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Light treatment of seasonal affective disorder in Switzerland

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ABSTRACT – Seasonal Affective Disorder (SAD) has been characterised by two or more depressive episodes in autumn or winter (with remission the following spring or summer), decreased energy, increased sleep, increased appetite, weight gain and carbohydrate craving. SAD patients were identified in a Swiss-German population; 22 participated in a light-therapy protocol (1 week bright white light 2500 lux or dim yellow light 250 lux, from 06–08h and 18–20h). Both observer and self-ratings indicated a significant diminution of depressive symptoms with both lights. One week after withdrawal from yellow light, depression ratings relapsed to previous values; remission lasted longer after bright white light. Global VAS self-rating scales for “mood” and “well-being” however, and the Hamilton scale for atypical SAD symptoms, differentiated clearly between bright and dim light: only bright light showed an improvement that persisted after withdrawal. These results suggest that even though a placebo effect cannot be excluded, 4 h explicit light exposure/day may not be a negligible quantity. Light treatment promises to be a useful non-pharmacological intervention in certain forms of depressive illness.

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A relationship between the season and affective illness has been postulated since ancient times, and systematically investigated for more than 100 years (review 1). Epidemiological studies concur in the finding that depression is most frequent in spring, with a lesser peak in autumn, and that mania tends to occur more often in summer. A study of hospitalisations in the Psychiatric University Clinical Basel for affective illness during the years 1920–1947 provides a local population that follows this pattern:

“Depression...tends to begin in spring and autumn... In contrast, the manic phases of circular psychoses tend to manifest themselves in summer. In particular, it is the less severe, periodic – possibly annually recurring – manic-depressive phases that show this typical seasonal onset.” (2).

The recently described syndrome of “Seasonal Affective Disorder” (SAD) fits the pattern of a condition characterised by recurrent depressive episodes that occur in autumn and winter and remit in spring or summer (3–7). Additionally, these patients characteristically manifest hypersomnia, overeating and carbohydrate craving with weight gain, and depressive symptoms that respond to climate and latitude. The seasonal depressive phase also responds to bright artificial light (3–7). These clinical findings support the hypothesis that geophysical factors of photoperiod (daylength) or light intensity (hours of sunshine) are coupled to the psychopathological phenomena (4–9).

We report a European replication study of seasonal affective disorder and its response to artifi-

cial light, using the methods and rating scales developed by the NIMH group (4). We addressed two questions: whether a group of SAD patients could be identified in a Swiss-German population, and whether their depressive symptoms could be alleviated by light treatment.

Subjects and methods

Subject recruitment

The relatively rare occurrence of seasonal depression (estimated by Kraepelin to be only about 4–5% of manic-depressive patients) led the NIMH group to seek out a large population by means of a newspaper article describing the syndrome. Our approach was similar. We began with a personal letter to all registered physicians in the canton of Basel; this was extended to an article in the Swiss Medical

Journal (1984:65:1812) that reached physicians throughout the country. However, only 12% of the total number of persons replying were referrals by doctors thus informed. Of 322 replies, 88% came after publication of a scientific article in five Swiss-German newspapers. The seasonal screening questionnaire was returned by 159 subjects; those with at least 2 consecutive years in which depression had developed during autumn or winter with remission in spring and summer, and with at least three of the six symptoms defined by Rosenthal et al. (depressive mood, decreased energy level, increased appetite, weight, carbohydrate craving and sleep time) (4) were considered to fulfil the criteria of SAD. "Atypical SAD" were those patients with a seasonal course but less than three of the six symptoms defined for SAD. These diagnoses (from the questionnaire) are shown in Fig. 1.

Prospective patients were interviewed at least

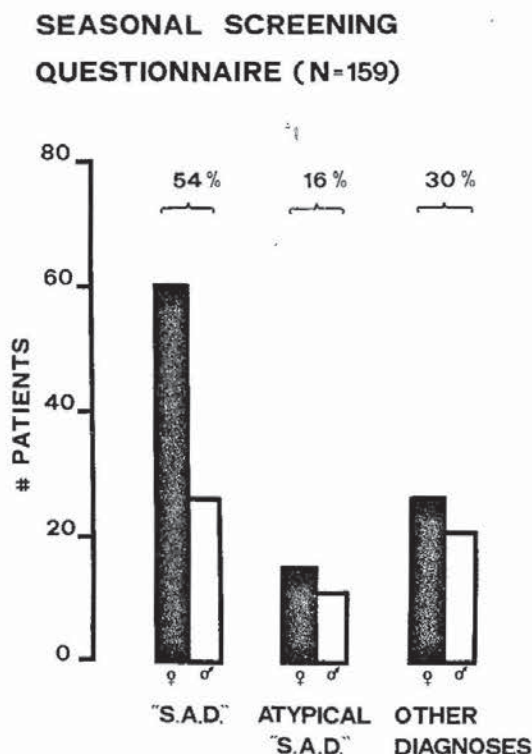


Fig. 1. Diagnoses of SAD (seasonal course plus three of the six main symptoms (4)) or atypical SAD (seasonal course with less than three of the six main symptoms) from the seasonal screening questionnaires returned. Half of the replies had a history of the SAD syndrome; of these 70% were women.

