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Research report

Winter and summer outdoor light exposure in women with and without seasonal affective disorder

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Abstract

Background: The annual decrease of daylight duration initiates a depressive phase in patients with seasonal affective disorder (SAD), and light therapy treats it. How much bright light exposure in winter and summer these patients actually receive may help understand the pathogenetic factors initiating SAD. *Methods*: During a week in winter and summer, women with and without SAD kept daily logs of the time spent outdoors, subjective sleep, and self-ratings of mood and alertness. *Results*: Compared with the winter depressive state, mood, alertness, and sleep of SAD patients improved in summer to control values, but did not correlate with the amount of light exposure. In summer, patients with SAD spent more time outdoors than controls. *Limitation*: Light logs – in comparison with light monitor measurements – may overestimate light exposure outdoors. *Conclusion*: Women with SAD do not spend less time outdoors in winter than controls, but spend more time outdoors in summer. *Clinical Relevance*: Patients with SAD show a high amplitude seasonal difference in outdoor light exposure. The susceptibility to winter depression may arise not from behaviourally-related lack of sufficient light exposure, but an increased vulnerability to the amount of light received. They may require more light than controls to remain euthymic (higher light exposure in summer, light therapy in winter). © 1999 Elsevier Science B.V. All rights reserved.

Keywords: Seasonal affective disorder; Light exposure; Seasons; Mood; Alertness; Sleep

1. Introduction

The seasons are still important for human behaviour and physiology, even though our 'civilized' life style usually masks its manifestation (Aschoff, 1981; Wehr et al., 1993). Many, if not all endocrine and behavioural functions are also modulated by the seasons (e.g. Lacoste and Wirz-Justice, 1989). The ancient subject of seasonality in human behaviour has regained scientific interest since the description of seasonal affective disorder (SAD) (Rosenthal et al., 1984). Patients with SAD appear to be sensitive to the decrease in available light after the autumn equinox, and can be successfully treated with bright artificial light (evidence summarised in Wesson and Levitt, 1998) or with regular exposure to natural light outdoors (Wirz-Justice et al., 1996). These findings lead to the assumption that clinical state in

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SAD patients is closely related to their light-oriented behaviour.

Classical light therapy uses light intensities of 2500–10,000 lux for a period of 0.5 h–2 h per day (Lam and Levitt, 1998). These intensities rapidly suppress melatonin secretion (Lewy et al., 1980) and phase shift the human circadian clock (Honma and Honma, 1988; Czeisler et al., 1989; Minors et al., 1991). Longer exposure, critical timing, and/or simulation of the dawn signal is required for lower intensities to have similar effects (e.g. Boivin et al., 1996; Wirz-Justice et al., 1998).

The amount of light indoors varies between 50 and 500 lux, much lower than that measured outdoors even with cloudy skies or rain in winter (at least 1000 lux). Depressed patients in general tend to retreat from social contact and thus spend much time indoors, receiving less bright light exposure. Our hypothesis concerning the photoperiodic decrease of light availability in autumn and winter as being a trigger for depression was that patients with SAD spend less time outdoors than controls. It was assumed that both SAD patients and controls spend more time outdoors in summer than in winter. We collected empirical data to test whether light-oriented behaviour is linked to mood, alertness and sleep in both winter and summer. These correlations could help understand the pathogenetic role of light in initiating winter depression.

2. Methods

2.1. Subjects

Patients were screened for seasonal symptoms with the Seasonal Pattern Assessment Questionnaire (SPAQ) (Rosenthal et al., 1987) and diagnosed as SAD according to DSM-III-R (American Psychiatric Association, 1987). They participated in a study (1988/89) of the antidepressant effect of one week of exposure to 'natural' light therapy (in the form of a daily 1 h morning walk outdoors, $n = 20$) or 'low dose' artificial light (0.5h, 2800 lux, $n = 8$), described in detail elsewhere (Wirz-Justice et al., 1996). During their depression in winter (before treatment) and, when possible, during spontaneous remission in summer, patients were interviewed by a psychiatrist and completed one week's self-ratings. Only data from the female SAD patients are included ($n = 22$).

