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Emotion coping strategies and dysfunctional sleep-related beliefs are associated with objective sleep problems in young adults with insomnia

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Abstract

Background: Though insomnia is associated with affected emotion regulation and dysfunctional ideas about sleep, little is known about the relation of these problems with objective sleep disruption. We aimed to explore this relationship in young adults with and without insomnia.

Methods: Twenty young adults with diagnosed [insomnia disorder](#) (aged 27.7 ± 8.6 years) and twenty age-matched individuals without insomnia (26.7 ± 7.0 years) completed questionnaires, measuring sleep-related thoughts and emotions and [emotion regulation](#). Objective sleep measurements were collected through 10-days actigraphy [as a representative sample of nights](#), and analyzed for sleep onset latency, sleep efficiency total sleep time. T-tests and multivariate analyses of variance (MANOVA) were conducted for sample characterization and analysis of the association of sleep-related thoughts and emotions and [emotion regulation](#) with objective sleep data.

Results: As expected, young people showed more dysfunctional sleep-related thoughts and emotions (all $ps \leq 0.025$) and dysfunctional emotion regulation strategies (all $ps \leq 0.040$). Surprisingly, MANOVA results showed that only emotion coping strategies after a stressful event ($p = 0.017$) and dysfunctional beliefs about sleep ($p = 0.012$), but not other factors of arousal or sleep reactivity, were associated with overall worse sleep, especially sleep onset latency (all $ps \leq 0.012$) and sleep efficiency (all $ps \leq 0.010$).

Conclusions: Maladaptive emotion coping strategies after a stressful event and dysfunctional sleep-related beliefs and attitudes affect objective sleep onset latency and sleep efficiency in young adults, highlighting the importance of targeting these features in the prevention and treatment of chronic insomnia and improving actual sleep quality.

Keywords: Insomnia, cognitive control, emotion regulation, sleep reactivity, arousal

1. Introduction

[Insomnia disorder](#) is a common sleep disorder affecting about 6 to 10% of the general population with important health consequences, such as functional impairments, psychological and medical risk, and reduced quality of life [1,2]. Insomnia etiology is however not well understood: a large number of somatic, cognitive and emotional factors seem to play a major role in insomnia [3], but how these factors interfere with actual sleep loss still needs further investigation.

Stress is one of the most important precipitating factors of insomnia [4,5]. Many studies show a significant relationship between major stressful events (e.g., family, health, work) and the onset of insomnia [6,7]. However, it seems that the relationship between stress and sleep depends on the type, duration and severity of the stressor [8,9]. Some studies show that individuals with insomnia report a greater frequency of negative life events but also a higher impact of daily stressful events [10,11]. This is further reflected in strong insomnia prevalence increases in the COVID-19 outbreak situation, with many people have developing insomnia in this unprecedented stressful situation of unknown duration [12–14]. Others suggest that chronicity and severity of stress exposure are significant predictors of future sleep disturbance, and that emotional reactivity to stressful events remains the same between younger and older adults, despite the diminished exposure to stressful events with age [8,9].

Insomnia is characterized by heightened levels of physiological, cognitive, and emotional arousal across the 24-hour day [4,15], which mediates the relationship between perceived daytime stressors and sleep disturbances [10, 16–18]. Indeed, psychosocial or artificial (e.g., [Trier Social Stress Test](#), caffeine) stress induces somatic (e.g., heart rate), cognitive (e.g. rumination) and emotional (e.g., negative affect) arousal during the day and at bedtime, which disrupts subjective and polysomnographic sleep, through longer sleep onset latency, frequent nocturnal awakenings, shorter sleep duration and lower sleep efficiency [10,16,19–23]. In return, sleep difficulties can influence subsequent emotional and cognitive responses during the next day and night [16,23,24]. More

specifically, enhanced cognitive and emotional reactivity, including increased attention to sleep deprivation and its consequences, as well as misconceptions about sleep and harmful compensatory behaviour can further increase physiological arousal which then disrupts sleep, leading to further dysfunctional cognitive and affective activity (rumination, worry) [25,26]. Over time, this cognitive and emotional reactivity perpetuates sleep disturbances and reinforces the vicious circle of insomnia and affected day- and nighttime functioning [4,27,28].