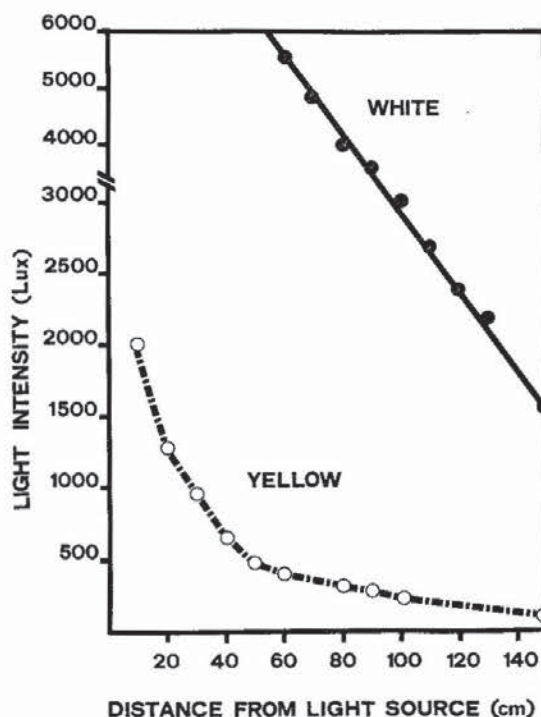


Fig. 2. Light intensity (Lux, Gossen Panlux electronic lightmeter) measured at 10-cm intervals in front of the white and filtered yellow light source used in the study. Light intensity increased rapidly with proximity to the yellow light.

once; in these interviews ($n = 72$) details of the individual SAD syndrome were elucidated. Informed consent and an extensive medical history were obtained from those who agreed to participate in the study, and DSM III criteria for major depressive episodes were applied.

Experimental design

A single-blind crossover design in an outpatient study was planned. Two kinds of light were used: bright, white full-spectrum fluorescent light (Vita-lite, 2500 lux at 100 cm), and dim yellow fluorescent light (250 lux at 100 cm, using Rosco Sun #3 filter) (Durotest Corporation, New Jersey) (Fig. 2).

The light source from eight plastic-screened fluorescent tubes mounted on a stand was placed on a table in the patient's home. Patients were randomly assigned to one or the other lighting condition. They were instructed to sit facing the lights at a distance of 100–150 cm, glancing at them from time to time, for 2 h (from 06–08h and 18–20h) keeping a logbook of their activities and vigilance state during this time. Each light treatment was continued for at least 5 days, with 1 week withdrawal after each, unless the clinical state indicated immediate transfer to the other light source (no improvement), or no further treatment (no relapse).

The study did not begin until October 1984 (newspaper articles published in September), and because of this delay most patients were already depressed at the time of the first interview. They were seen by the same psychiatrist (C.B.) on a weekly basis in the three centres of Basel, Bern und Zürich; the 21-item Hamilton Rating Scale (HRS) and the von Zerssen self-rating depression scales (10) were administered. All patients with an HRS score greater than 15 were offered light treatment immediately or after a further week, depending on clinical judgement and completion of an ophthalmological investigation. This consisted of slit-lamp biomicroscopy, dilated funduscopy, dark adaptometry, and stereo-colour photography of the retina. After light treatment, 14 subjects completed the ophthalmological examination a second time.

In addition to the weekly interviews, patients

Table 1

Clinical and demographic features of patients with SAD

	Swiss study ($n = 22$)	NIMH study ($n = 29$)
Women:Men	16:6	25:4
Age \pm SD (years)	41.7 \pm 10	36.5 \pm 11
Age at onset (years)	27 \pm 13	26.9 \pm 13.2
Mean length of depressive episode (months)	5.4 \pm 1.4	3.9 \pm 1.5
Cycles more severe over time or longer in duration	15 (75%)	35%
Previous treatment because of SAD		
None	6 (27%)	31%
Antidepressants/lithium	15 (68%)	42%
Hospitalisation	3 (14%)	10%
Thyroid problems	0 (0%)	21%

completed a daily sleep log and twice daily self-ratings (visual analogue scales of the items: mood, well-being, tiredness, energy level, anxiety, and appetite) and a weekly self-rating scale (translations of the NIMH scales). After completion of the light-therapy protocol patients were given the option of continuing with the light of their choice as long as they wished. Many continued their daily self-ratings throughout the summer as a preparation for early treatment next winter.