We recruited 54 healthy control women to be in the same age range (1991/92). These women were asked to completed the SPAQ and one week of the same self-ratings in winter (November–March) and summer (June–mid-September). The subjects had 6 months$\pm$31 days between seasonal measurements.

2.2. Instruments

The following measures were collected in winter and summer:

1. Self-ratings of depression (von Zerssen and Koeller, 1976) to exclude any subject with high values in the control group, and to compare the clinical state of SAD in winter and summer.
2. Daily light logs throughout the entire week, noting when and how long they were outdoors during the daytime (accuracy of 15′ intervals).
3. Throughout an entire day (SAD patients over two days, averaged), mood and alertness ratings with a 100 mm visual analogue scale (VAS) (Aitken, 1969) from awakening (first rating), then every 2 h until they went to bed (last rating).
4. Daily sleep and nap logs throughout the entire week to estimate subjective sleep latency, sleep onset and wake-up time, frequency and duration of awakenings, as well as sleep quality.

2.3. Statistics

For analysis of the light logs, raw data were averaged in half-hourly values to create a diurnal profile and summed over the whole week (including weekends). The influence of light exposure on VAS and sleep variables was calculated by analysis of covariance with light exposure as covariate. The analysis was checked by a factorial ANOVA using light as a factorial variable (median division). Separate calculations were carried out for light logs in winter, summer, and the winter–summer difference. Power analysis was calculated for the outdoor light exposure (Cohen, 1988).

Pearson's product-moment correlation (r) was calculated and controlled by Spearman's rank correlation (Rho) for variables not showing normal distribution.

Differences between the groups and the influence of the seasons were calculated by 2- or 3-way ANOVA with the factors: group (controls vs. SAD), season (winter vs. summer), and in the case of VAS the repeated factor (time course over the day). Huynh-Feldt statistics were used to adjust the covariance matrix for violations of sphericity. Huynh-Feldt's p-values were based on corrected degrees of freedom, but the original degrees of freedom are reported. Post-hoc comparisons were performed with Duncan's multiple range test. Only results with significance level $p < 0.05$ are reported.

3. Results

3.1. Populations studied

Twenty two women diagnosed as SAD (age range, 18–80 yr; 44.6 yr±16.4) participated in both seasons. These drug-free patients did not differ from the complete sample of the study ($n = 28$) with respect to age, initial depth of depression, or clinical response to light (Wirz-Justice et al., 1996). Their SPAQ score for seasonality was 14.1±3.9 ($n = 21$; one patient missing).

Forty seven healthy women (age range, 19–70 yr; 45.5 yr±18.0; no difference from SAD ($F_{(1,67)} = 0.04$, n.s.)) participated in both winter and summer. Their SPAQ score for seasonality was 6.8±4.0 in winter, 7.0±4.3 in summer ($F_{(1,46)} = 0.7$, n.s.). Depending on the variable and the completeness of the data collected, the value of n varied. In particular, the more difficult two hourly self-ratings of mood and alertness were not always entirely filled out, so that the number of completed paired ratings for the winter-summer comparison was reduced by half.

As expected, SAD patients showed a seasonal difference in the von Zerssen depression self-rating scores, whereas controls were not depressed (SAD: winter, 22.2±9.9; summer 8.4±7.0, $F_{(1,19)} = 24.3$, $p = 0.0001$; Controls: winter, 4.2±2.9; summer, 4.8±3.6, $F_{(1,40)} = 2.4$, n.s.).

3.2. Light logs

Fig. 1 shows the time spent outdoors per 15′ bin throughout the day over a week in winter and summer in control subjects and SAD patients. A 3-way ANOVA revealed a significant interaction ($F_{(31,1953)} = 2.53$, $p = 0.002$). Post-hoc comparisons showed no significant differences between controls and SAD in winter (power = 0.57). In summer, patients with SAD spent more time outdoors in the afternoon than controls ($F_{(1,64)} = 9.68$, $p = 0.003$; power = 0.83). As expected, both groups spent more time outdoors in summer than in winter ($F_{(1,64)} = 61.8$, $p = 0.0001$; power = 0.80).

3.3. Mood and alertness

Fig. 2 compares the diurnal profiles of 2 h-ratings of mood and alertness throughout the day between the seasons.

