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Seasonal Affective Disorder: SAD or Fad?

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Seasonal affective disorder (SAD) and phototherapy have recently been the subject of a great deal of interest in the psychiatric literature. First described in 1984 (1), SAD is now defined as "a cyclic illness characterized by recurrent episodes of fall/winter depression alternating with periods of spring/summer euthymia (normal mood) or hypomania (mild elation and behavioral activation)" (2). Recent findings indicate that there may be at least two additional patterns of seasonal depressions, one characterized by annual summer depressions with euthymic, hypomanic or manic symptoms in the winter, and the other characterized by depressive episodes occurring in both winter and summer (3-6).

As defined by Rosenthal et al. (3), SAD diagnostic criteria include:

- 1) a history of at least one major depressive episode, according to RDC (7) criteria;
- 2) regularly occurring fall-winter depressions (at least two occurring during consecutive winters) alternating with nondepressed periods during spring and summer;
- 3) no other major psychiatric disorders; and,
- 4) no psychosocial variables accounting for the regular changes in mood.

SAD has gained increased acceptance with the inclusion of "seasonal pattern" criteria for a number of different diagnoses within the Mood Disorder section of DSM III-R (8).

Recently a number of reports of additional "seasonal" diagnostic entities have appeared in the scientific literature, including, "reverse seasonal affective disorder" (3), "seasonal affective disorder in children and adolescents" (9), "seasonal premenstrual syndrome" (10), seasonal battering of women (11) and a hypothesized seasonal obesity disorder (12). Furthermore, the lay press has reported extensively on SAD and phototherapy (13-19), with phototherapy currently being touted as potentially beneficial for depression, multiple sclerosis, AIDS, premenstrual syndrome, school absenteeism, low work productivity, poor morale, jet lag, and dental cavities (13-20).

While others have written to express concern about the validity of these diagnoses and their proposed etiologies (21-23), the validity of the diagnosis of SAD is the focus of this paper.

LITERATURE SUPPORT FOR SAD

Although numerous papers have been published over the years that support the existence of seasonal differences in psychiatric disorders (24-39),

many of these early papers do not seem readily applicable to support current notions of the entity of SAD. Although, Rosenthal et al. briefly allude to the numerous discrepancies in these early studies (40), Christensen et al. in the "Myths of Mid-Winter Depression," present a detailed literature review of the conflicting nature of the empirical data that relates to seasonal peaks of psychiatric disorders (41). On closer review many of these seasonal study populations do not appear to relate to the current populations being described as having the SAD diagnosis. For example, the early research was done almost exclusively with psychiatric inpatients and with few exceptions (42), SAD is described as a mild disorder found primarily in outpatient populations (1-3,43-45). Many of the early studies did not differentiate the specific "seasonal" diagnoses, or if diagnoses were provided, they did not discuss the specific symptomatology. In addition, although not specifically stated, one must assume from the current clinical descriptions of SAD that most patients do not display suicidal thoughts or suicidal behavior, therefore, excluding many of the subject populations reported on in the papers on seasonality of suicides.

A related issue is the lack of current epidemiologic evidence for SAD and the absence of literature support for determining its reliability, validity or prevalence as a psychiatric diagnosis. The patient population upon which the SAD diagnosis was developed was a partly self-selected research population at NIMH, which was included in what appears to be a non-controlled phototherapy trial (1). Unfortunately, the majority of the follow-up studies, by NIMH and other groups, have used this same method of subject selection to develop their study populations, to test the effects of phototherapy and to establish their demographic data. An exception to this approach was a recent comparison of seasonal and nonseasonal affective disorders (46). This study used a psychiatry clinic population as its source of subjects, did not incorporate a phototherapy protocol, and used a control population to test their hypothesis. They reported a "prevalence of seasonal depression" of 38% in their study population (46), whereas the most frequently quoted prevalence estimate for SAD has been "about 4-5% of manic-depressive patients" (47). Unfortunately, Garvey et al. use their own idiosyncratic criteria to diagnosis SAD (46). In fact, this type of diagnostic variance is a common problem throughout the SAD literature. The significance of this variability in describing psychiatric syndromes has been discussed by Spitzer (49). He stated that "criterion variance" occurs when there are differences in the criteria that are used to make a certain diagnosis and this is one of the major sources of difficulty with reliability in psychiatric diagnoses.

