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Research report

Seasonal depression
The dual vulnerability hypothesis revisited

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Abstract

Objective: In DSM-IV, winter seasonal affective disorder (SAD) is classified as a seasonal pattern of recurrent major depressive episodes in winter with full remission of symptoms in summer. However, other groups with ''winter depression'' have been identified, including patients with incomplete summer remission (ISR) and subsyndromal SAD (sub-SAD, winter depressive symptoms that do not meet criteria for major depression). In this study, we compare the clinical characteristics of these three seasonal groups and their response to light therapy. *Method*: 558 patients assessed at a specialized SAD Clinic were diagnosed using DSM-III-R or DSM-IV criteria. Clinical information was recorded using a checklist at index assessment. A subset of patients ($N=192$) were treated with an open, 2 week trial of light therapy using a 10 000 lux fluorescent light box for 30 min per day in the early morning. Patients were assessed before and after treatment with the 29 item modified Hamilton Depression Rating Scale and clinical response was defined as greater than 50% improvement in scores. *Results*: The rates of some melancholic symptoms, anxiety, panic, suicidal ideation, and family history of mood disorder were lowest in the sub-SAD group. The clinical response rates to light therapy were highest in the sub-SAD group ($N=32$, 78%), intermediate in the SAD group ($N=113$, 66%), and lowest in the ISR group ($N=47$, 51%). *Limitations*: This was a retrospective study of patients seen in a specialty clinic, although information was obtained in a standardized format. The light therapy trial had an open design so that placebo response could not be determined. *Conclusions*: There are differences in both the patterns of clinical symptoms and the response to light therapy in these three groups with winter depression. These results are consistent with a dual vulnerability hypothesis that considers these groups to result from interaction of separate factors for seasonality and depression.

Keywords: Seasonal affective disorder; Depression; Seasonal; Light; Light therapy; Diagnosis

1. Introduction

In the DSM-III-R and DSM-IV, winter seasonal affective disorder (SAD, or winter depression) is

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classified as a seasonal pattern or ''course specifier'' for recurrent major depressive episodes. Although so-called atypical features of depression are prominent in SAD (Rosenthal et al., 1984; Oren and Rosenthal, 1992; Lam, 1998), the diagnostic criteria only include the pattern of recurrent depressive episodes. These criteria include regular onset and regular remission (or a switch into mania or hypomania) of depressive episodes in a particular season, and exclusion of seasonal psychosocial stressors. If any nonseasonal episodes of depression have occurred in the patient's lifetime, the seasonal episodes must greatly outnumber any nonseasonal episodes. In the DSM-IV, the last two seasonal episodes must have occurred in the last two years. Using these criteria, the prevalence of SAD has been estimated at less than 1% in the United States (Blazer et al., 1998) and at 1% to 3% in Canada (Levitt and Boyle, 1997). Light therapy has been shown to be effective in the treatment of SAD in both large randomized controlled trials (Eastman et al., 1998; Terman et al., 1998) and in meta-analyses (Terman et al., 1989; Lee and Chan, 1999; Thompson et al., 1999). Recent clinical consensus guidelines have recommended light therapy as a first-line treatment for SAD (Lam and Levitt, 1999).

Other diagnostic groups with winter depression, however, have also been identified. ''Subsyndromal'' SAD is the term used to describe people with significant winter depressive symptoms that do not meet criteria for DSM-IV major depressive episode (Kasper et al., 1988). These patients usually have the vegetative symptoms described in SAD (hypersomnia, hyperphagia, weight gain) but either do not have depressed mood or do not have continuous symptoms for more than 2 weeks. Although patients with subsyndromal SAD are regarded as having a less severe form of SAD, they have considerable psychosocial impairment during the symptomatic winter months (Schlager et al., 1995). Preliminary studies suggest that light therapy may also be effective for subsyndromal SAD (Kasper et al., 1989a; Norden and Avery, 1993).

