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Associations of Sleep With Everyday Mood, Minor Symptoms and Social Interaction Experience

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Summary: Although there are strong popular beliefs about the value of a good night's sleep, there is very little documented evidence of day-to-day relations between sleep and well-being. In this study, covariations between sleep and both prior and subsequent daily states of well-being were studied in a healthy, employed sample. Thirty volunteers used pocket computers to complete a daily sleep diary and self-rating scales of mood, minor symptoms and social interaction experience. These were recorded every 2 hours for 14 days except during sleep periods. A pooled regression analysis showed small but significant relationships between many of the sleep and well-being measures. Sleep appeared to be more strongly related to subsequent well-being than prior well-being. An earlier onset of sleep was associated with better mood and social interaction experience the following day and was a better predictor than sleep duration. This result was interpreted to be consistent with the phase angle model of chronobiologic mood disorders. In general, the results suggest that the sleep disturbances found in affective disorders may not be pathological but instead represent the extremes of normal relationships between sleep and well-being. **Key Words:** Sleep—Mood—Symptom—Social interaction—Circadian rhythm.

Is there any truth in the proverbs "One hour's sleep before midnight is worth two after", or "Early to bed, early to rise, makes a man healthy, wealthy and wise"? Common wisdom suggests the importance of a good night's sleep for well-being the next day, and yet there is very little scientific evidence that either supports this assumption or characterizes how it might work. This study, therefore, investigates the associations between naturally occurring changes in sleep behavior and both prior and subsequent self-reports of aspects of well-being.

Much of our current understanding of the psychological effects of sleep in humans is derived from studies of either total sleep deprivation, sleep reduction, sleep disturbances arising from external demands or sleep disturbance in psychiatric disorders. With the exception of the last, such studies have often concentrated on physical and performance effects rather than changes in well-being.

Horne (1) summarizes the findings of total sleep deprivation (TSD) studies as indicating that most of the effects of TSD are on the brain—particularly the cerebrum—and behavior, but that as yet there is no conclusive evidence that sleep is restorative for the brain. Sleep following TSD recovers most of the lost slow-wave sleep (SWS) and some of the lost rapid eye movement (REM) sleep. This led Horne (2) to propose that only the first 5–6 hours of sleep, which contain nearly all the total SWS and about half of total REM sleep, are essential for the brain; this he refers to as core sleep. The remainder is referred to as optional sleep and is considered dispensable. However, although optional sleep is not essential to cerebral functioning, loss of optional sleep does appear to affect motivation adversely, so that increased effort—and usually incentive—is therefore required to maintain performance. Also, despite the fact that the health consequences of sleep deprivation appear minor, many studies have demonstrated consistent decrements in a range of variables associated with well-being (3). For example, in a study of four men undergoing 205 hours of continuous sleep deprivation, Pasnau et al. (4) reported that the men exhibited increased irritability; anger; temporal and cognitive disorganization; regressive behavior; and decreased social cooperation, interest and competence.

Studies of sleep reduction over one or a few nights have consistently demonstrated impaired performance and alertness (5,6). The long-term effects of gradual sleep reduction are less clear but one study (7), in which two adults reduced their sleep by 30 minutes every 2

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weeks, demonstrated mood problems when sleep was restricted to below 5 hours. There is also indirect evidence that sleep reduction or extension may have serious long-term consequences for health. Two independent studies (8,9) have shown that mortality rate is significantly higher among adults who habitually sleep for less than 7 or more than 8 hours a night, even after controlling for such factors as age, gender, smoking, alcohol consumption and physical health.

Sleep disturbance is one of the major complaints of shiftworkers (10,11). The sleep of shiftworkers is usually shortened, particularly on morning and night shifts, and hence shiftworkers are likely to experience long-term partial sleep loss. Preferred sleep time and social pressures are probably two parts of the explanation for curtailed sleep in shiftworkers (12), but the changed timing of sleep is also important because ease of falling asleep (13) and duration of sleep (14) are known to be under circadian control. Shiftworkers also commonly experience reduced sleep quality and an increase in awakenings, and REM sleep often appears earlier in the day sleeps of nightworkers (15). It has been suggested that sleep deprivation, circadian desynchronization and social disruption are the major causes of the psychological and physical complaints of shiftworkers (16), such as increased fatigue, feelings of malaise, difficulty concentrating, interpersonal relationship difficulties and digestive problems.

