# Is bright light therapy effective for improving depressive symptoms in adults with Seasonal Affective Disorder (SAD)?

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**Abstract**

## INTRODUCTION

In 1984, Seasonal Affective Disorder (SAD) , now a well-recognized form of recurrent depressive disorder characterized by depressive symptoms that occur in a distinct, predictable, seasonal nature, was first described by a group of researchers at the National Institutes for Mental Health (1). Symptoms include depressed mood, loss of energy, carbohydrate cravings, hypersomnia, hyperphagia and weight gain in addition to significant impairment in social and/or occupational functioning (1, 2). The most common type is winter type with onset of symptoms in the fall-winter will full remission of symptoms in the spring-summer (with occasional switch to hypomania-mania). Summer type SAD is much less common than winter type with symptom onset in spring-summer with full remission in the fall-winter (3). Specific DSM classification will be discussed in further detail in the background section. Onset is typically in the third and fourth decades of life and the course of the disorder varies with each individual (4).

The exact cause of SAD is unknown; therefore, many hypotheses exist to explain the etiology and pathophysiology of the condition. Pituitary gland size and function, circadian rhythm shifts and bright light exposure to seasonal depressive symptoms have all been implicated. It is from these scientific pursuits that treatment options for SAD are derived. A few treatment options other than light therapy have been used for SAD and include dawn simulators, ion generators and antidepressants (1, 3). Selective Serotonin Reuptake Inhibitors (SSRIs) have demonstrated the best evidence for pharmacological symptom improvement in SAD especially fluoxetine and sertraline (2,3). These treatment options are associated with multiple side effects leaving many researchers and clinicians searching for alternative treatments with a smaller side effect profile (3). Early researchers of SAD also suggested that melatonin was "depressinogenic" after small, simple studies of SAD showed greater improvement of depressive symptoms in individuals receiving bright light versus dim light(7). This idea has shaped the duration, intensity, and timing of bright light exposure designed to suppress the effects of melatonin. Other treatment modalities reported anecdotally include moclobemide, L-tryptophan and St. Johns wort (3).

Ideas that circadian rhythms have some role in affective disorders such as SAD arose from clinical observations of altered sleep-wake cycles and cyclic nature of symptoms in patients with SAD but early attempts to identify circadian rhythm shifts had been disappointing and inconsistent (6). Bright light therapy research has suggested that decreased exposure to bright light can contribute to or cause depressive symptoms and that increased light exposure may ameliorate depressive symptoms. Despite the growth of this treatment option in the psychiatric community, there is still a lack of well-established evidence for the SAD and a lack of support and training in bright light therapy for healthcare providers. In 2004, the American Psychiatric Association assigned a taskforce to address the research on bright light therapy and SAD as well as other mood disorders to determine its efficacy in the adult population (7). To date, over 100 studies have addressed the effectiveness of bright light therapy in improving symptoms of SAD (6,7).

This Evidence-Based Medicine paper will address whether bright light therapy is effective in improving symptoms of SAD in adults.

## BACKGROUND

Seasonal Affective Disorder (SAD) is chronic with recurrent symptoms most commonly in the fall-winter that result in occupational and social dysfunction. The disorder characteristically remits in the spring-summer season and is of unknown etiology. Onset is typically in patients 20-30 years old, occurs more commonly in women and young adults and its course varies between patients (1, 3). The DSM IV outlines SAD as:

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 (eg. Regular appearance of major depressive episode in the fall or winter). Do not include cases in which there is an obvious effect of seasonal-related psychosocial stressors such as 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 (eg. Depression disappears in the spring).

In the last 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationship 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 nonseasonal major depressive episodes that may have occurred over the individual's lifetime. (2)

There are two types of SAD; winter and summer. Winter type is more common with predictable onset of depressive symptoms in late September-October and remission in late March-April where summer type has an onset of depressive symptoms in late April-May with remission of symptoms in late August-September (1, 3, 4, 6).

