Early light treatment can prevent an emerging winter depression from developing into a full-blown depression


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Summary

The administration of light at the development of the first signs of a winter depression appears to prevent it from developing into a full-blown depression. Not a single patient from a group of 16 treated this way became severely depressed during the remaining part of the winter season, whereas 5 out of 11 from the non-treated control group did.

Key words: Seasonal affective disorder; Prevention; Depression; Light therapy

Introduction

Seasonal Affective Disorder (SAD), winter type, is a depressive syndrome characterized by the occurrence of depression in autumn and winter, followed by a complete recovery in spring and summer (Rosenthal et al., 1984). Hypersomnia, increased appetite and weight gain are frequent concomitants.

The prevalence of SAD is estimated to be 4.3–10% in Maryland (USA) (Kasper et al., 1989), 4.3% in Iceland (Magnusson et al., 1991), 3.4% in Finland (Hagfors et al., 1992) 2.2% in Switzerland (Wirz-Justice et al., 1992) and 9.2% in Alaska (Booker and Hellekson, 1992). In Japanese outpatient university clinics, SAD was mostly reported in 1–3% of depressed patients who consulted a psychiatrist for the first time (Sakamoto et al., 1992).

In most patients suffering from SAD, the symptoms do not recur every winter (Rosenthal et al., 1984). Without prophylaxis or treatment, about two thirds of the SAD subjects become depressed in the following winter (Thompson, 1989). The depression usually takes from 2 to 6 weeks after the onset of the episode to reach maximum severity (Winton and Checkley, 1989).
The syndrome may well be caused by seasonal fluctuations in light intensity. Several studies have shown that light therapy is a highly effective form of treatment for SAD (Terman et al., 1989). In the present study, the effects of early light treatment on the course of mood during the winter season are investigated. For that purpose, we compared the course of mood in a group of winter depressives who received light therapy at the appearance of the first signs of a depression with that in a control group of winter depressive patients who did not receive any therapy. Preliminary findings of this investigation were presented before (Meesters et al., 1991).

Methods

All participants were outpatients meeting the criteria for winter depression as described by Rosenthal et al. (1984). They had been drug free for at least a month (in most cases this was since the previous winter season). In the winters of 1989-1990 and 1990-1991, the subjects' mood was assessed at weekly intervals from September until early April. The self-rating instrument used was the Beck Depression Inventory (BDI, 21 item version; Beck et al., 1961; Beck et al. 1979; Bouman et al., 1985). The BDI contains items to be scored from 0–3. Subjects are allowed to choose more than one answer per item. In those instances, we averaged the scores for that item. To make measurement of the atypical complaints by means of self-ratings possible, we created an addendum to the BDI (BDIadd) in analogy to the addendum to the Hamilton Rating Scale for Depression (HRSDadd; Rosenthal and Heffernan, 1986). This BDIadd correlated highly with the HRSD-Addendum (Meesters and Jansen, 1992a).

The first appearance of a BDI score of ≥13 was considered to represent the first sign of winter depression. This cut-off score has to be compared to a cut-off value of 17 which Beck et al. (1961) have used for discriminating between no depression and mild depression. The reason for this discrepancy is that most SAD people will not score on the items concerning sleep, eating and weight. So, a score of 13 seems a reasonable criterion of a very mild (beginning) winter depression. Similarly, a score of 22 (instead of 26) was chosen as the cut-off point for severe depression in the SAD subjects in this study.

Patients who reached a BDI score ≥13 were randomly assigned to either the treatment or the control group. For both groups the number of patients selected in each winter month is indicated in Fig. 1. Patients completed the following self-rating scales three times a day (7.30 a.m.; 3.00 p.m. and 11.00 p.m.) during a subsequent period of 24 days: the Adjective Mood Scale (AMS; Von Zerssen, 1986; Elsenga, 1988), the Activation Deactivation Adjective Checklist, which measures 4 components of activation: energy, tiredness, tension and calmness (AD-ACL; Thayer, 1976; 1978; 1986), a Visual Analogue Scale for depression, elation, anger and anxiety (VAS; Albersnagel, 1988), and the Stanford Sleepiness Scale (SSS; Hoddes et al., 1973.). The Sleep Quality scale which measures a patient's subjective sleep quality (SQ, range 0–14, with a high score indicating a poor sleep quality; Mulder-Hajonides v.d. Meulen et al., 1980), was completed once a day at 7.30 a.m. and the BDI once a week. After a 4-day baseline period (called 'before'), light therapy was given during a period of 5 consecutive days, from 9.00–12.00 a.m. (this period is henceforth labelled 'during'). The light was produced by a set of four full-spectrum fluorescent light tubes (Philips TL 58 w/95, 2500 lux). Mood assessments were continued during the withdrawal period. This latter period was

![Fig. 1. Cumulative number of subjects who became depressed (BDI ≥13) during the season, and were assigned to the treatment or the control group.](image-url)
split into a 10-day period ('after I') and a subsequent 5-day period ('after II'). At the end of the 'after I' interval, the severity of depression was rated by means of the 21-item Hamilton Rating Scale for depression (HRSD; Hamilton, 1967). The atypical symptoms were assessed by means of seven questions added to the HRSD (HRSDadd; Rosenthal and Heffernan, 1986). Interviewers were members of the research team who were not blind to the conditions. Taken together, the four periods mentioned above constitute the 'experimental period'. The subsequent part of the winter season is referred to as the 'follow-up period'. Here mood was assessed on a weekly basis (BDI).