Results

Swiss SAD population

Clinical history. A core group of 22 SAD patients with HRS scores greater than 15 at the start of light therapy form the data base of this report, and will be compared with the first NIMH SAD patient population ($n = 29$) (4). These 22 patients all fulfilled DSM III criteria for major depressive episodes.

Clinical and demographic features are given in Table 1: there are many similarities between the Swiss and U.S. patient populations.

Three-quarters of the patients were women, and many reported seasonal cycles from their earliest years, although few had recognised this aspect as being important before reading the

newspaper article. The events shortly before the first manifestation of SAD were varied: no clear memory (6 patients) or no special events (1 patient), hormonal-related phenomena (puberty (3 patients), birth, and menopause (each in 1 patient)), change in situation (first school year (1 patient), moving house (6 patients), new job, divorce, or pronounced reduction of spare time light exposure (each in 1 patient)).

The psychological and psychosomatic history of childhood and adolescence showed the following incidence (when remembered): menstrual-related problems (16 patients), school fears (12), shyness (9), sleep disturbances (8) or sleepwalking (2), bedwetting (5), headache (7), overweight (6) or underweight (3), overactivity (4) or temper tantrums (4), abdominal pains (3) or cramps (2).

Medical histories showed no thyroid abnormalities in our group, in contrast to the NIMH group (21%) (4), and no diabetic or kidney problems. Noteworthy was the relatively high incidence of low blood pressure (12 patients compared with high blood pressure in two), rheumatic problems (10) and allergies (8). Other observations were: a history of infections (7), heart (4) and liver problems (4), and asthma (2). Diverse ophthalmological status was found in six, with nine patients subjectively considering themselves to be more sensitive to light (and wearing sunglasses more often), only one less sensitive to light. 16 of the 22 patients woke up at first light in the morning.

With respect to the SAD case histories, 20/22 noted a change in SAD symptoms over the years. Of these, 13 had an increase in intensity of symptoms with time, four a decrease, and one variable; additionally, changes in the number of symptoms in the last few years: five had more and three less. Sometimes changes in the duration of the depressive phase with time had also occurred, tending more towards longer (8) than shorter cycles (1). One quarter of the patient population had had no treatment for previous depressive phases, 11 had had psychotherapy, and 15 antidepressants or lithium, three of whom had been previously hospitalised. According to their life history, 17 could be diagnosed as bipolar II, four probably bipolar II, and one male patient as unipolar.

Table 2
Family history of SAD patients (first-degree relatives)

	Swiss study (<i>n</i> = 18)	NIMH study (<i>n</i> = 29)
Negative family history	1 (6%)	
Affective disorder	12 (67%)	69%
SAD	3 (17%)	17%
Suicide	3 (17%)	
Alcoholism	5 (28%)	7%
Schizophrenic disorders	0 (0%)	

An important motivation for volunteering was the dissatisfaction with psychopharmacological agents and the hope for a non-drug alternative. Patients were without medication at the time of the study, except for one on lithium carbonate and another on fluvoxamine.

Many subjects reported changes related to travel, in particular positive responses to going south. Three subjects considered a change of meridian to have initiated their seasonal depression: return to Switzerland after years in a more temperate clime (two from California and one from Australia). Furthermore, on questioning, most subjects could differentiate "good" and "bad" days within the depressive or healthy phases, dependent on the actual weather conditions. This importance of local weather was noted by those patients whose illness began with the move

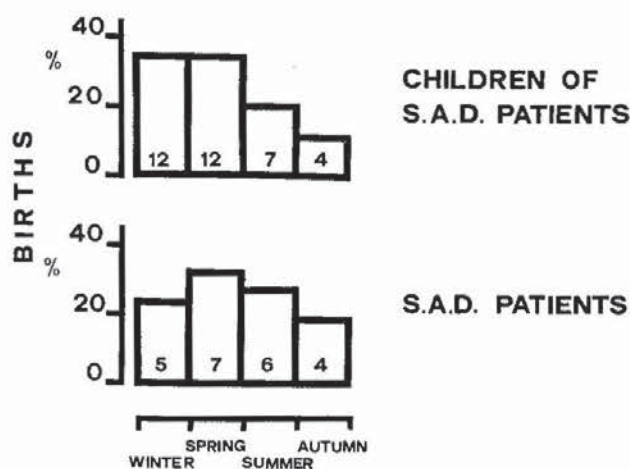
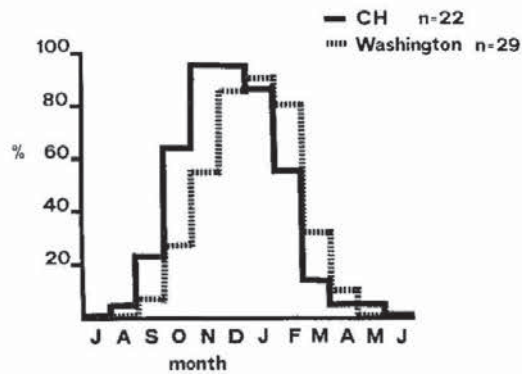
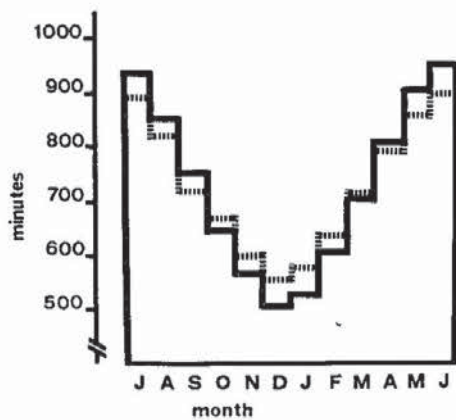


