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SCIENTIFIC INVESTIGATIONS

Worry and rumination predict insomnia in patients with coronary heart disease: a cross-sectional study with long-term follow-up

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Study Objectives: Insomnia is highly prevalent and associated with anxiety and depression in patients with coronary heart disease patients. The development of effective psychological interventions is needed. Worry and rumination are potential risk factors for the maintenance of insomnia, anxiety, and depression that may be modified by psychological treatment grounded in the Self-Regulatory Executive Function model. However, the relationships between worry, rumination, anxiety and depression, and insomnia are not known. Therefore, we investigated these relationships both cross-sectionally and longitudinally among patients with coronary heart disease.

Methods: A cross-sectional study consecutively included 1,082 patients in 2014–2015, and 686 were followed up after mean of 4.7 years. Data were gathered from hospital records and self-report questionnaires comprising assessment of worry (Penn State Worry Questionnaire), rumination (Ruminative Responses Scale), anxiety and depression (Hospital Anxiety and Depression Scale), and insomnia (Bergen Insomnia Scale).

Results: Insomnia correlated moderately with all other psychological variables (R 0.18–0.50, all P values < .001). After adjustments for anxiety and depression, odds ratios for insomnia at baseline were 1.27 (95% confidence interval 1.08–1.50) and 1.60 (95% confidence interval 1.31–1.94) per 10 points increase of worry and rumination, respectively. Corresponding odds ratios for insomnia at follow-up were 1.28 (95% confidence interval 1.05–1.55) and 1.38 (95% confidence interval 1.09–1.75). Depression was no longer significantly associated with insomnia after adjustments for worry and rumination, but anxiety remained significant.

Conclusions: Worry and rumination predicted insomnia both cross-sectionally and prospectively, even after controlling for anxiety and depression, although anxiety remained significant. Future studies may test psychological interventions targeting these factors in patients with coronary heart disease and insomnia.

Keywords: insomnia, coronary heart disease, worry, rumination, anxiety, depression, metacognitive therapy

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Worry and rumination are potential risk factors for the development and maintenance of insomnia among patients with coronary heart disease, and these processes may effectively be modified by psychological treatment grounded in the Self-Regulatory Executive Function model. However, the relationships between worry, rumination, anxiety, depression, and insomnia in coronary heart disease are not known.

Study Impact: Worry and rumination are significant predictors of insomnia both cross-sectionally and prospectively, even after factoring out anxiety and depression. Psychological interventions targeting these factors could be developed and tested for insomnia in patients with coronary heart disease.

INTRODUCTION

Insomnia is defined as difficulty initiating and maintaining sleep, often followed by nonrestorative sleep, and waking up too early, with impairment of daytime functioning requiring a symptom duration of at least 4 weeks according to the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition criteria.[1] Chronic insomnia is found among 10% of the general population[2] and is associated with daytime fatigue, increased risk of depression,[3] increased health care utilization and cost, and reduced quality of life. There is also recent evidence that significant symptoms of insomnia are particularly prevalent in patients with coronary heart disease (CHD) with 4 of 10 patients with CHD reporting significant symptoms[4,5] or a diagnosis of insomnia.[6] Insomnia is associated with psychological distress (ie, anxiety and depression), diabetes, subclinical inflammation,[6] and increased mortality.[7–9] These data indicate a need to identify and manage insomnia among patients with CHD.

Cognitive behavior therapy for insomnia (CBT-I) is the most effective treatment for insomnia,[10] also in those with comorbid psychiatric and medical disorders.[11] However, we are only aware of 3 treatment studies of insomnia in patients with CHD. Two studies tested traditional CBT-I vs placebo and compared 2 methods of self-help CBT-I, respectively, in older adults with mixed chronic medical illnesses including CHD. These studies reported some effectiveness.[12,13] A recent feasibility study of a web-based CBT-I reported feasibility of the intervention in older patients with CHD.[14] Furthermore, a review of CBT-I implemented in primary care and community settings reported small to moderate effect sizes for insomnia symptoms.[15] Finally, a Cochrane review

of the CBT-I for older adults (> 60 years) concluded that there is only a mild effect on sleep problems.[16] In general, only 30–40% of insomnia patients achieve full remission posttreatment, and the rate of reoccurrence is particularly high for those who still report elevated presleep arousal after treatment.[17] These results emphasize that a significant proportion of patients do not improve or continue to have significant sleep disturbances despite symptom improvements.[11,14] Thus, increasing the effectiveness of psychological treatment is of great importance.

