

Canadian Consensus Guidelines for the Treatment of Seasonal Affective Disorder:

A Summary of the Report of the Canadian Consensus Group on SAD

Editors - Raymond W. Lam, MD, FRCPC and Anthony J. Levitt, MD, FRCPC

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APOLLO

Seasonal affective disorder (SAD) is a subtype of major depression that is characterized by onset at a certain time of year, usually the winter. These guidelines were arrived at by consensus, and have undergone both internal review by the 14 members of the consensus group and by external, international consultants. The executive summary of these guidelines for the clinician and this journal was chosen to ensure the widest distribution of this information to physicians across Canada. We hope that the information presented in this executive summary will assist clinicians to better identify patients with SAD and to manage the disorder more effectively in their practices. We also hope the guidelines will help patients and their family members.

Canadian Consensus Guidelines for the Treatment of Seasonal Affective Disorder

This document represents a summary of the report of the Canadian Consensus Group on SAD. The purpose of the consensus guidelines was to systematically review all available evidence regarding the diagnosis, clinical features, epidemiology, and treatment of SAD. The guidelines are based on a series of recommendations that were formulated based on the best available evidence on which the recommendations are based. The level of evidence on which the recommendations are defined below. Level 1 describes the highest level of evidence and level 5 describes the lowest level.

Guideline Panel Members

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Introduction to This Document

Seasonal affective disorder (SAD) is a subtype of major depression that is characterized by onset at a certain time of year, usually the winter. These guidelines were arrived at by consensus, and have undergone both internal review by the 14 members of the consensus group and by external, international consultants. The executive summary format was chosen to provide a quick reference for the clinician and this journal was chosen to ensure the widest distribution of this information to physicians across Canada. We hope that the information presented in this executive summary will assist clinicians to better identify patients with SAD and to manage the disorder more effectively in their practice. We also hope the guidelines will help physicians to explain some of the many questions that patients and family members ask about SAD.

This document represents a summary of Consensus Guidelines for the Treatment of Seasonal Affective Disorder (SAD) developed by a group of Canadian researchers and clinicians. The purpose of the consensus guidelines was to systematically review all available evidence regarding the diagnosis, clinical features, epidemiology, pathophysiology and treatment of SAD, and to produce a series of recommendations that were clinically and scientifically meaningful. The levels of evidence on which the recommendations are based appear after each recommendation. These levels of evidence are defined below. Level 1 describes the highest level of evidence and level 5 describes the lowest level.

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Levels of Evidence for Data

Level 1: Randomized controlled trials (RCT's) with sufficient numbers or good quality meta-analyses based on RCT's.

Level 2: RCT's with smaller numbers (therefore insufficient power).

Level 3: Non-randomized, controlled or cohort studies, case series, case-controlled or cohort studies, cross sectional or high quality retrospective studies.

Level 4: Evidence based on the published opinion of expert committees, for example consensus / guidelines committees.

Level 5: Evidence which expresses the opinion of the committee member(s) who have reviewed the literature and guidelines, following discussion with peers.

Diagnosis and Epidemiology**How is a "seasonal pattern" of depression defined?**

Seasonal affective disorder (SAD), first described in 1984, is also referred to as seasonal depression, winter depression or major depression with a seasonal pattern. Each of these terms refers to a subtype of major depressive disorder. SAD is characterized by the following four central features:

Recurrent major depressive episodes that start around the same time each year (e.g., September-October) and end around the same time each year (e.g., March-April).

Full remission of symptoms during the unaffected period of the year (e.g., May-August).

Over the lifetime course of the illness there are relatively more seasonal depressive episodes than non-seasonal episodes.

Seasonal depressive episodes occur in at least 2 consecutive years.

The DSM IV criteria for SAD are shown below. There is some argument about the definition for "the same time each year", but most investigators and clinicians allow for a 60-90 day "window" for the timing episode onset and timing of remission. In general, the patient should have an "absence of symptoms" for at least two months to be considered in remission from the seasonal episode. The most common type of SAD is winter depression. "Summer" SAD is uncommon in northern latitudes and much less is known about it.

DSM IV Criteria for "Seasonal Pattern" Specifier (SAD)

With Seasonal Pattern can be applied to the pattern of Major Depressive Episodes in Bipolar I Disorder, Bipolar II Disorder, or Major Depressive Disorder, Recurrent.