Full remission of symptoms in the summer is also required for the DSM-III-R and DSM-IV diagnosis of winter seasonal pattern. However, some patients with clear winter depressive episodes continue to endorse some depressive symptoms in the summer, hence do not meet this criterion for SAD. Preliminary studies have suggested that these patients with incomplete summer remission (ISR) may be less likely to have atypical depressive symptoms compared to SAD patients (Danilenko and Putilov, 1996) and may be less responsive to light therapy in the winter (Lingjaerde and Foreland, 1999). These studies have been limited, however, by small sample sizes ($N=32$ and 14, respectively).

The objective of this study was to further clinically characterize patients with subsyndromal SAD and ISR, compared to that of SAD. In a subset of patients, we also compared clinical responses to a standard, open trial of light therapy. We also propose a dual vulnerability hypothesis for seasonality and depression to explain the results.

2. Methods

The Seasonal Mood Disorders Clinic was established as a ''subclinic'' of a larger Mood Disorders Program in 1989 at the University of British Columbia Hospital in Vancouver, BC, Canada (latitude 49°N). The UBC Hospital is a tertiary teaching hospital located on a university campus. Referrals to the clinic were usually made by family physicians or psychiatrists; a smaller number were self-referred in response to advertisements for studies. Patients were assessed by experienced, board-certified psychiatrists and clinical diagnoses are assigned using DSM-III-R or DSM-IV criteria based on all available information. Clinical information was recorded for each patient using a symptom checklist (available on request). Patients were classified as SAD if they met criteria for major depressive disorder (MDD) and reported full remission of symptoms during the summer. If patients met criteria for MDD in the winter but did not experience full summer remission, they were classified as having incomplete summer remission (ISR). If patients did not meet criteria for MDD, but had significant symptoms during the winter and full remissions in the summer, they were classified as subsyndromal SAD (sub-SAD).

A subset of these patients were treated in an open design using a standard light therapy protocol described in previous controlled studies (Terman et al., 1990; Magnusson and Kristbjarnarson, 1991; Terman

et al., 1998). A 10 000 lux fluorescent light box with an ultraviolet filter was used (Daylight Technologies, Inc.). After receiving instructions, patients used the light box at home for 30 min in the early morning, as soon as possible after waking (between 7:00 and 9:00 am). Patients were rated after 2 weeks of treatment, and compliance was checked by inquiry. Only patients who used the light box for at least 5 days of each week were considered to have an adequate trial. Patients were rated pre- and post-treatment with the Structured Interview Guide for the Hamilton Depression Rating Scale, Seasonal Affective Disorder version (Williams et al., 1988), which generates a 21 item Hamilton Depression Rating Scale (Ham-21) (Hamilton, 1967) and an 8 item addendum that rates the atypical vegetative symptoms (Ham-8). Overall severity is rated with all 29 items (Ham-29). Patients taking antidepressant medications were only treated with light therapy if the medication dosage had been stable for at least 4 weeks and they were still clinically depressed. Medication doses were not changed during the light therapy trial.

Parametric data were analyzed using ANOVA, with post hoc pairwise comparisons using Tukey's tests to account for multiple comparisons. Non-parametric data were analyzed using chi square tests; if the overall chi square was significant, post hoc pairwise comparisons used a bonferroni correction. The analysis was performed using SPSS v.8.0 (SPSS Inc., 1998). All results are reported as percentages of available data or as mean ±S.D.

3. Results

Clinical information was available on 558 patients: 454 patients with SAD, 55 patients with sub-SAD, and 49 patients with ISR (Table 1). There were no significant differences between the three groups in the proportions of men and women, age, total number of depressive episodes, or number of winter depressive episodes. As expected, the ISR group had a significantly greater number of nonseasonal episodes than the other two groups.

Table 2 shows the clinical features of the three diagnostic groups during the winter depressive episode. All groups had a majority proportion of ''atypical'' vegetative symptoms, with no differences in the rates of hypersomnia or weight gain. There were significant differences in the proportions of patients with insomnia and weight loss, although the rates were low overall in all groups. The sub-SAD group had much lower rates than the SAD and ISR groups in experiencing other core symptoms of depression including anxiety and suicidal ideas. The sub-SAD and SAD groups had lower rates than the ISR group for panic attacks and past suicide attempts. Similarly, the sub-SAD group had a smaller proportion of patients with a first degree relative suffering from depression (of unspecified seasonality) than the SAD or ISR groups, although the rates for having a first degree relative with alcoholism were similar across the three groups.