The limited effect of CBT-I has led to recent attempts to develop models of insomnia as a basis for more effective treatment.[18] Potentially modifiable risk factors for the development and maintenance of insomnia are important to target in psychological treatment. One potential target for more effective treatment may be repetitive thinking, as this is a crucial element across all the most widely accepted models of insomnia.[19–21] Repetitive thinking was originally studied in the context of anxiety and depression.[22,23] Worry and rumination are 2 particular types of repetitive thinking that have been described as playing an important role in a transdiagnostic model of anxiety and depression[22] as well as in insomnia.[24] Worry is regarded as a coping strategy comprising the engagement in abstract repetitive thinking focused on uncertain events in the future,[25] whereas rumination is a coping strategy that consists of repetitive thinking on the causes and consequences of negative emotions.[23] According to Wells and Matthews model,[22] emotional disturbance is caused by a particular pattern of responding to inner experiences that maintain negative emotions (eg, anxiety and depression). This pattern of thinking is called the cognitive-attentional syndrome, and it is characterized by worry, rumination, fixated attention, and unhelpful self-regulatory or coping strategies. According to this model, this pattern of thinking (where worry and rumination form an integral part) causes anxiety and depression, which are known to be closely linked to insomnia, because insomnia itself is associated with such emotional disturbance. However, this model has not been tested in patients with insomnia yet.

To date, there is some evidence supporting the relationship between repetitive thinking and sleep problems.[10] However, few studies have investigated both worry and rumination in clinical settings.[10] Two recent studies exploring the relationship between worry, rumination and clinical insomnia identified a weak association between sleep problems and rumination, but not worry.[26,27] Furthermore, Lancee et al[10] reported that nighttime sleep-related worry was associated with sleep impairment, but failed to find a relationship between general worry, rumination, and insomnia. On the other hand, a study of psychological characteristics of elderly people with insomnia concluded that excessive worry was the most important psychological feature of elderly people with insomnia.[28] However, the relationship between worry, rumination, and insomnia in patients with CHD remains unknown.

The present study aimed to investigate the cross-sectional and prospective relationships between worry, rumination, and insomnia among patients with CHD. Given that a large proportion of these patients also have significant symptoms of anxiety and depression,[6] we also included measures of anxiety and depression to determine the individual contributions of worry and rumination over and above anxiety and depression. Based on the Wells and Matthews' model,[22] we hypothesized that worry and rumination would correlate with insomnia 1) cross-sectionally after adjusting for baseline (T1) anxiety and depression and 2) prospectively as predisposing factors for insomnia at follow-up (T2) after adjusting for anxiety and depression.

METHODS

Design and population

This is a prospective follow-up of the NORwegian CORonary (NOR-COR) prevention study, which is comprehensively described elsewhere.[29,30] Participant flow, losses, and exclusions are shown in **Figure 1**. In brief, 1,127 patients from 2 secondary care hospitals in Norway (Vestfold, n = 542 and Drammen, n = 585), aged 18–80 (median age 64.8, interquartile range 57.6–70.3) years were consecutively included in a cross-sectional baseline study (T1) in 2014–2015, on average 16 months (range 2–36) after a first or recurrent CHD event (defined as acute myocardial infarction and/or a revascularization procedure [coronary artery bypass grafting or percutaneous coronary intervention]). All patients filled in a comprehensive self-report questionnaire covering sociodemographic and psychological factors. In 2019, all patients at baseline were evaluated for taking part in a questionnaire-based follow-up study (T2), mean 4.7 years after baseline.

Drammen and Vestfold hospitals combined have a catchment area of 7.4% of the Norwegian population (380,000 inhabitants) with a blend of urban and rural districts that representatively reflects Norwegian education, economy, age distribution, morbidity, and mortality.[31,32]

Ethics

The NOR-COR baseline and follow-up studies were approved by the Regional Committee for Medical and Health Research Ethics in the South East Region of Norway (REC South-East) (2013/1885,2018/2007). All patients signed a written informed consent in advance of participation.

