



Original Article

Daily stress, presleep arousal, and sleep in healthy young women: a daily life computerized sleep diary and actigraphy study



Katja Winzeler^a, Annette Voellmin^a, Valérie Schäfer^a, Andrea H. Meyer^b, Christian Cajochen^c, Frank H. Wilhelm^d, Klaus Bader^{a,*}

^a Psychiatric Hospital of the University of Basel, Center for Specific Psychotherapy, CBT Unit, Wilhelm Klein-Strasse 27, 4012 Basel, Switzerland

^b University of Basel, Department of Psychology, Division of Clinical Psychology and Epidemiology, Missionsstrasse 60/62, 4055 Basel, Switzerland

^c Psychiatric Hospital of the University of Basel, Centre for Chronobiology, Wilhelm Klein-Strasse 27, 4012 Basel, Switzerland

^d University of Salzburg, Department of Psychology, Division of Clinical Psychology, Psychotherapy, and Health Psychology, Hellbrunnerstrasse 34, 5020 Salzburg, Austria

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ABSTRACT

Objective: Our study aimed to further elucidate the mediating role of presleep arousal in the relationship between daily stress and sleep by investigating subjective sleep quality and actigraphy-assessed sleep efficiency (SE) on both within- and between-participant levels in a sample of healthy young women.

Methods: Multilevel modeling was applied on electronically assessed data comprising 14 consecutive nights in 145 healthy young women to assess the relationship between daily stress, presleep (somatic and cognitive) arousal, and sleep on both levels between participants and within participants across days.

Results: Higher levels of daily stress were consistently and significantly associated with higher levels of somatic and cognitive arousal. Somatic arousal mediated the relationship between daily stress and worsened subjective sleep quality on the between-participant level, while cognitive arousal mediated the relationship between daily stress and worsened subjective sleep quality on the within-participants level. Unexpectedly, healthy young women showed higher SE following days with above-average stress with somatic arousal mediating this relationship.

Conclusions: Our data corroborate the role of presleep arousal mediating the relationship between daily stress and subjective sleep quality. Interestingly this effect was restricted to somatic arousal being relevant on interindividual levels and cognitive arousal on intraindividual levels. For young and healthy individuals who experience high stress and arousal, well-established cognitive-behavioral techniques could be useful to regulate arousal and prevent worse subjective sleep quality.

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1. Introduction

Sleep disturbances are widely prevalent and represent a momentous health problem in the general population. The point prevalence of primary insomnia is estimated to lie between 2% and 6%, though self-reported sleep disturbances in healthy populations range up to more than 40% [1–5]. The impact of insomnia and other sleep disturbances is known to be severe and includes reduced quality of life and well-being as well as impaired daytime-functioning and working ability, and thus is a potential risk factor for subsequent health problems [1,2,6]. Accordingly, insomnia and sleep difficulties are associated with increased work absenteeism and healthcare costs [2–4,7].

Various psychological factors, such as stress, daily hassles, rumination, and hyperarousal have been found to play an essential

role in the development of sleep disturbances [8–11], but the search for the specific roles and interplay among these factors is still ongoing. Our study aimed to further investigate the relationship between daily stress and hyperarousal and the influence of both factors on sleep and sleep disruptions.

Stress is one of the most common and well-known antecedents of insomnia and has been associated with impaired sleep in a variety of ways. Previous research shows that minor and major stressful events correlate with more sleep disturbances [12–15]. Major stressors usually are described as life events, such as severe illness or significant losses (e.g., death, divorce, work loss), and have been found to occur with greater incidence in the time preceding the onset of insomnia or to be associated with increased risk for the development of sleep problems [14–17]. Minor stressors usually appear with higher frequency and more likely on a daily basis (e.g., arguments, time pressure, work demands), and they have been associated with more disturbed sleep [12,13,18,19]. Additionally long-term stressors such as childhood adversities have been

* Corresponding author. Tel.: +41 61 325 5119; fax: +41 61 325 5671.

E-mail address: klaus.bader@upkbs.ch (K. Bader).

found to predict sleep problems several years later [20–24]. On a more acute daily basis, the experience of acute stress during the day was associated with impaired sleep the following night [25–30].

Well-established theories about hyperarousal and sleep postulate that physiological and cognitive arousal before bedtime is detrimental for sleep and contribute to the worsening of sleep problems [31,32]. Cognitive arousal consists of intrusive cognitions experienced as being uncontrollable, and physiological or somatic arousal is described as the perception of vegetative arousal (e.g., elevated heart rate, sweating [33]). In an integrative model, Morin [31] indicated that hyperarousal has a causal influence on sleep disturbances. In this model, the balance between sleep and wakefulness is regulated by the amount of arousal, and only low levels of arousal are compatible with sleep. Espie [32] further proposed that the inhibition of de-arousal processes in particular leads to the development of insomnia. Based on these theoretical frameworks, various studies tested the association between arousal and sleep and have confirmed that hyperarousal plays a major role in insomnia and sleep disturbances [10,11,34]. High arousal is more prevalent in poor than in good sleepers and can be measured on various physiological levels, such as sympathetic nervous system activation, hormone secretion, and high-frequency electroencephalogram activation [10,11,34]. In addition to the higher prevalence of arousal in insomniacs, there is evidence that high physiological and cognitive arousal also are prevalent in healthy populations and might constitute a preceding factor in the development of sleep disorders [35–37]. Even deliberately induced stress in the laboratory and the following increase in arousal at bedtime acutely worsen sleep in both poor and good sleepers [25,38,39].

Empirical studies on the relationship between stress and sleep on a day-to-day basis using within-participant data measured over time are still scarce. Garde et al. [40] found evidence for a bidirectional association between stress and sleep, indicating a self-reinforcing vicious circle. In a representative sample of the Danish population, higher ratings of stress at bedtime were associated with ratings of poor sleep the following night. In addition, higher ratings of poor sleep in the morning were associated with higher ratings of stress during the subsequent day [40]. In a study by Hanson and Chen [41], the daily number of stressors reported by healthy young adults was associated with subsequent sleep time when moderated by family risk. On days with elevated levels of stress, sleep time was significantly reduced the following night. Akerstedt et al. [30] studied the relationship between stress and sleep over a period of 6 weeks in 50 healthy adults. They found bedtime stress and worries to be the two main predictors of subjective sleep quality. Still the potentially mediating effect of arousal between stress and sleep was not tested in those studies.