Results from the standardized, open trial of light therapy were available for a subset of these patients

Table 1
Demographic information for the three diagnostic groups (Total $N=558$)

	Diagnosis		
	SAD[a] $N=454$	Sub-SAD[b] $N=55$	ISR[c] $N=49$
Proportion male (%)	26	26	29
Proportion female (%)	74	74	71
Age±S.D.	37.7±10.8	36.5±11.4	38.5±9.6
No. of total episodes ±S.D.	10.3±8.0	10.3±11.0	8.7±8.1
No. of winter episodes ±S.D.	9.8±7.9	10.1±9.4	8.5±8.0
No. of summer episodes ±S.D.[d]	0.4±1.3	0.2±0.6	1.3±2.4

[a] SAD = seasonal affective disorder.
[b] sub-SAD = subsyndromal seasonal affective disorder.
[c] ISR = incomplete summer remission.
[d] $F = 5.9$, $df = 2,467$, $P < 0.003$; post hoc Tukey's test: ISR > sub-SAD, SAD.

Table 2
Clinical information for the three diagnostic groups (Total $N=558$)

All data presented as %	Diagnosis			Post hoc comparisons[d]
	SAD[a] $N=454$	Sub-SAD[b] $N=55$	ISR[c] $N=49$	
Sleep				
–hypersomnia	68	66	75	No differences
–insomnia	24	11	17	SAD>Sub-SAD
Appetite				
–increased	55	46	38	No differences
–decreased	27	20	28	No differences
Carbohydrate craving	77	52	61	SAD>Sub-SAD, ISR
Weight				
–gain	51	58	45	No differences
–loss	14	2	17	SAD, ISR>Sub-SAD
Anxiety	87	56	87	SAD, ISR>Sub-SAD
Panic attacks	7	2	19	ISR>SAD, Sub-SAD
Suicidal ideas	45	27	55	SAD, ISR>Sub-SAD
Past suicide attempt	10	5	21	ISR>SAD, Sub-SAD
Family History–Mood	56	35	54	SAD, ISR>Sub-SAD
Family History–Alcohol	39	37	47	No differences

[a] SAD=seasonal affective disorder.
[b] sub-SAD=subsyndromal seasonal affective disorder.
[c] ISR=incomplete summer remission.
[d] Post hoc comparisons, if the overall chi square was significant, using Bonferroni correction for familywise $p<0.05$.

Table 3
Results from a standard, open 2 week light therapy trial (10 000 lux for 30 min per day in the early morning) for a subset of the patients ($N=192$)

	Diagnosis			Post hoc comparisons[a]
	SAD $N=113$	Sub-SAD $N=32$	ISR $N=47$	
Baseline Ham-29 score	31.6	26.4	26.8	SAD>sub-SAD, ISR
(±S.D.)	7.3	7.5	6.3	
Baseline Ham-21 score	18.5	14.7	15.8	SAD>sub-SAD, ISR
(±S.D.)	5.1	5.2	4.9	
Baseline Ham-8 score	13.1	11.7	11.1	SAD>ISR
(±S.D.)	4.7	3.7	3.7	
Ham-29 % improvement[b]	58	67	48	SAD, sub-SAD>ISR
(±S.D.)	23	29	22	
Ham-21 % improvement	60	71	45	SAD, sub-SAD>ISR
(±S.D.)	26	33	29	
Ham-8 % improvement	54	59	47	No differences
(±S.D.)	29	27	23	
Ham-29 response rate %[c]	66	78	51	SAD, sub-SAD>ISR

[a] Post hoc comparisons, if the overall ANOVA was significant, using Tukey's tests, $P<0.05$.
[b] Percent improvement from baseline to post-treatment.
[c] Response rate defined as greater than 50% improvement from baseline to post-treatment.