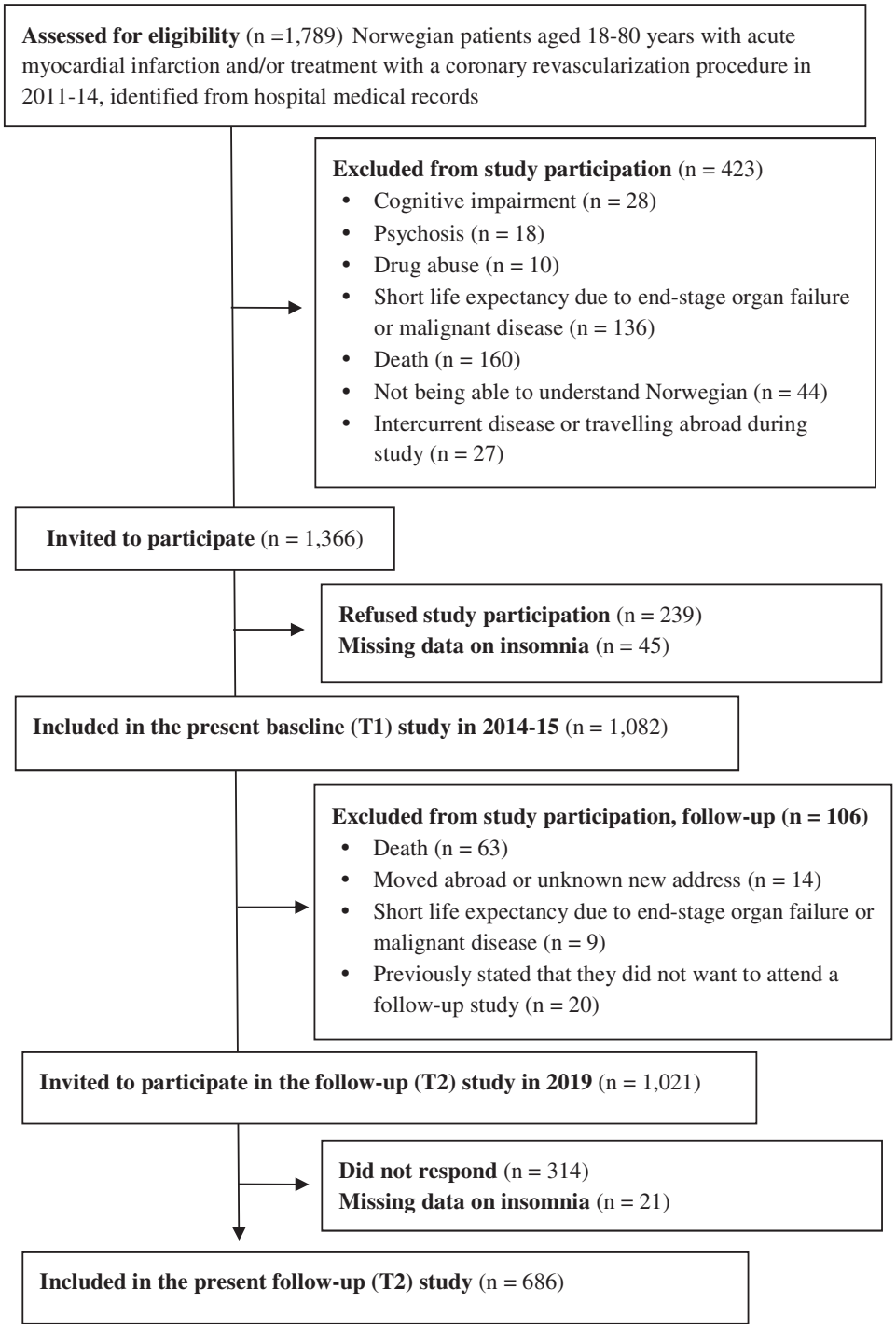
Variables

Information about age, sex, coronary multivessel disease, Charlson comorbidity score,[33] and coronary index diagnosis and treatment was collected from hospital records at the time of inclusion in the baseline study. The baseline self-report questionnaire included the following: level of education (with low education defined as 12 years or less), living alone (yes/no), risk of obstructive sleep apnea[34] and psychological factors (insomnia, anxiety, depression, worry, and rumination). A good test-retest reliability has been reported for this baseline questionnaire.[35]

The Bergen Insomnia Scale

Insomnia was assessed using the Bergen Insomnia Scale (BIS),[36] which is a 6-item questionnaire based on the criteria for the clinical diagnosis of insomnia described in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition.[1] The first 4 items inquire about difficulties with sleep initiation,

Figure 1—Study flow chart.