Insomniacs also appear to experience reduced well-being as a consequence of sleep disturbance. Insomniacs either cannot initiate or maintain sleep and are liable to suffer secondary mood problems (17). Attempts to treat insomnia frequently focus on either reducing pre-bedtime anxiety or on regulating bedtime and waketime behavior (18).

Mood disturbance may also be a cause, as well as a consequence, of sleep disturbance. For example, in a study of the relationship between sleep and mood disturbing events, Cartwright (19) found that women who were more traditional in their gender role were more depressed and had shorter REM latency following a divorce.

Sleep disturbance has also been associated with a range of psychiatric complaints. In a study of the sleep and mood of 375 psychiatric out-patients, Crisp and Stonehill (20) found that sleep onset, but not bedtime, was later and sleep duration was shorter in most diagnostic groups including neurotic depression, but that the sleep period was earlier in endogenous depression. One of their conclusions was that there was a striking relation between sleep changes in the first half of the night and disturbances of mood.

Endogenous depressives, in particular, often show a shortened latency to the first onset of REM (21), and their sleep is usually shallower and more fragmented (22). These differences in sleep characteristics are thought to be part of the pathophysiology of depression, and not just secondary symptoms, because a number of interventions involving sleep such as sleep deprivation (23) and advancing sleep onset (24) have been found to alleviate depression, at least temporarily. Manic-depressive patients have also been shown to spontaneously advance their time of awakening as they emerge from their depressive phase (24).

Various theories have been proposed to account for these abnormalities in sleep regulation in affective disorders. The phase advance hypothesis, for example, asserts that during depression certain circadian rhythms, such as REM sleep, peak earlier relative to the sleep–wake cycle (24). Internal coincidence theory (25) added to the phase advance hypothesis by proposing that there is an early morning depression switch that is activated if it is brought within the sleep period. This accounted for the therapeutic benefits of sleep deprivation during the second half of the night. However, studies of seasonal depression have shown that the circadian rhythms of sufferers may be phase delayed—not advanced—during winter compared to normals. This is supported by the fact that morning bright light, which produces a phase advance of circadian rhythms relative to sleep, is an effective treatment for seasonal depression. This led Lewy and colleagues (26) to propose a phase typing hypothesis in which mood and sleep disorders are classified according to abnormalities in the phase angle between sleep and the endogenous circadian pacemaker. A more complex explanation has also been proposed that requires consideration of the separate phase angles of a circadian pacemaker consisting of a morning and evening oscillator (27).

However, other explanations of affective disorders have focused on different circadian parameters. For example, circadian rhythms may be less stable rather than changed in phase (28), or there may be a deficiency in the buildup of sleep pressure (process S) during wakefulness (29).

Taken together these studies strongly suggest that there are associations between sleep and well-being and that these associations may be mediated by the circadian system. In particular, sleep length, sleep timing, sleep quality, awakenings and REM latency have all been associated in one or more studies with changes in mood, social behavior, physical symptoms, cognitive symptoms and performance. However, these associations have only been found in abnormal and extreme conditions such as sleep restriction, altered work schedules and clinical disorders. There have been surprisingly few studies of the relations between sleep and mood in healthy subjects under normal conditions.

Kramer et al. (30) found significant relationships

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between physiological sleep parameters and changes in mood from night to morning. Significant correlations with sleep were found for the friendly, aggressive, clear-thinking, and sleepy subscales of mood, but not for the unhappy and dizzy subscales. The nonrapid eye movement parameters were more predictive of mood change than the REM parameters or total sleep time. A study by Berry and Webb (31) examined the relations between mood states and the previous and following night's sleep in a sample of aging women. Only sleep efficiency (% bedtime asleep) and REM latency were found to be reliably related to mood, and this was taken to indicate that sleep and mood are insulated from each other except in pathological conditions. The design of both these studies may have limited the results: the participants had limited choice over their sleep onset because they were all put to bed between 2300 and 2400 hours (in order to obtain electroencephalogram records); important variations in mood during the day may have been missed because mood was measured twice a day at most; analyses were based on between rather than within subject differences; and no account was taken of the contribution of yesterday's mood to today's mood or yesterday's sleep to today's sleep before looking at the covariation between sleep and mood.

