Psychology Research and Behavior Management

Dovepress
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Open Access Full Text Article

REVIEW

Seasonal affective disorder, winter type: current insights and treatment options

This article was published in the following Dove Press journal:
Psychology Research and Behavior Management
30 November 2016
Number of times this article has been viewed

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Abstract: Seasonal affective disorder (SAD), winter type, is a seasonal pattern of recurrent major depressive episodes most commonly occurring in autumn or winter and remitting in spring/summer. The syndrome has been well-known for more than three decades, with light treatment being the treatment of first choice. In this paper, an overview is presented of the present insights in SAD. Description of the syndrome, etiology, and treatment options are mentioned. Apart from light treatment, medication and psychotherapy are other treatment options. The predictable, repetitive nature of the syndrome makes it possible to discuss preventive treatment options. Furthermore, critical views on the concept of SAD as a distinct diagnosis are discussed.

Keywords: seasonal affective disorder, review, light treatment, medication, psychotherapy, prevention

Introduction

Seasonal affective disorder (SAD), winter type, is an almost yearly recurrent depression with the onset of symptoms in autumn/winter followed by a spontaneous recovery in spring/summer. In their now classical paper, Rosenthal et al[1] described the syndrome and presented the first study using light treatment (LT) for SAD. Since then, the syndrome has been described in several editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and is still included in the most recent fifth edition (DSM-5).[2] In this paper, an overview of the syndrome, its prevalence, etiology, assessment procedures, treatment modalities, and some criticisms about the concept of SAD are presented.

The syndrome

The SAD syndrome, winter type, or winter depression has been described and studied since the early 80s of the previous century. In the last three editions of the DSM, SAD was either formulated as a specifier of a major depression or as a specifier of a bipolar I or II disorder, but not as an independent entity. Despite the arguments of Rosenthal to formulate SAD as a distinct entity, it is still a specifier in the DSM-5.[3]

Arguments for and against the choice for SAD as a specifier and not a distinct category can be found, and both have consequences for the recognition and treatment.

Classification systems are consensus based, which is not always identical to evidence based. The advantage of inclusion in the classification systems is the recognition of the symptoms, which makes it easier or more acceptable to treat these patients within different health care systems. Therefore, it is important to formulate distinct categories.

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Dovepress
http://dx.doi.org/10.2147/PRBM.S114906

The disadvantage to formulating the definition of SAD as a specifier of another depressive category, and not as a distinct entity in itself, in classification systems is that SAD becomes less clear and therefore less recognizable for clinicians. That may influence their choice of treatment and keep them from offering the treatment of first choice to their patients.

One argument in favor of an independent diagnosis is that the clinical presentation of the symptoms of SAD differs from that of other depressions and that treatment options also differ. SAD is defined as a distinct syndrome with specific symptoms and a characteristic treatment of first choice. The symptoms occur in autumn/winter and disappear in spring/summer on an almost yearly basis. SAD sufferers experience some atypical symptoms during wintertime, such as hypersomnia, increased appetite, overeating and weight gain, a preference of carbohydrate food, extreme loss of energy, and loss of social activity.[1] Women are more often affected by SAD than men (ratio 4:1),[4,5] and SAD is also more common among younger people.[5]

Prevalence

Epidemiological studies show that the prevalence of SAD is between 1% and 10% of the population and that it seems to be related to latitude. Since a shortage of daylight is involved, it would seem logical that prevalence is higher at more northern latitudes. The influence of latitude is sometimes unclear, though,[6] but prevalence in North America is two times higher compared to Europe.[7] A significant correlation between latitude and prevalence was found in North America, but in Europe only a trend in the same direction was found. Studies in some northern European countries have shown more mixed results.[6]

Many of the epidemiological studies mentioned use the Seasonal Pattern Assessment Questionnaire (SPAQ). Using this questionnaire may overestimate the prevalence of SAD.[6,8]

Working mechanism and etiology

SAD patients – only winter type is considered here – are obviously vulnerable to the effects of the changing seasons and suffer from a lack of (day)light in the winter season. In most cases, LT is effective in these patients. The etiology of SAD and the working mechanism of light are still unknown, however.