Morin et al. [8] tested the relationship between all three variables and found a significant relationship between daytime stress and nighttime sleep, with presleep arousal playing a mediating role. The authors collected prospective daily paper and pencil measures for 21 consecutive days in men and women aged 19–60 years with insomnia in addition to good sleepers. Data showed that subjective stress during the day was a significant predictor of self-reported subjective sleep quality the following night for both groups and higher levels of presleep arousal mediated this relationship. Objective sleep measures were not used in this study [8].

Our study aimed to extend these findings on the relationship between stress, presleep arousal, and sleep considering various important aspects at the same time in a large healthy sample. Therefore, both subjective and actigraphic sleep measures were assessed and computerized diaries were used to enhance compliance and reliability compared to paper and pencil data [42]. Furthermore, multilevel modeling was used to evaluate the relationship

on both levels (between participants and within participants across days). More specifically, it was hypothesized that participants reporting a higher level of stress compared to others also would experience a higher level of presleep arousal and comparably worse sleep (between-participant level). On the within-participant level, we expected that participants reporting a higher level of stress on a specific day compared to their own mean would experience higher presleep arousal and worse sleep the following night compared to days with a lower level of stress. It was further expected that presleep arousal would mediate the relationship between daily stress and sleep.

2. Methods

2.1. Participants

Data were collected in the context of a larger ongoing study about acute stress, emotion regulation, and sleep in young adults. Data for our analysis included a 2-week ambulatory assessment of sleep with actigraphic sleep measures and sleep diaries. The sample included young and healthy women (mean age, 21.7 ± 1.6 [standard deviation {SD} years]) who were recruited using flyers posted at two schools for healthcare professionals in Basel, Switzerland, or by e-mails within the schools. Potential study participants contacted the study office by e-mail or phone. They were first sent a screening questionnaire with the following inclusion criteria: female sex, age range between 18 and 25 years, German speaking, and good health.

Exclusion criteria for all participants included physical or psychiatric illness, pregnancy, regular and heavy tobacco use (>5 cigarettes a day), use of illegal drugs, use of any medication interfering with sleep, and night shift work. In a first office appointment, participants were further screened on inclusion and exclusion criteria and provided written informed consent. All remaining study participants were of either Swiss or German (86.9%) or other European nationality (13.1%), who received monetary compensation for their participation. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee.

Out of 246 individuals who responded to the advertisements, 38 (15.4%) were excluded because they did not meet the inclusion requirements (men [$n = 5$]; not meeting age criterion [$n = 4$]; physical illness [$n = 6$]; psychiatric illness [$n = 3$]; medication [$n = 7$]; no regular sleep-wake cycle [$n = 7$]; heavy tobacco use [$n = 5$]; and not German speaking [$n = 1$]). Further 24 (9.8%) did not return the screening questionnaire. Of the 184 participants who were invited for the first appointment, 23 (12.5%) dropped out due to time restrictions or personal reasons and 12 (6.5%) did not respond to repeated invitations. Out of the 149 individuals who came to the first appointment, another three had to be excluded due to physical illness ($n = 1$), psychiatric illness ($n = 1$), and dropout ($n = 1$). The remaining 146 participants were finally eligible for the study and started the 2 weeks of assessment.

All of the 146 participants completed the study and returned their material after 2 weeks, which corresponds to a total of 2044 actigraphy-recorded nights. The data set of one individual could not be used due to incomplete information about sleep and wake times (sleep parameters could not be reliably calculated). Three participants had two nights each for which sleep parameters could not be reliably calculated, in which case the data of those two nights were excluded. Nine participants reported illness during the 2-week assessment. Therefore, all nights affected by illness including one night of convalescence were excluded from analysis (a total of 49 nights). This response left data of 145 participants with 1976 nights (96.7%).

2.2. Procedure

All appointments took place in the laboratory of the CBT outpatient clinic of the Psychiatric Hospital of the University of Basel, Switzerland. Study volunteers were asked to wear the actigraphy device on their nondominant wrist and to complete their sleep logs every morning immediately after rising. Additionally daily stress and presleep arousal were measured every evening immediately before bedtime. After completion of the 2 weeks of ambulatory assessment, participants returned all material and were given their monetary compensation.

2.3. Measures

2.3.1. Clinical interview

A structured clinical interview for psychiatric disorders (SKID I for DSM-IV; Structured Clinical Interview for *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, [43]) was used to assess the absence of psychiatric illness.

2.3.2. Actigraphy

Participants wore an ambulatory wrist actigraph (Micro Mini-Motionlogger, Ambulatory Monitoring Inc., Ardsley, NY) for 14 successive days and nights on the nondominant arm. The Micro Mini-Motionlogger is capable of detecting arm movement through the use of an accelerometer and represents a useful instrument for detecting sleep–wake cycles [44]. Data were analyzed using the Software Package Action4 (Version 1.05) and the ACT Millennium (Version 3.47.0.3) software (Ambulatory Monitoring Inc., Ardsley, NY). For the calculation of sleep parameters, the Action4 scoring algorithm provided by the producers (Ambulatory Monitoring, Inc. Ardsley, NY) was used. Sleep efficiency (SE) was derived from this analysis and was calculated as the ratio of total time asleep to time in bed. Additionally daily sleep logs were completed by the participants to cross-validate sleep start and end times. Outcomes from wrist actigraphy have been repeatedly compared to polysomnography (PSG) measures and represent a validated and unobtrusive technique which provides accurate estimates of global sleep parameters and sleep–wake identification [44–46].

2.4. Daily self-report measures

A menu-driven computerized questionnaire was developed to repeatedly assess subjective estimates of sleep quality, daily stress, and presleep arousal. Palm Tungsten E handheld computers were used as recording devices. Questionnaires were programmed and displayed using Pendragon Forms 5.0 software (Pendragon Software Corporation, Buffalo Grove, IL).

2.4.1. Subjective sleep quality

To assess subjective estimates of sleep quality, participants were instructed to fill in a computerized Likert-type scale ranging from 1 (very good sleep quality) to 5 (very poor sleep quality). For statistical analyses, the scale was reversed to have higher values for higher sleep quality. Participants completed this question in their handheld computers every morning after rising. Such Likert-type scales are widely used to assess subjective sleep quality and have been shown to be highly correlated with multi-item measures [47].