Given these facts, the present study was designed to answer three questions. First, can associations between sleep and subsequent well-being be demonstrated in normal spontaneous daily behavior? If associations can be demonstrated then, second, which are the key factors in these associations? For example, is the timing of sleep more important than the amount of sleep in determining subsequent well-being, as suggested by the proverbs described above. Third, does well-being affect subsequent sleep instead of, or as well as, sleep affecting subsequent well-being?

These three questions were examined by assessing the relative strength of relationship of several parameters of daily sleep, such as timing and quality, with both prior and subsequent self-reports of well-being, such as mood and symptoms, provided by an employed sample every 2 hours during a 2-week period.

METHODS

Participants

Thirty volunteers, 16 females and 14 males, were recruited to the study via advertisements at their workplaces. The study was described accurately as a diary-based investigation into the everyday factors affecting mood and symptoms. They were paid £50 for taking part. The participants all had full-time day jobs, which were either clerical, managerial or professional. The average age of participants was 31.6 years (range 20–59). A prescreening ensured that none of the participants scored above the threshold for clinical symptoms (over 16) on the Beck Depression Inventory (32) and that none of them were taking medication that might affect their responses.

Procedures

The study covered 14 consecutive days, started on a Saturday and took place during January and February. The participants were trained in groups of five or less. Each participant recorded all their responses on a Psion pocket computer (Psion pLc, London, UK), which he/she carried around with them for the duration of the study. Totterdell and Folkard (33) gave details of the instrument and its use in intensive time sampling studies.

The pocket computer was programmed to give an auditory reminder to the participant, every 2 hours on the even hour during waking hours, to complete a series of tasks including self-ratings of current mood, symptoms and social interaction experience. These tasks took up to 5 minutes to complete. At the start of each day the participants were also required to complete a sleep diary on the pocket computer. The variables reported in this study were part of a larger battery of measures used in the study; further details of these measures are available from the corresponding author on request.

Correction of responses during the tasks was not possible after the *Enter* key had been pressed. All data were given a time stamp to record the time of entry. A pilot study, which used the four investigators as participants, was conducted prior to the main study to assess the practical aspects of using the pocket computer and completing the tasks.

Measures

Sleep diary. At the start of every day the participants completed a sleep diary, which included a number of questions about their previous night's sleep and required them to complete a visual analog scale (with 20 cursor positions) rating their quality of sleep. Although subjective records of sleep are not as reliable as polysomnographic records, subjective estimates of sleep length have been shown to correlate highly with polysomnographic estimates (34), and self-rated sleep quality has been shown to correlate negatively with activity during sleep (35).

A number of summary measures were derived from the sleep diary: sleep latency, number of awakenings,

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TABLE 1. *Descriptive statistics for sleep variables*

	Mean	SD	Minimum value	Maximum value
Latency (minutes)	18.45	23.20	0	225
Sleep onset	23.77	1.15	20.25	4.5
Sleep offset	7.76	1.54	4.18	14.5
Duration (hours)	7.98	1.39	4.68	12.92
Awakenings	1.76	2.26	0	13
Quality	12.51	4.43	1	20
Deviation from 8 hours	1.05	0.90	0	4.92
Delay in onset (hours)	0	1.09	−3.08	4.5
Prior wake length (hours)	16.07	1.43	11	20.25
S′	13.96	0.17	13.24	14.25

n = 414 (pooled observations).

sleep quality, sleep onset, sleep offset and sleep duration. Some secondary measures of interest were also constructed: absolute deviation from 8 hours of sleep, delay in sleep onset from one day to the next (delay = onset − previous night's onset), length of prior wakefulness (prior wake length = onset − previous night's offset), and the hypothesized recovery of process S during sleep [$S' = 14.3 - (14.3 - 7.96) * e^{-0.381 \cdot \text{duration}}$]. The parameters for S′ were taken from Akerstedt and Folkards' sleepiness model (36).

Mood. Self-ratings of current mood were measured using a series of visual analog scales labeled "0" at the extreme left and "++" at the extreme right, where 0 represented no experience and ++ maximum intensity of experience. There were 20 possible cursor positions along the scale. The mood adjective was centered above the scale. A positive and a negative item were selected from the three dimensions of mood identified in the University of Wales Institute of Science and Technology Mood Adjective Checklist (UMACL) (37): energetic arousal (alert, tired), hedonic tone (cheerful, depressed) and tense arousal (calm, tense). A further dimension for engagement was added (involved, disinterested). A single score for each dimension was obtained by subtracting the score for the negative item from the score for the positive item. For ease of identification, we have labeled the four dimensions by their positive items: alert, cheerful, calm and involved.

