Seasonal Affective Disorder: A Review of the Syndrome and Its Public Health Implications

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Abstract: Seasonal affective disorder (SAD) is a disturbance of mood and behavior which resembles some seasonal changes seen in lower mammals. Like these animal seasonal changes, SAD is thought to be related to decreased sunlight during winter months. [SAD has been successfully treated with exposure to bright artificial light of higher intensity than is usually present in the home or workplace. Many people not suffering from SAD may nonetheless have seasonal changes which could be helped by environmental light supplementation. Lighting standards in the home and workplace should be re-evaluated on the basis of new knowledge of the psychobiological effects of light.] We review the literature on SAD and discuss its public health implications in the context of a typical case presentation. (*Am J Public Health* 1987; 77:57–60.)

Introduction

Animal seasonal behaviors such as activity, reproduction, and migration are triggered by changes in the duration of daily sunlight (photoperiod).[1–3] Humans also show seasonal patterns, for example, in the growth rate of children, the incidence of menarche, hemoglobin and hormonal levels, birthrate, mortality, and suicide.[4] Like circannual rhythms in animals, human seasonal changes may be influenced by the duration of sunshine.[4]

While numerous studies have investigated the effects of indoor workplace lighting on such measures as productivity,[5–10] the public health implications of seasonal changes in environmental light have been largely ignored. We have recently characterized a recurrent fall/winter human syndrome, termed seasonal affective disorder, which resembles some seasonal changes found in lower mammals and which can often be effectively treated with light known to be biologically active in humans.[11] In this article we review the literature regarding SAD and discuss its potential public health relevance.

Seasonal Affective Disorder

After identifying a patient who manifested recurrent seasonal mood changes, we attempted a therapeutic manipulation based on the animal model of seasonal photoperiodism. In that patient, who developed depressive symptoms each fall as the days became shorter, we showed that lengthening the natural photoperiod with daily exposure to six hours of bright artificial light led to a reversal of his depressive symptoms.[12] Subsequently, we described a syndrome of seasonal mood changes called seasonal affective disorder and have treated over 100 patients with light.[11,13–17] Our findings have now been replicated by several other groups.[18–21]

Seasonal affective disorder (SAD) is a cyclic illness characterized by recurrent episodes of fall/winter depression alternating with periods of spring/summer euthymia (normal mood) or hypomania (mild elation and behavioral activation)[11] Diagnosis of SAD is based on the validated Research Diagnostic Criteria (RDC) for major depression,[22] from which the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (3rd edition) is derived.[23] SAD diagnostic criteria are:

1) a history of depression fulfilling RDC criteria for major affective disorder, depressed;
2) a history of at least two consecutive years of fall/winter depressive episodes remitting in the spring or summer;
3) the absence of other major (Axis I) psychiatric disorder or psychosocial explanation for the seasonal mood changes.

While SAD patients fulfill standard diagnostic criteria for major depression, they also frequently manifest the so-called "atypical" depressive[24] features of hypersomnia, increased appetite, weight gain, and fatigue, which are relatively uncommon in major depression.[25] Moreover, many SAD patients report symptoms (daytime drowsiness, carbohydrate craving) which are not diagnostic of either major or atypical depression, but may occur in sleep disorders[26] or eating disorders.[23] Table 1 shows the percentages of SAD patients studied at the National Institute of Mental Health from 1981–85 who reported specific typical and atypical depressive features.

Untreated SAD depressive episodes generally end in the spring and are frequently succeeded by a constellation of symptoms opposite to those seen in the winter. Summer manifestations are usually mildly elated or irritable mood; increases in energy, libido, creativity, and social behavior; decreased appetite and need for sleep; and loss of winter weight. However, a small percentage of patients (7 per cent) have a history of being more severely affected and becoming manic during the spring or summer, while 10 per cent have had winter depressions without summer symptoms (Table 2).

SAD typically begins in the second or third decade of life, although we have identified several children with the disorder.[15] It is four times more common in women than men, a female predominance greater than for most other psychiatric disorders.[23] The majority of SAD women also experience some of the above mentioned "atypical" depressive symptoms in association with the premenstrual phase of their menstrual cycles,[27,28] a condition which has been termed premenstrual syndrome (PMS).[29] Moreover, in many SAD women affected by premenstrual difficulties there appears to be a clear exacerbation of premenstrual symptoms during the winter.[30]

Indirect assessment of reproductive function by analysis of birthrate data has revealed that SAD women have a large seasonal variation, with a peak birthrate of children to SAD

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TABLE 1—Symptoms Reported by SAD Patients during Winter (by per cent of patients, N = 156)

Variables	Symptom Reported	% of Patients
Activity	Decreased	96
Affect	Sadness	94
	Irritability	79
	Anxiety	84
Appetite	Incresed*	66
	Decreased	19
	Mixed	14
	No change	1
	Carbohydrate craving**	67 (N = 123)
Weight	Increased*	72
	Decreased	13
	Mixed	1
	No change	15
Libido	Decreased	62
Sleep	Increased duration*	79
	Later wakening**	69
	Earlier onset**	60
	Change in quality	77
	Daytime drowsiness**	82 (N = 123)
Other	Symptoms milder nearer equator**	74 (N = 71)
	Menstrual difficulties**	61 (N = 116)
	Work difficulties**	88
	Interpersonal difficulties**	94