Vulnerability to develop insomnia later in life has also been associated with the presence of several cognitive and emotional factors: pre-sleep cognitive and somatic arousal, rumination, worry, general arousability, neuroticism, negative emotional affect and emotion-oriented coping [29–34]. This notion of vulnerability suggests that premorbid characteristics may contribute to insomnia disorder, including personality traits, sleep reactivity, or emotional and cognitive hyperarousal [30,35–37]. For example, people with insomnia tend to manage stress and conflict through the internalization of emotions and distress [38]. Other studies show that cognitive–emotional arousal negatively influences the relationship between sleep reactivity (lying awake because of arousing events the previous or next day) and stress deregulation [29,30], thus increasing the risk of insomnia’s development or its persistence [39]. Similarly, Vgontas and colleagues showed that the phenotype of shorter sleep duration is associated with cognitive-emotional and cortical arousal as well as stress system activation [37].

Other factors such as age further increase the risk of developing insomnia, due to changes in health, circadian rhythm and hormone levels [40]. In adults, total sleep time, sleep efficiency and deep sleep significantly decrease with age, while wake after sleep onset significantly increases with age [41]. However, insomnia features such as stress responsiveness and arousal predisposition seem to affect older people less [9,42,43]. Older adults may be less affected by stress or negative emotions and cognition, perhaps due to habituation and improved stressful coping strategies developed over time [9,43]. These differences in the regulation of emotional and cognitive processes with age must

therefore be taken into consideration in order to better understand the mechanisms of insomnia, its natural history and evolution throughout lifetime, and provide customized interventions for insomnia.

Though several studies have thus examined the presence of sleep reactivity and cognitive factors in the development of insomnia [15,27,28], few studies have added objective measures of sleep [28]. Given that insomnia with short sleep duration however typically shows more severe symptoms than insomnia without short sleep duration [44], investigating those factors that influence objective sleep measures might enable us to provide more focused treatment and reduce relapses. Furthermore, most studies investigating factors at play in insomnia either investigate cognitive or emotional factors, not combinations of both. Examining several factors simultaneously in the same sample can provide a more precise and exhaustive description of the profile of individuals with insomnia disorder compared to that of good sleepers. Specific factors are typically investigated in isolation, such as rumination, neuroticism or negative and positive affect. To our knowledge, the association of emotional-cognitive strategies involved in the etiology of insomnia and objective sleep measures, in a young adult population, has not yet been investigated. Results could guide the development of effective early intervention programs to prevent the development of chronic insomnia and its comorbid mental health problems.

The current study thus explores the association between objectively measured sleep difficulties, emotion regulation and sleep-related emotion and cognition in young adults with insomnia disorder, as compared to those without insomnia disorder. Based on previous findings, we hypothesize that: 1) young adults with insomnia disorder will show more comorbid symptoms to insomnia, such as anxiety and depression and arousal levels, but also show more dysfunctional emotion regulation strategies and more dysfunctional sleep-related thoughts and emotions compared to those without insomnia disorder and that 2) higher cognitive arousal levels and sleep reactivity levels will explain differences in sleep onset latency, sleep efficiency and total sleep time more than other emotion-related factors.