A number of different hypotheses have been formulated to unravel etiology and working mechanism, but to date no sufficient evidence has been found in support of these hypotheses. The most prominent hypothesis is that a disturbance of SAD patients' biological clock underlies the complaints. In early days, Lewy et al[9–11] described their phase-shift hypothesis, which postulates that the biological clock is out of phase (mostly delayed) with the natural day–night cycle. In a minority of patients, the circadian system is phase advanced. Lewy et al[9,10,12] assumed that LT in the morning would cause a phase correction and therefore be beneficial to SAD sufferers. Several studies have confirmed LT in the morning to be superior to LT at other times of the day,[13] but other studies have shown the effectiveness of LT in the evening or at midday,[14–16] and have confirmed a positive treatment effect without inducing phase shifts in the circadian system.[17,18] Since no study is available to show that evening LT is superior to morning LT, only that it is equally effective, and studies showing that morning light is superior do in fact exist, it became clinical practice to offer LT in the morning. More recently, Lewy et al[19] refined their phase shift hypothesis and showed support for the assumption that an internal desynchronization between rhythms driven by the biological clock and the sleep–wake rhythm are causal in the pathology of SAD, and they also support morning light therapy as the most effective treatment.

The exact timing of LT in the morning for the treatment to be optimally effective may differ between chronotypes. Chronotype refers to the actual timing of sleeping and activity in individuals, which results in a broad distribution of different groups ranging from extreme evening-types to extreme morning-types, with intermediate types in the middle. Evening-type individuals in particular are seen to have a higher risk of getting depressed.[20] Chronotypes in the SAD population differ in their baseline phase of melatonin onset (as a marker of the timing of their circadian clock) by up to 5–6 hours. Terman et al[21] showed that due to differences in the timing of light, smaller or larger phase shifts of melatonin were found, with larger responses accompanying a larger phase advance of melatonin onset. Assessing melatonin onset, however, is not always feasible. The scores of the Morningness Eveningness Questionnaire[22] have been shown to highly correlate with melatonin onset, making it a practical tool in the assessment of the optimal LT time.[21,23] Contrary to these studies, Knapen et al[24] show in a retrospective study that exposure at a fixed time in the morning does not show different treatment outcomes for SAD patients with different Morningness Eveningness Questionnaire sores, although these outcomes were not necessarily the most optimal response.

Light exposure has to be at the level of the retina to affect the circadian system, as assessed by means of a shift in dim light melatonin onset. A small study of Wehr et al[25] on

SAD treatments showed a superior effect of light exposure through the eyes compared to exposure to the skin. Based on behavioral similarities in plants and animals, Oren[26] proposed another way of photo transduction than through the eyes only. He supposed that the signal of light could be transported to the brain through the hemoglobin in the red blood cells. In this line of reasoning, it is thought to be possible to influence the human circadian system in healthy people by administering light at the back of the knees (where large blood vessels are present near the surface).[27] A logical next step was to investigate the antidepressant effects of light at the back of the knees in SAD patients. The effects of light at the back of SAD sufferers' knees were comparable to those of a placebo condition.[28] Also, in a replication of the first study claiming some positive results on circadian rhythms from light exposure to the back of the knees, and several other studies, no support for these finding was found.[29,30] This does not mean that we are absolutely certain that the positive results of antidepressant LT can only be reached by means of exposure to the eyes. In a recent paper, Oren et al[31] described an evolutionary-based theoretical model of humoral phototransduction and the possible roles of hemoglobin and bilirubin in addition to the role of rods, cones, and melanopsin. In a study from Finland, positive results were described after exposure to light through the ears.[32] This study can be criticized owing to the lack of a real placebo condition, however. Two studies showed that light therapy through the ears did not suppress melatonin production, whereas light exposure at the retina did.[33,34] Recently, it was shown that passing light through the ears was not effective in shifting the sleep–wake rhythms.[35] In a study comparing the prevalence of SAD in severely visually impaired subjects (n=4,099) to subjects with full eyesight (n=2,781), it was found that the prevalence of SAD in the former group was significantly higher. The authors argue that these results support the hypothesis that decreased retinal light input plays a role in the pathogenesis of SAD.[36]