Symptoms. Using the same scale format described above, participants were asked to rate how much they had experienced a number of physical and cognitive symptoms over the previous 2 hours. Because the data were nonindependent, they required treatment prior to factor analysis. A daily average was calculated from each day's 2-hour responses for each of the 10 symptoms. The data from all the subjects were then pooled. Using multiple regression a residual series was derived for each symptom series, having first removed the following: variance due to differences in level of response between subjects by entering a dummy variable for each subject; serial dependency by entering the first order lag of the symptom; and linear practice effects by entering a variable for the number of days into the study. A principal components factor analysis with varimax rotation on the residual series converged on two factors with single items loading above 0.5 on only one factor: cognitive symptoms (difficulty concentrating, difficulty making decisions, difficulty with memory, absent-mindedness, and clumsiness) and physical symptoms (back pain, bodily aches, eyestrain, sick or nauseous and cold symptoms). Cronbach's reliability coefficient, alpha, was 0.8 for cognitive symptoms and 0.75 for physical symptoms.

Social interaction experience. Social interaction experience was measured by asking participants how much time they had spent alone in the last 2 hours, using scales labeled "0" and "2 hours" at the two ends, and then using the same scale format asking what would have been their ideal amount. Taken together these two items give us a fairly precise quantification of coping by avoidance. An extensive factor-analytic investigation by Amirkhan (38) found that avoidance was one of three fundamental coping strategies; the other two were problem solving and seeking social support. Items loading on the avoidance factor included "avoided being with people", and "wished that people would just leave you alone". Although these items are similar to our own, we decided on using our own format because we consider that the relevance of this experience may depend on the actual time spent with people. A supplementary measure—wanting more or less time alone—was constructed by taking the absolute difference between the actual and ideal score. This difference or mismatch score, therefore, represents a general or nondirectional dissatisfaction with the amount of time spent alone.

Alcohol. Participants were also asked to record the number of units of alcohol consumed in each 2-hour period. This measure was summed for each day. Alcohol is the most widely used psychoactive drug and is known to have effects on both mood and sleep (39), and hence this measure was included as a control variable.

Compliance

In total, the participants completed each of the 2-hour ratings 2,752 times and the sleep diary 417 times. Assuming that they could potentially complete the ratings eight times a day and the sleep diary once a day for 14 days, this represents an average compliance of 82% for the ratings and 99% for the sleep diary. The minimum compliance for any task for any participant was 50%.

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TABLE 2. *Results of pooled regression analyses with sleep variables as predictors of well-being*

Independent variable	Cheerful	Alert	Calm	Involved	Cognitive symptoms	Physical symptoms	Time alone	Want more time alone	Want more or less time alone
Latency									
Beta	−0.12	−0.06	−0.05	−0.04	0.02	0.04	−0.05	0.02	−0.01
Partial	−0.18**	−0.09	−0.07	−0.07	0.05	0.06	−0.06	0.03	−0.02
Onset									
Beta	−0.12	−0.17	−0.05	−0.06	0.06	−0.01	0.03	0.13	0.17
Partial	−0.13*	−0.19***	−0.06	−0.08	0.10	−0.02	0.03	0.16**	0.18**
Offset									
Beta	−0.08	−0.12	−0.06	−0.15	0.05	0.10	0.12	−0.05	0.12
Partial	−0.07	−0.11	−0.05	−0.15**	0.06	0.09	0.08	−0.04	0.10
Duration									
Beta	0.04	0.05	0.01	−0.05	−0.01	0.07	0.05	−0.13	−0.05
Partial	0.04	0.06	0.01	−0.06	−0.02	0.09	0.05	−0.16**	−0.06
Awakenings									
Beta	−0.12	−0.13	−0.05	−0.1	0	0.15	−0.03	0.01	0.03
Partial	−0.13*	−0.15**	−0.05	−0.13*	0	0.18***	−0.03	0.01	0.03
Quality									
Beta	0.12	0.25	0.08	0.09	−0.12	−0.17	0.01	0.01	0.02
Partial	0.17**	0.33***	0.11	0.15**	−0.24***	−0.27***	0.01	0.01	0.02
Deviation from 8 hours									
Beta	0	0.01	0.01	−0.02	0.04	0.08	−0.09	−0.03	0.02
Partial	0	0.01	0.01	−0.04	0.07	0.13*	−0.10	−0.04	0.03
Delay in onset									
Beta	−0.06	−0.09	−0.03	−0.05	0.04	−0.05	0.04	0.02	0.05
Partial	−0.09	−0.14*	−0.04	−0.08	0.08	−0.09	0.05	0.03	0.08
Prior wake length									
Beta	−0.07	−0.19	−0.01	−0.04	0.07	0.03	0.02	0.08	0.16
Partial	−0.08	−0.22***	−0.02	−0.06	0.12*	0.04	0.02	0.09	0.18**
S′									
Beta	0.03	0.05	0	−0.04	−0.03	0.02	0.09	−0.09	−0.05
Partial	0.04	0.06	0	−0.06	−0.06	0.03	0.09	−0.12*	−0.06
Prior R^2	0.633	0.611	0.643	0.714	0.825	0.700	0.456	0.651	0.578