2. Methods

2.1. Participants

Participants were recruited through local advertising (e.g., newspapers) and included after telephone screening [45,46]. Participants were excluded if they had a chronic psychiatric, neurological or other medical condition (including other sleep disorders), were taking psychotropic or hypnotic medication, had done or were doing shift work, were pregnant or lactating, or showed drug or alcohol dependence, except smoking. Participants stopped over-the-counter [sleep aids \(valerian, herbal treatments\)](#) throughout the study period. [Insomnia disorder](#) was diagnosed based on DSM-5 criteria during a clinical interview, including sleep and daytime functioning complaints for at least three nights during at least the last three months. For the control group, absence of sleep complaints were verified, also through a clinical interview. Twenty young adults with [insomnia disorder](#) (aged 27.7 ± 8.6 years) and twenty age-matched control participants (26.7 ± 7.0) without [insomnia disorder](#) were included, and all provided written informed consent. The study was approved by the local ethical committee (Comité de Protection des Personnes Sud-Ouest et Outre Mer III [CPP]) and registered as a clinical trial (ClinicalTrials.gov.identifier: NCT02821234). All experimental procedures were conducted according to the Helsinki Agreement (World Medical Association General Assembly, 2014).

2.2. Experimental Procedures

Participants visited the SANPSY laboratory (CHU of Bordeaux, France) where a semi-structured interview for sleep and psychiatric disorders (MINI), as well as a set of questionnaires was administered: the Pittsburgh Sleep Quality Index (PSQI) [47], the Insomnia Severity Index (ISI) [48], the Beck Depression Inventory Second Edition (BDI-II) [49], the Beck Anxiety Inventory (BAI) [50], the Arousal Predisposition Scale (APS) [51], the Coping Inventory for Stressful Situation (CISS) [52], the Ford Insomnia Response to Stress Test (FIRST) [35], Dysfunctional Beliefs and Attitudes about Sleep (DBAS) [53], Difficulties in Emotion Regulation Scale (DERS) [55], Presleep Arousal Scale (PSAS) [56], and Thought Control Questionnaire Insomnia-revisited (TCQI-R) [57]. [Actigraphy](#)

(Actiwatch-64; Phillips Respironics, US) was recorded during 10 days, applying the high sensitivity setting for actigraphy scoring. Sleep diaries were filled out by participants, where participants had to report every notable event that would influence recording (e.g. showers, naps). This prevented any automatic misdetection from misuse of the actimeter (i.e., unintentional event marker, forgetting to put back the actimeter after a shower etc.), as well as quality control and artefact detection. Three sleep parameters derived from computer-generated actigraphy data based on sleep diary information are analyzed in this study, which are described as most characteristic to insomnia [41]: total sleep time (TST), sleep efficiency (SE) and sleep onset latency (SOL) [58].

2.3. Questionnaires

The **Pittsburgh Sleep Quality Index** (PSQI) [47] is a validated 10-item questionnaire asking about sleep habits and their frequency in the last month, for which a cut-off score of more than 5 is indicative of sleep problems in adults (global scores: 0 to 21).

The **Insomnia Severity Index** (ISI) [48] is a validated 7-item (5-point Likert scale) questionnaire asking about difficulties sleeping, daytime functioning, and distress about sleep in the last month. A score of 15 or higher is indicative of clinical insomnia (global scores: 0 to 28).

The **Beck Depression and Anxiety Inventory Second Edition** (BDI-II/BAI) [49,50] is a validated 21-item (4-point Likert scale) self-report questionnaire to assess the presence and severity of depression or anxiety symptoms in the two last weeks. A score of 17 or higher is indicative of clinical depression or anxiety (global scores: 0 to 63).

The **Arousal Predisposition Scale** (APS) [51] is a validated 12-item (5-point Likert scale) questionnaire whether the respondent considers her/himself a stressful and emotionally reactive person (e.g. I am a quiet person). A higher score indicates a greater predisposition to activation (global scores: 0 to 48).

The **Coping Inventory for Stressful Situations** (CISS) [52] is a validated 48-item (5-point Likert scale) self-reported measure assessing stress coping strategies and actions through five subscales (scores: 16 to 80): task (16 item), emotional (16 item), avoidance (4 item), distraction (8 item) and social diversion (4 item).