Although the study of the effects of transcranial light on depressive symptoms has been criticized for its lack of a placebo condition, the real problem in clinical studies on the effects of ocular LT is that it is impossible to create a methodologically justified placebo condition (or LT without the supposed effective part without the participant noticing this).

In light research, various placebo-like conditions have been used, such as imaginary light,[37] invisible light,[38] extraocular light,[28] low-intensity light,[39] or another placebo condition totally unrelated to light, such as a deactivated ion generator.[40] Responses to these "placebo" conditions have been found to vary from 36% to 46%.

There are nonetheless some promising developments in the search for convincing evidence in the diagnosis and etiology of SAD.

Hypocretin/orexin neuropeptides (hcrt) play a role in controlling patterns of sleep and wakefulness. In a Danish study on 227 subjects suffering from hypersomnia, a seasonal variation in hcrt was found in cerebrospinal fluid, correlating with day length. This finding might be helpful in the understanding of a physiological basis for fatigue in wintertime or SAD.[41] The preliminary conclusion of a small study on SAD patients is that it is possible to find useful biomarkers with gene expression in white blood cells.[42] Another recent progress in the search for biomarkers was the identification of a serotonin transporter binding with a potential role in LT of SAD[43] and higher levels of serotonin transporter binding in the whole brain, but specifically in areas involved in the regulation of affect in SAD.[44,45] In the longitudinal study by Mc Mahon et al,[45] the seasonal change in serotonin transporter binding was observed to be positively associated with the change in depressive symptom severity, suggesting that the development of depressive symptoms in winter is associated with a failure to downregulate serotonin transporter levels, a phenomenon observed in healthy controls. Finally, a recent paper focused on a possible relationship between the immune system and SAD. Song et al[46] found that SAD patients have enhanced inflammatory responses in winter in their depressed state compared to healthy controls. Light therapy improved mood and normalized their immune function.

Assessment

A clinical interview using a structured and standardized questionnaire is often been seen as the gold standard to diagnose a psychiatric disorder or to rate symptom severity. In depression research, the Hamilton Rating Scale for Depression (HRSD)[47] has often been used from 1960 onward for this purpose. The need arose to improve the interrater reliability of this clinical interview. This led to the development of structured interview guides. In addition, there was a clear need to rate the atypical symptoms.[48] These two aspects were combined in the *Structured Interview Guide for the Hamilton Depression Rating Scale – Seasonal Affective Disorder* version (SIGH-SAD).[49] Rohan et al[50] have recently presented a protocol and rules for the scoring of the HRSD and have reported high interrater reliability scores. A self-rating version of the SIGH-SAD rating scale is also available. The latest version of this instrument has a slightly different title, since atypical symptoms are not exclusive for SAD: Structured Interview Guide for the Hamilton Depression

Rating Scale with Atypical Depression Supplement (SIGH-ADS).[51] The HRSD, SIGH-SAD, and SIGH-ADS assess the severity of the depression but are unsuitable for use as a diagnostic instrument. Instruments to diagnose depression are the Structured Clinical Interview for DSM-IV axis I disorders (SCID-I)[52] or the Mini-International Neuropsychiatric Interview (M.I.N.I.).[53]

The severity of a depression with a clinical diagnosis can vary from mild to very severe. The severity of depression can be assessed by means of self-ratings, but in more severe depressions, a standardized structured clinical interview would certainly be preferable.

