Sleep Polysomnography and Reported Stress Across 6 Weeks

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Abstract: Despite the common notion that stress impairs sleep there is little published data showing that sleep (polysomnography (PSG)) is impaired across several sleep episodes in individuals who complain of daily stress during the same period. The present paper aimed at investigating such a connection. 33 subjects had 3 sleeps recorded with PSG at home across 6 weeks and kept a sleep/ wake diary each day, including 3-hourly ratings of stress (scale 1–9). The stress ratings and the conventional PSG parameters were averaged across time. A stepwise multiple regression analysis showed that the best predictors of stress were Stage 1 sleep (beta = 0.49), latency to Stage 1 sleep (0.47) (adjusted for anxiety and age). Other sleep continuity variables had significant correlations with stress (reversed) but did not enter the multiple regression analysis. The correlation between stress before the start of the study and PSG data was not significant. It was concluded that moderately increased stress over a longer period of time is related to moderate signs of disturbed sleep during that period. This may be of importance when considering stress as a work environment problem.

Key words: Polysomnography, Subjective ratings, EEG, Diary

Introduction

The international classification of sleep disorders (ICSD[1]) lists stress as the main aethiologicial factor behind primary (psychophysiological) insomnia. A relationship between sleep and stress is also supported by cross-sectional questionnaire studies[2, 3] as well as by prospective studies[4, 5].

With regard to polysomnography (PSG − electroencephalography, electro-oculography, and electromyography) and stress the state of knowledge is less clear. Some early studies involving unpleasant films before sleep did not show much effect other than intensifying rapid eye movement (REM) sleep[6–8]. Among studies of real life stress, the effect of losing a life partner involved increased REM intensity[9], an impending exam resulted in reduced TST[10, 11] (and SWS in the latter study) or increased REM[12] and an impending coronary by-pass operation led to increased sleep fragmentation[13]. Beaumaster *et al.*[14], on the other hand, found no effect on PSG variables in unexperienced sky divers the night before the first jump.

Only a few studies have looked at real work stress. For example, it has been shown that marine officers being on call (on nights without actual calls to duty) have reduced SWS and increased stage 2 as compared to being off duty[15]. In another study early morning flights shortened sleep duration and reduced SWS. The latter was related to self-rated apprehension of the early awakening (measured at bedtime)[16, 17]. In a field study in a real life setting a day

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with moderately increased stress ratings showed reduced sleep efficiency, and increased Stage 0, and latency to stage 3[18]. In a similar real life study a week with moderately increased work stress (compared to a low stress week) showed decreased sleep efficiency[19].

All available studies thus far have focused on effects of acute stress, whereas the stress behind the diagnosis of insomnia refers to extended periods of stress. Thus, it seems necessary to investigate whether individuals that report stress across a longer period of time also exhibit indications of impaired sleep. The present study was designed to provide such information on real life stress and sleep and focused on three home PSG recordings over a period of six weeks and stress ratings daily across the same weeks. To the best of our knowledge, this is the first study of its kind.

Methods

A total of 52 subjects were approached, 46 accepted to participate and 33 subjects completed the full protocol. The loss of the 13 subjects were mainly due to not completing the polysomnographic recordings because of minor disease, travel, work schedules, technological problems, etc that interfered with participation. All were recruited through advertisements and contacts. All subjects were in good health and did not complain of insomnia. They received an economic compensation of approximately $180. The ethical committee of Karolinska Institute approved the study. All participants gave written informed consent.

Every participant filled in a self-administered questionnaire which included questions about social and demographic background, working/not working, smoking (yes/ no), alcohol consumtion (no, occasionally, 2–4 times/ month, 2–4 times per week or more, scored 0–3), use of pain killers / fever reducing medication (never − almost every day, range 0–4), physical activity (almost never, seldom, light physical activity, heavy training, competition level training, range 0–4), subjectively rated health (SRH) range 1–5, very poor − excellent)[20]. No participant used sedatives/hypnotics or antidepressants while four used anti-hypertensive medication. They also filled out the Hospital Anxiety/Depression scale[21], which is a less clinically oriented scale than other similar scales, and better suited for normal populations. It still retains a good correspondence with traditional clinical scales[21]. In the questionnaire was also included a rating scale for habitual (last 6 weeks) work stress and home stress on a scale from 1–5 (from none to very stressful). The two stress scales were averaged to form a habitual overall stress value. In addition, the well-established work demand scale of Karasek and Theorell[22] was used. The latter included questions on having too much to do, having to exert too much effort at work, etc.