($N = 192$, Table 3). The baseline Ham-D scores were significantly different between groups ($F = 11.5$, $df = 2,189$, $P < 0.001$), with post hoc tests showing that the SAD group had significantly higher baseline scores than the other two groups (Tukey's test, $P < 0.05$). All three groups had good responses to light therapy. The ISR group, however, had significantly lower mean percentage improvement in Ham-21 and Ham-29 scores than the other two groups, although no differences between groups were found in the Ham-8 scores. Also, there were differences between groups in the clinical response rates to light therapy, defined as 50% or greater improvement in Ham-29 scores at post-treatment compared to baseline. The sub-SAD and SAD groups had significantly higher response rates (78 and 66%, respectively) compared to the ISR group (51%).

As for medication status, none of the sub-SAD patients, 13% of the SAD patients and 35% of the ISR patients were taking antidepressants during light therapy. However, there were no significant differences in response rates with medication status in either group (data not shown).

4. Discussion

The results of this study must be interpreted with some caution because of the nature of patient selection and the light therapy protocol. Patients were referred to the clinic because of seasonal problems, so there may be a selection bias. For example, the sub-SAD patients may be more severely affected than a community sample since they were actively seeking help. Because the light therapy trial was conducted with an open design, placebo responses and expectation effects cannot be excluded. However, the light treatment protocol in our clinic is more likely to represent ''real world'' results and the 66% response rate in the SAD group is consistent with other controlled and open studies of light therapy (Lam et al., 1997; Eastman et al., 1998; Terman et al., 1998).

Our main results indicate that there are certain clinical differences between the three putative diagnostic groups. Contrary to the results of Danilenko and Putilov (1996), the ISR and SAD groups had similar rates of the atypical vegetative symptoms. Although the rates of melancholic symptoms were low overall, the sub-SAD group had lower rates of insomnia and weight loss than the other two groups. Of interest, though, is that the sub-SAD group also had significantly lower rates of other core depressive symptoms such as anxiety, panic attacks, and suicidal ideation, and lower rates of family history of mood disorder, compared to the SAD and ISR groups. This suggests that subsyndromal SAD may be qualitatively different from SAD (and ISR) rather than being simply a milder form of SAD.

The light therapy results showed that all three groups improved, but the percentage improvement and response rate was significantly lower in the ISR group compared to the sub-SAD and SAD groups. Medication status did not affect the response rates. Atypical symptoms can predict response to light therapy (Nagayama et al., 1991; Oren et al., 1992; Krauchi et al., 1993; Lam, 1994; Terman et al., 1996), but the baseline Ham-8 score, representing atypical symptom items, was not significantly different between groups, suggesting that the differential response was not due to baseline differences in atypical symptomatology. These results are similar to those in a smaller group of ISR patients ($N = 12$) reported by Lingjaerde and Foreland, 1999. The ISR group in the present study had a higher mean improvement in depression score (48% versus 38%, respectively) but we treated the patients with 2 weeks of light treatment, compared to only 6 days in the other study. Again, because we did have a placebo control condition, we cannot exclude the possibility that the differences in light response reflect differences in the placebo response rates among the three groups.

The present study shows that, while winter depressive symptoms (and particularly the atypical vegetative symptoms) are common to all three diagnostic groups, there are clinical differences between the sub-SAD, SAD and ISR patients. The sub-SAD group appears to have fewer of the associated symptoms of depression (anxiety, panic, suicidal ideation, family history) that are found in the SAD and ISR groups. The sub-SAD group, however, had the best response rate to light therapy, whereas the ISR group had a more limited response.

Regardless, the results of this study challenge the DSM-III-R and DSM-IV concept of SAD as a

categorical diagnosis, where a patient either has a seasonal pattern, or not. Our results, and those of the other studies cited, indicate that there are other groups with winter depression that do not meet DSM criteria for SAD, suggesting greater heterogeneity in the expression of winter depression.