The SPAQ is popular as a screening instrument and is often used in epidemiological studies.[54] This self-rating instrument consists of seven sections on seasonal changes in some key variables. A subscale assessing seasonal changes in mood, energy, social activity, sleep, weight gain, and eating habits is particularly suited to calculate a so-called Global Seasonality Score (GSS). The existence of seasonal complaints, their severity, and a certain global seasonality score enable this instrument used in epidemiological studies to estimate SAD prevalence. Although the SPAQ is widely used, its validity has been criticized.[55–60] A number of new self-rating instruments have been developed for the assessment of seasonal depression, such as the Seasonal Health Questionnaire (SHQ)[61] or more recently, the Seasonality Assessment Form.[62] The SHQ would seem to be more sensitive and specific in assessing seasonal depression than the SPAQ and be of more predictive value as a screening instrument.[63] Prevalence estimates for SAD when using the SHQ are lower than when using the SPAQ.[63,64] A high score on the SPAQ or other self-rating instruments is not identical to a SAD diagnosis, however. Direct structured interviewing remains superior.[8]

Light treatment

In a paper by Lewy et al,[65] the first detailed description of exposure to bright white light in the treatment of SAD was published in a case study. They reported a successful treatment of a single patient with exposure to bright light, lengthening the day light period. Rosenthal et al[1] published the first study in which exposure to bright light was successful in treating SAD. Ever since, a large amount of studies have been published in which the effect of LT as an antidepressant therapy has been shown. Nowadays, LT is the treatment of first choice for SAD sufferers.[23,66–70] Most studies investigate the effects of LT in adults, but in a number of small studies and case studies positive results after LT in children and adolescents have also been described.[71–74]

In the early days, it was common to use bright white light with an intensity of 2,500 lux for 3 hours; nowadays, LT with bright light intensity of 10,000 lux for 30–45 minutes per session has become more or less the standard.[23,56,75] In the first studies, effects were reported after 3 days of light exposure. Later on, the duration of the LT was lengthened. The effects of 2 weeks of LT were reported to be superior to those of 1 week,[76] but it was questioned whether this effect after the second week of LT was actually caused by the exposure to light. Meesters[77] described that the effects of LT as assessed 1 week after finishing exposure to light were superior to the results immediately after ending the 1-week treatment.

The duration of the treatment offered does in fact affect the response to the treatment. Levitt and Levitan showed that a shorter duration of LT (2 weeks) can be equally effective as a longer duration (5 weeks).[78] This suggests that the response rate in the group receiving shorter LT duration is faster. A retrospective study comparing the therapeutic effects between studies with 1 vs 2 weeks of LT arrived at the same conclusion.[79]

Because of the potential role of the circadian system in the pathology of SAD and the observed effects of LT, the discovery of a novel photoreceptor, the retinal ganglion cells containing melanopsin, has heavily influenced the field.[80–84] These non-image forming photoreceptors influence the circadian system and are most sensitive to light with a wavelength of 470–490 nm (blue light).[85] On the basis of these findings, several studies have been conducted studying the possible role of the melanopsin cell in the LT effects of SAD. Based on this knowledge, blue or blue-enriched light fixtures have become available for the treatment of SAD patients. In a study where extra blue light was added to the fixtures (17,000 K), the therapeutic outcome was compared to the conventional treatment outcome with bright white light. No differences were found between 30 minutes of conventional bright light exposure compared to 30 or 20 minutes of exposure to blue-enriched bright light; in all conditions, high responses were found.[86] Because of possible saturation effects, the study was repeated with low-intensity (750 lux) blue-enriched light compared to bright white light, with no differences in treatment outcome.[39]

With the invention of blue-light-emitting diodes (LEDs), narrow-band light sources fitting the maximum sensitivity of the NIF photoreceptors became available. These fixtures were also used in several studies. The therapeutic outcomes of exposure to blue light were found to be superior to those of red light;[87,88] blue-enriched LED light was superior to placebo,[40] but similar to blue light.[89] In studies comparing the therapeutic outcomes after exposure to low-intensity

narrow-band blue light with that of bright white light, no differences were found in the treatment effects in SAD[90] or subsyndromal SAD (winter blues).[60] In a recent study comparing the therapeutic results after exposure to blue light (~465 nm) vs exposure to blue-free light (595–612 nm) no difference in treatment outcome was found.[91] If this latter study can be replicated with a larger sample size, the role of the blue light as an essential ingredient of successful LT should be questioned.