There has been a regular temporal relationship between the onset of major depressive episodes in bipolar I or bipolar II disorder or major depressive disorder, recurrent, and a particular time of the year (e.g, regular

appearance of the major depressive episode in the fall or winter). Note: Do not include cases in which there is an obvious effect of seasonal related psychosocial stressors (e.g., regularly being unemployed every winter).

Full remissions (or a change from depression to mania or hypomania) also occur at a characteristic time of the year (e.g., depression disappears in the spring).

In the last 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined in Criteria A and B, and no nonseasonal major depressive episodes have occurred during that same period.

Seasonal major depressive episodes (as described above) substantially outnumber the non-seasonal major depressive episodes that may have occurred over the individual's lifetime.

Summary Diagnostic Criteria For Seasonal Affective Disorder

Recurrent major depressive episodes that start around the same time each year (e.g., September-October) and end around the same time each year (e.g., March-April)

Full remission of symptoms during the unaffected period of the year (e.g., May-August)

Over the lifetime course of the illness, there are relatively more seasonal depressive episodes than non-seasonal episodes

Seasonal depressive episodes occur in at least 2 consecutive years

What are the symptoms of depression in SAD?

SAD patients have the usual symptoms of depression, including low mood, reduced interest, decreased concentration, low energy and fatigue. SAD patients also tend to have a specific symptom cluster consisting of the so-called "reverse" or "atypical" vegetative symptoms of depression. These symptoms include increased sleep (70-90% of SAD patients), increased appetite (70-80% of SAD patients), unacceptable weight gain (70-80% and carbohydrate/sweets craving (80-90%).

Is the diagnosis of SAD stable over time?

Most long-term follow-up studies of SAD demonstrate that over 60% of patients diagnosed with the disorder continue to demonstrate a seasonal disturbance of mood and/or behaviour over time. However, only about half of these patients continue to have discrete seasonal depressive episodes. Approximately 20% of SAD patients can have complete remission within several years of first diagnosis. The stability of the diagnosis of SAD appears to be similar to the long-term stability of the diagnosis of major depression itself (i.e., 44-76% of patients with major depression retain the diagnosis over several years of follow-up), and similar to the stability of other subtypes of recurrent depression, such as melancholic and atypical depression.

What instruments are useful for the diagnosis and measurement of SAD?

The only widely used instrument to detect SAD is the Seasonal Pattern Assessment Questionnaire (SPAQ), a self-report questionnaire that retrospectively assesses the magnitude of seasonal change in sleep, social activity, mood, weight, appetite and energy. The SPAQ is a very simple, brief and useful screening questionnaire, but a careful clinical evaluation is still necessary to confirm the diagnosis. Instruments used to measure the severity of SAD include the Structured Clinical Interview Guide for the Hamilton Depression Rating Scale, SAD version (SIGH-SAD), a 29-item interviewer-rated scale that is

widely used in studies of SAD. There are also self report measures of severity of depression, such as the Beck Depression Inventory (version II), and a self-report version of the SIGH-SAD.

Do SAD patients frequently have bipolar illnesses?

The majority of patients with SAD have unipolar depression, but as many as 20% may have or go on to develop a bipolar course. Typically the manic or hypomanic episodes occur in the spring and summer, and these must be carefully distinguished from the improved mood that is associated with recovery from the winter depression. There are important treatment differences for patients with bipolar as compared with unipolar illness.

Are there other diagnoses to consider in a patient presenting with winter difficulties?

Many people will complain of seasonal difficulties, but clinicians need to consider a variety of conditions in the differential diagnosis of SAD. Seasonally recurrent psychosocial stressors (e.g., fall/winter unemployment, anniversary grief reactions during the fall-winter) may produce some of the symptoms of depression. Some people experience marked changes in sleep, appetite, weight and energy during the winter but do not meet criteria for a major depressive episode - such patients are generally considered to have "subsyndromal" SAD. Preliminary studies suggest that such patients may also have a good response to light therapy. Finally, a number of reports indicate that conditions other than major mood disorders may be subject to significant seasonal influences. These conditions include eating disorders, premenstrual syndromes and anxiety disorders (panic disorder, obsessive-compulsive disorder).

What is the prevalence of SAD?