maintenance of sleep, awakenings in the morning, and nonrestorative sleep, while items 5 and 6 assess daytime impairment and satisfaction with sleep. A delay in sleep initiation or awakenings during the night or in the morning of 30 minutes is the cut-off value for the first 3 items. All items are scored as number of days per week (0–7), yielding a continuous sum score from 0 to 42 (BIS sum score) increasing with insomnia symptoms severity. Three days or more on items 1, 2, 3, or 4 combined with 3 days or more on item 5 or 6 indicate a diagnosis of insomnia. Those who fulfilled these criteria were categorized with "insomnia", whereas those who did not fulfill these criteria were described as "no insomnia". The BIS has adequate psychometric properties and there are normative Norwegian data for comparison.[36] In the baseline study, the 4-week test-retest reliability of the BIS was 0.92.[35] The Cronbach's alphas were 0.88 (T1) and 0.89 (T2).

The Hospital Anxiety and Depression Scale

Depression and anxiety were assessed using the Hospital Anxiety and Depression Scale (HADS).[37] This is a 14-item self-report questionnaire consisting of 2 7-item subscales that assess anxiety (HADS-A) and depressive (HADS-D) symptoms. A cut-off value of HADS-A or HADS-D greater than or equal to 8 has been reported to represent clinically significant symptoms of anxiety or depression.[38] The HADS has demonstrated good psychometric properties in a number of studies, including in patients with CHD.[38] The Norwegian version of the HADS has shown good internal consistency and acceptable validity across studies.[38] The 4-week test-retest reliabilities were 0.92 for HADS-A and 0.94 for HADS-D in the baseline study.[35] The Cronbachś alphas were 0.84 (T1) and 0.87 (T2) for HADS-A and 0.76 (T1) and 0.73 (T2) for HADS-D.

The Penn State Worry Questionnaire

The Penn State Worry Questionnaire (PSWQ) aims to measure the trait of worry and has an ability to differentiate patients with generalized anxiety disorder from other anxiety disorders.[39] PSWQ consists of 16 items, each rated on a 5-point Likert scale, from 1 ("not at all typical of me") to 5 ("very typical of me"). The total score ranges from 16 to 80. In general, the PSWQ has good psychometric properties and in the baseline study the 4-week test-retest reliability of the PSWQ was 0.91.[35] The Cronbach's alpha was 0.91 (T1).

The Ruminative Responses Scale

The Ruminative Responses Scale (RRS) is a measure of rumination (ie, describing one's responses to depressed mood) and consists of the 3 factors: depression, brooding, and reflection).[40] RRS is a 22-item questionnaire, each item rated on a 4-point Likert scale, from 1 ("almost never") to 4 ("almost always"). The total score ranges from 22 to 88. In the baseline study, the test-retest reliability of the RRS was 0.88.[35] The Cronbach's alpha was 0.96 (T1).

Statistics

The descriptive measurements are presented as means with standard deviations for continuous variables and frequencies and percentages for proportions. Chi-square tests, independent and paired *t*-tests were used to test group differences. Multicollinearity was tested with standard variance inflation factors. For odds ratios, we performed bivariate and block-wise hierarchical logistic regressions. *P* values less than .05 were considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics for Windows, version 25 (IBM Corp., Armonk, NY).

At baseline, the observed level of "caseness" (45.1%) for the dependent variable (insomnia) and for the dichotomous independents HADS-anxiety (21.1%) and HADS-depression (14.7%) implied that N values above 300 and 90 are required to detect "small" or "medium" odds ratios,[41] respectively, with a power of 80% ($P < 5$) for HADS-anxiety. For HADS-depression, N values approximately above 100 and 310 are required. For the linear covariates of worry and rumination, detecting "moderate" and "small" effect sizes[42] with a power of 80% ($P < .05$) would require N values approximately higher than 105 and 290. Thus, with N values of 899 at baseline and 577 at follow-up, this study is sufficiently powered.

RESULTS

Figure 1 shows the study flow chart. Among 1,789 patients identified from hospital discharge lists with myocardial infarction and/or a coronary revascularization procedure, 423 patients were excluded and 1,366 were eligible for study participation. Of these, 239 patients declined participation, and 1,127 (83% participation rate) were included in the baseline study. In all, 45 (4%) had missing data for insomnia, thus a total of 1,082 patients were included in the baseline analyses (T1). In the follow-up study (T2) conducted a mean of 4.7 (range 4.1–5.3) years later, 1,021 were eligible for participation after excluding 106 patients. Of those invited, 314 did not respond, whereas 21 patients (3%) had missing data on insomnia. Thus 686 patients (67% participation rate) were included in the study.

