineate the long-term influences of insomnia on HRQoL and the roles of mood and somatic symptoms in mediating this relationship. A few studies with small sample size have suggested that treatment of insomnia can improve patients' HRQoL.^[15] Further studies taking mood and somatic symptoms into consideration will help to understand the mechanisms underlying the treatment of insomnia and improvement of HRQoL.

Strengths and limitations

The main strengths of the current study included the recruitment of community-based sample, ascertainment of diagnosis of insomnia disorder by clinical interview, and a relatively large sample size. However, several limitations should be noted when interpreting our findings. First, although we constructed a mediation analysis to delineate the complex relationships among insomnia severity, HRQoL, and mood and somatic symptoms, the nature of a cross-sectional design in the current study precluded us from determining the causal relationship among these variables. Second, participants in the current study included adolescent probands and their parents and sibling. In this regard, the age distribution in the current study was not normally distributed. Third, objective sleep parameters were not included in the current study. Fourth, the response rate in parents was slightly lower than that in adolescent group, which may have led to response bias. Finally, the information about participants' treatment history and help-seeking for insomnia was not available for the analyses. It is unclear whether hypnotic use also influences the relationship between insomnia severity and HRQoL. The previous study has shown that hypnotic use is not likely to affect the HRQoL in patients with insomnia.^[37]

Conclusions

The close associations between insomnia severity and impaired HRQoL are largely mediated by psychological symptomatology and personality dimension. Further

prospective study is warranted to investigate the long-term impact of insomnia symptoms on HRQoL and the roles of mood and somatic symptoms.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