In addition to fixed light equipment, portable light fixtures have become available, such as head-mounted devices (light visors),[92] and since the introduction of LEDs other portable devices.[40,60] The commonality among all these light therapies is that patients are awake and exposed to light at the retina by sitting in front of a light fixture or via a head-mounted light device. Another way of LT is the use of dawn simulation. In the wintertime, a gradual increase of morning light is simulated just prior to wake-up time (mostly from 0 to 300 lux in 30 minutes). Positive results have been reported.[93,94] An advantage of dawn simulation is that it is less time-consuming than more traditional LT. People receive the light when they are still sleeping until they wake up. Patients who are less motivated to spend time in front of a light fixture can reap the benefits of LT this way.[95] Dawn simulation has also been shown to have positive effects on waking up, reducing sleep inertia in healthy subjects, without shifting the biological clock.[96,97] When comparing the therapeutic results of bright light vs dawn simulation, the results of exposure to bright light have been better.[98,99] In a study with exclusively hypersomnic SAD patients, exposure to dawn simulation was shown to be superior to exposure to bright white light.[100]

Although LT is highly effective, it sometimes has side effects or contraindications. Regularly reported side effects are mild visual complaints, nausea, dizziness and headaches, tired eyes, agitation, sleep disturbances, or, very rarely, (hypo) manic decompensation.[23,101,102] (Relative) contraindications are preexisting retinal diseases, the use of photosensitizing drugs, and recent eye surgery.[23] If there is any doubt about eye health, it is recommended people consult an ophthalmologist before starting LT. Although detrimental effects on eye health caused by LT are rarely reported, some eye specialists warn against the effects of high-intensity light on the eyes,[103] against the effects of narrow-band blue light in particular.[104]

Medication

The number of studies on antidepressant medication in the treatment of SAD is not very high. In a placebo-controlled study, the effects of fluoxetine did not differ from that of the placebo treatment, both were equally effective.[105] In a placebo-controlled study, Ruhrmann et al[106] found no difference in treatment outcome between the use of LT compared to fluoxetine, with remission rate after LT tending to be superior.

In a large placebo-controlled study (n=187), it was shown that sertraline was superior to placebo and was well tolerated in the treatment of SAD.[107] In a comparison between LT, fluoxetine, and placebo, Lam et al[108] found that at the end of the treatment both light and medication had been effective, with LT having an earlier onset of recovery and fewer adverse effects than fluoxetine (n=96). These two studies are the largest ones investigating the effects of medication in the treatment of SAD. In a meta-analysis of the effects of second-generation antidepressants, only three studies fulfilled the inclusion criteria, all using fluoxetine as medication; little evidence for a treatment effect based on this small number of trials is presented.[109]

There are several very small studies or open-label studies claiming some positive effects of other antidepressants.[110] One of these open-label studies is by Pjerk et al[111] who reported positive effects of the use of agomelatine (n=37), but to our knowledge no controlled studies supporting this conclusion have been published yet. Partonen and Lönnqvist[112] found that the use of moclobemide was equally effective compared to the use of fluoxetine in treating SAD (n=32), but no placebo-control condition was available. In a larger study (n=282), it was found that after one successful week of LT, the use of citalopram was more effective at preventing a relapse during the next 15 weeks than placebo.[113] The effect of bupropion has not been investigated very thoroughly in treating the acute phase of SAD.[114] Since it has a positive effect in the prevention of SAD, bupropion is the only licensed drug used in the treatment of SAD.