Sample characteristics

Mean age at index event among all participants in the baseline-study (T1) was 61.5 (standard deviation 9.6) years and 226 (20.9% were women). Acute myocardial infarction was the index coronary event in 79% (n = 858) of the patients and stable/

Table 1—Sociodemographic and psychosocial characteristics at baseline and follow-up for participants in both studies.

	Baseline T1 (n = 1,082)	Follow-up T2 (n = 686)
Age at the index coronary event, mean (SD), y	61.5 (9.6)	-
Sex female, n (%)	226 (20.9)	-
Years since the index coronary event, mean (SD)	1.5 (0.9)	6.2 (0.9)
Low education (≤ 12 years), n (%)	750 (70.0)	-
Living alone, n (%)	210 (17.8)	173 (15.3)
Coronary multivessel disease, n (%)	416 (38,5)	254 (37,1)
Charlson comorbidity score, mean (SD)	4,1 (1,4)	4,1 (1,3)
Bergen Insomnia Scale sum, mean (SD)	13.9 (10.8)	12.5 (10.8)
Insomnia, n (%)	488 (45.1)	258 (37.6)
HADS-A sum score, mean (SD)	4.8 (3.7)	4.0 (3.8)
HADS-A ≥ 8, n (%)	221 (21.1)	115 (17.4)
HADS-D sum score, mean (SD)	3.9 (3.3)	3.7 (3.4)
HADS-D ≥ 8, n (%)	156 (14.7)	98 (14.7)
Worry PSWQ, mean (SD)	38.0 (12.6)	-
Rumination RRS, mean (SD)	32.6 (11.6)	-

HADS-A = Hospital Anxiety and Depression Scale–anxiety subscale, HADS-D = Hospital Anxiety and Depression Scale–depression subscale, PSWQ = Penn State Worry Questionnaire, RRS = Ruminative Responses Scale, SD = standard deviation.

Table 2—Cross-sectional and longitudinal correlations of psychological factors including insomnia for participants in both studies (n = 686).

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Insomnia, T1	.78 *	.39*	.35*	.34*	.22*	.34*	.37*	.47*	.54*	.30*	.25*	.27*	.18*
2. BIS sum, T1	-	.50*	.43*	.43*	.30*	.46*	.49*	.55*	.69*	.43*	.34*	.38*	.29*
3. HADS-A, T1		-	.79*	.63*	.46*	.73*	.69*	.37*	.41*	.62*	.46*	.44*	.34*
4. HADS-A ≥ 8, T1			-	.52*	.41*	.61*	.59*	.29*	.34*	.49*	.46*	.34*	.30*
5. HADS-D, T1				-	.76*	.53*	.63*	.29*	.32*	.42*	.35*	.57*	.46*
6. HADS-D ≥ 8, T1					-	.39*	.46*	.22*	.24*	.30*	.26*	.42*	.40*
7. PSWQ, T1						-	.61*	.39*	.38*	.54*	.40*	.36*	.31*
8. RRS, T1							-	.34*	.41*	.53*	.42*	.50*	.40*
9. Insomnia, T2								-	.80*	.48*	.38*	.36*	.25*
10. BIS, T2									-	.56*	.45*	.45*	.31*
11. HADS-A, T2										-	.79*	.67*	.55*
12. HADS-A ≥ 8, T2											-	.57*	.53*
13. HADS-D, T2												-	.79*
14. HADS-D ≥ 8, T2													-

*P < .001. Pearson's r and point biserial (for dichotomies). BIS = Bergen Insomnia Scale, HADS-A = Hospital Anxiety and Depression Scale–anxiety subscale, HADS-D = Hospital Anxiety and Depression Scale–depression subscale, PSWQ = Penn State Worry Questionnaire, RRS = Ruminative Responses Scale.

unstable angina treated with a revascularization procedure in 21% (n = 244). Compared to BIS responders at T1, BIS nonresponders were older (mean years 66.0 vs 61.5, P value < .001), while no significant differences in sex, level of education, or time since the index coronary event were found.

A comparison of baseline (T1) characteristics between follow-up (T2) participants (n = 686) and follow-up (T2) nonparticipants (n = 409) is shown in **Table S1** in the supplemental material. Compared to participants at T2, nonparticipants were more likely to be younger, female, and report lower education and higher baseline (T1) scores on insomnia, worry, rumination, and depression.

Characteristics of participants at baseline (T1) and follow-up (T2) are presented in **Table 1**. The proportion with insomnia and clinically relevant symptoms of anxiety was numerically slightly lower at follow-up, whereas only minor differences were found for the other factors.