POOR RELIABILITY DUE TO CRITERION VARIANCE

Development of diagnostic criteria would provide a means to verify reliability of SAD as a diagnosis and possibly establish its descriptive validity. However, while most researchers have attempted to describe the diagnosis of SAD based on the Research Diagnostic Criteria (RDC) for major depression (7),

there is a recurring problem with various authors changing the SAD criteria they report in their individual studies. While Rosenthal et al. have reported that other researchers have used similar criteria (3), a review shows that the current SAD criteria differ significantly from those initially presented in 1984 (1) and those presented by other groups (2,47,48). Sometimes the changes are quite extensive, such as, diagnosing SAD without a major affective diagnosis as a criteria (47), leaving out criteria pertaining to psychosocial influences (1), or not ruling out other psychiatric disorders (48). Therefore, failure to specifically define how these individuals are being diagnosed could decrease the reliability of SAD. Perhaps this accounts for Yerevanian et al. reporting that SAD patients usually have unipolar disorder (50), Rosenthal et al. reporting that SAD patients usually have bipolar disorder (1,3), and Wirz-Justice et al. reporting that SAD patients usually give a history consistent with bipolar disorder, but actually fail to show any evidence of mania or hypomania on longitudinal follow-up into the spring/summer (47).

ETIOLOGIC HYPOTHESES

Rosenthal and Fishman (51) recently reviewed current theories of the reported antidepressant effect of light on SAD patients and discussed four:

THE MELATONIN THEORY: Melatonin is secreted in a circadian pattern with nocturnal release from the pineal gland in response to environmental light input to the retinas. The pathway of information flow is reported as; light input to the retinas is passed along the retino-hypothalamic tract to the suprachiasmatic nuclei of the hypothalamus, which in turn stimulates the specific pattern of melatonin release from the pineal gland. In SAD it is postulated that "the symptoms of SAD are due to an abnormality in melatonin secretion or, perhaps, an abnormal brain response to melatonin" (51). While not being totally excluded, this theory seems less plausible now that report data indicates that melatonin suppression is not necessary to demonstrate beneficial effects of light treatment (48).

THE PHASE-SHIFT HYPOTHESIS: A theory discussed by numerous authors (51-56), who postulate that "light corrects an abnormality of circadian phase either relative to real clock time or internally (between different circadian rhythms)" (51). These authors have also speculated that exposure to light in the morning brings the SAD patient's rhythms back to normal, thus stabilizing their mood. Conversely they suggest that phototherapy in the evenings should delay circadian rhythms, thus exacerbating the patient's symptoms. The most important arguments against this theory are the more recent reports that the time of day of phototherapy application does not alter the antidepressant effect of phototherapy (9,48,57).

THE DIRECT PHOTO-CHEMICAL HYPOTHESIS: This is a general theory that postulates an abnormality existing in the brain, perhaps in the hypothalamus, that is probably genetic and which produces the symptoms of

SAD in the absence of sufficient light. Phototherapy functions by reversing this brain deficiency (51).

THE PLACEBO THEORY: A major concern with all of the SAD literature lies in the difficulty of controlling for placebo and positive expectancy effects. This is particularly important because most of the literature that bears on the biologic theories seems to argue by inference that response to phototherapy treatment constitutes reasonable evidence of the validity of the SAD diagnosis (1-5,47,52). Further, a number of SAD papers do not consider the placebo effect at all (3-5,42,55), while papers that do, provide a one-sided argument against it (1-2,44-45,47-48,51,53,58).

Proponents of SAD and light treatment accept that the consistent two- to four-day time lag for improvement with phototherapy and the likelihood of relapse occurring over a similar time span when the lights are withdrawn mitigate against the placebo hypothesis. Also, SAD researchers report that the beneficial effects of phototherapy persist throughout the treatment course and reliably return with subsequent treatments during relapses. Furthermore, a fair degree of consistent findings in phototherapy responses from year to year and between different investigative groups (1,47,59) has been claimed, as well as a dose-response relationship. To bolster arguments against the placebo theory, proponents also cite the evidence of a circadian rhythm in response sensitivity to treatment (i.e. "some studies have shown a greater efficacy of light treatment in the morning than in the evening") (61-62). Additionally, SAD proponents invoke the notions about light's multiple biological effects (61-68) and the superiority of light therapy compared to sham light control treatments (1,47,53,69,70) as evidence to refute the placebo effect.

However, accumulating evidence seems to refute a number of these arguments: In regard to the reported consistent time lag until symptom relapse when lights are withdrawn, at least one group reports that symptom relapse is not consistent and may occur within one day of the withdrawal of lights or not occur at all (47). Also, recent data suggests that a circadian sensitivity to response to timing of phototherapy is not present, as the timing of the phototherapy is not critical for producing the antidepressant effect (i.e. applications at various times throughout the day and evening have resulted in beneficial effects) (48,57,69-70).