n = 367.
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Data analyses

The 2-hour self-rating measures were averaged for each day of the study to provide one data point per day per participant for each measure. A pooled time series method was used for the analysis, using a least squares dummy variable regression model. See West and Hepworth (40) for details of the use of this type of analysis with daily experience data. For each dependent variable, the following variables were entered into the regression equation prior to entry of the independent variable(s):

1. A set of n − 1 dummy variables to represent each participant. This eliminates differences in level between different subjects' series and transforms the analysis into a within-subjects analysis.

2. A variable for the lag (previous day's value) of the dependent variable. This eliminates serial dependency in the series by assuming a first-order autoregression in the series.

3. A variable for the number of days into the study. This eliminates any linear effects arising from repeated response to a measure.

4. A set of six dummy variables for (n − 1) days of the week. This eliminates variance attributable to particular days of the week; for example, differences between weekends and weekdays.

5. A variable for the number of units of alcohol consumed. This controls for the possible effects of alcohol.

6. If the dependent or independent variable was for the ideal amount of time spent alone, then the variable for the actual amount of time alone was also entered. This controls for the level of social interaction, so that in the model the variable for the ideal amount actually represents wanting more (or less if negative) time alone.

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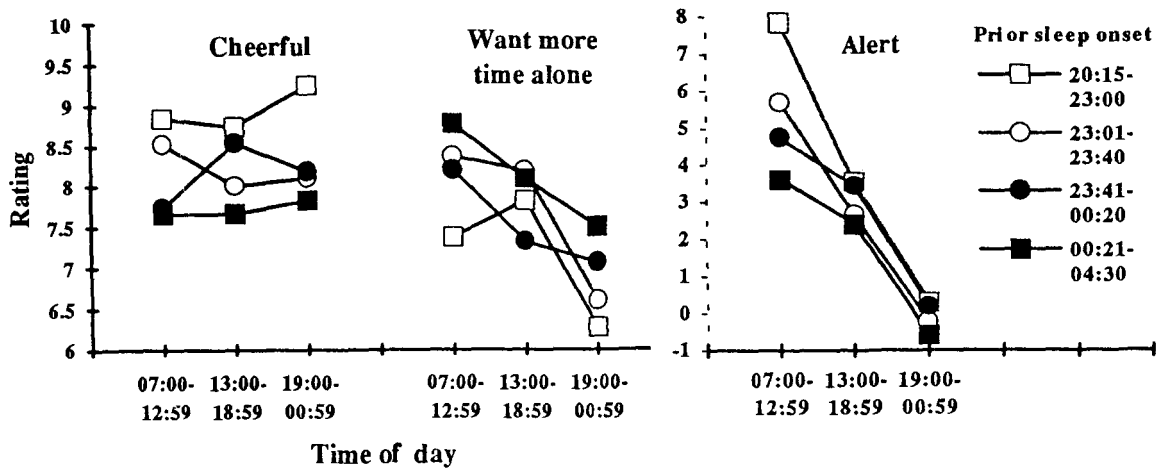

FIG. 1. Mood and social interaction experience at different times of day following different times of sleep onset.