There have been more than 15 studies that have examined the prevalence of SAD in various population groups. Community-based surveys in North America have reported the prevalence of SAD between 0.8% and 9.7%. European community-based studies have estimated the prevalence of SAD between 1.3% and 3% of the population and studies in Asia report rates of 0 to 0.9%. The discrepancy in these findings may be attributed to a variety of methodologic differences between the studies. However, the most important factor that appears to account for the relatively wide range of prevalence estimates is the diagnostic instrument that was employed in each study. Most studies use the SPAQ (see section on diagnosis), which tends to over-estimate the prevalence. Studies that have used a more accurate structured diagnostic interview and standard diagnostic criteria suggest that the prevalence of SAD in Canada is between 2% to 3%, and that the rate in the US is less than 1%. By comparison, bipolar disorder or manic depression occurs in just over 1% of the population.

What is the female to male ratio?

Virtually all studies of the prevalence of SAD report that women are more likely to suffer from SAD. In some studies the female to male ratio is close to 4 to 1, but the average ratio across all studies is approximately 1.8 to 1.

What is the prevalence with respect to age?

Epidemiological studies report that the lifetime prevalence of SAD increases with age until the sixth decade. After the age of 50-54 the prevalence declines dramatically, such that the prevalence of SAD over 65 is very low. Nonetheless, patients over 65 may still present to clinics for treatment and clinical experience suggests their response to treatment does not differ from that of younger patients with SAD.

Does latitude impact on prevalence?

Some earlier studies in the United States that used the SPAQ reported a significant effect of latitude on prevalence, with an increase in prevalence with increasing latitude. However, more recent studies using structured interviews have shown that the latitude effect is not as robust as previously suggested.

Do other factors impact on the prevalence of SAD?

The recall of lifetime episodes of seasonal depression is affected by the time of year the interview takes place; that is, patients interviewed during the fall or winter are more likely to report lifetime seasonal difficulties as compared to patients interviewed in the summer. One USA study, which used a structured diagnostic interview, reported that SAD patients were more educated than non-SAD patients were and that it was more common in rural settings. However, a Canadian study, which used a similar diagnostic interview, found no urban-rural or educational effects.

PATHOPHYSIOLOGY OF SAD

Research has focused on the relationships between light and SAD, but the etiology and pathophysiology of SAD remain unknown. When SAD was first described in 1984, the obvious link between SAD, seasonal changes in photoperiod and the response to bright light inspired hope that an underlying photoperiodic mechanism would soon be found. Subsequent research has shown that SAD is likely a heterogeneous condition, since no one factor has been found to account for SAD. Several promising theories, not necessarily mutually exclusive, have emerged, including disturbances in circadian rhythm and serotonin regulation.

What is the role of circadian rhythm disturbance in SAD

The original model of SAD as a simple disturbance in photoperiodic regulation has not been confirmed. Another hypothesis considers that SAD results from phase-delayed circadian rhythms that are corrected by appropriately timed light therapy. Several studies have supported these findings, and bright light clearly has effects on the human circadian system. Not all studies, however, have shown circadian rhythm disturbances in SAD or that light-induced changes in circadian rhythms are necessary for an antidepressant effect. Melatonin was of interest in SAD because it mediates many seasonal animal behaviours and bright light has direct suppressive effects on nocturnal melatonin secretion. It is unlikely that melatonin is directly involved as a cause of SAD, because studies have not shown consistent changes in melatonin levels and melatonin suppression is not always required for the antidepressant effect of light.

What neurotransmitters are involved in SAD?

Various neurotransmitters (e.g., dopamine, noradrenaline) have been implicated in the etiology of SAD. The strongest evidence involves serotonin. Of all of the neurotransmitters of interest in depression, only serotonin has a distinct seasonal pattern of metabolism in normal humans, with the lowest levels of serotonin generally occurring in the winter and spring, and the highest levels in the summer and fall. Many neuroendocrine studies have shown abnormal hormonal responses to serotonergic drugs in SAD, suggesting dysregulation at the level of the post-synaptic serotonin receptor. Rapid depletion of plasma tryptophan, the amino acid precursor of serotonin, has been shown to reverse the antidepressant effect of light therapy in SAD, which suggests that bright light acts through serotonergic mechanisms. Drugs that affect serotonin via different mechanisms, including serotonin synthesis (L-tryptophan), neuronal serotonin release (d-fenfluramine), and serotonin reuptake blockade (sertraline, fluoxetine), are beneficial in SAD. Twin studies shown that a genetic factor accounts for between 29% and 65% of the variance in seasonality scores. Recent studies show that changes in genes affecting serotonin metabolism (e.g., the serotonin transporter gene and the tyrosine hydroxylase gene) may differentiate SAD patients from normal subjects.

What other abnormalities are found in SAD?