2.4.2. Presleep arousal

The Pre-Sleep Arousal Scale (PSAS) [33] contains 16 items with eight symptoms of cognitive (e.g., intrusive thoughts) and eight symptoms of somatic (e.g., sweating) arousal experienced at bedtime. Ratings range from 1 (not at all) to 5 (extremely). A total score from 8 to 40 is computed for both subscales with higher

scores indicating higher arousal. We used a German translation similar to that used by Giesemann et al. [48]. The PSAS has been broadly used and has shown satisfactory internal consistency and test–retest reliability [33]. Study volunteers completed the PSAS on their handheld computers every evening before bedtime.

2.4.3. Daily stress

The Daily Stress Inventory (DSI) [49] (German version [50]) is a 58-item self-report questionnaire assessing the occurrence and the impact of 58 possible daily stressors. Participants specify which events occurred and, in case of occurrence, the impact of every event is rated on a Likert scale (1 = occurred, but was not stressful; 7 = caused me to panic). Three scores can be derived: the actual number of events that occurred during the day (frequency), the sum of impact ratings (total impact of all events), and the average impact rating (sum of all ratings divided by the frequency). Considering that daily stress levels fluctuate, internal consistency and test–retest reliability are adequate [49,50]. The participants completed this questionnaire on their handheld computers every evening before bedtime.

2.4.4. The Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index (PSQI) [51] (German version [52]) was used to descriptively assess subjective sleep quality and potential sleep problems. Global scores of >5 distinguish poor sleepers from good sleepers [51,52]. The PSQI was administered twice: once at the beginning of the study period, examining the weeks prior to the study participation; and once at the end of the study period, examining the weeks of the study duration. This 2-fold application was used to investigate if sleep was significantly influenced by the study participation (i.e., if sleep changed during the study period).

2.5. Data analysis

Our study used daily data from 14 consecutive days. Because of the hierarchical structure of the data (days nested within participants) and with the aim to be able to predict fluctuations from night to night in the variables, a multilevel modeling approach was used. Multilevel models are an extension of the general linear model and do not require observations to be independent. Because of their autoregressive nature and hierarchical structure, multilevel models are especially useful to study time-dependent changes [53–56]. By applying this approach, we were able to examine the relationship between daily stress, presleep arousal, and sleep within and between participants. Analyses on the between-participants level addressed the question if participants who experienced higher levels of daily stress also reported higher levels of arousal and worse sleep compared to participants reporting lower levels of daily stress and arousal.

Analyses on the within-participant level addressed the question if individuals reported higher arousal and worse sleep on days when they also reported above average stress levels compared to their individual average level. Here we used a multilevel structural equation model (MSEM), which represents an extension of the multilevel model (for details see [54]). MSEM has been shown to lead to nonconflated estimates of between- and within-level components of indirect effects, thereby avoiding biased estimates which can occur when using more traditional multilevel models [54]. Note that in MSEM model based participant mean centering is used by default, i.e., the involved variables on the within-participants level denote deviations from each individual's mean. Our MSEM confers to a fixed slopes model, i.e., only intercepts were allowed to vary between participants but not slope parameters. Allowing random slopes would have led to a more complex model, typically requiring more time points per participant. Note that we

included time as additional predictor variable in all analyses to account for temporal trends in the mediator and outcome variables during the 14-day period (Fig. 1).

The following steps were required to establish mediation in our study: (1) the predictor (stress) positively affected the mediator (arousal), i.e., a was significantly higher than zero; (2) the mediator (arousal) negatively affected the outcomes SE and subjective sleep quality after controlling for the predictor (stress), i.e., b was significantly lower than zero; and (3) the indirect effect ab was significantly lower than zero. In contrast to the common and well-known approach by Baron and Kenny [57] in 1986, this method of establishing mediation does not require the total effect of the predictor on the outcome variable to be significant, i.e., c need not be significantly different from zero. This method enabled other mediating factors to influence the outcome in an opposite direction, which then could result in the total effect c to be equal zero, thereby obscuring the assumed mediating effect [58–61].

Preliminary analyses were performed using SPSS (version 19.0; SPSS, Chicago, IL) and R (version 2.15.2; R Foundation for Statistical Computing, Vienna) software packages. The MSEM was performed with Mplus (version 6.12; Mplus, Los Angeles, CA), which allowed assessment of the total, direct, and indirect effects on both hierarchical levels. Prior to analysis, data were checked for multiple outliers and were transformed to meet distributional assumptions. To identify highly influential data within our hierarchical dataset we used the R package Influence.ME [62]. For each analysis outliers defined by the Cook distance criterion were separately assessed and excluded. The number of outliers varied between 2 and 7. Subjective sleep quality and the stress variables number of events and sum of impact ratings were square root transformed, SE was log transformed, and cognitive and somatic arousal were reciprocally transformed.

There were missing data for 70 nights (12 participants) due to technical failure of actigraphs and for 27 nights (13 participants), as participants either forgot to wear the actigraph or to report their bedtimes and wake times. In addition, there were missing data for 39 nights regarding subjective sleep parameters, for 32 nights regarding arousal measures, and for 30 nights regarding stress measures, as participants forgot to fill in the questionnaires on their electronic diaries. This response left 1855 nights (90.8%) with complete data for all analyses with actigraphy-assessed sleep measures and 1916 nights (93.7%) with complete data for all analyses with subjective sleep measures.

3. Results

3.1. Stress, arousal, and sleep characteristics

Table 1 shows the means and standard deviations for age, daily stress, and presleep arousal, as well as the subjective and actigraphic sleep variables. The average PSQI score of 4.10 ± 1.8 (SD)

and a mean SE of $93.7\% \pm 5.1\%$ (SD) both indicate good sleep during the study period in our sample of healthy young women [51,52]. PSQI scores before the study period and at the end of the study did not significantly differ from one another ($P > .5$), indicating that sleep was not influenced on average by the study participation.