The mean age was 44 years (range 28–69), 75% worked (the remainder were students or retired), 87% were married/cohabiting and 28% had small children (<7 years old), 51% lived in their own house (49% in an apartment), 40% had a university education, 66% were blue collar workers, all except one person worked daytime (5 participants occasionally worked evenings). The occupations represented were: nurse (3), physician (1), teacher (2), minister, social worker (2), research assistants (4), administrator (8), guard (2), and factory worker (2), students (adult education − 4), unemployed (2), retired (2). Mean bedtime was 23:22 (range 22:40–00:30) and mean time of rising 07:01 (range 5:00−9:24). 25% were smokers: Means for physical activity was 2.0 ± 0.7 (mean ± SD; range: 0–3), alcohol intake = 1.7 ± 0.9 (0–3), pain killers = 3.6 ± 1.1 (1–5), Body mass index (BMI) = 24.5 ± 0.45 (20.5–29.7), subjective health = 3.9 ± 0.9 (1–5), habitual stress = 3.4 ± 1.2 (1–5).

Diary data were collected daily over a period of 6 weeks. To obtain information on stress levels participants filled in a scale of momentary stress every three hours during wakefulness[23]. The scale (one item) ranges from 1 to 9, with 1 = no stress whatsoever and 9 = maximum stress imaginable. This scale has also been used in a study of longitudinal everyday work stress and cortisol[24].

Ambulatory polysomnographic recordings were carried out in the subject's home on two different occasions between two working days and one night after a working day and before a day off. Sleep was recorded using "Embla" recorders (Flaga HF®) with two EEG derivations C3–A2 and C4−A1, one chin electromyographic (EMG) derivation and two electro-occulogram (EOG) oblique derivations. Ag/AgCl electrodes were used. The sampling rate was set at 100 Hz. To reduce the impact of low frequency artifacts a 0.8 Hz high-pass filter was applied for one channel during scoring. This was carried out for all recordings to ensure that the amount of SWS would not be affected. Epoch-to-epoch correlation with the filter set at 0.5 and at 0.8 Hz respectively was r=0.94, range: 0.91–0.97. The changes mainly included less stage 4, replaced with slightly more stage 3 epochs, and also slightly more REM instead of wake. Sleep stages were scored visually in 30-s epochs according to Rechtschaffen and Kales[25].

Arousals were scored using the ASDA (American Sleep Disorders Association) criteria[26]. For an arousal to be
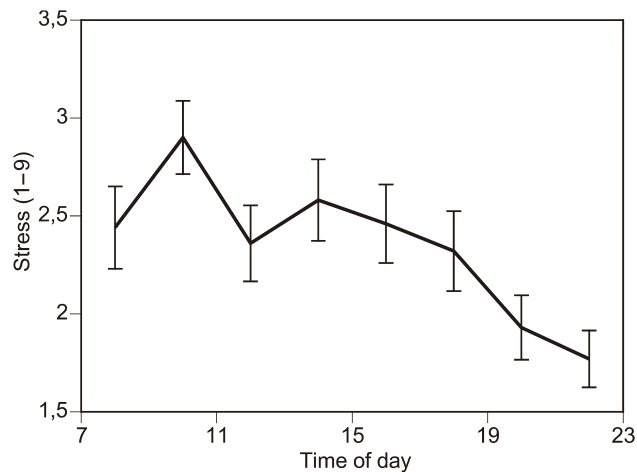
Fig. 1. Mean diurnal pattern of three-hourly stress ratings across all days of the six weeks (± SE).

