Seasonality in human sleep

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Abstract. The timing of sleep and sleep EEG parameters in 10 healthy male subjects were investigated in four seasons under controlled conditions. The phase of nocturnal sleep was delayed about one and a half hours in winter as compared to that in summer. The duration of stage 4 sleep decreased and REM sleep increased significantly in winter compared with summer. The seasonality in the timing of sleep can be explained by photoperiodic time cues, but the changes in sleep EEG parameters are difficult to explain in terms of photoperiod.

Key words. Human sleep; slow wave sleep; REM sleep; circadian rhythm; photoperiod; seasonal variation; timing of sleep.

A number of functions in humans are known to vary on an annual basis [1–3]. However, in most cases it is not clear whether the seasonality is a direct response of the body to environmental factors such as ambient temperature, which is also changing throughout the year, or is based on an annual rhythm regulated by an endogenous mechanism. An endogenous annual rhythm may either be due to the circadian rhythm whose entrainment is systematically affected by photoperiodic time cues, or to a circannual clock. Seasonal changes have been found in the circadian period of core temperature rhythm and in the duration-of-sleep fraction in subjects under temporal isolation [4].

Recently much attention has been paid to the seasonality of human biology, especially in the field of psychiatry. For instance, photoperiod is postulated to be involved in seasonal affective disorder (SAD)[5,6]. In the present study we investigated the timing and architecture of sleep in four seasons under controlled conditions.

Materials and methods

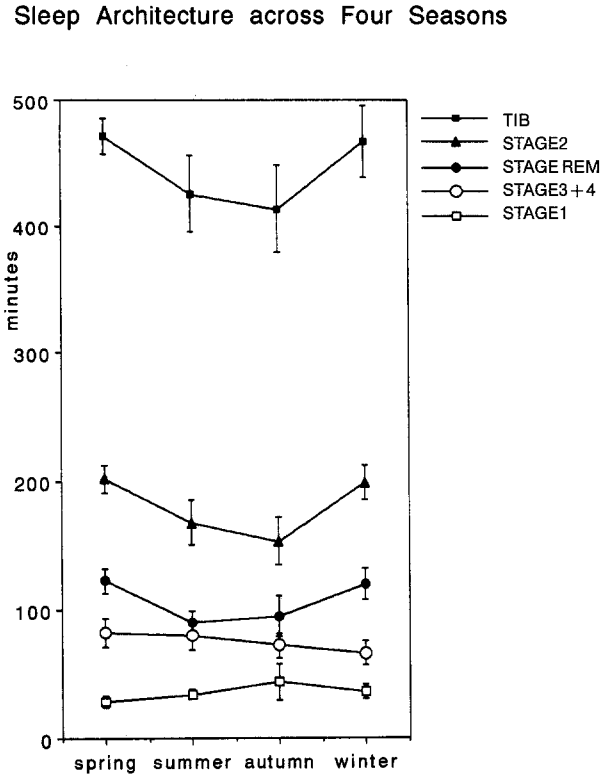
Ten male subjects (20–28 years old) who had lived in Sapporo City (latitude: North 43°, longitude: East 141°) for at least 3 years underwent a medical examination to exclude those with sleep or psychiatric problems. They stayed in an experimental facility which consisted of a living room, bedroom and bathroom, where temperature and humidity were kept within a relatively narrow range (20–24 °C; 60 ± 10%), from Friday evening to Monday morning in the winter of 1987, and in 1988 in the spring (March–April), summer (June–July), autumn (September–October), and winter (December). Natural daylight came into the living room through a large window facing south. The sound-proof bedroom had no window. The subjects were examined in pairs. They shared a living room during the daytime and for a couple of hours after supper, and spent their time reading books, listening to music, watching TV, playing games, etc. The subjects knew the time of day. No special time schedule was imposed on them. The bedrooms were separate. They went to bed when they felt sleepy, although daytime naps were not permitted. We instructed them not to communicate to each other after the attachment of EEG electrodes (about 20.00–21.00 h), nor to interfere with the sleep of their partner. They took meals (without alcohol) at their preferred times. The total calories per day (ca 2500 kcal) and the nutritional composition (carbohydrate, fat, protein etc.) were strictly controlled. They were allowed to turn artificial lights of about 300 lux on and off whenever they wanted. The lights were turned off during sleep. Polysomnography was recorded with an ambulatory cassette EEG system on the first and second nights [7]. Sleep EEG records of the second night were scored according to Rechtschaffen and Kales' criteria [8]. Rectal temperature was measured continuously by thermisters with a line. On the third day blood sampling was performed for 24 h. For statistical analysis, Friedman's nonparametric analysis of variance by ranks was used for comparison among four seasons, and the Wilcoxon signed rank test for comparison between two seasons.