Psychotherapy (CBT)

Psychotherapy, which is successful in nonseasonal depression, might prove to be useful in treating SAD as well. There is some support that SAD can be explained by underlying dysfunctional attitudes.[115] Since 2007, Rohan et al[116] have described positive results of Cognitive Behavioral Therapy (CBT) by itself or in combination with LT. Six weeks of CBT with two 90-minute sessions per week were equally effective as LT for 30 minutes every morning.[117] In an exploratory study, Sitkinov et al[118] found that SAD patients with more cognitive problems at baseline had more complaints in the next winter if they were offered LT as a monotherapy compared to those who received CBT or CBT combined with LT.

Depending on a correct baseline diagnosis, these findings may play a role in the choice of first treatment.

Other treatments

Besides LT, medication, and psychotherapy, other treatments are sometimes offered to SAD sufferers. From the herbal therapies, only some evidence for a therapeutic effect is described after the use of St. John's Wort (*Hypericum perforatum*),[119–121] but not for Kava (*Piper methysticum*).[121] Neither has any difference been found between the use of *Ginko biloba* extract and placebo in the prevention of SAD symptoms.[122]

There is some weak evidence that the use of tryptophan has some benefits in the treatment of SAD[123,124] and probably also in those responding poorly or not at all to LT.[125]

Physical exercise, either by itself, or in combination with LT, has also been shown to have a positive effect on mood in SAD patients.[126,127]

Prevention

Since winter depression is by definition a recurrent disease, efforts have been made to find treatment options for the prevention of the next full-blown depressive episode. Preventive treatment options are based on exposure to light, medication, and CBT.

In one study, it was shown that LT offered at the first signs of the depressive episode prevented the development of a full-blown depressive episode in the remaining part of the season.[128] In a more or less replication study, it was shown that LT offered at the first signs of the episode could postpone the development of the next episode.[129] Offering LT at the start of the autumn, when no complaints of depressive symptoms were present, did not prevent a depressive episode in the following winter period.[130]

Starting with LT before SAD patients show symptoms and continuing this treatment into the period in which patients were having complaints in former winters may lead to the prevention or early treatment of a depressive episode.[131]

The use of light visors (wearable light fixtures on the head) during the winter made it possible to prevent a depressive episode.[92] This last study was the only one to fulfill the criteria of a Cochrane review about the possibilities of LT in preventing the development of a depressive episode in SAD patients,[132] and so the evidence to use LT as a preventive method has to be investigated further in order to find more support for this treatment modality.

In a large study, n=1,042, the use of bupropion in preventing the recurrence of SAD symptoms was investigated.[133] These authors showed that the use of bupropion, started before the depressive episode in wintertime, was superior to placebo in preventing a depressive episode in the following winter. In a review about the use of bupropion in treating SAD, based on the results of previous studies and some open-label studies showing that the use of this medication had some effect, the authors concluded that the use of bupropion is a treatment option well-tolerated by patients, but they also stated that for a definitive conclusion more placebo-controlled studies are needed.[114] In a Cochrane review regarding the use of second-generation antidepressants to prevent SAD, only the Modell et al[133] studies were accepted for inclusion. Their conclusion was that bupropion is effective in the prevention of SAD, but they cautioned that these effects are not large, since 4 out of 5 patients did not benefit at all and a next depressive episode was not prevented. Because of the side effects of bupropion, the authors suggest that this treatment option be carefully discussed with patients, considering the advantages, disadvantages, and other potentially effective interventions.[134]

No controlled studies of the use of melatonin or agomelatine in the prevention of SAD are available.[135]

In a Cochrane review on psychological therapies for the prevention of SAD, no studies fulfilled the inclusion criteria.[136] After publication of this review, a study of Rohan et al[137] was published which reported that the effects of CBT were superior to LT for two consecutive winters after acute treatment. This suggests that CBT has greater durability in treating SAD and leads to less relapse compared to LT.