Fig. 3 Seasonal distribution of birth in the SAD patient group (*n* = 22) and their children (*n* = 35). Seasons defined according to the equinox and solstice. The children of SAD patients tend to have been born in the first half of the year.

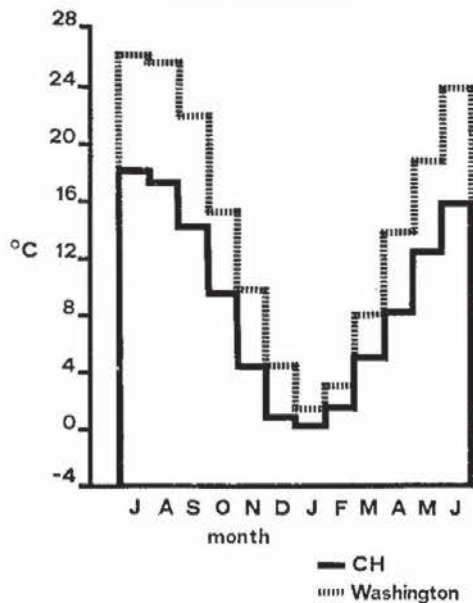
A. PERCENT OF SUBJECTS EXPERIENCING DEPRESSION (per month)



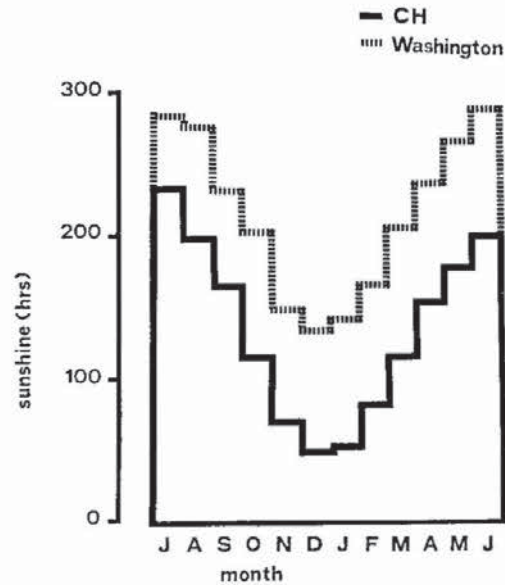
B. PHOTOPERIOD AT MID-MONTH FOR WASHINGTON (39°) AND SWITZERLAND (47°)



C. AVERAGE MONTHLY TEMPERATURE from 1965–1984



D. HOURS OF SUNSHINE/MONTH



E. AMOUNT OF RAIN/MONTH

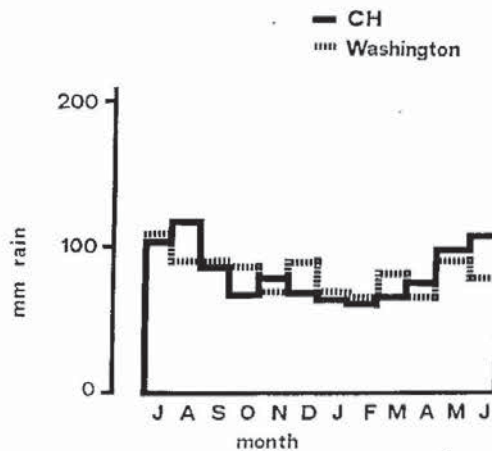


Fig. 4. Comparison between Switzerland and Washington in seasonal patterns. Washington data from (4) and the National Climatic Data Center (Washington National Airport local climatological data summary); Swiss observatory data from Basel, Bern, and Zurich (averaged from 1965–1984).