The **Difficulties in Emotion Regulation Scale** (DERS) [55] is a validated 36-item (5-point Likert scale) questionnaire comprised of six subscales assessing different dimensions of emotion regulation: non acceptance of emotion (6 item); difficulties engaging in goal-directed behaviors (5 item); impulse control difficulties (6 item); lack of emotional awareness (6 item); limited access to emotion regulation strategies (8 item); and lack of emotional clarity (5 item). Higher scores reflecting greater difficulties regulating emotion (global scores: 36 to 180).

The **Presleep State Arousal Scale** (PSAS) [56] is a validated 16-item (5-point Likert scale) questionnaire assessing whether the respondent experienced somatic (8 item) or cognitive hyperarousal (8 item) at bedtime. A higher score indicates a greater level of somatic or cognitive arousal (subscale scores: 8 to 40).

The **Ford Insomnia to Stress Scale** (FIRST) [35] contains validated 9-item (4-point Likert scale) assessing whether past and future stressful events are likely to affect sleep quality. A higher score indicates a greater level of stress reactivity (global score: 9 to 36).

The **Dysfunctional Beliefs and Attitudes about Sleep Scale** (DBAS-16)[53] is a validated 16-item (10-point Likert scale) questionnaire that assess maladaptive sleep-related beliefs and attitudes about sleep. Higher scores indicate a stronger endorsement of dysfunctional beliefs and attitudes concerning sleep (global scores: 0 to 160).

The **Thought Control Questionnaire Insomnia-Revisited** (TCQI-R)[57] is a validated 35-item (4 point Likert scale) self-reported measure assessing what strategies people use to control thoughts that may be interfering with their sleep at night (global scores: 0 to 32). Six subscales are identified:

aggressive suppression (8 item); social avoidance (3 item); cognitive distraction (5 item); behavioral distraction (5 item); worry (7 item); reappraisal (7 items).

2.4. Statistical Analysis

The data manipulation and analyses were conducted using SPSS 11. Data distributions were checked for normality, outliers and missing data, of which the latter were imputed. Questionnaires and actigraphy encompass different concepts on emotions and sleep that have been grouped in five categories: subjective sleep (PSQI, ISI), objective sleep (actigraphy averages for each variable), comorbid symptoms (BDI, BAI, APS), emotion regulation ('ER'; DERS, CISS), and sleep-related thoughts and emotions ('STE'; TCQI-R, PSAS, FIRST, DBAS-16). Independent sample t-tests on each dependent variable (i.e., questionnaires, actigraphy) were conducted to characterize people with and without insomnia (independent variable; cf. hypothesis 1). Effect size and confidence intervals are reported for intragroup differences. For predictive variables, alpha inflation was addressed by applying False Discovery Rate (FDR) correction, implementing the adaptive Benjamini-Hochberg procedure. Only those results surviving FDR correction are reported.

To investigate the association between emotion regulation and objective sleep (cf. hypothesis 2), a multivariate analysis of variance was applied to results of those questionnaires measuring emotion regulation (ER) or sleep-related thoughts and emotions (STE) that showed differences between insomnia patients and controls, and investigate their effect on objective sleep parameters. Specifically, the analyses include the sleep variables as dependent variables (sleep efficiency [SE], sleep latency [SOL] and total sleep time [TST]), and the predictors as covariates in the SPSS command. In order to test multicollinearity problems, a correlation between our different predictors ($r < .70$) was performed to include our variables in the MANOVA. Aiming to analyze multiple dependent variables with multiple predictors, we performed a MANOVA, whilst ensuring that our multiple predictors were parametrized as continuous predictors in the statistical model, meeting the relevant MANOVA assumptions of 1) absence of multivariate outliers in dependent variables; 2) linearity and 3) absence

of multicollinearity in dependent variables. (Tabachnick, B. G., & Fidell, L. S., 2013; Tatsuoka, M. M. 1988). Anxiety and depression scores (BAI, BDI) were not included in the analyses as they are part of the insomnia disorder symptoms, while participants with clinical depression or anxiety symptoms had already been excluded from participation.

Estimates of effect sizes and parameters are used to discuss even non-significant contributing factors and to discuss the direction of the association, respectively.