Correlation between psychosocial characteristics

Correlations between insomnia, BIS sum-score, HADS-A, HADS-D, worry (measured at T1 only), and rumination (also measured at T1 only) at baseline and follow up are presented in **Table 2**. All variables were significantly correlated (P values < .01), with coefficients ranging from 0.18 to 0.80. PSWQ and RRS correlated 0.61, and both variables correlated moderately with insomnia and BIS at baseline (T1) and follow-up (T2). Moderate correlations were also found for insomnia and BIS with HADS-A and HADS-D, both at baseline (T1) and follow-up (T2). Moreover, HADS-A and HADS-D were highly correlated with both PSWQ and RRS, with the exception that worry correlated only moderately with HADS-D at follow-up (T2).

Anxiety and depression adjusted logistic regressions

Crude and multi-adjusted odds ratios for insomnia at baseline (T1) and follow-up (T2) by demographic and psychological factors are presented in **Table 3**. Tests for multicollinearity were nonsignificant. In the first step of the multi-adjusted analysis, both anxiety (HADS-A ≥ 8) and depression (HADS-D ≥ 8) were significantly associated with insomnia in the cross-sectional analysis (T1) after controlling for age, sex and HADS-D and HADS-A. Noteworthy, the effects of both HADS scales attenuate compared to their bivariate odds ratios. In the second step of the analysis, when worry and rumination (per 10 points increase) were entered, depression (HADS-D ≥ 8) was no longer significantly associated with insomnia and the association with anxiety (HADS-A ≥ 8) was substantially reduced. Worry and rumination remained significantly associated with baseline insomnia, indicating that the association between HADS depression and insomnia was completely confounded by worry and rumination, whereas the association between HADS anxiety and insomnia to a large extent was confounded (odds ratio reduced from 4.18 to 1.65).

In the prospective analysis, insomnia at follow-up (T2) was entered as the outcome variable (**Table 3**). At the first step of the multi-adjusted analysis, depression (HADS-D ≥ 8) was no longer significant and anxiety (HADS-A ≥ 8) was substantially attenuated. At the second step, when worry and rumination were entered into the analysis, anxiety still remained highly significant and was further attenuated. Relevant for the purpose of this study, worry and rumination assessed at baseline (T1) significantly predicted insomnia at follow-up (T2) in the multi-adjusted model, even after adjusting for anxiety (HADS-A ≥ 8) and depression (HADS-D ≥ 8) at follow-up (T2).

DISCUSSION

Overall, in this study comprising patients with CHD, worry and rumination were significantly associated with insomnia both

Table 3—Hierarchical multiple block-wise logistic regressions predicting insomnia at T1 and at T2.

	OR	95% CI	adjOR[a]	95% CI	adjOR[b]	95% CI
Insomnia at baseline (T1), n = 899						
Age (age/10 years)	0.78**	0.57–0.84	0.87*	0.76–1.00	0.88 ns	0.75–1.02
Sex (female)	1.44**	1.21–1.70	1.36**	1.16–1.61	1.24*	1.02–1.50
HADS-A ≥ 8 (T1/T2)[c]	5.70**	3.93–8.26	4.18**	2.89–6.06	1.65*	1.02–2.67
HADS-D ≥ 8 (T1/T2)[c]	4.32**	2.83–6.60	2.15**	1.41–3.27	1.38 ns	0.83–2.29
Worry (PSWQ sum / 10) (T1)	1.88**	1.66–2.12	-	-	1.27**	1.08–1.50
Rumination (RRS sum / 10) (T1)	2.27**	1.95–2.66	-	-	1.60**	1.31–1.94
Insomnia at follow-up (T2), n = 577						
Age (age/10 years)	0.61**	0.50–0.74	0.70**	0.57–0.88	0.73**	0.58–0.91
Sex (female)	2.85**	1.84–4.43	1.59**	1.25–2.02	1.45**	1.13–1.86
HADS-A ≥ 8 (T1/T2)[c]	10.46**	5.97–18.35	7.35**	3.88–13.92	4.89**	2.50–9.54
HADS-D ≥ 8 (T1/T2)[c]	4.16**	2.50–6.94	1.54 ns	0.80–2.97	1.08 ns	0.54–2.14
Worry (PSWQ sum / 10) (T1)	1.85**	1.59–2.16	-	-	1.28*	1.05–1.55
Rumination (RRS sum / 10) (T1)	2.08**	1.72–2.51	-	-	1.38*	1.09–1.75