The theory behind dim (250-300 lux) light as a control versus bright (2,500 lux) light as the active treatment was initially based on the finding that at 500 lux, light suppresses production of melatonin in normal volunteers (61). However, a subsequent study demonstrated that the suppression of melatonin is not necessary for phototherapy to produce its antidepressant effect in SAD patients (48). This finding casts doubt not only on the melatonin hypothesis, but also on the use of dim light as a "nonactive" control and bright light as an "active" treatment. Wehr et al. have reported that dim "control" lights have repeatedly not shown significant antidepressant effects (48) and that other

studies have confirmed these findings (47,59). However, a review of the other studies they reference reveals that Wirz-Justice et al., reports antidepressant effects with dim light, at levels as low as 250 lux, in 83% of their SAD population (47,71). One of the other papers, by Hellekson et al., was a non-controlled treatment trial of six SAD patients that used only bright light (2500 lux) (59). Furthermore, Wirz-Justice et al. (47) note that 30 to 40% of SAD patients in previous phototherapy trials have responded to dim light (1,57,58). These authors speculate that with the small number of subjects in all of these SAD studies, such differences in response may be related to the selection criteria (47). Doubts are further increased by reports that, in addition to less light intensity, antidepressant responses are seen with less time of exposure to phototherapy (9,47).

SAD advocates note that "patients become depressed in a regular and predictable way" (3), yet almost 40% of Rosenthal et al.'s initial group of SAD patients failed to develop any evidence of depression when followed during the fall/winter of 1984 (1). Further Rosenthal et al. report that >90% of SAD patients have a bipolar disorder (3), yet two other groups have reported that none of their SAD patients displayed evidence of mania or hypomania on follow up (47,50). Furthermore, while "winter depression" is the term that is used with the diagnosis, the actual onset of the reported depressions can occur anytime between July and January (5).

Additional arguments for light being an "active" agent, and not a placebo effect were presented by Wirz-Justice et al. (47). The authors stated that placebo effects are more evident in mild or neurotic depressions and report that their SAD patients were severely depressed. This argument is problematic for two reasons: First, there is little empirical evidence to support this assertion because placebo reactions are commonly demonstrated in normal, neurotic and psychotic individuals (62). Second, as stated before most SAD patients are reported to have "mild" depressions (1-2,43-45). Further, they argue that the "use of lights under self-selected conditions led to repeated improvement in the same individual." While we would agree this probably suggests the individual is deriving some type of benefit from phototherapy, it is not clear if it serves to differentiate "active" from "placebo" origins of that benefit, particularly in light of the reports of conditioned placebo effects (72-73). Lastly, they assert that "relapse after withdrawal is considered a criterion for an active agent." The authors did not provide a reference for this statement, however there are reports that individuals have displayed several years of chronic dependence with withdrawal symptoms on placebo alone (74). Further, drug withdrawal symptoms may occur as a psychologically conditioned responses, rather than as biologically induced symptoms (72-73).

The placebo effect has been the subject of extensive research and there is little doubt that it occurs in both experimental and clinical studies (75). Some of the findings about placebo responses that are applicable to the current discus-

sion of SAD include: A positive therapeutic response of almost 100% has been reported with placebo alone (72–75), this could account for the 80% positive response rate most frequently reported for phototherapy (3). In addition, the most frequently cited symptoms associated with the placebo reaction are “depression, anxiety and emotionality” (75), whereas Rosenthal et al. report the three affects associated with SAD are “sadness, anxiety and irritability.” Nausea, headaches, and nervousness are side effects that are frequently seen with placebos (75). Wirz-Justice et al. report phototherapy side effects consist of nausea, headaches, “hypomanic activation,” and/or irritability (42).

Experimenter bias explains why uncontrolled studies report success more frequently than controlled studies (75). While it has been demonstrated that the transfer of researcher bias to patients can occur subtly (75), in the phototherapy trials written presentation of the researchers’ SAD theories and past “successful” use of light treatments are used as pre-conditions for subject selection (1,9,44,45,49,59).

DISCUSSION

We have been unable to find any clinical, demographic, family history, laboratory study, or controlled light therapy research that has validated SAD as a distinct syndrome. Yet despite this lack of support we now see a flurry of additional “seasonal” diagnostic entities appearing in the scientific literature (3,9–12,20) and the lay press (13–19). One must question whether seasonal variation in mood is a characteristic found in the general population, including psychiatric patients generally, or if there is actually a distinct subgroup with extreme seasonal differences in psychiatric symptoms.

Furthermore, if SAD represents some type of specific biologic mechanism, similar to hibernation in animals (3), why is there so much variability in the onset, presentation, and response to treatment? These differences occur within individual patients and when comparing different SAD patients.

As recently pointed out by Winokur et al., “making up new sets of diagnostic criteria in American psychiatry has become a cottage industry with little attempt at quality control” (76). The history of psychiatry and medicine is marked with clinicians and researchers alike embracing new disorders and uncontrolled treatments, which for a while, seem to provide dramatic progress. Only after further clinical experience and judicious application of the scientific method, do these breakthroughs assume a more modest position in the understanding of psychiatric disorders and their treatments. Although face validity may be the first step toward identifying a psychiatric disorder (77), we must be able to establish more powerful types of diagnostic validity, such as descriptive and predictive validity. Only if such conditions can be met can we justify presenting SAD as a distinct behavioral syndrome, rather than a random collection of clinical features commonly seen in individuals with other types of mental disorders and in individuals without mental disorders.

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