*"atypical" depressive symptom.
**not diagnostic of typical or atypical depression.

women in May and a nadir between August and December.[30] The distribution of birthrate by months stands in contrast to that of the US population, which peaks in September and manifests a much smaller seasonal variation. The decreased August-to-December birthrate implies decreased conception between December-through-March, precisely the interval during which SAD patients suffer from decreased libido, lack of energy, and depression. Similarly, the May birthrate peak signifies an increased conception rate during the summer, a time when many SAD patients describe increased libido, energy, and social activity.

Family history data suggest that SAD may be genetically related to other forms of affective illness since more than two-thirds of SAD patients have a first degree relative with a major affective disorder (Table 2). Moreover, more than a third of the SAD patients we have surveyed have a first degree relative with seasonal affective disorder. These results are consistent with epidemiological studies showing increased rates of affective disorder in first degree relatives of depressed patients.[31]

Numerous biological correlates of SAD have been investigated. Physical examinations and laboratory tests of serum cortisol and the dexamethasone suppression test,[11] thyroid function tests (T3, T4, and TSH),[11] serum glucose and the glucose tolerance test,[30] and profiles of melatonin secretion,[16,32] have been normal. We have recently presented evidence that during their winter depressions SAD patients have higher levels of prolactin than age- and sex-matched volunteers.* While SAD patients' mean prolactin levels are 50–100 per cent higher than controls, they are within the normal human range. This finding is intriguing since prolactin triggers seasonal changes in lower mammals such as reproduction[33] and coat coloration.[34]

*Jacobsen FM, Sack DA, Wehr TA, Rogers S, Rosenthal NE: Prolactin is elevated in seasonal affective disorder. New Research Programs & Abstracts NR119, p 76, 139th Annual Meeting, American Psychiatric Association, Washington, DC, 1986.

TABLE 2—Clinical and Demographic Features of SAD (N = 156)

Age (years): 40.2 ± 10.1		
Age of onset (years): 22.8 ± 10.9		
Sex ratio (F/M): 127:29 (4:1)		
Length of depressions (months): 4.3 ± 1.4		
		%
Psychiatric Diagnosis:*	Bipolar II	83
	Bipolar I	7
	Unipolar	10
		%
Family history (at least one affected first degree reltive):	Major Affective Disorder	69
	SAD	37
	Alcohol Abuse	19
Previous treatment history:	No treatment	46
	Antidepressants	30
	Lithium	13
	Thyroid	9
	Hospitalization	12
	Electroconvulsive therapy	0

*By Research Diagnostic Criteria.

Electroencephalographic (EEG) all-night sleep monitoring has confirmed patients' reports of an increase in total sleep time during winter depressive episodes compared with summer periods of euthymia/hypomania. Slow wave sleep may also be significantly reduced in SAD patients during their winter depressions compared with levels of this sleep parameter in normals.

Phototherapy of Seasonal Affective Disorder

The lights which have been used for the treatment of SAD differ from those generally found in the home or workplace in two ways: spectrum and intensity. Most published studies on SAD have used full-spectrum fluorescent lights in an attempt to mimic sunlight's effects on the human visual system. However, incandescent light of higher than usual intensity has also been useful in an uncontrolled outpatient clinical trial,[35] suggesting that full-spectrum light may not be essential for reversal of seasonally related symptoms. While the relative benefits of full-spectrum versus incandescent light remain to be determined, we should caution that the safety of bright incandescent lights in the treatment of SAD has not been investigated.**

The light intensity used as an active condition in all studies of SAD to date (2500 lux: about the same intensity of light that one would be exposed to when standing at a window on a bright spring day) is known to have central nervous system activity in the suppression of human melatonin secretion[36] and the entrainment of human circadian rhythms.[37] Several double-blind random-ordered crossover studies have shown that this high intensity light is significantly more effective than light of ordinary intensity in reversing depressive symptoms in SAD patients.[11,13,14] The lights used as controls in these studies have been of intensities commonly found in the home and office (100–300 lux), which appear bright to the human eye but are of insufficient intensity to suppress human melatonin secretion[36] or affect human circadian rhythms.[38] We believe that the bright light's reversal of SAD symptoms is due to its specific biological activity. However, the possibility of a placebo response such

**The full-spectrum fluorescent lights (Vitalite®) used in our studies were tested for safety of short-term (two week) usage by the National Eye Institute. While we have noticed no long-term side effects of the lights, it is premature to conclude that there are none since long-term safety studies have not been performed.

as the well known "Hawthorne effect" described years ago in industrial studies[6] cannot be completely rejected due to the difficulty of administering light in a double-blind fashion.*** Nevertheless, a number of factors argue against a placebo effect:

- unlike a placebo response, relief from SAD symptoms does not occur immediately, but generally takes from three to five days;
- this symptomatic improvement is maintained until the lights are withdrawn, and remains effective year-after-year;
- phototherapy can be used prophylactically to prevent fall declines;[35]
- a biological mechanism is further suggested by the fact that relapse in patients withdrawn from phototherapy before springtime does not occur immediately, as might happen with a placebo, but takes about three days (about the same amount of time as for treatment to take effect).