Other hypotheses for SAD, with preliminary supporting evidence, include other hormonal dysregulation (cortisol, thyroid), reduced retinal sensitivity to light, psychological mechanisms and personality factors.

LIGHT TREATMENT

Light therapy, also called light treatment or phototherapy, involves daily scheduled exposure to bright artificial light. The therapeutic use of light in SAD arose from basic research showing that exposure to room light (less than 500 lux, a unit of illumination intensity) could alter circadian and seasonal rhythms in animals. Some circadian effects of light are mediated via suppression of nocturnal melatonin secretion. In 1980, it was shown that higher intensity light (>2,000 lux) was required to suppress human melatonin secretion. This observation led to the first controlled study of light therapy in SAD in 1984.

Is light therapy an effective treatment for SAD?

More than 60 controlled studies of light therapy have been conducted by researchers around the world. Although there are general limitations to each study (e.g., small sample size, brief treatment periods), several qualitative reviews have concluded that light therapy is an effective treatment for SAD, with response rates of 60% to 90% in controlled studies. Two meta-analyses also confirm the efficacy of light therapy against plausible placebo controls. In large series, the clinical response rate for light therapy is approximately 65%.

The most studied light device is the fluorescent light box. The fact that the light box has proven effective in almost every study, regardless of sample size, has placed the light box as the "gold standard" light device. Other light devices include head mounted units, or incandescent light visors. Studies of the head mounter units have shown good clinical response rates (comparable to those of light box studies) but the bright light conditions were no better than dim light, putting into question whether visors are superior to placebo. Dawn simulators are devices that slowly increase the room illumination while subjects are sleeping, to simulate a "summer dawn" during the winter. Early results suggest a beneficial effect of dawn simulators in SAD, but other studies show superiority of light boxes over dawn simulators. Although efficacy has not been established for head mounter units and dawn simulators, these devices may be helpful for some patients when light boxes are not available or not convenient.

What are the relevant parameters of light therapy?

Parameters for light therapy generally include intensity, wavelength, duration of daily exposure and timing of light exposure during the day. Intensity is usually expressed in "lux", a photometric unit of illuminance that corrects for the visual spectral responsiveness of the eye. As reference, indoor lighting is usually less than 500 lux, outdoor light on a cloudy day ranges from 1,000 lux to 5,000 lux, and midday summer sunlight can reach 50,000 lux or higher. The antidepressant effects of light therapy are thought to be mediated through the eyes, not through skin exposure.

The usual "dose" of light therapy used in studies was 2,500 lux for at least one to two hours per day, but recent studies showed similar efficacy for 30 minutes of 10,000 lux exposure. Because of the convenience of briefer daily treatments, the 10,000 fluorescent light box has become the clinical standard. Although there as been controversy about the importance of timing of light exposure, new studies have confirmed that, on average, morning light therapy is superior to evening light exposure. The wavelength or type of light (incandescent, fluorescent) is not as important as intensity, but white light may be superior to narrow band wavelengths. Ultraviolet wavelengths are not necessary for the antidepressant response, and should be avoided because of long term toxicity.

What is an adequate length of time for a trial of light therapy?

Response to light therapy generally occurs within two to four days, and measurable improvement is often seen in one week. Most patients (but not all) experience rapid recurrence of symptoms after discontinuing light therapy. Longer trials have shown increasing response after two weeks, and incremental

improvement response at three or four weeks. The atypical depressive symptoms (hypersomnia, increased appetite, carbohydrate craving, and weight gain) are associated with favorable response to light therapy, while the presence of melancholic symptoms or a personality disorder is associated with poor response.

What practical tips are there for using light therapy?

Commercial light devices are now widely available in medical supply stores or via mail order. A light device should meet government electrical safety standards, have a filter for the ultraviolet wavelengths and have been tested in reputable clinical trials. Patients must maintain proper distance and positioning to ensure the correct dose of light exposure. Because of the rapid response and relapse with light therapy, patients can become involved as active participants in determining their optimal dosing of light. For example, if patients respond to early morning light exposure, but the time is inconvenient for them, they can try shifting the exposure time to afternoon or early evening. Alternatively, they can try to reduce the duration of exposure to 15 minutes for maintenance.

What are the side effects of light therapy?

The common side effects of light therapy reported by patients in clinical trials include eye strain or visual disturbances (19%-27%), headache (13%-21%), agitation or feeling "wired" (6%-13%), nausea (7%), sweating (7%) and sedation (6%-7%). These side effects are generally mild and subside with time or by reducing the dose of light. Hypomania and mania have also been reported as uncommon but serious side effects of light therapy.