Results

Table 1 shows the timing of sleep in four seasons. The average times of going to bed (go-to-bed) and getting-up (get up) were significantly different among the four seasons ($p < 0.005$, $p < 0.01$, respectively). Not only the

Table 1. Timing of sleep in four seasons. Friedman's analysis of variance by ranks (repeated measure) was performed during 4 seasons in ten subjects. A significant difference detected between two seasons (Tukey's test) is indicated by: [a] $p < 0.05$, [b] $p < 0.01$. The values are expressed by the mean (clock time; hr:min) with the standard error in minutes.

	Spring	Summer	Autumn	Winter	p
Go-to-bed	0:19 ± 13.2	0:07 ± 13.2[b]	1:00 ± 22.2	1:11 ± 14.4[b]	0.0043
Get-up	7:44 ± 18.6[a]	7:23 ± 16.8[b]	7:44 ± 17.4	8:49 ± 22.8[a,b]	0.0058



Averaged TIB and duration times of each sleep stage at the second night in four seasons. The values are expressed as means with standard error (n = 7).

Table 2. Sleep EEG parameters in summer and winter

		Summer	Winter	Significance
TIB (min)		428.9(75.4)	478.8(77.2)	NS
SPT (min)		412.6(75.1)	457.9(70.3)	NS
TST (min)		398.4(72.4)	445.5(68.1)	NS
SE (%)		92.9(3.8)	93.2(2.3)	NS
Sleep latency	(min)	13.5(18.2)	17.1(9.7)	NS
Stage W	(min)	3.5(-2.7)	5.1(6.4)	NS
MT	(min)	6.8(4.1)	5.9(1.9)	NS
1	(min)	34.5(9.1)	35.8(14.3)	NS
2	(min)	170.0(43.3)	210.6(47.1)	NS
3	(min)	59.7(19.8)	50.7(17.1)	NS
4	(min)	20.0(11.5)	13.4(12.2)	$p < 0.05$
3 + 4	(min)	79.7(25.8)	64.1(24.6)	$p = 0.054$
REM	(min)	95.5(27.2)	123.4(30.6)	$p < 0.05$
Percentage of SPT				
Stage W	(%)	1.7(1.6)	1.4(1.3)	NS
MT	(%)	1.7(0.7)	1.3(0.4)	NS
1	(%)	9.9(2.3)	8.4(2.7)	NS
2	(%)	44.4(4.6)	47.8(5.6)	NS
3	(%)	14.5(4.0)	11.1(4.0)	NS
4	(%)	4.8(2.9)	3.1(2.9)	$p < 0.05$
3 + 4	(%)	19.3(5.3)	14.2(6.1)	$p < 0.05$
REM	(%)	23.0(4.1)	26.9(5.4)	NS
REM latency	(min)	74.3(39.2)	71.2(34.4)	NS

TIB, time in bed; SPT, sleep period time; TST, total sleep time; SE, sleep efficiency, MT, movement time. Wilcoxon signed rank test (two-tailed). The values are expressed by means with standard deviations in parentheses (n = 8).

time of go-to-bed but also of get-up were more phase advanced in summer than in winter ($p < 0.01$). The time of get-up in spring was also earlier than that in winter ($p < 0.05$).

Five sleep EEG parameters across four seasons are presented in the figure (n = 7). Because of technical failures, a complete set of EEG records was not obtained in two subjects (case 8 and 9). They missed switching off the cassette data recorder in spring (case 8) and in summer (case 9). One subject (case 10) had a sleep problem during the experiments. Therefore the results of these subjects were omitted from the analysis.

TIB (time in bed) which is the duration from getting into bed till getting out of bed, stage 2 and stage REM were short in summer and autumn as compared to those in spring and winter. By contrast, stage 3 + 4 (slow wave sleep) was short in winter as compared to that in summer. However, Friedman's non-parametric analysis of variance did not reveal statistically significant difference among the four seasons in any sleep parameters. The failure is partly due to the shape of seasonal variation. Therefore we compared the above sleep EEG parameters between summer and winter (table 2). For this comparison, we added the results of case 8 to the analysis (n = 8). TIB, sleep period time (SPT), total sleep time (TST) and sleep efficiency (SE) were not significantly different between two seasons (Two-tailed Wilcoxon signed rank test)[9]. However, stage REM increased significantly in winter ($p < 0.05$) and the duration of stage 4 decreased significantly in winter ($p < 0.05$). The time of stage 3 + 4 tended to decrease in winter also. Percentage REM sleep showed no difference between two seasons, but the percentages of stage 3 + 4 and stage 4 significantly decreased in winter. REM latency was not different.