Patients in the control group who reached a score of ≥ 22 during the experimental or follow-up period were considered to be severely depressed, and were offered light treatment for that reason. Once they received treatment, they served no longer as control subjects in the present study. Subjects who became severely depressed within the first 5 days of the protocol (this is the duration of the baseline period in the treatment group) were excluded from analysis. In such cases a

### TABLE 1

Average scores of four time intervals of treated vs. control subjects

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<td>11 14.8 10.1</td>
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Contr, control group; Treat, treatment group; BDI, Beck Depression Inventory; AMS, Adjective Mood Scale; VAS, Visual Analogue Scale; AD-ACL, Activation Deactivation Adjective Check List; SSS, Stanford Sleepiness Scale; SQ, Sleep Quality scale.
full-blown depression already existed before light treatment, so light treatment could no longer be applied to prevent the development of complaints. Fifty-four patients, 10 men, 44 women, were monitored from September onward. Thirty-two subjects were known from previous studies to suffer from SAD (Richter et al., 1992; Meesters et al., 1993). The remaining 22 SAD subjects were recruited through media publicity. All subjects had BDI scores below 13 at the beginning of the study and had no depressive complaints during the part of the winter season before the start of this study.

Thirty-eight participants, all outpatients, (70.4% of the total group) obtained a BDI score ≥ 13 and were therefore considered to be developing a depression. Their mean age was 39.1 (± 11.5 SD). Twenty of them were given preventive light treatment (4 men, 16 women), the others constituted the control group (5 men, 13 women). The two groups did not differ in severity of depression during the baseline period. Two subjects of the treatment group and four of the control group had a BDI score ≥ 22 on day 5 and were excluded from further analysis *). After that, the treatment group numbered 18 subjects and the control group 14 subjects. In the experimental period of 24 days, 3 patients from the control group and 2 from the treatment group dropped out, for reasons other than severity of depression (mainly because of the unpleasant obligation of having to perform frequent mood ratings). Patients visited the clinic for light treatment and for the HRSD interviews and completed the self-rating scales at home. As statistical procedures MANOVA with repeated measures, Mann-Whitney U, and the Kaplan Meier survival analysis (Kaplan and Meier, 1958) were used. If subjects, for any reason, dropped out during the experimental period, they were excluded from the MANOVA analysis. However, drop-outs due to the development of a severe depression are relevant to the present study. Their
data should be taken into account at least up to the day at which they dropped out. This was done by means of Kaplan Meier analysis. This analysis contains data about the period which started the first week of the experimental period (the week in which the treatment group received light) and which lasted till the end of the season or till subjects dropped out. Each subject participated in this study for one winter season only.

### Results

#### Experimental period

The results are shown in Table 1. In the treatment condition there was a significant improvement over time ($P = < 0.05$) in all self-rated variables. In the control group there was no improvement at all. MANOVA with repeated measures revealed an interaction effect on almost every self-rated variable (see Table 2) between the treatment and the control group. Only the tension sub-scale of the AD-ACL merely showed a trend of an interaction effect between both conditions. The mean HRSD score of the treatment group on day 19 (in week 6) was 5.9

<table>
<thead>
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<th>TABLE 2</th>
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BDI, Beck Depression Inventory; AMS, Adjective Mood Scale; VAS, Visual Analogue Scale; AD-ACL, Activation Deactivation Adjective Check List; SSS, Stanford Sleepiness Scale; SQ, Sleep Quality scale.

* Eight control subjects and eight subjects in the treatment group in the present analysis participated in our previous preliminary study (Meesters et al., 1991). In that study, three additional subjects were included, due to a less strict inclusion criterion.
(±4.7 SD), the score of the control group 9.1 (±4.9 SD). This difference was not significant (Mann-Whitney U: 82.5, z = -1.7, P = 0.10).

Follow-up

After the experimental period of 26 days the patients were followed by means of weekly BDIs until the beginning of April. Five control subjects became severely depressed (BDI ≥ 22) during the experimental period or after. They were therefore given light treatment and did no longer contribute to the design.

In contrast, not a single patient of the treatment group ever exceeded the BDI value of 22, so severe depression was not observed in this group. Kaplan Meier Survival Analysis revealed a significant difference between the two groups in developing a severe winter depression (Chi-square: 8.98; df = 1; P = 0.0027).