Typical symptoms of SAD are considered "atypical depressive symptoms" not found in the majority of patients with other mood disorders and include increased sleep duration, increased appetite, carbohydrate cravings, weight gain, increased daytime fatigue and somnolence (4). Additional symptoms of SAD can include sadness, loss of interest in daily activities once found enjoyable, psychomotor retardation, fatigue, feelings of worthlessness or hopelessness, excessive or inappropriate guilt, inability to concentrate and excessive or recurrent thoughts of death (1,3,4). Not all of these symptoms must be present to make a

diagnosis of SAD. See Appendix 1 for prevalence of specific symptoms in SAD. Occasionally, the presenting complaint clinically is a somatic symptom such as pain (4). Many patients report varying degrees of amelioration of symptoms with traveling to different regions during the winter, working at different latitudes, living/working in areas with different illumination. Thirty-four percent of patients report symptoms of hypomania in the spring-summer including increased libido, elation, improved social activity, increased energy, reduced appetite and weight loss. True mania only occurs in approximately 2-4% of patients with SAD (2-4). Worsening of symptoms has also been reported by patients in reaction to spells of weather in which there are decreased hours of sunlight or intensity of light exposure (4). Approximately 10% of patients with SAD have symptoms severe enough to warrant hospitalization (4).

Comorbid conditions include but are not limited to bulimia nervosa, generalized anxiety disorder, non-seasonal major depressive disorder, simple phobias and social phobias. These conditions are reported to occur in 4-10% of patients with SAD (2). The presence of any of these conditions is not considered a risk factor for the development of SAD and treating these co morbidities does not necessarily improve the symptoms of SAD (3). Some patients report a family history of mood disorder, alcohol or drug abuse, or SAD itself and can be present in 5-24% of patients diagnosed with SAD (4).

Generally, there is a lack of physical exam and laboratory findings and the diagnosis of SAD is made clinically. This can make the diagnosis difficult and complicated due to a variety of other affective disorders as well as some general medical conditions that have similar symptoms (2). Seasonal symptoms can occur in patients will already documented major depressive disorder and bipolar disorder (2). A complete blood count, complete metabolic panel, thyroid function tests, liver function tests, urinalysis and a pregnancy test if female, should be obtained to rule out medical conditions such as hypothyroidism, metabolic derangements, diabetes, pregnancy, urinary tract infection and other infections and conditions that may cause symptoms similar to SAD (8). Bipolar disorder, major depressive disorder non-seasonal, bulimia nervosa and anorexia nervosa, generalized anxiety disorder, simple phobias, social phobias, obsessive-compulsive disorder and some psychotic disorders can share similar symptoms with SAD. Patients with SAD report less suicidal ideation and less severe depressive symptoms in the

morning hours than those suffering from other affective disorders such as depressive disorder, generalized anxiety disorder and bipolar disorder (4,5). SAD can be distinguished from major depressive disorder because patients suffering from SAD have an onset of symptoms with a specific seasonal pattern (usually fall-winter) (4). SAD can be differentiated from bipolar disorder because patients with SAD have a lack of manic symptoms (3,4). The "holiday blues," a short-lived psychosocial stress reaction occurring around the holiday season can be differentiated from SAD because the "holiday blues" symptoms occur over less than 1 month and are less severe than SAD(4). Other cyclical factors such as winter unemployment, bereavement or anniversaries of significant personal loss should alert the clinician to a diagnosis other than SAD. Subsyndromal SAD is a condition that shares many symptoms with SAD but does not impact social or occupational functioning to the same degree as SAD (4,5). See Appendix A for subsyndromal SAD classification.

SAD is a chronic disorder with 22-42% of patients continuing to experience symptoms 5-11 years after diagnosis and 33-44% developing non-seasonal symptoms although this data was collected from SAD patients who were being treated and therefore, does not represent the "natural" history of the disorder (4). The prognosis of SAD is not related to age at onset, duration or disease history; however, the development of non-seasonal symptoms does indicate a worse prognosis and increased susceptibility to developing major depressive disorder. In general, the existence of SAD does not predispose individuals to developing major depressive disorder or other mood disorders and SAD is not considered a prodromal or premature form of a more severe, chronic disorder (4-6).