3.2. Between-participant effects

Table 2 shows the results of the multilevel mediator model for subjective sleep quality and actigraphy-quantified SE on the between-participant level. A strong positive relationship was found between stress and arousal in general (i.e., significant results for parameter a in all 12 analyses performed; P value of at least $<.001$). A significant association between presleep arousal and sleep (parameter b) was only found for the relationship between somatic arousal and subjective sleep quality but not for any other mediator-outcome pair. As a consequence, there was only a significant mediating effect (ab significantly higher than 0) of somatic arousal for the relationship between stress (as expressed by all three types of measures) and subjective sleep quality. Thus participants who reported higher average stress compared to others also reported higher somatic arousal and worse subjective sleep quality.

All other indirect effects tested did not yield significance. In contrast to subjective sleep quality, somatic arousal did not appear to play a mediating role between stress and actigraphic SE, and cognitive arousal also did not appear to play a mediating role between stress and both SE and subjective sleep quality. Note that the total effect of stress on sleep (parameter c) was negative and significant in all 12 analyses performed, but the direct effect (parameter c') was not significant in 11 of 12 analyses. This finding suggests that, although most mediating effects tested were nonsignificant, they still had an impact in that their inclusion in the model considerably reduced the total effect (compare c with c' in Table 2).

3.3. Within-participant effects

Table 3 shows the results of the multilevel mediator model for subjective sleep quality and actigraphy-quantified SE on the within-participant level. As seen for the between-participant level, there was a strong positive relationship between stress and arousal on the within-participant level (see significant results for parameter a in all 12 analyses performed; P value of at least $<.001$). Thus both cognitive and somatic presleep arousal also were increased on days with reported increased stress.

Significant associations between presleep arousal and sleep were found for the relationship between somatic arousal and actigraphy-assessed SE, though in the unexpected direction of higher SE, as well as for the relationship between cognitive arousal and subjective sleep quality with increased cognitive arousal leading to decreased subjective sleep quality (see parameter b). As a consequence, there was a significant mediating effect (ab significantly higher than 0) of somatic arousal for the relationship between stress as expressed by the sum of impact ratings, average impact rating, and (on a trend level, short of being significant) by the number of events and actigraphy-recorded SE. Additionally there was a significant mediating effect of cognitive arousal on the relationship between stress as expressed by all three types of measures and subjective sleep quality. Thus subsequent SE also was increased on days with reported increased stress. This effect was significantly mediated by somatic arousal. On days with reported increased stress, subsequent subjective sleep quality was decreased, which was significantly mediated by cognitive arousal.

Additional analyses revealed that the observed positive relationship between stress and SE could be explained by the fact that

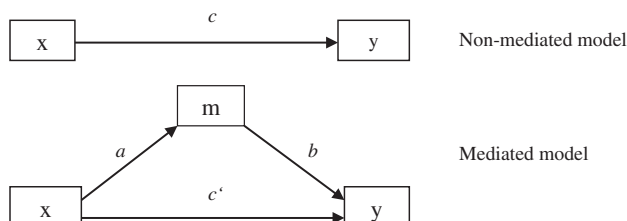


Fig. 1. Path diagram of the nonmediated (top) and mediated (bottom) effect of the predictor (stress) on the outcome (sleep). c denotes the effect of x on y in the absence of m ; c' denotes the effect of x on y , corrected for m ; a denotes the effect of x on m ; and b denotes the effect of m on y , corrected for x .

Table 1
Sample characteristics, including age, daily stress, presleep arousal, subjective sleep estimates, and actigraphic sleep efficiency.

N = 145	Mean	SD	Range
Age (y)	21.74	1.64	18–25
Daily stress inventory ^a			
Number of events	10.52	6.68	0–50
Sum of impact ratings	24.41	18.84	0–134
Average impact rating ^b	2.19	0.85	0–5
Pre-sleep arousal scale ^a			
Cognitive arousal	11.61	4.10	8–35
Somatic arousal	10.69	3.07	8–35
Subjective sleep estimates			
Sleep quality ^a	3.81	1.03	1–5
PSQI-Score prior to study	3.99	2.26	0–15
PSQI-Score at study end	4.10	1.86	0–11
Actigraphic sleep measure ^a			
Sleep efficiency (%)	93.66	5.11	54.7–100.0

Abbreviations: SD, standard deviation; y, years; PSQI, Pittsburgh Sleep Quality Index.

^a Mean values over all nights.

^b Sum of ratings divided by the number of events.

Table 2
Direct, total, and mediated effects of stress on subjective sleep quality and actigraphic sleep efficiency with mediators of cognitive and somatic arousal (between-individual level).

Sleep	Arousal	Stress	c (SE)	c' (SE)	a (SE)	b (SE)	ab (SE)
Actigraphic sleep efficiency	Cognitive	Number of events	−0.680 (.324) [*]	−0.402 (.393)	1.194 (.131) ^{***}	−0.233 (.230)	−0.278 (.279)
		Sum of impact ratings	−0.517 (.183) ^{**}	−0.429 (.267)	0.753 (.067) ^{***}	−0.117 (.264)	−0.088 (.199)
		Average impact rating	−1.133 (.497) [*]	−0.876 (.602)	1.462 (.253) ^{***}	−0.175 (.221)	−0.256 (.326)
	Somatic	Number of events	−0.685 (.323) ^{**}	−0.469 (.383)	0.848 (.125) ^{***}	−0.255 (.267)	−0.216 (.232)
		Sum of impact ratings	−0.526 (.181) ^{**}	−0.373 (.275)	0.557 (.063) ^{***}	−0.274 (.321)	−0.153 (.179)
		Average impact rating	−1.321 (.486) ^{**}	−1.107 (.622)	1.246 (.219) ^{***}	−0.172 (.266)	−0.214 (.336)
Subjective sleep quality	Cognitive	Number of events	−0.397 (.162) ^{**}	−0.199 (.204)	1.265 (.115) ^{***}	−0.156 (.109)	−0.198 (.137)
		Sum of impact ratings	−0.271 (.091) ^{**}	−0.193 (.133)	0.760 (.064) ^{***}	−0.103 (.125)	−0.078 (.095)
		Average impact rating	−0.818 (.258) ^{**}	−0.673 (.307) [*]	1.430 (.254) ^{***}	−0.102 (.101)	−0.145 (.148)
	Somatic	Number of events	−0.440 (.154) ^{**}	−0.040 (.210)	0.951 (.117) ^{***}	−0.421 (.142) ^{**}	−0.400 (.142) ^{**}
		Sum of impact ratings	−0.250 (.087) ^{**}	0.045 (.126)	0.602 (.061) ^{***}	−0.491 (.149) ^{**}	−0.295 (.094) ^{**}
		Average impact rating	−0.653 (.255) ^{**}	−0.234 (.278)	1.090 (.212) ^{***}	−0.385 (.110) ^{***}	−0.419 (.142) ^{**}

Abbreviation: SE, standard error.