The duration and timing of light exposure are important scientific and practical factors in the use of phototherapy. We initially used five to six hours of daily phototherapy given in divided doses at the beginning and end of the day, based on the hypothesis that winter depressive symptoms are an abnormal response to the natural seasonal diminution in day length. Recently, however, several groups have reported obtaining significant antidepressant effects using as little as a single two-hour "dose" of daily phototherapy[18–20] and a study contrasting long (summer) and short (winter) day lighting treatments found no difference in therapeutic response,[16] thereby casting doubt on the importance of timing for phototherapeutic effect. Further work is needed to clarify the mechanism of action of phototherapy in SAD.

The following is a typical case example of a patient with seasonal affective disorder:

> Betty was a 37-year old white female accountant who entered our program stating: "I don't want to get fired this winter." She related suffering since adolescence from recurrent winter depressions, consisting of daytime somnolence and fatigue, "restless" sleep with duration increased up to 12 hours daily, increased appetite with carbohydrate craving and a 10–20 lb weight gain, sadness and irritability, decreased libido, and social isolation. At her job, B experienced mid-afternoon drowsiness and concentration difficulties, leading to calculation errors and "probationary status." She began taking frequent "sick days" and developed fleeting suicidal ideation. We could not identify precipitating psychological events for B's depressions.
>
> B's depressive episodes began by mid-fall and worsened rapidly in the winter. They lasted until late March or April, then gradually lifted. During the spring and summer she was quite energetic and excelled at her job ("That's when I've received all my promotions"), slept less, no longer craved carbohydrates, and lost much of her winter weight. B noticed that her depressive periods were also improved by traveling south during the winter, but the improvement faded within days of returning north.
>
> B's mother suffered from similar winter depressions. Her father and several distant relatives were alcoholics. Medical history and physical examination were unremarkable.
>
> Light intensity measurements could not be made at the patient's workplace, but were said to be similar to the overhead fluorescent lighting of a clinic room, which measured about 300 lux at eye level. Her symptoms diminished after three days of exposure to six hours of bright (2500 lux) full-spectrum light per day at home and completely resolved when her home treatment was supplemented by bright full-spectrum lights at work. She remained well for the rest of that winter. Toward the end of the following summer she began to use phototherapy prophylactically and did not relapse (for the first time in years) while maintained on phototherapy.

Public Health Implications

Most SAD patients complain of daytime drowsiness, fatigue, and diminished concentration. Although their depressions are generally not severe enough to require hospitalization or stopping work, most patients (88 per cent) report significant disability at work as well as in their relations with others (94 per cent). Key symptoms of SAD such as daytime fatigue and somnolence are important not only for job performance, but also for public safety. A study of 1,502 industrial workers found a higher incidence of multiple work accidents among workers complaining of excessive daytime sleepiness compared with normals or workers with only nighttime sleep disturbances.[39] Another group recently found in an on-the-job survey in northern Norway that more than 23 per cent of people suffer from daytime sleepiness and a diminished work capacity during mid-winter, a condition partially reversible with exposure to bright artificial light.[40] Interestingly, the changes noted in the Norway study may not have been accompanied by depression and were ascribed to a seasonal sleep-wake disorder, "mid-winter insomnia".

The presence of daytime fatigue and hypersomnia, which affect job safety and performance adversely, may also predict response to phototherapy in SAD patients. Light-mediated improvement in these and other SAD symptoms (e.g., work productivity) may represent a physiological brain effect, since the high-intensity light used in the phototherapy of SAD has been shown to suppress melatonin secretion, entrain circadian rhythms, and may also increase arousal[41–43]† in humans.

Seasonal affective disorder is a disturbance of mood and behavior which resembles seasonal behavioral rhythms of lower animals. Based on animal models of light-induced seasonal rhythms, the modification of environmental lighting has been used to treat SAD patients. While the studies reviewed here represent samples of people who fit rigorous screening criteria for SAD and could participate in research, preliminary evidence from our group suggests that approximately 20 per cent of normal individuals complain of moderate to marked changes in mood and energy across the seasons, winter usually being the least favored season. Among SAD patients, complaints of SAD symptoms increased with increasing latitude in the US.†† Formal epidemiological surveys would be useful in determining the prevalence of seasonally related symptoms in the general population. The success of phototherapy in treating SAD patients suggests that it may be useful to identify people whose health and productivity suffer on a seasonal basis and study the effect of different ambient light levels on their mood and productivity.

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