**P < .01, *P < .05, ns: not statistically significant. Worry and rumination measured at T1 only. [a]Multivariate OR adjusted for age, sex, HADS-A ≥ 8, and HADS-D ≥ 8. [b]Multivariate OR adjusted for age, sex, HADS-A ≥ , HADS-D ≥ 8, worry, and rumination. [c]T1/T2: associations between HADS-A and HADS-D at T1 and insomnia at T1, associations between HADS-A and HADS-D at T2, and insomnia at T2. CI = confidence interval, HADS-A = Hospital Anxiety and Depression Scale–anxiety subscale, HADS-D = Hospital Anxiety and Depression Scale–depression subscale, OR = odds ratio, PSWQ = Penn State Worry Questionnaire, RRS = Ruminative Responses Scale.

cross-sectionally and prospectively even after adjusting for anxiety and depression. These results emphasize the importance of worry and rumination in insomnia, independent of anxiety and depression. Moreover, depression was no longer significantly associated with insomnia after adjustments for worry and rumination. However, anxiety remained significant.

Cross-sectional associations between worry, rumination, and insomnia

To our knowledge, this is the first study to investigate the role of worry and rumination in insomnia among patients with CHD. Results from the cross-sectional analyses indicate that worry and rumination play a role in insomnia independent of anxiety and depression. This is partly in line with the results of a previous cross-sectional study reporting worry and rumination to be higher in patients with chronic insomnia than in healthy controls and being cross-sectionally correlated with sleep indices derived from Pittsburgh Sleep Quality Index.[43] However, only worry remained significantly associated with sleep indices after adjustments for anxiety (State-Trait Anxiety Index) and depression (Beck Depression Inventory). In our study, both worry and rumination remained significantly associated with insomnia. The discrepancy in results may be due to differences in measures of insomnia, anxiety, and depression. Our results are also in line with a cross-sectional study that reported rumination to be associated with impaired sleep quality and insomnia.[44]

Prospective associations between worry, rumination, and insomnia

Our hypothesis that worry and rumination would predict future insomnia was supported by both variables prospectively, predicting insomnia at follow-up after 4.7 years even after adjustments for anxiety and depression. It has previously been described that the PSWQ may be considered a trait measure and the tendency to worry may be regarded as a core personality dimension.[28,45] Thus, in line with this, worry at baseline was strongly associated with insomnia both at baseline and follow-up. Furthermore, rumination may also be considered as a trait[46] and our results add to that of rumination being cross-sectionally as well as prospectively associated with insomnia and sleep problems.[44] However, both worry and rumination can be modified directly through metacognitive therapy.[47]

Overlap and differences between worry and rumination in insomnia

In general, previous research has shown that worry and rumination are 2 distinct cognitive processes being related to past or future events.[48,49] However, it is unclear if worry and rumination should be considered as 1 or 2 entities in their relationship with insomnia, since some studies have yielded contradictory evidence.[27,43,50] In the present study, we found a significant moderate correlation between worry and rumination of 0.61. However, when we tested for interactions between variables in the regression analyses, we did not find that worry and rumination related differently to our results. This may imply that while worry and rumination overlap and differ from each other, it is perhaps the similarities that they share that play a crucial role in their relationship with insomnia. Future studies are needed to explore this argument.

Implications of the metacognitive model and treatment in insomnia

Our study is not able to elucidate the exact mechanisms that link worry and rumination to insomnia. Ong and colleagues[17] argued

for their own perspective of a metacognitive model based on a conceptualization of insomnia from a framework of mindfulness. Others have suggested the Self-Regulatory Executive Function model[22,51] as an explanatory model on how worry and rumination might be linked to insomnia.[52] In line with this model, metacognitions (ie, beliefs about thoughts and processes) drive worry and rumination, which then contribute to the maintenance of anxiety and depression as well as insomnia. The metacognitive model is based on the Self-Regulatory Executive Function model, and a number of other metacognitive models have been developed and tested for a range of mood and anxiety disorders.[47] The core treatment derived from this model targets worry and rumination by modifying both positive (eg, "Worrying helps me prevent problems"; "rumination helps me understand what happened in the past") and negative metacognitive beliefs (eg, "My worrying or rumination is uncontrollable").