In SAD patients, mood was significantly lower in

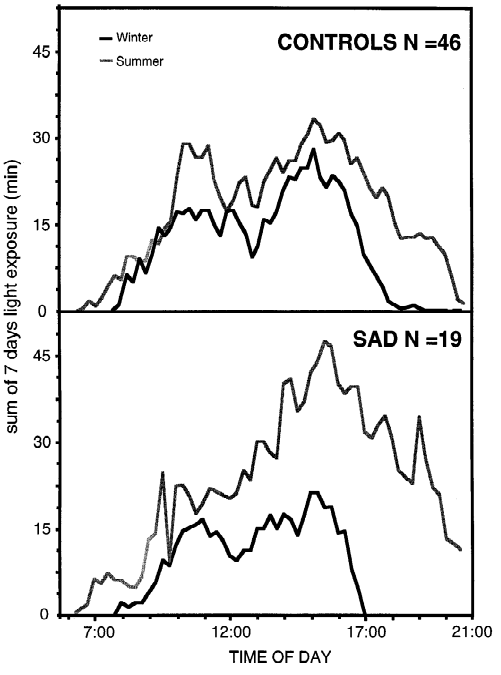

Fig. 1. Time spent outdoors (15′ bins summed over seven days in winter and summer) is plotted for control subjects (upper graph, $n = 46$) and SAD patients (lower graph, $n = 19$). For statistics see text.

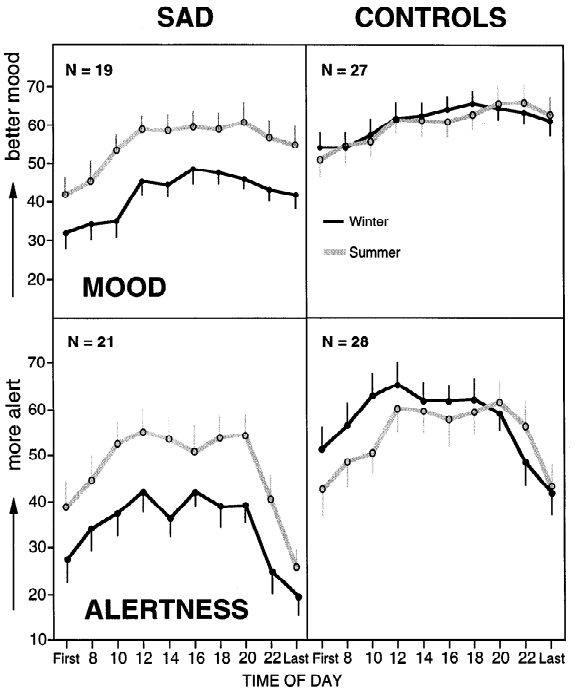

Fig. 2. Visual analogue scale ratings (in mm, 50 = normal; mean±1 s.e.m.) of mood (upper graphs) and alertness (lower graphs) measured 2-hourly throughout a day from awakening (first) to bed time (last) in winter (black line) and summer (grey line, open circles). For statistics see text.

winter than in summer (interaction group × season: $F_{(1,42)} = 11.8$, $p = 0.001$; SAD: winter < summer, $p < 0.01$), and increased in summer to control levels (summer: SAD vs. controls n.s.). There was no seasonal mood difference in control subjects.

A significant diurnal pattern in mood ratings was found (main effect: $F_{(9,378)} = 23.6$, $p = 0.0001$), with low mood in the morning (first rating and 8 h), rising thereafter (first, 8 h < 10 h, $p < 0.01$ and 10 h < 12 h, $p < 0.001$). Mood attained a stable plateau from 12 h to 20 h (n.s.). After 20 h, mood decreased slightly (20 h > last, $p = 0.09$).

Alertness was also significantly lower in SAD patients in winter than in summer (interaction group × season: $F_{(1,44)} = 8.2$, $p = 0.006$, SAD: winter < summer, $p < 0.01$) and increased in summer to levels of controls (summer: SAD vs. controls n.s.). There was no seasonal difference in alertness in control subjects (n.s.).