Fluorescent light therapy using 2,500 lux to 10,000 lux is considered relatively safe on the eyes. Two follow-up studies did not show any eye or retinal damage after five years of light therapy. Ophthalmologic monitoring is not considered necessary unless there are risk factors for light toxicity (Table 3).

Can light therapy be used in other populations and conditions?

Some studies have shown benefit of light therapy in children and adolescents with SAD, in adults who have "subsyndromal" symptoms of SAD and in adults with nonseasonal depression. There are also studies of light therapy in other psychiatric disorders, including bulimia nervosa and premenstrual dysphoric disorder. The circadian effects of bright light have been exploited to treat jet lag, shift work, circadian sleep disorders and behavioural disorders in dementia. These results are considered preliminary and beyond the scope of this summary.

Recommendations: Light Therapy

(Refer to a description of the levels)

Light therapy is an effective first line treatment for seasonal disorder (Level 1 evidence).

The fluorescent light box, with light intensities of greater than 2,500 lux, is the preferred device for light therapy (Level 1 evidence).

Some patients may respond to other light devices, such as head mounter units and dawn simulators (Level 5 evidence).

The starting "dose" for light therapy using a fluorescent light box is 10,000 lux for 30 minutes per day (Level 1 evidence).

Alternatively, light boxes emitting 2,500 lux require two hours of exposure per day (Level 1 evidence).

Correct positioning is important (e.g., sitting close enough to the light box) to obtain the correct illumination (Level 3 evidence).

Light boxes should use white, fluorescent light with the ultraviolet wavelengths filtered out (Level 3 evidence, Level 2 evidence).

Light therapy should be started in the early morning, upon awakening, to maximize treatment response, but exposure at other times of the day may be helpful for some patients (Level 1 evidence).

Response to light therapy often occurs within one week, but some patients require two to four weeks to show a response (Level 2 evidence).

Patients can be encouraged to become active participants in establishing an optimal light protocol (Level 5 evidence).

Common side effects of light therapy include headache, eyestrain, nausea and agitation, but these effects are generally mild and transient, or resolve with reducing the dose of light (Level 2 evidence).

There are no absolute contraindications to light therapy and no evidence that light therapy is associated with ocular or retinal damage (Level 3 evidence).

Patients with ocular risk factors should have a baseline ophthalmologic consultation prior to starting light therapy, and periodic monitoring (Level 5 evidence)

Recommendations For Ophthalmologic Consultation

Ophthalmologic consultation recommended for patients with the following risk factors for retinal toxicity to bright light exposure:

Pre-existing retinal or eye disease (e.g., retinal detachments, retinitis pigmentosa, glaucoma).

Systemic illnesses that affect the retina (e.g., diabetes mellitus).

Previous cataract surgery and lens removal.

Taking medications that have photosensitizing effects in humans.*

lithium

phenothiazines, such as thioridazine (antipsychotics, antiemetics)

chloroquine (antimalarial)

hematoporphyrins (used in photodynamic therapy for cancer)

8-methoxypsoralens (used in ultraviolet treatment for psoriasis)

melatonin

hypericum (St. John's Wort)

Older age, because of greater risk of age-related degeneration.

*Animal studies show retinal changes with drugs including beta blockers, tricyclic antidepressants, and tryptophan. The panel consensus opinion was that ophthalmological monitoring for patients on these drugs is not required unless patients have other risk factors.

MEDICATION TREATMENT

Are antidepressants effective in the treatment of SAD?

The best evidence for efficacy of antidepressants in SAD involve the selective serotonin reuptake inhibitors (SSRIs). Two multicentre, double-blind, randomized, placebo-controlled studies of sertraline (187 patients) and fluoxetine (68 patients) confirm that these medications are effective in the treatment of SAD. In the fluoxetine study, there was significant superiority of fluoxetine over placebo in the clinical response rates (59% versus 34%, respectively), but there were no significant differences between the two conditions in the actual depression scores. In the other study, sertraline was significantly superior to placebo in both the clinical response rates (62% versus 46% respectively) and the depression scores. The sertraline study had the advantages of a larger sample size, longer treatment duration (eight weeks versus five weeks) and flexible dosing design.

What about other antidepressants?