There is some evidence in the literature that Mindfulness Based Cognitive Therapy (MBCT) is useful in the prevention of depressive episodes in recurrent depressions when patients are in remission.[138] As SAD is a recurrent depression, it makes sense to offer MBCT to SAD patients in summer, when their depression is in remission. A small pilot study, however, concluded that MBCT was of no use in preventing a depressive episode in wintertime.[139]

Criticisms

Although there is a large amount of literature on SAD, and the positive results of light therapy and other treatments have been carefully described, some papers criticize the concept of SAD and the working mechanisms of LT. Stuhlmiller,[140] for example, stated that the effects of the seasons on psychological changes are inconsistent, controversial, and influenced by the appreciation of cultural perception and adaptation. More recently, Traffanstedt et al[141] have described the results of a cross-sectional survey using the Patient Health Questionnaire-8 Depression Scale. Their results do not support the construct of a seasonal modifier in major depression, and

so the authors suggest discontinuing seasonal variation as a diagnostic modifier of depression. They question the validity of the commonly used SPAQ because of its retrospective character (assessing complaints over at least 1 year) and the fact that the SPAQ does not assess the DSM criteria. Unfortunately, this study does not assess SAD itself, but the higher and lower prevalence of complaints of depression in a cross-sectional approach. In a cross-sectional cohort study, using the Kessler Psychological Distress Scale (K10), Winthorst et al[142] did not find a clear seasonal pattern in predefined depressive subgroups. Using the SPAQ in the same population, some seasonal variation was found in subgroups with depression and anxiety.[143] These two studies show that if the survey contains no specific relevant questions that relate to the concept of seasonal variations, no relevant answers can be found.

Considering the concept of the effects of light therapy in SAD, Martensson et al[144] reported the results of a meta-analysis using very strict inclusion criteria. They found an effect size of 0.54 for the effect of bright white LT in SAD and criticized the results of the meta-analysis of Golden et al[68] who reported a larger effect size (0.84) because of their inclusion criteria. Martensson et al[144] concluded that the evidence for the clinical use of bright white LT for SAD is weak at most. The differences between these two studies clearly show the weaknesses of meta-analyses: results can be heavily influenced because of the a priori formulated inclusion criteria. The problem with small studies is that their power is limited due to small sample sizes. Meta analyses have more power, but can be biased because of the a priori selection of criteria for inclusion or exclusion of studies. Epidemiological studies are mostly based on surveys or questionnaires and do not cover an actual diagnosis. A clinical diagnosis using an interview may clearly differ from the results of a survey based on questionnaires.[8]

As discussed earlier, it is still impossible to create a methodologically justified placebo condition to compare with the effects of LT. Theoretically, it remains possible that the effects of LT can be explained by a specific therapeutic variable unrelated to the light, such as an improvement of day–night structure, recognition of the complaints by a professional, or to placebo. Such variables definitely play a role, but it would seem unlikely that the quick and robust effects of LT can have existed for decades without having any intrinsic effects.

Conclusion

For more than three decades now, both for clinicians and researchers, SAD has been a source of inspiration in the discussion of the etiology and mechanisms underlying the treatment effect. For many years, a large number of SAD sufferers have benefited from the exposure to artificial bright light, which has become the treatment of first choice. Positive results have also been reported after the use of antidepressant medication, such as SSRIs and bupropion. The success of different treatment methods can be explained by means of the dual vulnerability model.[145–147] This model proposes that SAD patients are vulnerable to developing severe vegetative symptoms (eg, sleep, appetite, energy) and also have an additional vulnerability to develop psychological symptoms.

The knowledge about the etiology of SAD and the working mechanisms of LT is still growing, but not decisively. Some treatments are based on theories, but not all of the reported therapeutic outcomes can be explained by means of these theories. This does not mean that these theories or treatments are useless. Inconclusive results can be an inspiration for further research. The way the syndrome is diagnosed can be improved, as can the assessment, theories, and treatment procedures. This is by no means a negative conclusion degrading the concept of SAD and light therapy, since no diagnosis or treatment in the field of mental health, has perhaps shown such large improvements over a period of about 30 years. It is a stimulant to go on.

Acknowledgment

The authors are grateful to Josie Borger for the improvement of the English.

Disclosure

The authors report no conflicts of interest in this work.

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