RESULTS

Table 1 shows the mean, standard deviation and range of each of the sleep variables. Using the method of pooled regression analysis outlined above to examine selected associations among the primary sleep variables, it was found that a shorter latency [$\beta = -0.38$, R^2 change = 0.112, $F(1, 337) = 62.84$, $p < 0.001$], an earlier onset [$\beta = -0.15$, R^2 change = 0.01, $F(1, 337) = 5.01$, $p < 0.05$] and fewer awakenings [$\beta = -0.54$, R^2 change = 0.145, $F(1, 337) = 20.82$, $p < 0.001$] predicted a higher rating of quality of sleep; however, sleep duration did not predict quality of sleep [$\beta = 0.05$, R^2 change = 0.001, $F(1, 337) = 0.52$, ns]. It was also found that an earlier onset predicted a longer duration of sleep [$\beta = -0.67$, R^2 change = 0.203, $F(1, 336) = 208.84$, $p < 0.001$], which fits with previous findings on the relationship between sleep timing and sleep duration. No other associations were tested.

Sleep variables as predictors of well-being

Each of the sleep variables was regressed on each of the mood, symptom and social interaction variables. The sleep variables were analyzed in separate regression models because of the nonindependence of the sleep variables (partly demonstrated above). The results of the regression analyses are presented in Table 2. All of the well-being variables, except calmness and amount of time spent alone, were significantly associated with at least one of the sleep variables. Time of sleep onset, frequency of awakenings and sleep quality showed the greatest number (four or five out of a possible nine) of significant associations with well-being. In particular, a later sleep onset predicted lower cheerfulness and alertness, and the wish for more or less time alone the following day. Shorter latency, earlier onset, fewer awakenings and higher quality of sleep all predicted higher cheerfulness the following day. However, the largest partial regression coefficient was only 0.33.

The significant associations between sleep onset and cheerfulness, alertness, and wanting more time alone were examined in greater detail to see whether the effects were present during all or only parts of the following day. A multiple analysis of covariance was carried out on each of these three well-being variables, using the four quartiles of sleep onset time (2015–2300, 2301–2340, 2341–0020 and 0021–0430 hours) as levels of one factor, the three periods of the following day (0700–1259, 1300–1859 and 1900–0059 hours) as levels of a second factor, and all the sets of control variables that were used in the regression analysis, except the lag, as covariates. Cheerfulness showed a main effect of sleep onset [$F(3, 2,497) = 3.71$, $p < 0.05$]; wanting more time alone showed main effects for sleep onset [$F(3, 2,502) = 3.0$, $p < 0.05$] and time of day [$F(2, 2,502) = 14.61$, $p < 0.001$]; and alertness showed a significant interaction between sleep onset and time of day [$F(6, 2,497) = 4.28$, $p < 0.001$]. These results are illustrated in Fig. 1.

To determine if the associations between the primary sleep variables and the symptom and social interaction variables were mediated by mood, the relevant analyses were repeated but this time entering the four mood variables before the sleep variable. The significance of the regressions of sleep quality on cognitive symptoms and sleep onset on wanting more time alone dropped to $p < 0.1$; otherwise, there was no change. This suggests that the associations were largely independent of mood.

It is conceivable that sleep might affect the variability as well as the level of well-being. To examine this, the analyses for the primary sleep variables were repeated but this time using the within-day standard deviation of the well-being variables instead of the

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TABLE 3. *Results of pooled regression analyses with well-being variables as predictors of sleep*

Independent variable	Latency	Onset	Offset	Duration	Awakenings	Quality
Cheerful						
Beta	−0.16	−0.11	−0.06	−0.05	−0.22	0
Partial	−0.10	−0.01	−0.06	−0.05	−0.19***	0
Alert						
Beta	0.01	−0.06	−0.06	−0.06	−0.01	−0.11
Partial	0.01	−0.06	−0.07	−0.07	−0.01	−0.08
Calm						
Beta	−0.18	−0.06	−0.03	−0.03	−0.08	0.01
Partial	−0.13*	−0.06	−0.04	−0.03	−0.07	0.01
Involved						
Beta	0.04	0.10	−0.06	−0.08	−0.04	−0.03
Partial	0.03	0.08	−0.06	−0.08	−0.03	−0.02
Cognitive symptoms						
Beta	0.10	0.08	0.01	0	0.22	0.09
Partial	0.05	0.05	0.01	0	0.14**	0.05
Physical symptoms						
Beta	0.25	0.18	−0.03	−0.05	0.13	−0.05
Partial	0.19***	0.18***	−0.03	−0.06	0.12*	−0.04
Time alone						
Beta	0	0.03	0.06	0.06	0.13	−0.08
Partial	0	0.04	0.09	0.08	0.14**	−0.07
Want more time alone						
Beta	0.03	0.16	0.04	0.08	0.02	0.13
Partial	0.02	0.15**	0.04	0.07	0.02	−0.09
Want more or less time alone						
Beta	0.12	0.16	0	−0.02	0.06	0.07
Partial	0.09	0.15**	0	−0.03	0.06	0.05
Prior R^2	0.221	0.547	0.720	0.672	0.519	0.291