3. Results

Results of sociodemographic data are presented in *Table 1*. Groups were equivalent in sample size, age and gender. In line with the definition of insomnia syndrome and its co-morbid symptoms, the insomnia group reported worse quality of sleep (PSQI; $p < .001$; $d = 3.12$), higher severity of insomnia (ISI; $p < .001$; $d = 1.18$), more symptoms of depression and anxiety (BAI and BDI; $p < .001$; $d = 1.64$ and 1.28 respectively) and were more predisposed to increased arousal (APS; $p = .022$; $d = .77$) than the control group. Actigraphy data showed longer sleep onset latency ($p = .038$; $d = .90$), shorter total sleep time ($p = .004$; $d = .69$) and lower sleep efficiency ($p = .009$; $d = .97$) in these younger adults with insomnia compared to controls.

Results of the possible predictors of affected objective sleep (*Table 2*), show, with regard to stress coping strategies and actions (CISS), that participants with insomnia have poorer emotion coping strategies ($p = .012$; $d = .84$) compared to controls (see *Table 2*), but did not differ from controls on the other subscales. In this questionnaire, this implicates that they are more likely to worry about their problems or feel anxious in a situation that they cannot overcome. In terms of sleep-related thoughts and emotions, young adults with insomnia had a higher probability of experiencing sleep difficulties in response to stress (FIRST; $p < .001$; $d = 1.57$), had more dysfunctional beliefs and maladaptive cognitions about sleep (DBAS-16; $p = .009$; $d = .88$), and higher cognitive pre-sleep arousal (e.g. worries; $p < .001$; $d = 1.58$) and somatic pre-sleep arousal (e.g. elevated heartrate; $p = .002$; $d = 1.08$)

compared to controls. There were no differences on thought control strategies (TCQI-R) between the two groups.

Results of the multivariate analyses are shown in *Table 3*. Since the somatic subscale of the PSAS was strongly associated with the cognitive subscale of the PSAS ($r = .7, p < .01$), we here chose to include the PSAS cognitive scale only, in line with the focus for this particular study on sleep-related cognition and sleep-related emotion regulation processes.

The results demonstrate that poor emotion management (CISS-subscale emotion; $p = .013$; $\eta^2 = .28$) and dysfunctional beliefs and attitudes about sleep (DBAS-16; $p = .005$; $\eta^2 = .32$), but not sleep reactivity (FIRST) or pre-sleep cognitive arousal (PSAS cognitive) are associated with worse objective sleep. More specifically, *Table 4* shows that poor emotion coping (CISS- subscale emotion) was associated with lower sleep efficiency ($p = .009$; $\eta^2 = .18$) and longer sleep onset latency ($p = .010$; $\eta^2 = .18$). In addition, poor attitudes and beliefs towards sleep (DBAS-16) were associated with lower sleep efficiency ($p = .004$; $\eta^2 = .18$) and longer sleep onset latency ($p = .001$; $\eta^2 = .27$).

4. Discussion

The results of this study show that maladaptive coping strategies in stressful situations (i.e., CISS) and beliefs or attitudes about sleep (i.e., DBAS-16) are associated with lower sleep efficiency and longer sleep onset latency in young adults with or without insomnia. The results are contrary to our expectations in insomnia patients. Based on previous findings, we had anticipated a relation with other factors typical of insomnia, such as pre-sleep arousal or sleep reactivity. It underlines the importance of not only investigating those emotion regulation measures and sleep-related thoughts and emotions related to insomnia subjective complaints, but also those factors that determine actual objective sleep quality, as those factors may play a more vital role in disruptions of next day functioning in younger adults.