- Testa MA, Simonson DC. Assessment of quality-of-life outcomes. *N Engl J Med* 1996;334:835-40.
- Kyle SD, Morgan K, Espie CA. Insomnia and health-related quality of life. *Sleep Med Rev* 2010;14:69-82.
- Staner L. Comorbidity of insomnia and depression. *Sleep Med Rev* 2010;14:35-46.
- Uhde TW, Cortese BM, Vedeniapin A. Anxiety and sleep problems: Emerging concepts and theoretical treatment implications. *Curr Psychiatry Rep* 2009;11:269-76.
- Zhang J, Lam SP, Li SX, Tang NL, Yu MW, Li AM, *et al.* Insomnia, sleep quality, pain, and somatic symptoms: Sex differences and shared genetic components. *Pain* 2012;153:666-73.
- van de Laar M, Verbeek I, Pevernagie D, Aldenkamp A, Overeem S. The role of personality traits in insomnia. *Sleep Med Rev* 2010;14:61-8.
- Moussavi S, Chatterji S, Verdes E, Tandon A, Patel V, Ustun B. Depression, chronic diseases, and decrements in health: Results from the world health surveys. *Lancet* 2007;370:851-8.
- Kroenke K, Spitzer RL, Williams JB, Monahan PO, Löwe B. Anxiety disorders in primary care: Prevalence, impairment, comorbidity, and detection. *Ann Intern Med* 2007;146:317-25.
- Lahey BB. Public health significance of neuroticism. *Am Psychol* 2009;64:241-56.
- LeBlanc M, Beaulieu-Bonneau S, Mérette C, Savard J, Ivers H, Morin CM. Psychological and health-related quality of life factors associated with insomnia in a population-based sample. *J Psychosom Res* 2007;63:157-66.
- Zhang J, Li AM, Kong AP, Lai KY, Tang NL, Wing YK. A community-based study of insomnia in Hong Kong Chinese children: Prevalence, risk factors and familial aggregation. *Sleep Med* 2009;10:1040-6.
- Zhang J, Lam SP, Li SX, Li AM, Wing YK. The longitudinal course and impact of non-restorative sleep: A five-year community-based follow-up study. *Sleep Med* 2012;13:570-6.
- Zhang J, Lam SP, Li SX, Li AM, Lai KY, Wing YK. Longitudinal course and outcome of chronic insomnia in Hong Kong Chinese children: A 5-year follow-up study of a community-based cohort. *Sleep* 2011;34:1395-402.
- Wing YK, Zhang J, Lam SP, Li SX, Tang NL, Lai KY, *et al.* Familial aggregation and heritability of insomnia in a community-based study. *Sleep Med* 2012;13:985-90.
- Thorndike FP, Ritterband LM, Gonder-Frederick LA, Lord HR, Ingersoll KS, Morin CM. A randomized controlled trial of an internet intervention for adults with insomnia: Effects on comorbid psychological and fatigue symptoms. *J Clin Psychol* 2013;69:1078-93.
- American Psychiatric Association. Manual of Mental Disorders, (DSM-IV). 4th ed. Washington, DC: American Psychiatric Association; 1994.
- Riemann D. Nonrestorative sleep: Have we finally found it? *Sleep* 2010;33:417-8.
- Roth T, Zammit G, Lankford A, Mayleben D, Stern T, Pitman V, *et al.* Nonrestorative sleep as a distinct component of insomnia. *Sleep* 2010;33:449-58.
- Zhang J, Lamers F, Hickie IB, He JP, Feig E, Merikangas KR. Differentiating nonrestorative sleep from nocturnal insomnia symptoms: Demographic, clinical, inflammatory, and functional correlates. *Sleep* 2013;36:671-9.
- Ware JE Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30:473-83.
- Lam CL, Gandek B, Ren XS, Chan MS. Tests of scaling assumptions and construct validity of the Chinese (HK) version of the SF-36 health survey. *J Clin Epidemiol* 1998;51:1139-47.
- Bastien CH, Vallières A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med* 2001;2:297-307.
- Barsky AJ, Wyshak G, Klerman GL. Hypochondriasis. An evaluation of the DSM-III criteria in medical outpatients. *Arch Gen Psychiatry* 1986;43:493-500.
- Chan YF, Leung DY, Fong DY, Leung CM, Lee AM. Psychometric evaluation of the Hospital Anxiety and Depression Scale in a large community sample of adolescents in Hong Kong. *Qual Life Res* 2010;19:865-73.
- Leung CM, Ho S, Kan CS, Hung CH, Chen CN. Evaluation of the Chinese version of the Hospital Anxiety and Depression Scale. A cross-cultural perspective. *Int J Psychosom* 1993;40:29-34.
- Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behav Res Methods* 2008;40:879-91.
- Dixon S, Morgan K, Mathers N, Thompson J, Tomeny M. Impact of cognitive behavior therapy on health-related quality of life among adult hypnotic users with chronic insomnia. *Behav Sleep Med* 2006;4:71-84.
- Zhang J, Lam SP, Li SX, Yu MW, Li AM, Ma RC, *et al.* Long-term outcomes and predictors of chronic insomnia: A prospective study in Hong Kong Chinese adults. *Sleep Med* 2012;13:455-62.
- Luo C, Zhang J, Pan J. One-year course and effects of insomnia in rural Chinese adolescents. *Sleep* 2013;36:377-84.
- Alsaadi SM, McAuley JH, Hush JM, Lo S, Bartlett DJ, Grunstein RR, *et al.* The bidirectional relationship between pain intensity and sleep disturbance/quality in patients with low back pain. *Clin J Pain* 2014;30:755-65.
- Wu MP, Lin HJ, Weng SF, Ho CH, Wang JJ, Hsu YW. Insomnia subtypes and the subsequent risks of stroke: Report from a nationally representative cohort. *Stroke* 2014;45:1349-54.
- Anothaisintawee T, Reutrakul S, Van Cauter E, Thakkinstian A. Sleep disturbances compared to traditional risk factors for diabetes development: Systematic review and meta-analysis. *Sleep Med Rev* 2016;30:11-24.
- Sofi F, Cesari F, Casini A, Macchi C, Abbate R, Gensini GF. Insomnia and risk of cardiovascular disease: A meta-analysis. *Eur J Prev Cardiol* 2014;21:57-64.
- Bobic J. Subjective estimation of the quality of life in relation to neuroticism. *Arh Hig Rada Toksikol* 2012;63 Suppl 1:17-22.
- Dubayova T, Nagyova I, Havlikova E, Rosenberger J, Gdovinova Z, Middel B, *et al.* Neuroticism and extraversion in association with quality of life in patients with Parkinson's disease. *Qual Life Res* 2009;18:33-42.
- Samartzis L, Dimopoulos S, Manetos C, Agapitou V, Tasoulis A, Tseliou E, *et al.* Neuroticism personality trait is associated with quality of life in patients with chronic heart failure. *World J Cardiol* 2014;6:1113-21.
- Scalo J, P, Rascati K. Insomnia, hypnotic use, and health-related quality of life in a nationally representative sample. *Qual Life Res* 2015;24:1223-33.