n = 374.
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

mean. Variability in cognitive symptoms was predicted by sleep latency [$\beta = 0.08$, R^2 change = 0.004, $F(1, 329) = 4.09$, $p < 0.05$], onset [$\beta = 0.11$, R^2 change = 0.006, $F(1, 329) = 5.32$, $p < 0.05$] and quality [$\beta = -0.08$, R^2 change = 0.005, $F(1, 329) = 4.24$, $p < 0.05$]; physical symptoms by sleep quality [$\beta = -0.11$, R^2 change = 0.008, $F(1, 329) = 7.89$, $p < 0.01$]; and alertness by sleep onset [$\beta = 0.13$, R^2 change = 0.008, $F(1, 329) = 5.34$, $p < 0.05$].

Well-being variables as predictors of sleep

The lag (previous day's value) of each of the well-being variables was regressed on each of the primary sleep variables. The results are shown in Table 3. There were no significant predictors of sleep duration, quality or time of offset, but there was at least one significant predictor of sleep latency, onset and frequency of awakenings. In particular, a higher level of physical symptoms predicted longer latency, later onset and more awakenings. However, the largest partial regression coefficient was only 0.19.

Although not central to this study, there were some interesting associations between the number of units of alcohol consumed and subsequent sleep. As expected, consumption of more alcohol was associated with later sleep onset [$\beta = 0.20$, R^2 change = 0.025, $F(1, 337) = 18.65$, $p < 0.001$] and reduced quality of sleep [$\beta = -0.12$, R^2 change = 0.010, $F(1, 337) = 4.53$, $p < 0.05$]. However, on controlling for sleep onset, the association between alcohol and sleep quality was non-significant. Alcohol was, however, significantly associated with shorter latency [$\beta = -0.18$, R^2 change = 0.018, $F(1, 337) = 8.92$, $p < 0.01$] and longer duration of sleep [$\beta = 0.08$, R^2 change = 0.004, $F(1, 337) = 4.23$, $p < 0.05$].

DISCUSSION

This study provides evidence that a number of measures of sleep are related to both prior and subsequent states of well-being—including mood, symptoms and social interaction experience—in a healthy, employed sample. This extends the findings of previous studies

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of the relationship between sleep and mood (30,31). For example, we found associations between sleep and both social interaction experience and alertness that may correspond to the associations Kramer et al. (30) found between sleep and mood subscales for friendly and sleepy. Unlike Kramer et al. (30), we also found an association between sleep and cheerfulness, but this seemed to depend on the timing of sleep, which was restricted in their study. Although Berry and Webb (31) found only two sleep variables related to mood, there are similarities with their results, because in both studies more significant relationships were found between sleep and the following day's well-being than between sleep and the previous day's well-being, and many of the relationships tested were nonsignificant and those that were significant were small in size.

Summary of well-being variables

Calmness was the only mood variable that was not predicted by any of the sleep variables; calmness did, however, predict reduced sleep latency. A higher level of cognitive symptoms was only predicted by reduced quality of sleep, but variability in cognitive symptoms was predicted by sleep onset and latency, as well as reduced sleep quality. Surprisingly variability in most of the other well-being variables was not predicted by the sleep variables. The amount of time spent alone was not predicted by any of the sleep variables, whereas the variables that represented wanting a different amount of time alone were predicted by at least one sleep variable. This suggests that the experience, and not the amount, of social interaction was being influenced by sleep. The amount of time spent alone did, however, predict the number of subsequent awakening episodes. The experience of physical symptoms was the best predictor of subsequent sleep disturbance as shown by increased latency, later onset and more frequent awakenings. This is presumably because physical symptoms keep people awake or wake them up. Importantly, the results demonstrated that the reports of symptoms and social interaction were not just a function of mood.