This heterogeneity may be explained by a dual vulnerability hypothesis as first proposed by Young et al., 1991 for SAD. They suggested that people develop SAD when they have significant primary seasonal physiological symptoms (e.g., vegetative symptoms and energy disturbance) coupled with a vulnerability to develop secondary depressive symptoms (e.g., guilt, anxiety and rumination, hopelessness, suicidal ideation). In essence, they attribute SAD to a combination of a seasonal factor and a depression factor. This hypothesis emerges from the concept of seasonality as a dimensional trait, with SAD perhaps representing the extreme end of a seasonality dimension (Blehar and Lewy, 1990; Bauer and Dunner, 1993; Avery and Norden, 1998). Studies using questionnaires that assess seasonality (e.g., the self-rated Seasonal Pattern Assessment Questionnaire (Rosenthal et al., 1987) that measures seasonal variation in mood, sleep, appetite, weight, social activity, and energy) have shown that there is a spectrum of seasonality. For example, seasonality scores in the general population show a continuous distribution with no evidence for a discontinuous construct like SAD (Kasper et al., 1989b; Jang et al., 1997).

Extending the dual vulnerability concept, we propose that there may be different combinations or loadings of seasonality and depression factors in any given individual that may be expressed in one of four diagnostic categories (Fig. 1). Subsyndromal SAD results when a person loads primarily on the seasonality factor with little or no loading on the depression factor. These patients primarily have vegetative symptoms (e.g., hypersomnia, hyperphagia, weight gain) and anergia, without the cognitive symptoms required for a diagnosis of major depression. At the opposite spectrum, a person loading primarily on the depression factor with little or no contribution from the seasonality factor will experience a depressive illness that has no seasonal component, a ‘‘true’’ nonseasonal depression. These patients may have more typical melancholic depressive symptoms such as insomnia and weight loss. Between these two extremes, however, are people with intermediate loadings on seasonality and depression factors. Higher loadings on seasonality than

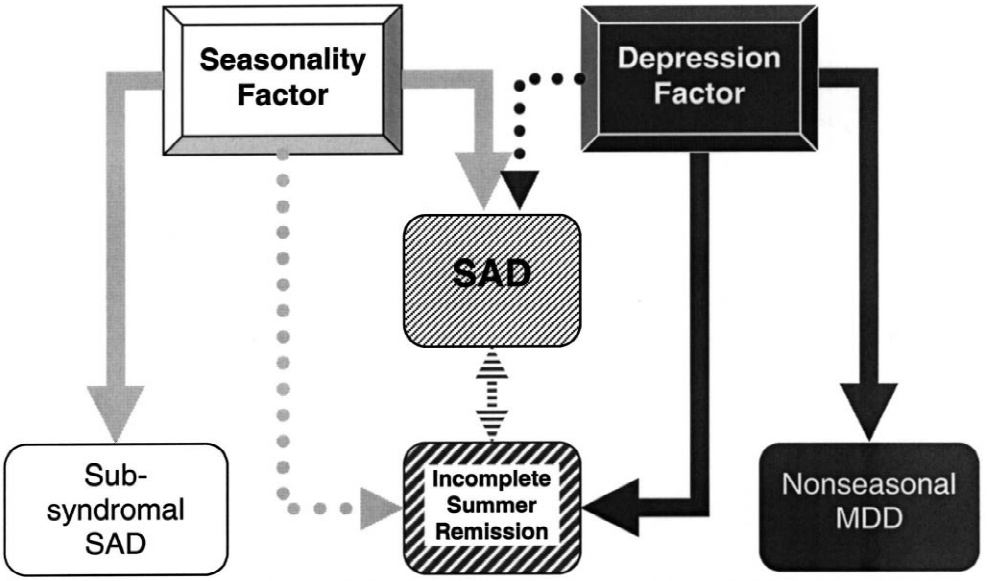

Fig. 1. A dual vulnerability model for seasonal and nonseasonal depressive diagnoses. Solid lines indicate greater loading on a factor, while broken lines indicate lesser loading (see text for further explanation).

depression factors result in SAD with full summer remission of symptoms. Conversely, higher loadings on depression than seasonality factors result in winter depressive episodes with incomplete summer remission.

This dual vulnerability model would explain some of the inconsistent findings reported in the literature. Prospective, longitudinal follow-up studies of SAD show that many patients diagnosed with SAD do not continue to have seasonal courses of illness (Leonhardt et al., 1994; Sakamoto et al., 1995; Thompson et al., 1995; Schwartz et al., 1996). Pooling the results from these studies, about a third of SAD patients (28–44%) go on to have complicated episode patterns suggesting a more nonseasonal course that would meet our criteria for ISR. A similar proportion (14–38%) either have subsyndromal episodes or went into clinical remission. Only a third (22–42%) continued to have definite, recurrent seasonal major depressive episodes on follow up.