A few studies show that erroneous beliefs and inappropriate attitudes towards sleep predict insomnia in adolescents and middle-aged adults, influenced by negative events [53,59]. These maladaptive sleep beliefs and behaviour have been associated with longer sleep onset latency, lower sleep efficiency and poorer sleep quality as reported in a sleep diary in middle-aged adults with insomnia [60]. Otherwise, significant reductions in dysfunctional sleep-related beliefs following CBTI in older adults also predicted improvement in several outcomes of sleep and daytime functioning [61], suggesting the importance of sleep-related beliefs in insomnia. On the contrary, emotion-focused coping did not predict insomnia in middle-aged adults [62] and was not associated with lower subjective sleep efficiency [10]. Interestingly, the emotion-focused coping indirectly influenced subjective sleep efficiency by increasing the stress impact and by higher cognitive pre-sleep arousal [10]. Concerning the studies using objective sleep measures, previous studies did not indicate a correlation between sleep difficulties and sleep-related thoughts and emotions in middle-aged and older people [28,53]. Only one study shows that thinking about sleep and the anticipated consequences of poor sleep are the strongest predictors of objective sleep onset latency on actigraphy [63]. This again emphasizes the importance of investigating this relation in younger insomnia patients.

However, it is surprising that responsiveness to pre-sleep arousal and sleep reactivity has not been associated with the sleep disturbances measured with actimetry. Indeed, previous studies show that worry, stress, rumination and pre-sleep arousal were associated with subjective or objective disturbed sleep in middle-age and older people [19,28,64,65]. Worry or stress levels were related to more wake after sleep onset and less total sleep time, sleep efficiency and percentage of REM sleep [19,64], whereas rumination, negative thoughts and cognitive pre-sleep arousal were associated with longer sleep onset latency and lower sleep efficiency [64–66]. In young adults, sleep difficulties are characterized by a longer sleep onset latency compared to older people [46,67], supporting the hypothesis that an association is possible between pre-sleep arousal and sleep onset latency. However, other studies did not find an association between pre-sleep arousal and sleep measures [63,68]. The methodological heterogeneity of studies (e.g., age, stressor type) does not make it possible to draw

definite conclusions on the cognitive-emotional processes involved in insomnia disorder, particularly in young adults whose insomnia is very often recurrent. Further research on the relationship between these different phenomena in the young adult population will allow us to understand the cognitive-emotional processes and ameliorate the treatment of young patients with insomnia.

As expected, young adults with insomnia had overall poorer quality and quantity of sleep compared to good sleepers in the current study, as measured by questionnaires and actigraphy [63,69,70]. Similarly, the presence of anxiety and depression symptoms, as well as predisposition arousal in young participants with insomnia is not surprising. Numerous studies show that symptoms of anxiety and depression and a higher arousal predisposition characterize patients with insomnia compared to those without insomnia [27,62,71].

Overall, previous studies suggest that poorer sleep quality and quantity is associated with more negative affect and less positive affect [74–76]. Only few studies have demonstrated that certain emotions (impulsivity, hostility, insecurity) were associated with poorer sleep efficiency, higher numbers of awakenings, shorter total sleep and higher sleep latency in adolescents and adulthood during polysomnography [77,78]. Other studies show that adults with insomnia (\pm 40 years old) have more difficulty regulating their emotions than good sleepers and this emotion dysregulation is associated to rumination trait and REM sleep macrostructure[34,79]. We did not find similar strong effects in the current younger population although a similar, non-significant effect was found here. This could be due to the fact that younger adults, independent of insomnia, may have fewer adequate emotion regulation strategies, and that these strategies improve with age, but only in those without insomnia. Stress resilience and improved stressful coping strategies developed over time have in fact been emphasized in the elderly, while a higher frequency of stress and negative emotions have been reported in young adults [9,43].

Several authors have shown that particular personality traits (e.g., neuroticism) or states (e.g., anger) are associated with sleep reactivity and arousal predisposition [27,31,34]. These moderate the

relationship between cognitive-emotional processes (e.g. rumination, intrusion) and sleep difficulties [29,31], suggesting they are premorbid characteristics of participants vulnerable to insomnia. One study demonstrates that people with insomnia and low worry report predominantly thoughts about sleep and the consequences of sleep loss, while people with insomnia and high worry report more heterogeneous thoughts about sleep and other issues (e.g., work, social) [80]. As such, future studies may provide more insight into the emotional-cognitive factors that characterize sleep disturbance by age and may contribute to the phenotyping of insomnia.