Table 3
Depressive symptoms (%)

	Swiss study (n = 22)	NIMH study (n = 29)
Affect		
Sadness	91	100
Anxiety	86	72
Irritability	82	90
Physical activity		
Decreased	100	100
Appetite		
Increased	45	66
Decreased	45	28
Mixed	0	3
No change	9	3
Carbohydrate craving	77	79
Weight		
Increased	55	76
Decreased	23	17
Mixed	0	3
No change	23	3
Sleep		
Earlier onset		79
Later waking		76
Increased sleep time	82	97
Change in quality (interrupted, non- refreshing)	82	90
Daytime drowsiness		72
Decreased libido	77	69
Difficulties around menses	67	71
Work difficulties	100	97
Interpersonal difficulties	86	100

from a sunny to a cloudy part of Switzerland. Many commented on the improvement induced by winter holidays in the alps, or on the well-being after visits to the solarium.

Family history. A high incidence of affective disorder in first degree relatives was found (Table 2). Noteworthy was the parallel history of SAD in a pair of male dizygotic twins, who both responded to light treatment (# 06, # 19).

A comparison of the birth dates of SAD patients and those of their children in terms of seasonal distribution is made in Fig. 3. Although too small a population for statistical analysis, the children tend to have been conceived in spring and summer, when, characteristically, the patients reported euthymic or hypomanic states.

Seasonality. The percentage of the patient population suffering from depression in any 1 month was calculated from the clinical history of the previous years. This pattern was found to be highly correlated with the length of the photoperiod (-0.94) and with mean monthly temperature after a shift of 1-2 months (-0.91) (a direct correlation gave a value of only -0.67) (Fig. 4 A-C). Comparison of the Swiss and NIMH data show that depression lasted 1.5 months longer in our population, because of an earlier start. The latitude related differences between Switzerland and Washington in monthly mean temperature and photoperiod cannot alone account for the earlier onset of SAD in the former population. We therefore calculated the actual number of hours of sunshine/month and monthly rain at both localities, averaged over the last 20 years, the time span retrospectively cov-

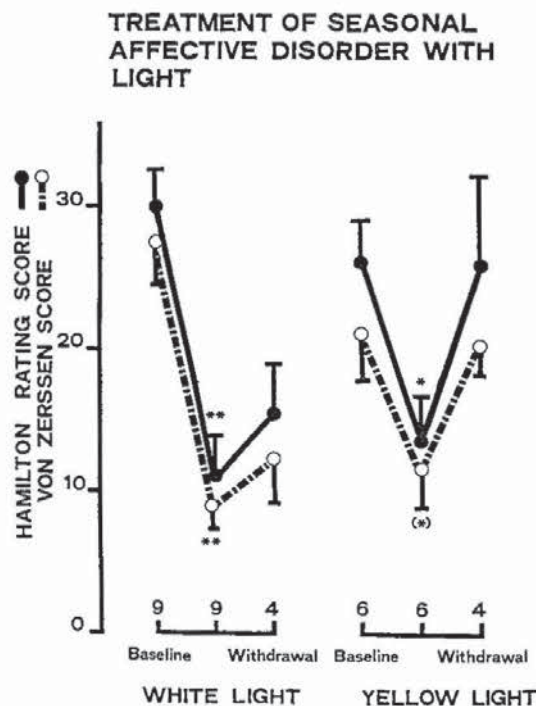


Fig. 5A. Psychiatrist's and subject's self-rating depression scores (mean \pm SEM) for SAD patients at baseline, after light treatment (white or yellow light > 5 days), and after 1 week withdrawal. Paired t test for baseline vs. treatment $**P < 0.01$; $*P < 0.05$; $(*)P < 0.1$. Not all ratings were available for all patients at the given times. After 2 weeks withdrawal from white light (data not shown) mean HRS scores had still not reached baseline values (20.8 ± 7 , $n = 5$).

Table 4
Hamilton rating scores for individual patients in the study

Patient			Light		
Sex	Age	Baseline	W = White		1 week withdrawal
			Y = Yellow		
			1 = 1st treatment		
			2 = 2nd treatment		
F	34	33	1 W	3	interview only after 2 weeks
F	28*	22	1 W	12	20 (crossover)
F	34*	27	2 W	12	17
+ M	43*	37	2 W	3	refused to stop lights
F	46	37	1 W	9	19
F	32	40	1 W	3	hospitalised on day 3
F	41	30	2 W	stopped after 1 day	
F	56*	18	2 W	14	continued lights
+ M	43	34	1 W	24	continued lights
M	44	22	1 W	11	interview only after 2 weeks
M	47	20	1 W	snowstorm	5
F	28*	20	2 Y	8	28
F	34*	26	1 Y	27	crossover immediately
+ M	43*	39	1 Y	14	37 (crossover)
F	41	29	1 Y	4	30
F	56*	20	1 Y	15	went skiing
M	70	24	1 Y	13	8
M	44	28	2 Y	stopped after 4 days	