In a dual vulnerability hypothesis, the seasonality and depression factors may have variable expression. The seasonality factor could result from an interaction of a vulnerability trait and external environmental influences, such as photoperiod or amount of available light in the winter. Similarly, the depression factor could also be affected by a vulnerability trait, and by external effects such as stressful life events. A complex interplay of these factors could account for the different illness patterns seen from year to year as found in the follow-up studies. The vulnerability traits for both factors could be genetic, consistent with the findings in twin studies that a substantial proportion (29–69%) of the variance in seasonality scores can be attributed to a genetic factor (Madden et al., 1996; Jang et al., 1997). In fact, this hypothesis is testable using twin studies, in that different heritability factors for seasonality and major depression can be investigated within the same cohort.

The dual vulnerability model may also account for some of the inconsistent findings in the neurobiological study of SAD. There may be different pathophysiological mechanisms for the seasonality and depression factors. For example, phase-delayed circadian rhythms found in SAD (e.g., (Lewy et al., 1987; Sack et al., 1990; Dahl et al., 1993) may be related to the seasonality factor (and thus more correctable by morning bright light exposure), while monoamine dysregulation may be associated with the depression factor. In any given cross-sectional sample of identified SAD patients, differential loading of the two factors can result in biological heterogeneity leading to inconsistent results between studies, explaining why, for example, that not all studies are able to demonstrate phase-delayed circadian rhythms in SAD (e.g., Eastman et al., 1993; Wirz-Justice et al., 1993; Oren et al., 1996).

The relevance of this hypothesis is that it will be important to study seasonality as a dimension separate from, or in addition to, the categorical diagnosis of SAD. Following this approach, some investigators have shown seasonal patterns in mood and behavioural symptoms in longitudinal studies of the normal population (Harmatz et al., 1999; Murray et al., 1999). A separate seasonality factor would also be consistent with reports of seasonal variation in symptoms in other depressive and non-depressive disorders including bulimia nervosa (e.g., Hardin et al., 1991; Lam et al., 1991, 1996; Blouin et al., 1992; Levitan et al., 1996), premenstrual depression (Maskall et al., 1997), panic disorder (Marriott et al., 1994), obsessive–compulsive disorder (Yoney et al., 1991), and post-traumatic stress disorder (Solt et al., 1996).

Another possibility is that light therapy may be specifically responsive for the seasonality factor. Studies of light therapy in ''nonseasonal'' depressed patients have reported inconsistent results. Heterogeneity in the loading for the two factors in these patients may explain why some studies show statistically significant positive results (Levitt et al., 1991; Kripke et al., 1992; Yamada et al., 1995), while others are negative (Yerevanian et al., 1986; Mackert et al., 1991; Thalen et al., 1995).

Indeed, treatment may need to be targeted more specifically at each of the two factors. A partial response to light therapy in a SAD or ISR patient may, in fact, indicate a complete response for the seasonality factor, but still require adjunctive treatments (e.g., medications, psychotherapy) for the depression factor. Similarly, this hypothesis provides a rationale for the use of light therapy as adjunctive treatment in other seasonal disorders, including bulimia nervosa (Lam et al., 1994; Blouin et al.,

1996), premenstrual depressive disorder (Parry et al., 1993; Lam et al., 1999), and ''nonseasonal'' depression.

In summary, this study found that the clinical features of subsyndromal SAD patients were qualitatively different than those of SAD patients, and that the patients with incomplete summer remission had lower response rates to light therapy than SAD or subsyndromal SAD patients. Even though the ISR group had lower response rates, it should be noted, however, that a response rate of 51% is still clinically meaningful, especially since many of these patients had not shown adequate response to an antidepressant medication. We have interpreted these results within a dual vulnerability hypothesis for seasonality, seasonal depression (including sub-SAD, SAD and ISR) and nonseasonal depression. Further research will be required to test this dual vulnerability hypothesis.

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