Estimated values and standard errors for direct, total, and mediated effects.

c: total effect of stress and arousal on sleep; c': direct effect of stress on sleep, corrected for arousal; a: direct effect of stress on arousal; b: direct effect of arousal on sleep, corrected for stress; and ab: mediated effect of stress via arousal on sleep.

^{*} P < .05.

^{**} P < .01.

^{***} P < .001.

Table 3
Direct, total, and mediated effects of stress on subjective sleep quality and actigraphic sleep efficiency with mediators of cognitive and somatic arousal (within-individual level).

Sleep	Arousal	Stress	c (SE)	c' (SE)	a (SE)	b (SE)	ab (SE)
Actigraphic sleep efficiency	Cognitive	Number of events	0.813 (.197) ^{***}	0.815 (.198) ^{***}	0.489 (.056) ^{***}	−0.003 (.083)	−0.002 (.041)
		Sum of impact ratings	0.311 (.103) ^{**}	0.312 (.108) ^{**}	0.410 (.033) ^{***}	−0.003 (.088)	−0.001 (.036)
		Average impact rating	0.328 (.178)	0.273 (.192)	0.719 (.072) ^{***}	0.076 (.095)	0.055 (.069)
	Somatic	Number of events	0.815 (.197) ^{***}	0.735 (.203) ^{***}	0.494 (.054) ^{***}	0.160 (.097)	0.079 (.048)
		Sum of impact ratings	0.309 (.104) ^{**}	0.251 (.108) [*]	0.307 (.033) ^{***}	0.189 (.096) [*]	0.058 (.029) [*]
		Average impact rating	0.333 (.180)	0.249 (.184)	0.359 (.066) ^{***}	0.235 (.097) [*]	0.084 (.037) [*]
Subjective sleep quality	Cognitive	Number of events	−0.190 (.117)	−0.131 (.119)	0.488 (.054) ^{***}	−0.119 (.057) [*]	−0.058 (.029) [*]
		Sum of impact ratings	−0.097 (.060)	−0.047 (.062)	0.418 (.034) ^{***}	−0.121 (.056) [*]	−0.050 (.024) [*]
		Average impact rating	−0.033 (.111)	0.064 (.111)	0.715 (.076) ^{***}	−0.136 (.056) [*]	−0.097 (.042) [*]
	Somatic	Number of events	−0.178 (.118)	−0.147 (.121)	0.494 (.055) ^{***}	−0.062 (.060)	−0.030 (.030)
		Sum of impact ratings	−0.098 (.061)	−0.080 (.065)	0.319 (.032) ^{***}	−0.059 (.062)	−0.019 (.020)
		Average impact rating	−0.042 (.109)	−0.011 (.114)	0.404 (.065) ^{***}	−0.077 (.060)	−0.031 (.025)

Abbreviation: SE, standard error.

Estimated values and standard errors for direct, total, and mediated effects.

c: total effect of stress and arousal on sleep; c': direct effect of stress on sleep, corrected for arousal; a: direct effect of stress on arousal; b: direct effect of arousal on sleep, corrected for stress; and ab: mediated effect of stress via arousal on sleep.

^{*} P < .05.

^{**} P < .01.

^{***} P < .001.

increased stress was negatively related to wake time i.e., on days with elevated stress, as expressed by higher sum of impact ratings and number of events, participants exhibited significantly shorter wake time the following night ($P < .001$ for both stress measures) compared to days with lower stress. At the same time, increased stress compared to other days was not associated with total sleep time or time in bed the following night. Note that the total effects (parameter c) for the predictors number of events and sum of impact ratings were only slightly reduced, as shown by the corresponding significant direct effects (parameter c'). Therefore, additional mediators are likely to account for the relationship between stress and SE.

A significant positive temporal trend across the 14-day period was found for subjective sleep quality ($P < .001$), whereas no such amelioration in the course of the study was detected for SE and for somatic and cognitive arousal, the two mediator variables ($P > .05$ for all three variables).

4. Discussion

In our study, we examined the relationship between daily stress, presleep arousal, and sleep in a cohort of 145 healthy young women using multilevel structural equation models. Our results indicate and further extend previous findings that presleep arousal plays an important role in mediating the effects of daily stress on sleep quality and SE in healthy young women. On the between-participant level, our results confirmed a mediating role of somatic arousal but not cognitive presleep arousal between daily stress and subjective sleep quality. Healthy young female participants who experienced higher levels of daily stress compared to other young female participants also experienced higher somatic presleep arousal and reported worse subjective sleep quality. However, there was no mediating role of presleep arousal between daily stress and actigraphy-assessed SE.

On the within-participant level, results showed a mediating role of cognitive but not somatic presleep arousal between daily stress and subjective sleep quality. Participants reported worse subjective sleep quality after days with above-average stress and cognitive arousal relative to their own mean. Further, results showed a mediating role of somatic but not cognitive presleep arousal between daily stress and actigraphic SE in an unexpected direction. On days with higher levels of daily stress relative to their own mean, participants experienced higher somatic presleep arousal and showed subsequent higher SE during the following night as indexed by reduced wake time during sleep.

The mediating role of cognitive arousal between daily stress and subjective sleep quality on the within-participant level, as well as the mediating role of somatic arousal between daily stress and subjective sleep quality on the between-participant level, are in accordance with our hypothesis. However, Morin et al. [8] also found a mediating role of somatic arousal on the within-participant level, which we could not corroborate. On the between-participant level, previous findings showed that not only somatic but also cognitive arousal was associated with sleep disturbance [20,35,39]. Possible explanations for this discrepancy may be due to the fact that our study volunteer sample of healthy women differed from the sample of good sleepers of Morin et al. [8] regarding age and gender distribution.