Epidemiological studies have attempted to identify the exact prevalence of SAD. Due to a large variety of measurement techniques used to determine prevalence in the general population; exact, accurate data are not available. Such measurement techniques have included hospital admissions, practitioner consultations for depression, suicide rates, questionnaires (of both patients and providers) and antidepressants prescribed. (4). Random community sampling of the general populations of the United States and Canada report rates between 0.4%-2.7%. SAD is found more commonly in young adults and women (3). One study attempted to compare rates of SAD between Alaska (65□) and New Hampshire (42.5□) to determine whether there is a

latitude gradient to the disorder with more depressive symptoms occurring at more northern latitudes (4). The end result of the study indicated that many other factors contributed to the prevalence of SAD regardless of the latitude at which the participants lived. These other factors include but are not limited to: occupation/employment, time spent in daylight, possible ability to adapt to longer, arctic winter and other individual variability (3-4). In patients with documented major depressive disorder, seasonal exacerbation of symptoms severe enough to have a qualifier of SAD (based on DSM-IV criteria) and is reported in up to 20% of patients based on chart review and questionnaires (4). The presence of a pre-existing mood disorder either diagnosed or undiagnosed also complicates the accuracy of epidemiological measures of SAD.

Neuroendocrinological investigations of other psychiatric disorders such as major depressive disorder have suggested that pituitary abnormalities in size and function may be related to mood disorders. In fact, studies of major depressive disorder have shown hyperactivity of the hypothalamic-pituitary axis and an adrenal inability to suppress cortisol release in response to dexamethasone challenge (5). Such research has opened up avenues of research for SAD and the relationship of symptoms to the pituitary gland. Thus far, the relationship of pituitary function to SAD is complicated by the fact that it has not been well researched or documented.

The incidence of SAD is thought to be 0.04%-2.7% of the general population with an unknown etiology (4). Prominent theories regarding the etiology of SAD are related to exposure to varying amounts of light during the year and circadian-rhythm shifts based on light exposure (3-7). Treatment options for SAD include bright light therapy, antidepressant therapy, negative ion stimulators, dawn simulators, St. Johns Wort, moclobemide and L-Tryptophan (3). Antidepressants most commonly used for SAD include the SSRI's which are also used for major depressive disorder and improve depressive symptoms by blocking the reuptake of serotonin allowing serotonin to remain in the synapse for a longer period of time. This allows serotonin to be taken up by the neurons which results in more serotonin in the brain. For SAD, fluoxetine and sertraline have been studied extensively and have shown to be more effective than placebo at improving depressive symptoms (3). Side effects of SSRI's can be undesirable and include suicidality, nausea, headache, weight gain, insomnia, nervousness, anxiety,

asthenia, diarrhea, anorexia, dizziness, dry mouth, decreased libido, rash, changes in vision, constipation and ejaculatory dysfunction. More serious adverse effects can include hypersensitivity reactions, serum sickness, hyponatremia, mania, seizures and hypoglycemia (8). In clinical trials, fluoxetine and duloxetine have been used for 6-20 weeks depending on the trial (3). Larger clinical trials for longer durations have not been undertaken.

In another form of treatment, dawn simulation uses a bedside machine that gradually delivers light at the same time as the summer sun rises. The light exposure begins before the individual is awake but continues to increase in intensity until awakening. This modality was first researched in rats which showed that the rat retina responded to naturalistic dawn simulation with accelerated shedding of "rod outer disk segments" compared to sudden onset light (9). In humans, dawn simulation with dim light that gradually increases has been reported to delay drift of circadian rhythms. Side effects of this method included increased reports of hypomania and premature awakening during the initial exposure to the rising signal (9).

Negative air ionization is another method under research for the symptomatic improvement of SAD. The mechanism of action of this modality is not understood. Multiple studies found improvement of depressive symptoms with an activated ion generator used for 30 minutes after awakening (9).

Bright light therapy is used for SAD based on the hypothesis that decreased light exposure leads to increase melatonin which has been called "depressinogenic" and that exposure to bright light will decrease melatonin levels (7). The mechanism by which bright light works to decrease depressive symptoms is complicated further by the changes in circadian rhythm with exposure to light. It has been proposed that SAD is related to phase delay of circadian rhythms and that light treatment can advance the rhythm by shifting the "time of sleep mid-point" with the use of bright light (1). The circadian rhythm timing system has the greatest propensity for advancement of light-elicited rhythm changes in the morning hours when melatonin levels are increased (9). Additionally, in the winter months at more northern latitudes, the early morning is also the period when it remains dark outside later in the morning. This has been implicated in increasing depressive symptoms in adults in these environments (7,9). These results are based on an intricate understanding of the optimal timing of light therapy after awakening and the ability of light therapy given in the morning hours (specifically a certain number of hours after the "sleep mid-point") can shift a human's circadian rhythm and result in a significant reduction in depressive symptoms. Numerous studies have been carried out that attempt to delineate the exact timing, duration, and intensity of light therapy for SAD based on these circadian rhythms. Human circadian rhythms are most sensitive to light in the range of 450-480 nm. Timing of bright light therapy was shown to be more effective at reducing depressive symptoms of SAD when used in the morning hours (630-830 AM) when compared to midday (12-2 PM) or evening hours (4-6 PM) (1,3,7). Additionally, bright light (3000 lux) was shown to be more effective at reducing depressive symptoms than dim light (30 lux).