Recent systematic reviews and meta-analyses demonstrate the effectiveness of metacognitive therapy for mood disorders as well as for anxiety disorders.[53,54] However, we do not know of any specific metacognitive model developed for insomnia. Positive beliefs about sleep-related worry have been described in insomnia patients[55] and a metacognitive questionnaire of insomnia has been developed.[52] Metacognitive beliefs about insomnia have been found to predict presleep hyperarousal, and there is increasing evidence for the role of such beliefs about sleep in insomnia.[56,57] Furthermore, general metacognitions, worry, and sleep have been significantly correlated in patients with generalized anxiety disorder, where the cardinal feature is worry.[58] These results together with those of our study speak in favor of future studies on the development and testing of a specific metacognitive model of insomnia to be undertaken. To understand the complexities of these relationships, insomnia-specific and general metacognitions should be included as well as an assessment of presleep arousal and objective indices of insomnia. To date, it is still unknown which cognitive treatment component should be targeted in insomnia.[59] We argue for further testing of the metacognitive model, particularly in patients with CHD and with the co-occurrence of insomnia, as anxiety and depression are also common in these patients, with negative associations to cardiovascular prognosis.[60] Furthermore, because the effectiveness of traditional CBT has been reported to have limited impact on CHD patients and metacognitive therapy has been described as a better fit with distress in these patients,[61] our results support an intervention to target worry and rumination as key factors in insomnia.

The relationship between emotional disturbance and repetitive negative thinking

A thorough discussion of the importance of anxiety and depression in insomnia is beyond the scope of this paper. However, there are some important points to be emphasized. Anxiety and depression may potentially influence worry and rumination as well as sleep.[62] In our cross-sectional study, the relationship between anxiety and depression with insomnia were attenuated or removed after adjustment for worry and rumination, hence suggesting that worry and rumination are either mediators or confounders of the associations between anxiety/depression and insomnia. However, the odds ratio for anxiety assessed at follow-up 4.7 years later was also attenuated (and that of depression eliminated), when adjusting for baseline worry and rumination, indicating that worry and rumination act as confounders.

Limitations

There are several limitations in the present study. First, worry and rumination were assessed only at baseline, since they are regarded as trait psychological factors. The absence of assessments at follow-up prevents us from detecting changes over time in worry and rumination. Second, insomnia was assessed with the BIS, and this should be interpreted as a self-report diagnostic measure for insomnia rather than an independent clinician-based diagnosis. Moreover, the BIS has not been specifically validated in patients with CHD. Third, an objective assessment of sleep (eg, actigraphy or polysomnography) was not included in this study. Nevertheless, insomnia is a clinical diagnosis and not based on objective assessments. Fourth, the nonparticipants at follow-up (T2) reported more psychological distress, including insomnia in the baseline study.[30] We are, however, not concerned that this may affect the representativeness of our sample, since the nonparticipants display profiles resembling those of more severe clinical populations. Fifth, we did not control for obstructive sleep apnea and use of sleep medication, which could have had a potential impact on our results. However, adjustments for obstructive sleep apnea risk did not affect the relationship between insomnia and anxiety/depression in multi-adjusted analyses.[6] Sixth, we did not include a control group consisting of good sleepers, or patients with other non-comorbid disorders. Therefore, we caution against generalizing our results to sleep in general and to that of other groups of patients with insomnia. Finally, we did not diagnose psychiatric disorders and control for the presence of anxiety and mood disorders that may also have affected our results. Despite the above limitations, this study has a number of strengths, particularly large sample sizes, high participation rates (79% at baseline and 67% at follow-up), patients from routine clinical practice, and a prospective design.

In conclusion, the results of our study support the existence of statistically significant relationships between worry, rumination, and insomnia both cross-sectionally and prospectively in a population with CHD. These relationships still hold even after factoring out symptoms of anxiety and depression. Future studies may develop and test a metacognitive model and therapy of insomnia based on the Wells and Matthews model, including general and insomnia-specific metacognitions in patients with insomnia with and without comorbidity (CHD, anxiety, and mood disorders).

ABBREVIATIONS

BIS, Bergen Insomnia Scale
CBT-I, cognitive behavior therapy for insomnia
CHD, coronary heart disease
HADS, Hospital Anxiety and Depression Rating Scale
HADS-A, Hospital Anxiety and Depression Rating Scale–anxiety subscale

HADS-D, Hospital Anxiety and Depression Rating Scale–depression subscale
PSWQ, Penn State Worry Questionnaire
RRS, Ruminative Responses Scale

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DISCLOSURE STATEMENT

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