Fig. 2. Plot of average stress vs Stage 1 sleep after adjustment for age, together with best fitting regression line.

scored it had to last for more than 3 s and for less than 15 s. At least ten seconds of uninterrupted sleep were required before every arousal for it to be scored, even if there were more than one arousal in the same epoch. An arousal was defined as an EEG shift to at least alpha activity from stages 2–4 or REM. During REM sleep also an increase in EMG-activity was required.

Multiple stepwise regression analyses were used to predict the average stress levels (mean of momentary stress ratings done every third hours awake for 6 wk). Only variables that showed a significant univariate relation to momentary stress levels were included. In addition, well-known confounders, such as age, depression, anxiety and gender were tried as predictors. Also, habitual stress at the start of the study was tried as a dependent variable to investigate if one early stress rating could substituted for the daily stress ratings.

Results

Figure 1 shows the mean diurnal pattern of rated stress for all days across the 6 weeks. The effect of time of day was highly significant ($F_{7,32} = 20.8$; $p<0.0001$; Huyhn-Feldt epsilon = 0.75), with highest ratings in the forenoon that fell towards the evening.

The mean momentary stress ratings were correlated with all background variables, but none of the correlations became significant except for HAD anxiety (positive) and subjective health (negative) (Table 1). None of the background variables correlated significantly with the PSG variables except for age and HAD anxiety (Table 1).
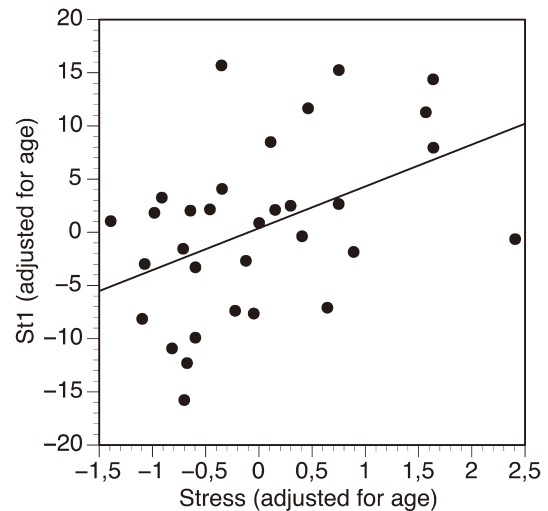
Table 1 presents the mean and dispersion measures for

all parameters in the multiple regression, as well as their intercorrelations with mean momentary stress ratings (controlling for age). Age was included as a control variable because of its strong relation to many sleep parameters[27]. The average stress levels showed significant correlations with total sleep time (TST), minutes of Stage REM, minutes of Stage 0, minutes of Stage 1, minutes of Latency to Stage 1, and HAD-anxiety.

Table 1 also shows that the stepwise multiple regression, after inclusion of the significant variables as predictors (age was forced into the analyses), yielded Stage 1 and latency to Stage 1, plus age and HAD anxiety as significant predictors ($F_{5,25} = 9.78$, $p<0.0001$). Higher stress was associated with higher levels of Stage 1, a longer Latency to Stage 1, higher anxiety, and lower age. Figure 2 illustrates the relation between Stage 1 and momentary stress ratings with control for age. The effect of age on each variable was removed through regression analysis and the resulting residuals around the regression lines (0 on the axes) entered in the new regression in the figure. The correlation amounted to $r=0.47$, $p<0.01$.

Since a single questionnaire question on habitual stress might be an economical way of predicting later polosymnography also this potential relation was investigated. Habitual stress was also tried as predictor in the multiple regression analysis in addition to the significant parameters but did not become significant. It was also tried as an alternative dependent variable with the PSG variables and age as predictors. The only significant correlation was with mean number of awakenings ($r=-0.36$, $p<0.05$) and in the stepwise multiple regression analysis the beta coefficient

Table 1.   Mean (± SE) and range for all variables, correlations of PSG variables with average momentary stress adjusted for age, and results from the stepwise multiple regression analysis of PSG vs. mean momentary stress