One small study compared fluoxetine with moclobemide and found similar response rates between the two drugs, but another study found no differences between moclobemide and placebo. Other antidepressant studies (including bupropion, tranylcypromine, citalopram and desipramine) are limited by small sample size and/or open design. Therefore, efficacy for these medications is not yet proven in SAD. Clinical experience suggests that patients with SAD better tolerate the newer, selective antidepressants, but it is possible that all antidepressants will be effective.

What is the usual effective dose of antidepressants in SAD?

The starting dose of the antidepressant depends on several factors. The clinician should start at a lower dose (e.g., sertraline 25 mg) and increase the dose cautiously (e.g., every two weeks) in: 1) patients with previous sensitivities to antidepressants; 2) adolescent or elderly patients; 3) patients with a concurrent medical illness; 4) patients who are taking other medications that interact and increase the blood levels of the antidepressant. The sertraline study was a flexible dosing study using doses of 50 mg or 200 mg per day. Most patients took 50 mg or 100 mg per day, and the average dose of sertraline was 111 mg per day. The fluoxetine study used a fixed dose of 20 mg per day. These doses are similar to the doses that are effective in nonseasonal major depression.

What are the side effects of antidepressants?

The only antidepressant studies to systematically report on side effects are the two double-blind studies involving the SSRIs sertraline and fluoxetine. Generally, these medications were well tolerated by the SAD patients. The most frequently reported side effects in the fluoxetine-treated patients were headache, flu-like syndrome, rhinitis, and pharyngitis, but less than 6% of patients terminated the study because of side effects. The most common side effects in the sertraline-treated patients were nausea, insomnia and diarrhea, and there was also a low rate of termination due to side effects. Antidepressant-induced mania or hypomania can occur, but is infrequent in patients with a unipolar depressive disorder. Although there are no direct comparisons, the side-effect profiles of these medications for SAD are similar to those reported in nonseasonal depression.

How long should an acute trial of antidepressant last?

Patients in the fluoxetine study were treated for five weeks, but the differences in depression scores between fluoxetine and placebo did not reach statistical significance during that time. The sertraline study treated patients for eight weeks and showed clear superiority of sertraline over placebo. Therefore, an adequate trial of antidepressants should last six to eight weeks, similar to that recommended for nonseasonal depression.

Have other medications been studied in the treatment of SAD?

Several controlled studies have examined non-antidepressant medications in the treatment of SAD, but these studies are limited by very small sample sizes. Preliminary positive results have been reported with drugs that affect melatonin secretion (e.g., melatonin, propranolol) or serotonin metabolism (e.g., d-fenfluramine, 1-tryptophan). A small study also found that hypericum (St. John's Wort) plus a dim light box had a similar positive effect compared to a pill placebo plus a bright light box. These results are too preliminary to recommend any of the drugs for the treatment of SAD.

Recommendations: Medication Therapy

(go to an description of the levels)

Sertraline and fluoxetine are effective, well tolerated, first-line treatments for SAD (Level 1 evidence).

The effective antidepressant dose ranges are likely similar to those recommended for the treatment of nonseasonal major depressive disorder (Level 2 evidence).

Other antidepressants may also be effective in the treatment of SAD, using doses similar to those recommended for non-seasonal depression (Level 3 evidence).

An adequate trial of antidepressants involves at least 6 weeks of treatment (Level 2 evidence).

Other medications (propranolol, 1-tryptophan, hypericum) require further study before they can be recommended for treatment of SAD.

MANAGEMENT ISSUES

How do you choose between light therapy and medications?

Some experts have suggested that light therapy is the treatment of choice for SAD, given that the response is rapid, that side effects are minimal, and that the effect sizes in light therapy studies have been greater than in antidepressant studies. However, it is difficult to compare these studies because of methodological differences. Since there is very little information comparing light therapy and medications, the selection of treatment relies on individual assessment of benefits and risks. Factors to consider are listed in Table 4. Note that none of these factors is absolute. For a given patient, the relative importance of each factor should be considered.

For patients with less severe depression and good compliance, light therapy can be considered as the first choice treatment. Patients with atypical symptoms of depression may have better responses to light therapy. For more severe depressions, antidepressants alone or combined antidepressants and light therapy, is recommended.

Patient preference and compliance are also very important factors. Many patients prefer a non-pharmacologic treatment. Women of child bearing age are particularly interested in non-drug treatments, even though there are no data on effects of light therapy during pregnancy, or on the fetus, or

with breast feeding. Other patients find that even 30 minutes a day required for light therapy is too inconvenient, and medications are a better choice for them.