*completed crossover

+ fraternal twins

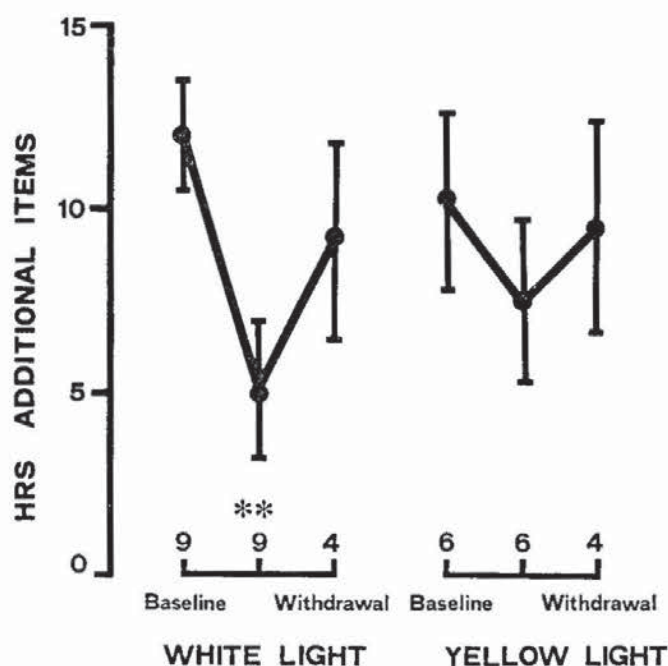


Fig. 5B. Psychiatrist's ratings of atypical symptoms of SAD. Conditions as in Fig. 5A.

ered by our respective patients populations in estimating their length of winter depression. Here a clear difference is seen in the amount of sunshine, but not in the amount of rain (Fig. 4 D, E), with no seasonal asymmetry that could account for the earlier start at a more northern latitude.

Depressive symptoms. The subjective retrospective rating of symptoms during the seasonal depressive phase is summarised in Table 3. There is again a close parallel to the NIMH patient population.

Light treatment

Twelve patients used light consistently for more than 5 days (4 completed the crossover). Both bright white light and dim yellow light improved depressive symptoms (defined as HRS change > 5: 8/9 and 5/6 responders, respectively). The mean observer and self-rating scores are shown in Fig. 5A: before treatment, after at least 5 days of light, and after 1 week's withdrawal.

There are no significant differences between the scores for white or yellow light before or after treatment. The only difference between the two lights is found after withdrawal: after yellow light, depression scores returned to a value similar to that before treatment, whereas after white light, the values remained low. Even 2 weeks after withdrawal from white light, the mean HRS scores had not reached baseline values. Individual HRS scores are given in Table 4.

In contrast to these classical rating scales for "melancholia" the global VAS ratings and the HRS "additional items for atypical symptoms scale" [4] (Fig. 5B) clearly differentiated between white and yellow light. In particular, the morning ratings for the VAS scales "mood", "well being", and "anxiety" showed a significant improvement with white light that continued after withdrawal, with yellow light showing no change in these ratings. Neither white nor yellow light significantly modified VAS ratings of "tiredness" or "energy".

Improvement, if any, was usually seen within 3 days; subjectively, the patients rated the white light effect as being sudden, whereas the yellow light effect was more gradual. The length of time required for a relapse after withdrawal varied from within 1 day to remaining free of symptoms for the rest of the winter. An example of the latter outcome is the case history of a woman with 10 years of SAD symptoms from October/November to February, often followed by hypomanic episodes in spring. White light treatment in October induced remission within 3 days, and she continued regular light use for 2 weeks. Self-ratings remained low until the stress of Christmas; in mid-January she used white light irregularly for 5 days with rapid improvement. Depressive symptoms remained absent for the rest of the winter, spring, and throughout the summer. At the first signs of depression at the beginning of October 1985 she used white light for 1 week only, from 06–08h, again with rapid improvement (26).

A number of side effects were reported, and five subjects interrupted the light treatment for this reason (2 white, 3 yellow). White light gave hypomanic activation, irritability, headache and nausea; yellow light gave headache and nausea,

agitation and irritability and worsening of depression. The latter light was also commented on: "this light is poison" "voodoo light", "feels like amphetamine". In contrast, one hypomanic patient refused to return the white lights after the study, inventing a new excuse each week for why he was dependent on using them, or not keeping his appointments.