Discussion

The two main findings of the present study are 1) seasonal changes in the timing of sleep, and 2) seasonal variation of certain sleep EEG parameters. The timing of sleep was phase-delayed in winter by 1–1.5 h as compared with sleep in summer. Similar phase shifts under natural conditions have been reported previously by Inoue et al.[10] and Binkley et al.[11]. These authors correlated the seasonal shift of sleep timing to the photoperiod, especially to the time of sunrise. Although these studies did not excluded a possible effect of ambient temperature, their interpretation was partly supported by the present experiment in which direct effects of ambient temperature was eliminated. In Sapporo city, the time of sunrise at the summer solstice is 3:55 h and at the winter solstice is 7:05 h. Although the wake-up time was not directly influenced by the sunshine in the present study, photointensity in the morning which varies seasonally[12] may act as a photoperiodic time cue. The subjects may have received brighter morning light through the window in summer than in winter. Bright light in the morning resets the human circadian pacemaker[13, 14].

Sleep EEG parameters varied in four seasons even though direct effects of the environment on sleep were

minimized. Stage 4 sleep decreased and REM sleep increased in winter. Although the subjects slept well in winter, their sleep did not proceed to deeper stages. SWS is known to be influenced by several factors. Among them, the duration of previous wakefulness is well established[15]. Although the daily wakening period was slightly shortened in winter time, the extent was too trivial to explain the decrease of stage 4 sleep. Stage 4 sleep is reported to be increased by hard physical exercise[16]. However, the effect of exercise was not consistent[17] and our subjects had no chance to exercise vigorously in the facility. On the other hand, REM sleep has been reported to increase on a carbohydrate-rich diet[18]. In the present study the nutritional composition and the calorie-content of the meals were controlled, so that the direct effect of meal content on sleep may be excluded.

There are few studies in which sleep architecture has been analyzed throughout the year. Weitzman et al.[19] could not find any seasonality of sleep parameters in 7 subjects living in northern Norway. In their study, the wake-up time and bedtime were controlled, a condition different from ours. An increase of SWS (stage 3 + 4%) has been reported in the Antarctic winter[20]. The result seems to be opposite to ours, but the interpretation is not necessarily inconsistent. The increase of SWS in winter could be an expression of an annual rhythm in the British subjects of this study, which was not phase-reversed in the Antarctic.

An annual rhythm is an expression either of a photoperiodically changing circadian rhythm or of an endogenous circannual rhythm. As discussed already, the seasonal variation in the timing of sleep is explained by a photoperiodic change in the entrainment of circadian rhythm. Could such a seasonal change in the circadian phase explain the seasonal variation in sleep parameters? It has been hypothesized that SWS is coupled with the behavioral sleep rhythm (rest-activity cycle) and REM sleep with the circadian rhythm in deep body temperature[21]. These two oscillations are assumed to be coupled internally and keep a stable phase-relationship which guarantees a stable sleep architecture. If the oscillatory coupling is subject to seasonal variation, sleep architecture would change seasonally. This is theoretically possible if one assumes that the circadian temperature rhythm, or REM sleep rhythm, either does not phase-delay as much as the SWS rhythm in winter or alternatively phase-advance in winter. In these cases REM sleep would increase and SWS would decrease in a given sleep episode. In fact, there is a report which suggests a phase advance shift of the circadian temperature rhythm in winter[22]. However, our own results did not confirm this finding, and the subjects' circadian rhythms of rectal temperature and plasma melatonin showed a phase-delay shift instead of phase-advance in winter[23]. Thus the seasonal variation in sleep EEG parameters, especially in SWS and REM sleep, is difficult to explain by photoperiodic time cues. An increase of stage REM in winter may partly be due to the extended sleep period, though the difference of TIB was not statistically significant.

The present findings are of special interest with respect to the pathophysiology of SAD, because similar changes in sleep EEG parameters have been found in SAD patients[24]. SAD patients show hypersomnia, hyperphagia and low physical activity in addition to depressed mood. Milder forms of these symptoms are thought to be found widely in the normal population[25]. It is not known whether or not the changes in sleep EEG parameters in SAD are specifically related to the etiology of the disorder. The present findings, however, indicate that at least the changes in sleep EEG parameters are not specific to SAD, but are found in healthy subjects during the winter time. It would be a further research target to find out whether or not the changes of sleep architecture in winter induce SAD in some predisposed individuals.

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