The sample of good sleepers of Morin et al. [8] consisted of 27 men and women of all ages (mean age, 33.7 years [range, 19–60 years]), while our cohort consisted of 145 young women (mean age, 21.7 years [range, 18–25 years]). Additionally in accordance with our results, the authors mentioned that associations between arousal and subjective sleep quality were low [8]. Furthermore, it is important to bear in mind that our sample consisted of young and

overall good sleepers with no clinical sleep impairment, as confirmed by the low PSQI scores. In addition, our women showed generally low levels of stress and arousal, which might further explain why we did not find arousal to significantly influence sleep in several of our analyses. The use of computerized diaries in comparison to paper and pencil format also constitutes a significant methodic difference. Stone et al. [42] found that computerized diaries enhance compliance compared to paper and pencil format, as participants are aware that times of diary entries are recorded.

Based on the results of our study it seems that it is not higher somatic arousal on a daily individual level that influenced subjective sleep quality, but rather higher somatic arousal on the interindividual level. On the other hand, it is not higher cognitive arousal on the interindividual level that influenced subjective sleep quality, but rather higher cognitive arousal on a daily individual level. This result is plausible considering the role of de-arousal processes introduced by Espie [32], assuming that good sleepers do not have the same kind of negative sleep-related cognitive intrusions compared to individuals with sleep disturbances or insomnia; in addition, they might be able to de-arouse more sufficiently than others with the result that presleep cognitive activity does not influence subjective sleep quality enough to be noticed on the between-participant level.

Although participants reported worse subjective sleep quality in association with higher stress and somatic arousal on the interindividual level and with higher stress and cognitive arousal on the intraindividual level, this relationship does not seem to apply for actigraphy-assessed sleep data. Still this finding is not necessarily contradictory, as it is well-known that the subjective perception of impaired sleep is not always objectively measurable in actigraphic or PSG sleep data [63–66]. Further, it might be possible that higher levels of stress and arousal might not yet influence actigraphic sleep measures in a young and healthy sample, but it could still constitute a factor preceding the development of subsequent sleep disturbances [35–37]. Therefore, it would be interesting to compare our findings with a group of healthy sleepers with a broader age range to investigate if stress and arousal do influence sleep more strongly with higher age (i.e., if age moderates this relationship). Finally subjective sleep quality and actigraphy-recorded SE measure different aspects of sleep, which are not exactly comparable. Actigraphy-assessed measures detect sleep-wake cycles by an accelerometer and objectively quantify sleep duration and number of awakenings, among others, in relation to the time spent in bed. Subjective sleep quality estimates include a variety of perceived sleep features, such as consciously perceived sleep disruptions, well-being, and sleep inertia on awakening, all entering the total perception of sleep resulting in a subjective rating. Interestingly participants even showed higher SE on days with above average levels of stress and somatic arousal, which may indicate an adaptive response to stress.

We consistently found higher levels of stress to be associated with higher levels of cognitive and somatic presleep arousal, which is in accordance with current models of insomnia, all including some sort of interplay between stress and arousal in the development of insomnia [10,31,32,67]. However, higher stress and arousal were not associated with lower actigraphic SE, which does not fit into insomnia models on the first sight. Still all models require some sort of dysregulation or malfunctioning of the homeostatic or regulatory processes in the development of insomnia (e.g., de-arousal processes, sleep habits, chronobiologic timing, attentional focus, coping strategies) [10,31,32,67]. Therefore, it fits into the models that these homeostatic processes are still intact and sleep is not automatically impaired after experiencing higher levels of stress and arousal in a sample of healthy women without clinically significant sleep impairment. The results on the level of subjective sleep quality did partially fit the assumption of higher stress

and arousal being associated with worse subjective sleep quality. Still it was only somatic arousal being relevant on the interindividual level and only cognitive arousal being relevant on the intraindividual level. This finding remains difficult to explain and could be a topic for further research.

It is important to note that there is evidence suggesting that the relationship between daily stress, presleep arousal, and sleep is bidirectional. Garde et al. [40] examined the relationship between psychological arousal and sleep and found a self-reinforcing vicious circle, with sleep and arousal as a bidirectional association. For our analysis, we decided to focus on the effect of stress and arousal on following sleep, and thus concentrated on the direction that Morin et al. [8] examined in their analysis. Still the inverse effect of sleep quality or quantity of the previous night on stress during the next day reports might be equally important to fully understand the relationship between stress, arousal, and sleep, and therefore could be a topic of further investigation. The positive temporal trend of subjective sleep quality over the 2 weeks of assessment could imply an initial reactivity bias to the start of the study habituating with time. Still time trends were included into the model and therefore did not significantly influence our results of mediation analysis.

Our study bears some limitations: the acquisition of sleep data was based on actigraphy and subjective sleep measures. Despite the value of those data, studies using PSG would be useful to confirm our findings due to the high validity of PSG and its ability to consider additional dimensions of sleep. We deliberately examined a sample of healthy young women in the context of our larger ongoing study about acute stress, emotion regulation, and sleep in young adults. To generalize these results to the whole population of healthy adults, it will be necessary to replicate the study with good sleepers and a broader age range. The assessment of daily stress and presleep arousal was based on subjective and self-report measures. Therefore, effects of memory and selective recall due to retrospective bias cannot be excluded in our study. Still participants assessed their stress and arousal levels on the same day to keep retrospective bias at a minimum and compliance with adequate time of entry was improved by the computerized diaries. Our sample consisted of young women attending schools for healthcare professions. It cannot be excluded that there is a sample bias in the direction that only individuals who are particularly resilient and capable of the demanding work in healthcare chose this kind of occupational career.

Compliance was extraordinarily high in our sample, which supports the assumption that the sample was resilient to additional stress and dedicated to social commitment. In addition, participants with any psychiatric diagnosis or psychopathology were excluded. This exclusion might further explain the low levels of presleep arousal and high SE in our sample. Finally it should be noted that five (within-participants) and three (between-participants) of the 12 statistical tests performed for indirect effects on each level yielded significance, which was 8.3 and 5 times higher, respectively, than the value 0.6 ($=12 \cdot .05$) to be expected by chance based on an α of .05 and independent tests.