The traditional mode of delivery for bright light treatment is a light box that often can often be cumbersome and awkward to use; therefore, one major goal of this field of research is producing more portable, compact light boxes to be used for shorter durations that are still equally as effective as larger models used over longer periods of time (1). Newer models are light-emitting diodes (LEDs) that can be selected to emit light only in the 450-480 nm range; they are more efficient than fluorescent and incandescent light boxes and can also be manufactured to be more compact. The most recent advances in bright light therapy have suggested that LED generated blue light of 398 lux emitting peak energy of 468 nm is more effective than LED generated red light of 23 lux emitting peak energy of 654 nm.

Common side effects of bright light therapy include jitteriness and headache. With its low side effect profile and promising clinical reduction of depressive symptoms, light boxes are being increasingly used for conditions other than SAD. In addition to few side effects, the delayed effects that are typical of antidepressant medications (usually 4-6 weeks for clinical improvement); light therapy has been shown to induce remission of depressive symptoms within 1 week of beginning treatment (5). Research on bright light therapy for the improvement of depressive symptoms of SAD is continuing to provide information regarding the efficacy of this treatment option and attempt to better delineate the etiology of the disorder.

The purpose of this Evidence Based Medicine paper is to determine if bright light therapy is effective in reducing the depressive symptoms of adults with SAD.

## METHODS

Potential studies for this review were obtained through Medline and CINAHL. Both databases were searched with a combination of keywords and phrases including "seasonal affective disorder," "seasonal affective disorder + treatment," "bright light therapy," "seasonal affective disorder + bright light therapy," and "treatment seasonal affective disorder." In addition, Current Orthopedics, Current Medical Diagnosis and Treatment and Harrison's Principles of Internal Medicine were utilized for information relating to seasonal affective disorder and treatment options.

The search was limited to studies published in English relating to SAD and treatment of the disorder. Further limitation included all full text articles published in peer-reviewed journals from 1998 to the present and retrospective and systematic studies published in the United States as well as abroad. The highest level of evidence considered when evaluating a treatment modality is a randomized- placebo controlled clinical trial, Level A/I. Placebo control is difficult when the treatment is bright light therapy because participants will know if they are or are not receiving light therapy. Therefore, control groups vary from study to study and include different methods to provide the best research environment and yield the most scientific results. Systematic reviews of randomized controlled clinical trials were also considered in the literature search to expand options.

Initially, 6 articles met the criteria listed above. Two were not available as full-text articles and one included participants under 18 years of age; therefore, they were excluded from the analysis. One article compared the use of bright light therapy and fluoxetine for SAD; therefore, it was excluded. Two remaining articles investigated the effectiveness of bright light therapy for SAD and were included. The two articles chosen were Level A evidence because one was a placebo-controlled clinical trial and the other was a meta-analysis of placebo controlled clinical trials.

## DISCUSSION

Study # 1: "A controlled trial of the Litebook light-emitting device (LED) light therapy device for treatment of seasonal affective disorder (SAD)." (1)