Summary of primary sleep variables

Higher self-rated quality of sleep was the best predictor of better mood and fewer symptoms the next day, but it did not predict social interaction experience. Perhaps the most interesting finding was that the timing of sleep onset was a better predictor of mood and interaction experience than either sleep duration or sleep offset. Specifically, a later onset was associated with reduced cheerfulness and alertness and increased dissatisfaction with the amount of time spent alone the following day. Figure 1 shows that the effects of sleep onset on cheerfulness and on wanting more time alone were maintained throughout the day, whereas the effect on alertness was only present in the morning.

Models of sleep and well-being

These results, namely that a modestly later sleep onset was associated with mild but adverse mood changes, are consistent with studies that have shown that delaying bedtime in healthy subjects produces REM and mood changes that mimic those of depression (41). One interpretation of these results is that a change in sleep onset alters the phase angle between sleep and the endogenous circadian pacemaker, and this is the source of changes in mood. A change in phase angle could also explain Berry and Webbs' finding (31) that reduced REM latency was associated with lower mood in a normal sample, because even though they did not vary sleep onset it is possible that circadian phase changed.

However, the change in phase angle in the present study would only occur when the time of onset changed because the endogenous circadian rhythms would quickly entrain to a specific time. Therefore, a phase angle interpretation would predict the delay in onset from one day to the next to be a better predictor than absolute time of onset: however, this prediction was not supported by the results. Possibly this was because large delays or advances in sleep were normally for social purposes that mitigated other effects on well-being.

Another possibility is that the effects on well-being were a consequence of third variables that also correlated with sleep timing. Specifically, people may go to bed later or earlier for reasons that are associated with changes in well-being. For example, going to bed earlier and getting up earlier are probably viewed as more virtuous in our culture—as implied by the proverbs—which may consequently uplift subsequent well-being. However, if this were true then sleep offset might have been expected to show similar results to sleep onset.

There have, however, been other explanations for the role of sleep in the affective disorders which may also be relevant to our results. For example, it has been suggested that circadian rhythms during depression are not phase advanced but instead are distorted or unstable (42) due to disruption of external social zeitgebers (43), hence explaining the similarities in symptoms to those experienced during enforced disrupted routines like shiftwork. In the present study, it is possible that going to sleep later was more disruptive (or perceived to be so) of social routines, such as work, and this reflected on well-being. It would have also

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been expected, however, that a change in duration of sleep, a change in sleep offset or a deviation in either direction from an expected 8-hour sleep would be similarly disruptive; this was not found to be so except for physical symptoms.

A deficiency in the buildup of process S during wakefulness has also been implicated in the changes in sleep seen during depression (29). The results of two experiments that reduced wakefulness in normals showed sleep changes consistent with this hypothesis (22). However, in one of the experiments the changes could have resulted from later sleep onset times. In the present study, the length of prior wakefulness (which should be tied to process S) was not a better predictor of well-being than sleep onset except for cognitive symptoms. However, it should be noted that the pattern of results for the two variables was very similar; this may reflect the fact that they are highly confounded.

Process S is thought to be reversed during sleep. However, neither total sleep duration nor the hypothesized recovery function for S was a better predictor of well-being than sleep onset. These results would also seem to argue against the view that sleep is depressogenic (23).

If, as we believe, a change in phase angle is the likeliest explanation of the relationship between sleep timing and subsequent well-being, then it is still unclear whether the frequently observed changes in REM sleep are merely a symptom of this phase angle or the source of changes in well-being.

Although we have found that an earlier sleep onset may have some benefits for well-being the next day, it is unclear what the cumulative effect of a number of "early nights" or "late nights" would be. This is a question that needs to be addressed in future studies, probably using an experimental intervention that requires the participant to engage in a series of "early" or "late" nights. Future studies might also address the possibility that the relationships found here show systematic intersubject differences (30), for example on gender or age.

Conclusion

The evidence from this study shows that there can be significant but small associations between changes in sleep and both prior and subsequent well-being on a day-to-day basis in a healthy sample. These associations are similar in kind, but much weaker than those found in many affective disorders. This suggests that the associations between sleep disturbance and reduced well-being found in psychological disorders may be epiphenomena rather than pathological. The evidence from this study also suggests that there may be a grain of truth to the proverbs referred to at the start of this paper; namely, going to sleep earlier, but not necessarily rising earlier, may show some subsequent benefits in well-being but the benefits are likely to be small and temporary.

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