Despite these limitations, our study provides important knowledge regarding the relationship between daily stress, presleep arousal, and sleep. It confirms that arousal plays a mediating role between stress and subjective sleep quality, even in a healthy sample of young women. This mediating role was restricted to somatic arousal being relevant on the interindividual level and cognitive arousal on the intraindividual level. To the best of our knowledge, our study is the first to investigate between- and within-participant levels, along with subjective and actigraphy-assessed sleep outcomes in young adults. Actigraphic SE was not impaired by stress and arousal in healthy young women, who might even be able to compensate for days with above average levels of stress

and arousal during the subsequent night. This finding suggests that it might be useful to further explore the mechanisms causing this adaptive regulation to derive useful strategies for prevention.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2013.09.027>.

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References

- [1] Hajak G. Epidemiology of severe insomnia and its consequences in Germany. *Eur Arch Psychiatry Clin Neurosci* 2001;251:49–56.
- [2] Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. *Am J Psychiatry* 1997;154:1417–23.
- [3] Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev* 2002;6:97–111.
- [4] Stoller MK. Economic effects of insomnia. *Clin Ther* 1994;16:873–97.
- [5] Morin CM, LeBlanc M, Daley M, Gregoire JP, Merette C. Epidemiology of insomnia: prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. *Sleep Med* 2006;7:123–30.
- [6] Kyle SD, Morgan K, Espie CA. Insomnia and health-related quality of life. *Sleep Med Rev* 2010;14:69–82.
- [7] Westerlund H, Alexanderson K, Akerstedt T, Magnusson Hanson L, Theorell T, Kivimaki M. Work-related sleep disturbances and sickness absence in the Swedish working population, 1993–1999. *Sleep* 2008;31:1169–77.
- [8] Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. *Psychosom Med* 2003;65:259–67.
- [9] Guastella AJ, Moulds ML. The impact of rumination on sleep quality following a stressful life event. *Pers Individ Differ* 2007;42:1151–62.
- [10] Riemann D, Spiegelhalder K, Feige B, Voderholzer U, Berger M, Perlis M, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Med Rev* 2010;14:19–31.
- [11] Bonnet MH, Arand DL. Hyperarousal and insomnia: state of the science. *Sleep Med Rev* 2010;14:9–15.
- [12] Kashani M, Eliasson A, Vernalis M. Perceived stress correlates with disturbed sleep: a link connecting stress and cardiovascular disease. *Stress* 2012;15:45–51.
- [13] Jansson M, Linton SJ. Psychosocial work stressors in the development and maintenance of insomnia: a prospective study. *J Occup Health Psychol* 2006;11:241–8.
- [14] Healey ES, Kales A, Monroe LJ, Bixler EO, Chamberlin K, Soldatos CR. Onset of insomnia: role of life-stress events. *Psychosom Med* 1981;43:439–51.
- [15] Vahtera J, Kivimaki M, Hublin C, Korkeila K, Suominen S, Paunio T, et al. Liability to anxiety and severe life events as predictors of new-onset sleep disturbances. *Sleep* 2007;30:1537–46.
- [16] Cartwright RD, Wood E. Adjustment disorders of sleep: the sleep effects of a major stressful event and its resolution. *Psychiatry Res* 1991;39:199–209.
- [17] Bastien CH, Vallieres A, Morin CM. Precipitating factors of insomnia. *Behav Sleep Med* 2004;2:50–62.
- [18] Bernert RA, Merrill KA, Braithwaite SR, Van Orden KA, Joiner Jr TE. Family life stress and insomnia symptoms in a prospective evaluation of young adults. *J Fam Psychol* 2007;21:58–66.
- [19] Hicks RA, Garcia ER. Level of stress and sleep duration. *Percept Mot Skills* 1987;64:44–6.
- [20] Gregory AM, Caspi A, Moffitt TE, Poulton R. Family conflict in childhood: a predictor of later insomnia. *Sleep* 2006;29:1063–7.
- [21] Bader K, Schafer V, Schenkel M, Nissen L, Schwander J. Adverse childhood experiences associated with sleep in primary insomnia. *J Sleep Res* 2007;16:285–96.
- [22] Schafer V, Bader K. Relationship between early-life stress load and sleep in psychiatric outpatients: a sleep diary and actigraphy study. *Stress Health* 2013;29:177–89 [Published online ahead of print June 15, 2012].
- [23] Chapman DP, Wheaton AG, Anda RF, Croft JB, Edwards VJ, Liu Y, et al. Adverse childhood experiences and sleep disturbances in adults. *Sleep Med* 2011;12:773–9.
- [24] Greenfield EA, Lee C, Friedman EL, Springer KW. Childhood abuse as a risk factor for sleep problems in adulthood: evidence from a U.S. national study. *Ann Behav Med* 2011;42:245–56.
- [25] Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, et al. Acute stress affects heart rate variability during sleep. *Psychosom Med* 2004;66:56–62.