The goal of this study was to conduct a multi-center, randomized, placebo-controlled, parallel group trial for SAD winter type to test the efficacy of a white LED device whose light emission was relatively concentrated in shorter wavelengths (using the Litebook, The Litebook company Ltd Alberta Canada). This device was tested for effectiveness at improving depressive symptoms in the SAD patient population and represents just one type of bright light treatment box. Since negative ion generators have been reported to be effective in treatment of SAD, a "credible placebo" design in which an inactivated negative ion generator was used as a "no light" control condition, was employed. The study was conducted at 5 sites: New Haven - CT, Vancouver - Canada, Montreal - Canada, Ottawa - Canada and Groningen – Netherlands. The study was approved by the IRB, and met the standards for the Helsinki declaration. Twenty-six participants signed informed consent and the trial was registered at the US National Institutes of Health. This study used the Structured Clinical Interview Guide for the Ham-D, SAD version (SIGH-SAD) containing 24 items. This assessment tool is Hamilton Depression Rating Scale (HDRS) modified to reflect symptomatology of SAD. This form of the questionnaire utilizes the first 17 items of the HDRS plus 7 atypical items. The SIGH-SAD was used to confirm the diagnosis of SAD, provide a baseline value for each participant. This assessment was repeated at four weeks after treatment. At both the baseline visit and at the 1,2 and 4 week visits, the questionnaire was administered by researchers who were blinded to which patients were in the treatment or placebo group. Data was collected and analyzed by another group of blinded researchers and reported to the research coordinators.

Participants were enrolled between October 1 and March 1 to reduce confounding effects of natural remission as expected in the spring months (late March-April). Recruitment was by advertisements or professional referrals. Potential participants were screened by trained phone interviewers and invited for a baseline visit if they were found to be appropriate. The study did not indicate who exactly performed these initial phone interviews except to say that they were trained to perform this specific interview. Twenty-six participants were found to be appropriate and invited for a baseline visit to be seen by a psychiatrist. Baseline visits included a full physical exam, psychiatric evaluation, urine toxicology and b-HCG for female participants. Participants were included in the study if they were 18-65 years old, had a DSM diagnosis of SAD (or major depressive disorder with seasonal variation), had a SIGH-SAD score of 20 or greater, and had habitual sleep onset prior to 1 AM and sleep end time prior to 9 AM (prior to time of trial). Additionally, to be included in the study,

participants had to agree to avoid other treatments for SAD such as alteration of sleep patterns due to change light exposure or travel to sunny destinations, to maintain stable sleep schedules, and if female and potentially fertile, to use an appropriate form of contraception during the trial. Participants also had to complete the Morningness-Eveningness Questionnaire (MEQ), a measure of preference for activity in the early or late part of the day. In this case, the MEQ questionnaire was an attempt to control for the treatment expectation of study participants.

Exclusion criteria included any of the following: significant medical illness, any retinal disease or medical disorder with retinal disease, pregnancy, use of photosensitizing medications, mood-altering medications, light therapy or other treatment for SAD within 1 week of the baseline visit (except within 4 weeks in the case of pharmacological antidepressant agents), initiation of psychotherapy within 3 months of the baseline visit (except when terminated by participant prior to this visit), current organic mental disorder, panic disorder, anorexia or bulimia nervosa, OCD, PTSD, history of psychotic disorder or Bipolar disorder, history of manic episode, history substance abuse not in full remission for 1 year, unstable sleep or mood patterns (such as severe PMS/PMDD), previous unsuccessful trial of light therapy with an accepted device for at least 2 weeks, inability to provide informed consent, poor likelihood of complying reliably with study requirements, suicidal risk or other factors making trial participation clinically inappropriate.

If participants met the inclusion criteria and did not have any exclusion criteria they were invited for a second visits called the randomization visit in 1 week and were issued either an active light treatment box or inactivated ion generator box by a blinded researcher. A separate, non-blinded clinician handed out the light box or ion generator, demonstrated how to use it and was available at subsequent visit for questions if necessary. Participants were also required to fill out a questionnaire regarding expectations of the study. A tape measure was provided to measure the appropriate distance from the participants chair to the device (20 inches). Participant s were instructed that (A) the study involved treatment with either a new bright light treatment device or a negative ion generator; (B) that both types of treatment were experimental; (C) the study was placebo-controlled and (D) one half of the devices in the study were modified in such a way that the investigators did not expect the device to be

efficacious. Most importantly, the participants demonstrated understanding that if they participated, they had a 1 in 2 chance of being assigned to treatment expected to be inactive for 4 weeks. The participants in the control group were instructed to wear a wrist band attached to the ion generator to assist in the transfer of ions and to sit exactly 20 inches away from the device. The active treatment group was given an LED light box that generated 1350 lux at 20 inches from the participant and did not receive a wrist band to wear with their treatment.