Side effects are another consideration. Although the newer medications are well tolerated by most patients, the side effects of light therapy are generally milder than those of antidepressants. Some patients have risk factors for using light therapy, such as retinal disease or use of photosensitizing medications, while others have risk factors for use of antidepressants, such as liver disease or potential drug-drug interactions.

Finally, cost is an issue for many patients, commercial light boxes that have been tested in reputable clinical trials cost between CDN\$300 and CDN\$500, about the cost of one season's treatment with antidepressants. Light boxes have a higher initial cost, but fewer ongoing costs, make them cost-effective over several seasons. Medications have less initial cost, but more ongoing costs. However, light boxes may not be covered under health insurance plans, while costs of medications are often reimbursed.

When should you combine medications and light therapy?

A clinical aphorism is to use one treatment at a time. Starting both treatments together leads to clinical confusion when trying to determine which treatment is beneficial, or which treatment is producing side effects. Combination treatment may be considered when there is only partial response to one treatment, or when side effects limit pushing the dose of a treatment.

How long should SAD patients be treated?

A trial of light therapy should last at least two to four weeks. A trial of antidepressants should last at least six weeks. Relapse generally occurs rapidly when light therapy is discontinued, and less rapidly when antidepressants are stopped. Most patients are treated only during the symptomatic winter months, and then discontinue their treatments during the spring and summer. The treatments are then restarted in the autumn. Some patients can wait until they have mild symptoms before restarting treatment, while others will opt to start treatment well ahead of their usual onset time (at least two weeks ahead for light therapy and four weeks ahead for antidepressants) to prevent an episode. Since the onset of action of light is rapid, continuous light therapy throughout the summer is not necessary, although some patients occasionally use light therapy for transient, mild symptoms during the summer. Continuous antidepressant treatment (i.e., throughout the summer) is indicated in patients who have problems with compliance, who take a long time to taper on and off medications, who have difficulty dating onset of symptoms in the fall, or who have occasional, transient symptoms in the summer.

Factors To Consider In The Choice Between Light Therapy And Antidepressant Medications

(note: none of these factors are absolute)

Consider light therapy as first-line when:

Less severe depression

Good compliance for light therapy

Warrants non-pharmacologic treatment (e.g., pregnancy, breast feeding)

Able and willing to make time commitment for light therapy

Relative contraindications to drug therapy (e.g., hepatic disease, allergies)

Intolerant to medication side effects

Assessing costs: greater initial cost but less expensive on-going costs

Assessing costs: light box covered by insurance?

Consider medications as first-line therapy when:

More severe depression
Low interest or motivation for light therapy
Light therapy too inconvenient
Unable to make time commitment for light therapy
Relative contraindications to light therapy (e.g., retinal disease, photosensitizing drug)
Intolerant to light therapy side effects
Assessing costs: less initial cost but greater on-going costs
Assessing costs: medications covered by insurance?

How do you manage patients who do not respond to light therapy or antidepressants?

The clinician usually differentiates between partial response and non-response. When patients are not responding, it is important to consider contributing factors such as patient compliance and unrecognized co-morbidity. In pursuing strategies for limited response, a stepwise approach with clear documentation of treatments and effects is essential.

For partial response to light therapy after two weeks of adequate treatment, there are several options to consider:

Increasing the dose, by increasing the duration of daily light exposure to 45 minutes or one hour, or by raising the intensity of light; changing the timing of light from morning to evening; or adding another treatment such as 1-tryptophan or antidepressants.

In patients who have no response after 2 weeks of adequate light therapy, many clinicians would switch to another treatment (e.g., antidepressants) instead of pursuing these options. For partial response to antidepressant medications after two to four weeks, the clinician can:

- 1) increase the dose of medication, or
- 2) add light therapy.

If these are unsuccessful then the usual pharmacologic approaches to drug-refractory depression should be considered, including adding an augmentation drug, switching to an antidepressant in a different therapeutic class, adding another antidepressant or using electroconvulsive therapy. Because of the (relatively) short winter season, these alternative strategies may need to be continued over to the next seasonal depressive episode.

Is psychotherapy effective in SAD?

Although many studies document the efficacy of short-term psychotherapies for nonseasonal depression (cognitive behavioural therapy, interpersonal therapy), there are no studies of psychotherapy in SAD. It is reasonable to assume that these treatments may also benefit those with SAD.

Recommendations: Management Issues

Recommendations: Light Therapy Or Antidepressants Or Both?

(Because of the lack of data, recommendations are based on clinical experience and panel consensus: i.e., Level 5 evidence).