- [26] Kecklund G, Akerstedt T. Apprehension of the subsequent working day is associated with a low amount of slow-wave sleep. *Biol Psychol* 2004;66:169–76.
- [27] Bader K, Bauer C, Christen R, Schafer V. Sleep after stress induction: the role of stressful memory reactivation. *Stress Health* 2011;27:E143–51.
- [28] Sadeh A, Keinan G, Daon K. Effects of stress on sleep: the moderating role of coping style. *Health Psychol* 2004;23:542–5.
- [29] Gross RT, Borkovec TD. Effects of a cognitive intrusion manipulation on the sleep-onset latency of good sleepers. *Behav Ther* 1982;13:112–6.
- [30] Akerstedt T, Orsini N, Petersen H, Axelsson J, Lekander M, Kecklund G. Predicting sleep quality from stress and prior sleep – a study of day-to-day covariation across six weeks. *Sleep Med* 2012;13:674–9.
- [31] Morin CM. *Insomnia: psychological assessment and management*. New York, NY: Guilford Press; 1993.
- [32] Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. *Annu Rev Psychol* 2002;53:215–43.
- [33] Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the pre-sleep state—the development of the Pre-Sleep Arousal Scale. *Behav Res Ther* 1985;23:263–71.
- [34] Pigeon WR, Perlis ML. Sleep homeostasis in primary insomnia. *Sleep Med Rev* 2006;10:247–54.
- [35] Fernandez-Mendoza J, Vela-Bueno A, Vgontzas AN, Ramos-Platon MJ, Olavarrieta-Bernardino S, Bixler EO, et al. Cognitive-emotional hyperarousal as a pre-morbid characteristic of individuals vulnerable to insomnia. *Psychosom Med* 2010;72:397–403.
- [36] Jansson-Frojmark M, Linton SJ. The role of psychological mechanisms to insomnia in its early phase: a focus on arousal, distress, and sleep-related beliefs. *Psychol Health* 2008;23:691–705.
- [37] Drake C, Richardson G, Roehrs T, Scofield H, Roth T. Vulnerability to stress-related sleep disturbance and hyperarousal. *Sleep* 2004;27:285–91.
- [38] Haynes SN, Adams A, Franzen M. The effects of presleep stress on sleep-onset insomnia. *J Abnorm Psychol* 1981;90:601–6.
- [39] Wuyts J, De Valck E, Vandekerckhove M, Pattyn N, Bulckaert A, Berckmans D, et al. The influence of pre-sleep cognitive arousal on sleep onset processes. *Int J Psychophysiol* 2012;83:8–15.
- [40] Garde AH, Albertsen K, Persson R, Hansen AM, Rugulies R. Bi-directional associations between psychological arousal, cortisol, and sleep. *Behav Sleep Med* 2011;10:28–40.
- [41] Hanson MD, Chen E. Daily stress, cortisol, and sleep: the moderating role of childhood psychosocial environments. *Health Psychol* 2010;29:394–402.
- [42] Stone AA, Shiffman S, Schwartz JE, Broderick JE, Hufford MR. Patient compliance with paper and electronic diaries. *Control Clin Trials* 2003;24:182–99.
- [43] Wittchen HU, Wunderlich U, Gruschwitz S, et al. SKID-I: Strukturiertes Klinisches Interview für DSM-IV – Achse I: Psychische Störungen. Göttingen: Hogrefe; 1997.
- [44] Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP. The role of actigraphy in the study of sleep and circadian rhythms. *Sleep* 2003;26:342–92.
- [45] Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. *Sleep* 1995;18:288–302.
- [46] Sadeh A. The role and validity of actigraphy in sleep medicine: an update. *Sleep Med Rev* 2011;15:259–67.
- [47] Rodenbeck A, Cohrs S, Jordan W, Wortelboer U, Rütther E. Zusammenhang zwischen Schlafqualität, Morgen- und Tagesbefindlichkeit bei schlafgestörten Patienten. Relation between Sleep Quality, Morning and Daytime Well-Being in Sleep-Disordered Patients. *Somnologie* 2001;5:129–33.
- [48] Gieselmann A, de Jong-Meyer R, Pietrowsky R. Kognitive und körperliche Erregung in der Phase vor dem Einschlafen. Die deutsche Version der Pre-Sleep Arousal Scale (PSAS). *Z Klin Psychol Psychother* 2012;41:73–80.
- [49] Brantley PJ, Waggoner CD, Jones GN, Rappaport NB. A Daily Stress Inventory: development, reliability, and validity. *J Behav Med* 1987;10:61–74.
- [50] Traue HC, Hrabal V, Kosarz P. Alltagsbelastungsfragebogen (ABF): Zur inneren Konsistenz, Validierung und Stressdiagnostik mit dem deutschsprachigen Daily Stress Inventory. *Verhaltenstherapie und Verhaltensmedizin* 2000;21:15–38.
- [51] Buysse DJ, Reynolds 3rd CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193–213.
- [52] Backhaus J, Junghanns K, Broocks A, Riemann D, Hohagen F. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J Psychosom Res* 2002;53:737–40.
- [53] Singer JD, Willet JB. *Applied longitudinal data analysis*. Oxford, UK: Oxford University Press; 2003.
- [54] Preacher KJ, Zyphur MJ, Zhang Z. A general multilevel SEM framework for assessing multilevel mediation. *Psychol Methods* 2010;15:209–33.
- [55] Kenny DA, Korchmaros JD, Bolger N. Lower level mediation in multilevel models. *Psychol Methods* 2003;8:115–28.
- [56] Bauer DJ, Preacher KJ, Gil KM. Conceptualizing and testing random indirect effects and moderated mediation in multilevel models: new procedures and recommendations. *Psychol Methods* 2006;11:142–63.
- [57] Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol* 1986;51:1173–82.
- [58] Zhao XS, Lynch JG, Chen QM. Reconsidering Baron and Kenny: myths and truths about mediation analysis. *J Consum Res* 2010;37:197–206.
- [59] Hayes AF. Beyond Baron and Kenny: statistical mediation analysis in the new millennium. *Commun Monogr* 2009;76:408–20.
- [60] Krause MR, Serlin RC, Ward SE, Rony YZ, Ezenwa MO, Naab F. Testing mediation in nursing research beyond Baron and Kenny. *Nurs Res* 2010;59:288–94.
- [61] MacKinnon DP, Fairchild AJ. Current directions in mediation analysis. *Curr Dir Psychol Sci* 2009;18:16–20.
- [62] TheRDevelopmentCoreTeam R: a language and environment for statistical computing. Vienna: R foundation for statistical computing. <http://www.R-Project.org/>. Published 2012. Accessed January 3, 2014.
- [63] Feige B, Al-Shajlawi A, Nissen C, Voderholzer U, Hornyak M, Spiegelhalder K, et al. Does REM sleep contribute to subjective wake time in primary insomnia? A comparison of polysomnographic and subjective sleep in 100 patients. *J Sleep Res* 2008;17:180–90.
- [64] Means MK, Edinger JD, Glenn DM, Fins AI. Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. *Sleep Med* 2003;4:285–96.
- [65] Tworoger SS, Davis S, Vitiello MV, Lentz MJ, McTiernan A. Factors associated with objective (actigraphic) and subjective sleep quality in young adult women. *J Psychosom Res* 2005;59:11–9.
- [66] Jackowska M, Dockray S, Hendrickx H, Steptoe A. Psychosocial factors and sleep efficiency: discrepancies between subjective and objective evaluations of sleep. *Psychosom Med* 2011;73:810–6.
- [67] Harvey AG. A cognitive model of insomnia. *Behav Res Ther* 2002;40:869–93.