Instructions for the participants included using the device for 30 minutes, prior to 8 AM daily, maintaining a stable sleep schedule and a sleep log. The blinded clinicians were instructed to reduce treatment time to 15 minutes in the event of jitteriness or overstimulation (this was not reported during the trial). After the randomization visit, participants were required to return at 1, 2 and 4 weeks after treatment to complete the SIGH-SAD questionnaire given by a blinded researcher (clinician) and to report any adverse reactions to treatment. No other outcome measures were reported at these visits.

Twenty-six patients were randomized; 15 in the treatment group and 11 in the control group. In the treatment group, 1 participant withdrew after the first week for reasons that were unclear and 1 participant withdrew from the control group due to missed treatments after a motor vehicle accident. Twenty-five participants total completed the trial; 15 in the treatment group and 10 in the control group and there were no episodes of accidental unblinding.

Multiple statistical analyses were performed and failed to show a significant difference in mean SIGH-SAD scores at randomization and there was no significant difference between groups in terms of age, gender, ethnic background, previous episodes of SAD, and age at first SAD episode, weight, BMI, expectation score or MEQ score. Significant differences in SIGH-SAD were found between treatment and control groups at 4 weeks after treatment with statistically significant remission of depressive symptoms. No differences were found between active and control groups for changes in sleep time. All treatments were completed from 610-740 AM and side effects were reported by 3 participants (2 in treatment group and 1 in the control group) which included dry mouth, jitteriness, headache and difficulty falling asleep. The study concluded that the LED Litebook device used for 30 minutes/day was statistically significant in reducing depressive symptoms of SAD over a

4 week period.

These results should be interpreted with caution because of the small study size and therefore, they should be confirmed with larger studies. Longer duration studies are necessary to determine if the conclusions drawn from this study can extrapolate from 4 weeks to 16-20 weeks. This is especially important because the clinical duration of SAD is from late September-late March which is 24 weeks in length.

This study only used subjective assessment of side effects but further studies using an ophthalmologic exam would provide more objective data on side effect profile. Recent research has questioned the physiologic effect of direct daily bright light therapy on the eyes resulting in cataracts, macular degeneration, uveitis and other ocular conditions that can be caused by or exacerbated by high intensity light exposure.

The placebo condition used in this study is also problematic because only the group given the ion generator received a bracelet whereas the bright light group did not. In addition, the bright light group could obviously see that they were being exposed to bright light on a daily basis. Participants given the ion generator neither saw nor heard any tangible output from the machines they were connected to. This may have led to an expectation bias of the participants exposed to bright light therapy.

Details about the phone interviews and some of the blinded researchers/clinicians were not consistently provided by this study including level of education (whether certain levels or degrees were required) and whether they were provided any detailed information about the study itself such as what was being researched.

The SIGH-SAD assessment tool was one positive aspect of this study as it has been used in other studies and is a specific tool for SAD to target certain symptoms. Since this was the only subjective outcome measure used in this case, it simplified the interpretation of the results of the study.

Study # 2: "The efficacy of light therapy in the treatment of mood disorders: a review and meta-analysis of the literature." (7)

This study is a review and meta-analysis of available literature on the efficacy of bright light therapy in the treatment of mood disorders including SAD. Because there are very limited double blind placebo-controlled clinical

trials on the use of bright light therapy for the treatment of SAD, a group of researchers at the American Psychiatric Association set out to review the available literature.

Researchers searched Pubmed for "phototherapy" with any of the following: "Seasonal affective disorder," "jet lag," "bipolar disorder," "sleep," "sleep disorder," "Alzheimer's disease," "circadian rhythm," "melatonin," "premenstrual dysphoric disorder," "premenstrual syndrome," "eating disorder," "bulimia," "obesity." Articles were restricted to those only in English. The same search was also done in MEDLINE.

Initial study inclusion mandated that participants be between 18-65 years old and have an established DSM III, III-R or IV diagnosis of a mood disorder. Any subsyndromal diagnoses were excluded. Studies were also required to randomized, controlled clinical trials in acute phase of treatment with "credible placebo" design. A minimum lux light treatment specification was complicated by a lack of standard adequate dosing. Researchers therefore consulted textbooks and experts in phototherapy treatment for mood disorders to come up with minimum standards for selection of research articles. Minimum standards included 4 days of 3000 lux-hours (1500 lux for 2 hours or 3000 lux for 1 hour) with placebo comparison maximum of 300 lux. Outcomes were measured by the Hamilton Depression Rating scale – SAD type. Any article whose design was altered during the course of the study was excluded. This initial search yielded 173 studies. Two independent researchers examined the articles that met the previously discussed criteria and used further agreed-upon exclusion criteria to analyze the articles.