During the experimental period and the consecutive follow-up period some subjects in both the treatment and the control group became mildly depressed (BDI ≥ 13 and < 22) for one or more weeks. On calculating the ratio of the number of weeks in which subjects reached a BDI score ≥ 13 and the total number of weeks of the recording period, we found 0.14 ± 0.24 for the treatment group and 0.45 ± 0.35 for the control group. This difference is significant (Mann-Whitney U: 55.0, z = -2.7, P = 0.007). Fig. 2 shows the course of mood of the two groups during the winter season after the first onset of depression.

Two of the severely depressed patients in the control group recovered after one series of 5 days of light treatment, two patients needed two series, and one did not recover at all in response to light. In addition, none of the four subjects who dropped out of the control group and who had successful treatment became severely depressed during the same winter season.

Fig. 2 shows that the number of patients decreased over time in both groups. This effect can partly be attributed to the different points in time at which patients entered the study: Since the end of the experiment was set at the beginning of April, the duration of the monitored time intervals varied considerably. In addition, the reduction of patients in the control group is also partly caused by the drop-out due to the severity of depression.

Discussion

The administration of light at the very first signs of a winter depression, evidently prevented it from developing into a full-blown depressive episode. The treatment improved mood, the level of subjective activation and sleep quality. The control group of patients, who had not been given any therapy, either showed a deterioration or no change of depressed mood. This is illustrated by all mood variables examined *).

The BDI scores suggest that, in general, the beneficial effects of light treatment were maintained until spring. Only a minority became mildly depressed again, and this lasted at most for only a few weeks. Not a single person of the treatment group. This selection fully accounts for the differences.

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*) A quantitative comparison of the results of the present analysis and the published preliminary analysis (Meesters et al., 1991), may give the impression that the increased number of subjects has substantially reduced the magnitude of the overall effects. This, however, is not the case. In the preliminary analysis, data of all subjects from the two groups after admission to the experimental period were taken into account, including those subjects who had a BDI ≥ 22 score on day 5. This selection fully accounts for the differences.
group became severely depressed in the part of the winter season after light treatment. This finding is at variance with the reported relapse period of 3-4 days (Rosenthal et al., 1985a; Rosenthal et al., 1985b; Terman et al., 1989). However, several authors have found no such fast relapses. Wehr et al. (1986) reported that five out of seven patients did not relapse completely to their baseline depression levels before they started a second treatment condition within 9 days. Yerevanian et al. (1986) found virtually no relapse and Grota et al. (1989) no relapse at all within a period of 14 days after treatment. Wirz-Justice et al. (1986a) described a case study of a patient who had no relapse up to 3 months after treatment. In another study, Wirz-Justice et al. (1986b) reported that the time interval between termination of treatment and relapse showed extreme variation: some patients relapsed within 1 day, others remained free of symptoms during the remaining part of the winter. Meesters et al. (1993) found a relapse rate of 54% within the same winter season after a successful treatment (with the relapses occurring in a period from 2 to 14 weeks after treatment). In that study, 46% of the subjects didn’t relapse in a period of 9–21 weeks after treatment (the remaining part of the winter season; the average period being 14.7 weeks).

Lasting remission was also noted for the 4 subjects from the control group, who had been successfully treated, after a BDI score ≥ 22 had been reached. We have no explanation for the differences between the studies. Perhaps the weekly contact of the subjects with the research centre (through sending self-rating scales) contributed as a placebo effect to the lasting remission (Eastman, 1990).

However, whereas treatment at the onset of symptoms seems to prevent the development of a full-blown winter depression, we have shown in a separate study that winter depression cannot be prevented by exposing SAD patients to light early in the winter season when there are no complaints yet (Meesters et al., 1992b). This discrepancy suggests that the precise timing of light therapy is of great significance to the therapeutic effects. Light therapy administered at the emergence of the first depressive symptoms has prophylactic effects, whereas its administration in a symptomless phase, long before, has not. Variations in timing may therefore explain differences in relapse rates between studies.

In interpreting our data, there is a point which has to be mentioned. Not only did the treatment condition imply the administration of light, but it also included visits to the clinic on 5 consecutive mornings. In contrast, the control patients carried on with their normal daily routines. Consequently, the conditions differ at least in these two aspects. Without proper control for these differences, it remains uncertain what factors are responsible for the beneficial effect of the treatment condition.

Yet, in the prolonged follow-up period, contacts with the clinic were similar for both patient groups. Therefore, it is obvious from our data that a 1-week period of light treatment, with all of its accompanying physiological and psychological aspects, when applied at the very first signs of a winter depression is sufficient to reduce these minor complaints and to prevent the development of a severe winter depression for a long period of time.

Acknowledgments

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References