Our study has several limitations and strengths. First, small sample size and homogeneous socio-demographic characteristics (i.e., gender) limit the type of statistical analyses possible, which limits statistical power and generalisation of results. Studies on a larger and heterogeneous sample could confirm our results in young participants and would make it possible to compare them with larger samples of older insomnia patients. In addition, measures of emotions were based on subjective reports only and did not include objective measures such as electrodermal activity [81]. However, the inclusion of objective sleep measures applied in a young adult population are study strengths to be highlighted. Most studies are conducted in older populations with an average age between 40 and 60 years [17,27,28,34]. Understanding the link between emotions and sleep difficulties in young adults is therefore important in order to prevent risks of chronic insomnia and its limiting comorbidities.

In conclusion, the results of this study show that specifically emotion coping strategies and dysfunctional sleep-related beliefs and thoughts are linked to affected objective sleep in younger insomnia patients. The results allow a better understanding of emotional processes involved in sleep difficulties in young adulthood, which can lead to better prevention and treatment adaptations of young insomnia patients. For example, the development of specific age-related emotion coping programs could be added to current treatments to reduce the risk of transient insomnia, which may in the long term prevent the maintenance of insomnia. Further studies are needed to examine the specific role of

particular emotion regulation and coping processes in the development of sleep difficulties in young adulthood.

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Conflict of interest

Charles Morin has served as a consultant on the advisory board for Eisai, Merck, Pear Therapeutics, Sunovion and Weight Watchers, and has received research support from Idorsia, Canopy, Eisai, and Lallemand. All other authors declare that they have no conflicts of interest.

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Table 1.

Descriptive sociodemographic data between young adults without (control) and with insomnia.

	<i>CONTROL</i>	<i>INSOMNIA</i>			
	(<i>n=20</i>)	(<i>n=20</i>)			
	<i>M (SD)</i>		<i>CI 95</i>	<i>p</i>	<i>d</i>
<i>Sociodemographic</i>					
<i>Gender (f/m)</i>	19/1	18/2			
<i>Age</i>	26.7 (7.04)	27.70 (8.62)	[-0.6; 4.0]	.690	.12
<i>Subjective sleep</i>					
<i>PSQI</i>	1.8 (1.5)	9.6 (3.2)	[-9.4; -6.2]	.000*	3.12
<i>ISI</i>	2.4 (2.4)	15.3 (4.2)	[-15.1; -10.8]	.000*	1.18
<i>Objective sleep</i>					
<i>TST</i>	423.3 (29.0)	396.3 (47.6)	[1.57; 52.4]	.004*	.69
<i>SOL</i>	5.8 (3.5)	22.9 (26.4)	[-29.6; -4.7]	.038*	.90
<i>SE</i>	89.7 (3.3)	83.6 (8.3)	[2.1; 10.1]	.009*	.97
<i>Comorbid symptoms</i>					
<i>BDI</i>	1.4 (2.6)	6.65 (5.2)	[-7.9; -2.6]	.000*	1.28
<i>BAI</i>	2.4 (2.4)	7.8 (4.0)	[-7.5; -3.3]	.000*	1.64
<i>APS</i>	27.5 (6.7)	33.1 (7.7)	[-10.2; -0.8]	.023*	.77

CI95 = confidence intervals. *Abbreviations:* PSQI = Pittsburgh Sleep Quality Index; ISI = Insomnia Severity Index; TST = Total Sleep Time; SE = Sleep Efficacy; SOL = Sleep Onset Latency; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; APS = Arousal Predisposition Scale

Table 2.