These exclusion criteria included: inadequate or absent placebo, inadequate intervention, participants outside the specified age range (either too young or too old), no acute treatment phase, DSM diagnosis not used, subjects used in larger version of same study and inadequate reporting of data. After application of these exclusion criteria, 20 articles remained. The remaining studies were then classified into 4 groups; Group 1: bright light for treatment of SAD; Group 2: bright light for treatment of non-seasonal major depression ; Group 3: dawn stimulator for treatment of SAD; Group 4: bright light as adjunctive treatment for non-seasonal major depression. For the purposes of this review, only group 1 will be considered as the other three groups do not relate to this evidence-based question.

Group 1 contains 11 articles whose results were pooled and

analyzed to provide the largest data sample available. For each study standardized mean differences and a confidence interval of 95% was calculated by homogeneity analysis and a Q score was calculated. Reported side effects included headache, nausea, eye strain and agitation. Outcome measures for all studies included the use of the SIGH-SAD assessment tool.

Results of the data pooling and statistical analysis showed statistically significant differences between treatment and placebo groups for the treatment of depressive symptoms of SAD. All of the studies included used bright white light as the treatment condition while control conditions were not uniform and included the use of red light, inactivated negative ion generators, or yellow light. Bright light therapy was shown to be more effective when used in the morning hours between 6-9 AM when compared to treatment at midday (12-2 PM) or during the evening hours (430-630 PM). While the results are encouraging for the remission of symptoms in SAD using bright light therapy, there are still some reservations in applying these results to patients in a clinical setting.

This meta-analysis highlights the fact that most research on the use of bright light therapy for symptom improvement in SAD and other mood disorders does not meet the rigorous criteria for clinical trial design. One problem is the difficulty in coming up with an acceptable placebo. In clinical trials for pharmacotherapy, it is relatively simple to put a filler in place of an active ingredient and still have treatments that are identical in appearance. In this case, the different treatment groups involve a variety of light conditions which the patient can see and which may create expectation biases among participants. Another problem is that there is variability between studies regarding selection of study groups, doses and forms of active treatment, and control treatment conditions. This makes equal interpretation of multiple studies impossible because there are no uniformly accepted treatment doses for bright light therapy. Unfortunately, this has perpetuated the impression that bright light therapy is not effective at improving the depressive symptoms of SAD because many studies can only draw limited conclusions.

This review addressed only the efficacy of bright light therapy for treatment of depressive symptoms in SAD and other mood disorders and did not ask any questions regarding the safety of the treatment. Safety and side effects of bright light therapy should be addressed in continuing research. The use of bright light therapy is limited for this reason and should be used with caution because long term side effects are not yet known. Continued research should look not only at subjective side effects but more objective methods to measure side effects therefore eliminating some of the bias of side effects reported by study participants.

The meta-analysis did confirm that bright light therapy is more effective than placebo conditions at reducing the depressive symptoms of SAD. Not enough research has been performed to draw strong scientific conclusions regarding exact specifications of bright light therapy including doses or exact duration.

Clinical application of bright light therapy should be limited by healthcare providers who are informed of all the risks and benefits associated with bright light therapy. The providers should also caution their patients that this treatment for SAD has only been suggested to be beneficial by some studies and that the treatment may not improve symptoms to all patients with SAD.

## CONCLUSION

Recent studies that examine the treatment of depressive symptoms in SAD can only provide limited concrete conclusions regarding the effectiveness of treatment modalities. Placebo conditions for bright light therapy are one major restriction of many studies. The "acceptable placebo" such as red light, yellow light or inactivated ion generator are commonly used and considered acceptable in the scientific community. However, questions still remain whether these placebo conditions cause expectation bias in study participants. Many researchers are still skeptical of the use of even dim light as a control because of the possibility that even low-lux light can have some effects on symptomatology. This would mean that the placebo conditions may actually be providing therapeutic benefits for participants in the control group.