	Mean ± SD	range	r	p<	b	Beta	R2
Stress momen.	2.26 ± 0.98	1–4.55					
Age	44.4 ± 12.6	28–69	–0.31	0.05	–0.035	–0.51	0.09
HADanxiety	6.23 ± 4.96	0–16	0.43	0.05	0.049	0.26	0.15
SRH	3.84 ± .85	1–5	–0.36	0.05			
TST min	377 ± 52.6	276–529	0.48	0.01			
Sleep Eff %	85 ± 6	70–93	–0.24				
Stage 0 min	60.4 ± 33.4	17–169	0.47	0.01			
St1 min	18.9 ± 9.3	5.3–43.0	0.5	0.01	0.047	0.49	0.21
St2 min	226.5 ± 31.1	159.6–287.9	0.35				
SWS min	35.3 ± 26.6	0–98.3	0.07				
REM min	96.4 ± 20.6	46.5–134.5	0.45	0.05			
MT min	9.9 ± 4.6	1.1–18.0	–0.22				
Arousals	17.0 ± 27.2	4.1–165.8	–0.09				
Awakenings	12.8 ± 3.5	5.3–22.8	0.18				
Lat St1 min	12.6 ± 8.5	3.4–50.9	0.5	0.01	0.049	0.47	0.1
Lat St2 min	3.4 ± 3.7	0.6–22.4	0.33				
Lat St3 min	23.1 ± 11.5	10.2–60.3	0.23				
Lat REM min	74.5 ± 19.9	49.2–131.4	0.17				
Sum							0.55

MT=movement time, SWS=slow wave sleep (stages 3+4), REM=Rapid Eye Movement sleep, Lat=latency, min=minutes, BMI=body mass indes, SRH=subjectively rated health, mom=momentary, r=unadjusted correlations, p=level of significance, b=adjusted regression weight, beta=standardized adjusted regression weight, R2=proportion of variance accounted for

was β=0.32 with age β=0.44. The measure of habitual stress correlated r=0.56 (p<0.001) with the average of momentary stress across the six weeks.

For further help in understanding the results, product moment correlations were computed for some variables. Thus an increasing age was correlated with a reduced TST (r=−0.38, p<0.05), more Stage 0 (r=0.50, p<0.001), more Stage 1 (r=0.47, p<0.001, less SWS (r=−0.74, p<0.0001), and less REM (r=−0.48, p<0.01), but not with Latency to Stage 1 (r=0.09). Latency to Stage 1 and amount of Stage 1 did not correlate significantly (r=0.23, ns) but the two correlated significantly with Stage 0 (r=0.60, p<0.0001 and r=0.51, p<0.01, respectively).

Discussion

Rated momentary stress showed moderate mean levels across time, with higher ratings during the morning and the afternoon, and reduced levels in the evening. The correlation analysis showed that the higher momentary subjective stress level was related to higher amounts of Stage 1 sleep, a longer latency to Stage 1, and anxiety. The higher amount of Stage 1 and the extended sleep latency seem to suggest that perceived momentary stress is related to a moderate sleep disturbance.

In a previous study we found increased levels of stage 1 sleep and increased sleep latency in burnout (stress related) patients on long term sick leave (together with increased sleep fragmentation and decreased SWS)[28]. Even if there is very little consensus on polysomnographical criteria for disturbed sleep[29], increased sleep latency is one of the most well established indicators of disturbed sleep according to the International Classification of Sleep Disorders (ICSD 10)[30]. In two meta-analyses it has been suggested that a self-reported sleep latency of >30 minutes is a reasonable indicator of disturbed sleep[31, 32]. It is not clear if there is a "critical level" of the amount of Stage 1, but the level is increased in many situations of disturbed sleep[33]. It should be emphasized that momentary stress was also related to more Stage 0 during sleep (or WASO) in the univariate analysis. It is thus possible that Stage 0 is an equally interesting sleep variable in relation to stress as is Stage 1. In our previous study comparing high and low stress nights, wake after sleep onset (WASO, which cor-

responds to the amount of Stage 0) and was significantly increased, and sleep efficiency decreased, on the stressed night[18]. However, there is a possibility that disturbed sleep could cause an increased perception of stress[34]. However, the direction of causality was not possible to address with the present design.