Factors to consider when deciding on a first-line treatment include: severity of depression, side effects, safety, patient preference, patient compliance and cost.

Generally, one treatment should be used at a time to minimize clinical confusion about the therapeutic effects and the side effects of treatment.

Situations in which combined light therapy and antidepressants would be considered include:

- a) partial response to light therapy alone,
- b) partial response to antidepressants alone,
- c) partial response to light therapy or antidepressants in past episodes, and
- d) severe or treatment-refractory depression that is associated with prolonged dysfunction.

Recommendations: Length of Treatment

(go to a description of the levels)

A therapeutic trial of light therapy should be at least 2 weeks in length (Level 2 evidence).

A therapeutic trial of antidepressants should be at least 6 weeks in length (Level 2 evidence).

Because of risk of relapse, patients should continue with treatment for the entire winter season, until the time of their natural spring or summer remission. Treatment is not generally recommended during the summer (Level 2 evidence).

When possible, antidepressants should be tapered instead of abruptly discontinued (Level 5 evidence).

Treatment should be restarted in the autumn, either with onset of mild symptoms, or in advance of the usual onset of symptoms (Level 5 evidence).

Light therapy may be helpful during the summer for occasional transient symptoms (Level 5 evidence).

Preventative year-round antidepressant treatment (including the summer) should be considered in situations where:

- a) patients are poorly compliant or motivated,
- b) they take a long time to taper off and on medications,
- c) they are unable to recognize early signs and symptoms of depression,
- d) they have very early onset or very late offset of symptoms, and
- e) they experience symptoms during the summer (Level 5 evidence).

Recommendations: Managing Limited Treatment Response

(Because of the lack of data, recommendations are based on clinical experience and panel consensus, i.e., Level 5 evidence).

Patients showing limited response to treatment should first be evaluated to ensure they have adequate dosing of treatment (light therapy or medications), they are compliant with treatment and they do not have unrecognized comorbid conditions.

Strategies for dealing with partial responses to light therapy include: increasing the dose, changing the timing and trying alternative therapies, such as 1-tryptophan augmentation or combining with antidepressants.

Strategies for dealing with partial responses to antidepressant medications include: increasing the dose, combining with light therapy, switching to another antidepressant, augmenting with another agent, combining other antidepressants and electroconvulsive therapy.

In dealing with patients with refractory illness, it is important to take a methodical, stepwise approach with clear documentation of treatments.

Psychological treatments, such as cognitive therapy, may be of benefit as adjunctive treatment in SAD.

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Information Resources for SAD

Society for Light Treatment and Biological Rhythms (SLTBR).

SLTBR is an international, not-for-profit society dedicated to fostering research, professional development and clinical applications in the fields of light therapy and biological rhythms.

Contact: Stephanie Argraves, Executive Director, SLTBR
842 Howard Avenue, New Haven, CT, USA 06519
e-mail: sltbr@yale.edu
web site: <http://www.websciences.org/sltbr/>
(includes a list of Corporate Members that manufacture and distribute light devices)

Web Site Links

Dr. Lam's SAD Page at the University of B.C.
http://www.psychiatry.ubc.ca/mood/md_sad.html

Dr. Terman's Winter Depression Research Program at Columbia University
Includes a FAQ (Frequently Asked Questions) about SAD.
<http://www.columbia.edu/~mt12/>

Center for Environmental Therapeutics
Includes info on recent research of treatment for SAD
<http://www.cet.org/cet2000/>

Lou Puls' SAD Page
An excellent consumer-oriented page with SAD information and links.
<http://www.geocities.com/HotSprings/7061/sadhome.html>

Books for Patients

Don't Be SAD. Your Guide to Conquering Seasonal Affective Disorder.
By Celeste A. Peters.
Script Publishing, Calgary AB, 1994, \$18.95

Winter Blues. Seasonal Affective Disorder and How to Overcome It.
By Dr. Norman E. Rosenthal.
Guilford Press, New York NY, 1998, \$22.95

Books for Clinicians and Researchers

Seasonal Affective Disorder and Beyond. Light Treatment for SAD and Non-SAD Conditions.
Raymond W. Lam (editor).
Washington DC, American Psychiatric Press Inc., 1998.

Seasonal Affective Disorders and Phototherapy.
Norman E. Rosenthal, Mary Blehar (editors).
New York, Guilford Press, 1989.

Seasonal Affective Disorders.
Chris Thompson, Trevor Silverstone (editors).
London, CNS (Clinical Neuroscience), 1989.