Possible predictors of affected objective sleep

		<i>CONTROL</i>	<i>INSOMNIA</i>			
		(<i>n=20</i>)	(<i>n=20</i>)			
		<i>M (SD)</i>		<i>CI 95</i>	<i>p</i>	<i>d</i>
<i>Emotion regulation</i>						
<i>CISS</i>	<i>Task</i>	61.1 (7.1)	56.9 (6.6)	[-0.2; 8.6]	.061	.61
	<i>Emotion</i>	33.2 (7.2)	40.5 (9.9)	[-12.9; -1.7]	.012*	.84
	<i>Avoidance</i>	48.0 (12.3)	45.9 (9.7)	[-4.9; 9.2]	.542	.19
<i>DERS</i>	<i>Total</i>	70 (18.4)	82.7 (19.1)	[-18.4; 19.1]	.039	.68
<i>Sleep-related emotion and cognitions</i>						
<i>FIRST</i>		19.8 (4.7)	26.5 (3.8)	[-9.5; -4.0]	.000*	1.57
<i>DBAS-16</i>		3.7 (0.8)	4.7 (1.4)	[-1.8; -0.3]	.009*	.88
<i>PSAS</i>	<i>Cognitive</i>	12.8 (4.5)	21.7 (6.6)	[-12.5; -5.2]	.000*	1.58
	<i>Somatic</i>	10.3 (2.3)	13.2 (3.0)	[-4.6; -1.2]	.002*	1.08
<i>TCQI-R</i>	<i>Total</i>	69.8 (11.5)	71.35 (7.2)	[-7.7; 4.6]	.613	0.16

CI95 = confidence intervals. *Abbreviations* CISS = Coping Inventory for Stressful Situations; DERS = Difficulties in Emotion Regulation Scale; FIRST = Ford Insomnia Response to Stress Test; DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep; PSAS = Pre-Sleep Arousal Scale; TCQI-R = Thought Control Questionnaire Insomnia-Revisited.

Table 3.

Multivariate test and effect size for each emotion and cognition variable to predict a linear combination of three objective sleep variables.

<i>Variables</i>		<i>F</i>	<i>p</i>	η^2
<i>CISS</i>	<i>Emotion</i>	4.20	.013*	.28
<i>FIRST</i>		0.83	.488	.07
<i>DBAS-16</i>		5.11	.005*	.32
<i>PSAS</i>	<i>Cognitive</i>	1.47	.240	.12

Note: * Significant $p < .05$. Eta-squared (effect size): small ($\eta^2 = .01$), medium ($\eta^2 = .06$), large ($\eta^2 > .14$). Dependent variables: set of objective sleep variables sleep efficiency (SE), sleep onset latency (SOL) and total sleep time (TST). *Abbreviations:* CISS = Coping Inventory for Stressful Situations; FIRST = Ford Insomnia Response to Stress Test; DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep; PSAS = Pre-Sleep Arousal Scale;

Table 4.

Regression coefficients and effect size between each emotion-regulation variable and each

Variables		SE			TST			SOL			objec tive sleep varia bles.
		<i>B</i>	<i>p</i>	η^2	<i>B</i>	<i>p</i>	η^2	<i>B</i>	<i>p</i>	η^2	
CISS	Emotion	0.40	.009*	.18	-0.21	ns	.00	-1.11	.010*	.18	
FIRST		-0.34	ns	.05	-0.71	ns	.01	0.52	ns	.02	
DBAS-16		-2.75	.004*	.18	-2.42	ns	.00	9.11	.001*	.27	
PSAS	Cognitive	-0.29	ns	.07	-0.92	ns	.02	1.11	.039	.12	

Significant $p < .05$. Eta-squared (effect size): small ($\eta^2 = .01$), medium ($\eta^2 = .06$), large ($\eta^2 > .14$). Dependent variables: sleep efficiency (SE), total sleep time (TST), sleep onset latency (SOL). ns = non-significant. *Abbreviations:* CISS = Coping Inventory for Stressful Situations; FIRST = Ford Insomnia Response to Stress Test; DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep; PSAS = Pre-Sleep Arousal Scale;