Another potential study limitation is the lack of standard dosing and duration of bright light treatment. Thirty minutes of bright light therapy is increasingly becoming accepted as the minimum duration of treatment but many studies still use 1 hour and others still use 2 hours. Differences between 30 minutes, 1 hour and 2 hours of treatment need to be examined in further studies.

Timing of therapy has repeatedly been shown to be more effective when given in the morning hours as compared to

midday or evening. For this reason, clinicians who are begin to incorporate bright light therapy into patient treatment plans should recommend that the treatment take place prior to 8AM.

As the scientific community continues to research and better understand the etiology and pathophysiology of SAD, the application of bright light treatment will also be better delineated. Factors such as duration, timing and intensity of treatment are beginning to be outlined by repeated studies of bright light therapy but exact specifications have yet to be developed. Larger, longer duration studies are necessary to confirm the reduction of depressive symptoms with bright light therapy in patients who suffer from SAD. Further avenues of research include comparing more distinct and narrow ranges of bright light (including blue lux light), comparing bright light therapy directly to other treatment modalities including pharmacotherapy, dawn simulators and negative ion generators. Currently, there is evidence that bright light therapy can cause remission of depressive symptoms in SAD when used over periods of 4-6 weeks during the winter months (6,7).

Based on early research, bright light therapy does result in improvement of depressive symptoms of adults with SAD. Bright light treatment is most beneficial when used for 30 minutes prior to 8 AM on a daily basis during the winter months when symptoms of SAD worsen. Risk and benefits of bright light therapy used on an annual basis to improve symptoms of SAD in adults have not been determined. This treatment should be used with caution in adults with SAD especially in regard to the potential side effect profile of bright light therapy. Patients wishing to begin this form of treatment should talk with their healthcare provider for a detailed discussion of this treatment option.

**Figure 1**

APPENDIX A. SYMPTOMS IN PATIENTS WITH
WINTER-TYPE SAD (4).

## TABLE 4. SYMPTOMS AND SIGNS IN PATIENTS WITH WINTER SAD*

| Frequent | Percent |
|---|---|
| • Sadness | 96 |
| • Decreased activity | 96 |
| • Social misfortune† | 92 |
| • Anxiety | 86 |
| • Irritability† | 86 |
| • Occupational misfortune | 84 |
| • Daytime tiredness† | 81 |

| Fairly frequent | Percent |
|---|---|
| • Increased sleep | 76 |
| • Poor quality of sleep† | 75 |
| • Increased weight | 74 |
| • Carbohydrate craving | 70 |
| • Decreased libido | 68 |
| • Increased appetite | 65 |

| Fairly infrequent | Percent |
|---|---|
| • Suicidal thoughts† | 35 |
| • Decreased sleep† | 31 |

| Infrequent | Percent |
|---|---|
| • Mixed or no change in appetite† | 17 |
| • Mixed or no change in weight† | 17 |
| • Decreased appetite | 15 |
| • Decreased weight | 7 |
| • Mixed or no change in sleep† | 5 |
| • No change in activity† | 2 |

* Data derived from the NIMH Seasonal Studies Program (N=662).
† N=366.
‡ From the Seasonal Mood Clinic at the University Hospital-UBC Site (N=46).*
SAD=seasonal affective disorder; NIMH=National Institute of Mental Health; UBC=University of British Columbia.

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## Figure 2

APPENDIX B. SUBSYNDROMAL SAD (4).

**TABLE 1. OPERATIONAL CRITERIA FOR SUBSYN-DROMAL SEASONAL AFFECTIVE DISORDER[4]**

- A history of some difficulty during the winter months that have occurred on a regular basis (at least 2 consecutive winters) and have lasted for a sustained period of time (≤4 weeks). Example: decreased energy; decreased efficiency at work (concentration, completing tasks); decreased creativity or interest in socializing; and change in eating habit (eating more carbohydrates); weight (gaining weight); or sleep patterns (more sleep)

- Subjects have to regard themselves as normal, not suffering from an illness or a disorder

- Subjects have not sought medical or psychological help specifically for the above difficulties, nor has anyone else suggested that they should do so

- People who do not know them well do not recognize that they have a problem, or if they do, easily attribute it to circumstances such as "flu" or "overwork"

- The symptoms experienced by the subjects have not disrupted their functioning to a major degree. Example: calling in sick several times per winter, or severe marital discord

- No history of major affective disorder in winter

- No serious medical illness

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