Ten SAD patients dropped out of the data analysis for the following reasons: irregular application; simultaneous start of new medication, or change in dosage ($n = 3$); light (yellow) stopped due to extreme anxiety; parallel family problems; regular use of light, but SAD phase related to son's suicide; regular use of light, but coinciding with the projected end of the depressive phase (according to comparison with previous years) ($n = 3$). There was no difference in attrition rate between yellow and white light.

Six further patients received light treatment but were not included for the following reasons: two did not fulfil the criterion of two or more consecutive years of seasonal depression; two had a history of seasonal depression but also seasonal work and financial problems; one did not fulfil DSM III criteria; and one had shown a case history of SAD but was now depressive the entire year and therapy resistant.

Ophthalmological investigation showed no changes in visual function after light treatment. The spectra of the white lights was that described by the manufacturer, with the exception of the UV portion (< 370 nm) that was absorbed by the plastic screens. The yellow lights had peaks at 540 and 580–590 nm only.

Of the 22 study patients, 15 could be interviewed again in spring or summer; none was clinically depressed, although two had extraneous reasons for HRS scores > 15 (work stress and marriage problems). None was hypomanic; this questions the life history diagnosis of bipolar II. 14 agreed to continue the self-ratings throughout the summer and wanted to use light treatment at the first signs of their depressive symptoms next autumn. For one SAD patient on lithium the winter with light treatment was the first in 30 years where she had not taken additional antidepressants.

Discussion

The main findings in this first European study can be summarised as follows:

1. In a Swiss-German population, we found a surprisingly large number of seasonal depressive patients who fulfilled the NIMH criteria; these patients could be more easily recruited via newspapers (recognising the description of their symptoms), than via their doctors.
2. We were able to replicate previous observations that light improves depressive symptomatology, even with less total hours of light exposure than the NIMH study ($2 \times 2\text{h}$ compared with $2 \times 3\text{h}$) (4).
3. We did not replicate the difference between bright white light and dim yellow light previously found using conventional rating scales for "melancholia" (4). Both had a positive effect; only in the withdrawal phase did the effects of bright white light appear to be longer-lasting. Examination of references (4, 6, 7) detailing individual HRS scores showed that some patients had responded to dim yellow (3/9) or dim white light (7/18, 4/9), even though not as often as in our study (5/6). With the small N in all these studies, such differences may be related to selection criteria: we applied the criteria for SAD (4) more stringently than the NIMH group itself.
4. We did find a difference between bright white and dim yellow light when rating the HRS "atypical items for SAD", as well as when patients self-rated "mood", "well-being" and "anxiety". Thus bright white light was found to be more effective than dim yellow light for certain specific SAD items as well as for more global improvement.
5. Sleep deprivation, a well-known, though short-lasting non-pharmacological antidepressant modality, has been shown to be neither necessary nor a sufficient explanation for the effects of light therapy (28). In our study also, the improvement after light treatment could not be attributed to a partial sleep deprivation effect, since neither light treatment reduced sleep duration (sleep logs, data not shown).

It could be argued that the improvement found with both lights is a placebo effect of the entire experimental procedure. This aspect has certainly

not been adequately tested and some placebo effect cannot be excluded. There is some evidence that light is an active agent. First, placebo effects are more evident in mild or neurotic depressions; our patient group was quite severely depressed (mean HRS score of 29). Second, in general, a first treatment is more likely to give a placebo response than a second treatment: the improvement with the second light was similar to that with the first light. Third, use of the lights under self-selected conditions led to repeated improvement in the same individual. Fourth, relapse after withdrawal is considered a criterion for an active agent: this was fulfilled by the yellow light.

If both lights are considered as active agents, we need to ask why a Swiss population behaved differently from the NIMH group. The demo-

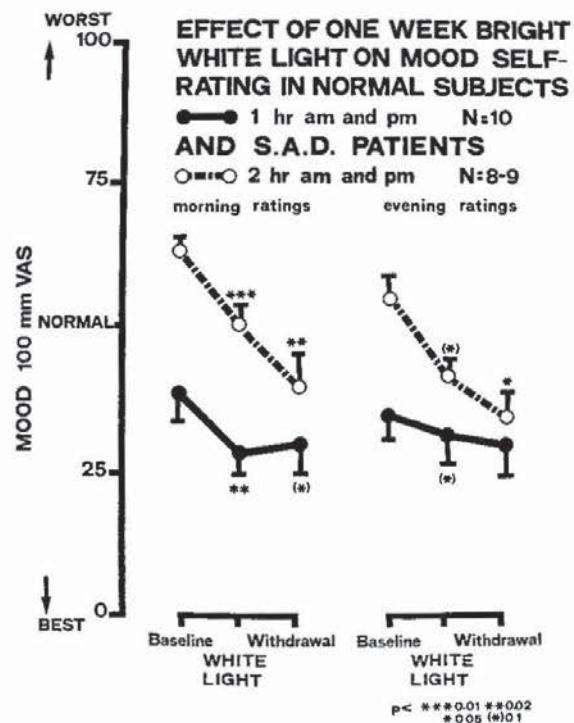


Fig. 6. Daily mood self-ratings (100mm VAS), averaged individually for a week baseline, the week of light treatment, and the week of withdrawal. Shown are the mean \pm SEM for 10 normal subjects and 8-9 SAD patients. Both groups show significant improvement in morning mood ratings after light. Additionally, SAD patients show significant diurnal variation of mood (t test between baseline values morning vs evening, $P < 0.01$). Neither group showed worsening of mood after withdrawal of the lights.