A significant diurnal pattern in alertness ratings was found (main effect: $F_{(9,396)} = 19.5$, $p = 0.0001$) with low alertness in the morning (first rating and 8 h), rising thereafter (first rating < 10 h, $p < 0.001$). Alertness attained a plateau from 10 h to 20 h (n.s.). After 20 h, alertness decreased rapidly (20 h > 22 h, $p < 0.001$; 22 h > last, $p < 0.001$).

A separate 2-way ANOVA of alertness in controls revealed a phase delay in summer compared with winter (Interaction: $F_{(8,192)} = 3.16$, $p = 0.01$). That is, they were more tired in the morning in summer than in winter, but more alert in the evening.

3.4. Sleep

A significant interaction of the factors group and seasons (all $p \leq 0.01$) permitted post-hoc comparisons to reveal seasonal differences in SAD patients' sleep: in winter they woke up later than in summer (7:48±14′ vs. 7:23±14′; $p = 0.01$), had a longer sleep latency (28.8±4.3′ vs. 17.2±2.8′; $p < 0.001$), more nocturnal awakenings (40.4 + 7.3′ vs. 20.5±4.7′; $p < 0.001$) and judged their sleep to be shallower ($p = 0.001$). Controls did not show winter-summer differences in any sleep variable.

In summer the only difference between controls and SAD patients was the persistent higher sleep need in SAD ($p = 0.002$) measured by the sleep log item 'Would you have liked to have slept longer? Yes/No'.

SAD patients took more naps than controls, independent of season (SAD vs. controls, winter/summer: 16.9±5.4′/16.9±4.9′ vs. 4.7±12.2′/6.3±10.8′; $p < 0.01$), but there was no significant correlation between naps and the sleep need in summer.

3.5. Relationship between light exposure and subjective variables

In the covariance analysis, neither mood or alertness ratings, nor sleep logs were dependent on the amount of light received (analysis not shown).

4. Discussion

This study addressed the question as to whether winter depression is correlated with the amount of

ambient light exposure. It is known that the prevalence of SAD and its subsyndromal form S-SAD (Kasper et al., 1989) are partially dependent on the latitude, i.e. the duration of daylight or photoperiod. Epidemiological studies using the SPAQ have revealed 1–10% SAD, 7–20% S-SAD in the USA, rising from south to north (Rosen et al., 1990); in Switzerland (47°N), 2.2% SAD and 8.9% S-SAD have been found (Wirz-Justice et al., 1992). There is, however, for unknown reasons, a much higher prevalence in the region of Basel: 9.9% SAD and 23.8% S-SAD (Wacker, 1995). We thus checked our selection of 46 healthy women for the presence of seasonality according to the SPAQ: 6 (13%) fitted the SAD criteria, 11 (23%) were S-SAD, but none had a clinical diagnosis of SAD. They had not been selected for absence of seasonality, but showed a similar prevalence as the representative sample of the same population in Basel. There were no significant correlations of the SPAQ seasonal score with the light logs. Thus, the control group can be regarded as adequately representative with respect to the central variable of light-oriented behaviour.

The winter–summer changes in mood, alertness, and sleep that patients with SAD undergo (and controls do not), reflected their clinical state changes – depression in winter and remission in summer. Improvement similar to that spontaneously occurring in summer is also induced by both natural and artificial light treatment (Wirz-Justice et al., 1996; Wesson and Levitt, 1998). However, the subjective improvement of sleep of the SAD patients in summer or after light therapy is not found in changes in the sleep EEG (Brunner et al., 1993, 1996; Anderson et al., 1994).

Our results showing no difference in the light exposure in winter between patients with SAD and controls do not confirm our hypothesis. This result must be interpreted cautiously, since power was only medium (0.57) with a high type II error risk, but it conforms to findings in the literature.

A prior investigation of SAD patients living at a similar latitude, using light logs, found nearly identical durations of time spent outdoors in winter to the present study (Eastman, 1990). The technique of light logs may over-estimate real light exposure. Particularly in winter, estimations of time spent outdoors are more than twice as long as the measured light exposure >1000 lux (Cole et al., 1995; Guillemette et al., 1998; Hébert et al., 1998).