SWS was not related to stress in the present study, nor in the two previous studies[18, 19]. Possibly more severe stress is required to affect SWS. Another observation is that REM sleep was positively correlated with stress in the univaariate analysis but was not strong enough to enter the multiple regression.

Total sleep time (TST) did not enter the multiple regression, but the initial correlation with momentary stress was rather high. This seems to be in conflict with the common notion that stress reduces sleep duration. But, there are no empirical data supporting the latter view. In fact there are some indications to the contrary. Thus, Dahlgren *et al.*[24] found increased during a week of increased stress, presumably, reflecting a need for sleep. Horne *et al.*[35] found increased levels of SWS after a day of visual and mental stimulation. It may also be of interest to consider the results of Meerlo *et al.*[36] who demonstrated increased "high intensity" sleep in rats after social stress. Kripke *et al.*[37] have argued that increased sleep duration is an indicator of problems or disease, whereas short sleep may be the opposite. This seems to suggest that the increased TST in the present study could be a compensatory response to stress, and its resulting impairment of sleep. However, one could also argue that the increased amounts of Stage 1 (and Stage 0) could result from an attempt to extend sleep beyond its natural limits. This might be true since such an extension would result in difficulties maintaining sleep, thus resulting in the appearance of superficial sleep, awakenings etc. One should remember that the setting was naturalistic and that the subjects could regulate their sleep duration at will, at least within the confines of work and other commitments.

The modest signs of disturbed sleep in this study may be due to the stress levels in the present population being rather moderate, and hence only having limited effects on sleep. As discussed previously, there is no well-established method of quantifying stress in absolute terms and there are no criteria for what levels of stress that might result in sleep impairment. This seems to be an important topic for future research.

Previous studies of stress and PSG also show rather modest links. This is probably in the nature of naturalistic studies; to be allowed into individuals' homes in periods of intense work stress (or other stress) may not be practically or ethically feasible. However, it has been demonstrated that being sick listed after long periods of work stress is associated with similar, but stronger sleep impairment[28]. Possibly, modest increases of reported work stress or disturbed sleep can be used as warning signals by employers.

One limitation of the present study is the modest number of participants and the study needs replication in a larger sample. Another problem is its cross-sectional character, which makes it impossible to infer causation. However, important factors like age, gender, anxiety, depression, physical activity, BMI, SRH, use of alcohol, smoking, and use of pain killers were controlled for or left out because of non-significant correlations with the dependent variable. Interestingly, baseline HAD anxiety added to the prediction of mean momentary stress of the sleep variables, without forcing them out of the regression. Thus, traitlike anxiety at the start of the study was not involved in the PSG relation to momentary stress. Habitual stress at the start of the study had a significant correlation with mean momentary stress, which was expected, and explained 25% of the variance. Still, it failed to enter the multiple regression against momentary stress. Furthermore it was only related to one PSG variable (awaknenings/h) and rather weakly. Thus, it seems unlikely that mean momentary stress represents trait-like stress, but rather temporary stress during a certain time period. And, levels during this time period seem to reflect physiological sleep impairment during the same time span.

The present study used a sample of convenience and is probably not representative of the average individual. However, care was taken to include a reasonable age range, both genders and to use no restrictions with respect to other background factors. Real-life studies of the present type will always require a compromise between feasibility and representativeness. The study also took into account a number of possible confounders. There might be other confounders not accounted for, but the results leave the impression that a state of (moderate) stress during a certain time period is associated with impaired physiological sleep during that same period. Causation cannot be determined with the present design but studies comparing sleep physiology between a single day with low stress with a day with moderate stress show similar impairment due to stress[18, 19].

In summary, the present study has shown that higher average stress across a 6-week period was related to a longer sleep latency, an increased amounts of Stage 0 and Stage 1 sleep, and a slight increase of total sleep time. Thus, a

modest increase of stress is related to a mild sleep impairment. Longitudinal studies are, however needed in order to describe the effect of varying periods of stress on sleep.

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