graphic and clinical variables were remarkably similar, with the exception of three factors that might be relevant: the patients had higher depression scores, they were given light treatment when already in the middle of their winter depression, not at the beginning, and were a more highly selected SAD group of patients than the NIMH group.

The rationale underlying bright white light treatment of SAD arose from the finding that humans require high intensity light (> 2000 lux) to suppress nocturnal secretion of the pineal hormone melatonin (11). Manic-depressive patients require less intense light to show this response (12, 13). If light sensitivity is related to depth of depression, our highly selected patient population could have been more sensitive to less intense light than the NIMH group. In addition, animals show a reciprocal intensity/duration relationship for light-induced suppression of melatonin (14). If such an intensity/duration relationship also holds in man, 4h explicit light exposure/day for 1 week is not a negligible quantity (27). The factor of individual trait- and not clinical state-dependent differences in visual sensitivity also need to be considered (13, 15): patients showed clear differences in their preferences for bright white or dim yellow light, and chose the same light again for further treatment. It should be noted that a few patients reported sitting nearer the yellow lights ("because they were dimmer"); in this outpatient study the actual distance from the light source could not be precisely quantified for each individual. That small differences in distance may have influenced the amount of light actually received is shown by the rapid increase in light intensity with proximity (Fig. 2). At 50 cm the yellow light intensity was 500 lux, which has been shown sufficient to suppress melatonin in manic-depressive patients (11). Pupil size varies with time of day and clinical state and may also, though to a lesser extent, have modified the amount of light impinging on the retina.

The parallel between depression and hibernation, very early proposed by Lange (16), is a potential neurobiological model for SAD. Seasonal rhythms of hibernation in many species are driven by the photoperiod, and this information

is transduced by melatonin: the duration of the nocturnal peak is a function of the duration of the dark phase (17). Even if it is not known whether such photoperiodic mechanisms are still relevant in humans, extension of the photoperiod by bright white light was the strategy used for seasonal affective disorders. And it worked.

However, recent studies now show that the same amount of bright white light given within the winter photoperiod (18), or only in the evenings (7, 19) or a pulse given only once a day (9, 19, 20), all have a therapeutic effect. Additionally, even though melatonin given to patients who improved with light treatment partially reversed certain symptoms (6) the inverse experiment, suppression of melatonin by the beta-blocker atenolol did not always improve depressive symptoms (6). Thus melatonin appears rather more indirectly involved in the pathogenesis of SAD than originally believed.

Thus, the amount of light (number of photons) may be as important (6) as the timing of the application itself (20). Somewhat indirect evidence for the amount of light being important comes from the comparison between two demographically similar populations in Washington and Switzerland. The shorter duration of SAD in a more southern latitude appears to correlate less with the photoperiodic differences than the greater amount of sunshine in Washington than in Switzerland. In this respect, the magnificent autumn of 1985 in Europe had modified the pattern found in 1984: as of mid-October, only two patients were sufficiently depressed to begin light therapy.

Investigations of the effects of bright white light in normal subjects are also important for defining the intensity/duration relationship. For example, we have found that mood and well-being can be significantly improved by 2×1 h white light/day for 1 week (21). The visual analogue scale self-ratings in normal subjects follow a similar pattern to that found in SAD patients. Both show a significant improvement in morning ratings (Fig. 6) (21).

In addition, $1 \times 1/2$ h/day for 1 week modifies the nocturnal core temperature rhythm (22). Even 2×3 h for a single day can change sleep architecture (23). In midwinter insomnia in Nor-

way, $1 \times 1/2$ h for 5 days shortens sleep latency (24). Thus, bright white light has biological and psychological effects in non-depressive subjects. This non-pharmacological intervention therefore has potential usefulness in treating not only depression (3–9, 18–20, 23) but also certain sleep disorders related to darkness in the northern winter (24), and perhaps also those arising from shift work and jet lag (25). Our follow-up study addresses the issue of a “dose-response” relationship for the antidepressant effect of bright white light in SAD patients.

Comment. The rating scales and questionnaires used in this study and modified on the basis of the above results (in German), are available on request for use by interested investigators.

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