More recent studies have used objective measurements of light exposure with light monitors. These too find no difference in light exposure between SAD (Oren et al., 1994) or S-SAD (Guillemette et al., 1998) and controls in winter. The Canadian study (Guillemette et al., 1998) is the only other investigation of seasonal differences: they did not find the marked higher summer light exposure in S-SAD that we found in SAD compared with controls. The clinical diagnostic difference may be the explanation, although comparison of SPAQ seasonality scores does not support this interpretation (our SAD patients: 14.1 ± 3.9, $n = 21$; S-SAD patients (Guillemette et al., 1998): 12.6 ± 3.0, $n = 11$; $t = 1.21$ n.s.). We could also find no significant correlation between light-oriented behaviour and the SPAQ seasonality score.

In control subjects, a seasonal difference in light exposure has been documented for intensities >1000 lux (Hébert et al., 1998), which would be equivalent to our seasonal differences in the amount of time spent outdoors. All studies show this obvious seasonal difference in behaviour.

We can perhaps interpret the surprising finding of higher summertime light exposure in SAD than controls in terms of 'amplitude'. The power of the difference was very high in the afternoon (0.93), higher than for the whole day (0.80). Patients with SAD show a greater seasonal difference in light exposure than controls, i.e. a larger amplitude. The winter-summer difference in overall average daily light exposure was 97′ in SAD and 46′ in controls. More dramatically, when expressed as a percentage of the summer value, patients with SAD received only 40% of this light exposure in winter, whereas controls received 67%. This means that patients with SAD *do* have *relatively* less light exposure in winter.

The available studies thus indicate no difference in light exposure between subjects with seasonal problems and healthy controls in winter. The explanation for a seasonal susceptibility to depression has to be explained on other levels: either at the retinal photoreceptor itself, in CNS sensitivity, or even psychological sensitivity to light.

Both retinal subsensitivity (Remé et al., 1990) and supersensitivity (Beersma, 1990) have been post-

ulated as a model for SAD vulnerability to light input, but experimental studies of ophthalmic function do not yet yield a clear picture (reviewed in Tam et al., 1998).

The light-induced suppression of melatonin can be used as an index of sensitivity of the entire retinohypothalamic-pineal tract. The experimental results are controversial, with some evidence for supersensitivity (Thompson et al., 1990), though this has not been replicated (Murphy et al., 1993).

If seasonal vulnerability to mood disorder and psychological distress were a trait, then SAD patients should also be less stable than healthy subjects in summer. There is some indication that long periods without sunshine can induce negative mood swings in SAD patients, which are less intense than the winter depression. Our SAD patients still have higher depression ratings in summer than controls, even though they are improved compared with their winter state (von Zerssen depression self-ratings in summer: 8.4 vs. 4.8; $t = 2.09$, $df = 56$, $p = 0.04$); similar results are found in the Canadian S-SAD sample (Guillemette et al., 1998). However, this 'residual depressivity' is not manifested in self-ratings of mood, alertness, or sleep. A further possibility is that the vulnerability might be at the psychopathological level. Yet there is no evidence that SAD patients have any abnormal personality traits (Schulz et al., 1988; Lilie et al., 1990; Schuller et al., 1993; Schüle, 1995).

The mechanisms underlying SAD are still poorly understood: there are no clear abnormalities of the circadian system or sleep regulation, in the EEG and brain imaging. However, serotonergic function, implicated in depressive syndromes, appears to be altered in patients with SAD (Tam et al., 1998). Serotonin is a major neurotransmitter in the circadian pacemaker that directly receives light input from the retina. It may be relevant in terms of this vulnerability concept that serotonin levels change with season in the human hypothalamus, dropping precipitously in winter, and that women (who suffer from SAD 2–4 four times more than men (Lam and Levitt, 1998)) have markedly lower serotonin synthesis rates (Brewerton, 1989). Our findings that women with SAD do not receive less bright light exposure in winter than control women, suggest that their decline into depression as the days get shorter is not related to light input per se. Less light in winter may impinge on an altered serotonergic system in SAD, triggering the depressive state. The same reduction of photoperiod does not affect healthy subjects. Since, SAD patients overcompensate light exposure in summer, perhaps requiring more light input than controls to adequately normalise their serotonergic function, their relative light exposure in winter is lower than that of controls, i.e. they show a larger seasonal amplitude.

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