Effects of Bright Light Intervention on Typical and Atypical Depressive Symptoms and Predictors of Response

By

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Abstract

The objective of the present study was to investigate within a prospective design the seasonality and depression predictors of light therapy response after controlling for treatment expectancy and pre-therapy functioning. Seasonality was indicated by the Global Seasonality Score (GSS) on the Seasonal Pattern Assessment Questionnaire (SPAQ). The depression predictors were measured with the Structured Interview Guide for the Hamilton Depression Rating Scale - Seasonal Affective Disorder Version (SIGH-SAD) that yielded severity levels for the typical and atypical depressive symptom. Seventeen participants received 30 minutes of bright light therapy at 10,000 lux daily for 14 days. All participants had a screening SIGH-SAD score of at least 22, were not receiving antidepressant treatment at least 4 weeks (8 weeks for fluoxetine) before the study, had no previous experience with light therapy, were free of pre-existing retinal or eye diseases, and were free of use photosensitizing medications or supplements. Their treatment expectancy, pre-therapy and post-therapy functioning on several variables were measured. Significant increase was found in sleep quality and significant decrease was found in carbohydrate craving, carbohydrate intake, atypical symptom score, typical symptom score, and total depression score. Higher expectations of treatment predicted lower carbohydrate craving following light therapy. Higher typical symptom score before light therapy predicted higher atypical symptom score and higher total symptom score following light therapy. The GSS bore no relationship to treatment outcome. The present study provides support for previous research findings that individuals with more severe typical depressive symptoms have a poorer response to light therapy. Findings also underscore the need for placebo control in light therapy studies.

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Introduction

Since the time of Hippocrates (460-370 BC), it has been recognized that the change of seasons has an influence on affective disorders (Wehr, 1989; Wehr & Rosenthal, 1989). This phenomenon, referred to as Seasonal Affective Disorder (SAD) in the research literature, is characterized by depressive episodes that occur around the same time every year (Rosenthal et al., 1984). Two types of SAD have been recognized. The first is summer SAD, which is identified by the onset of depression in the summer and its alleviation during the fall and winter (Wehr et al., 1989). The second, which is the more common variety, is winter SAD. It is characterized by fall or winter depression that remits in the spring or summer, with or without mania or hypomania (Faedda et al., 1993).

Winter depression is often accompanied by a distinctive cluster of atypical vegetative symptoms, such as hypersomnia, increased appetite, carbohydrate craving, and weight gain (Wirz-Justice et al., 1986) that are not commonly found in nonseasonal depression (Allen, Lam, Remick, & Sadnovick, 1993; Meesters et al., 1993; Sakamoto, Nakadaira, Kamo, Kamo, & Takahashi, 1995; Wehr & Rosenthal, 1989). Bright light therapy has been found to be an effective treatment for SAD (Partonen & Lönnqvist, 1998; Thalen, Kjellman, Morkrid, Wibom, & Wetterberg, 1995; Tam, Lam, & Levitt, 1995). The focus of the present study is on winter SAD, which hereafter, shall be referred to simply as SAD.

The prevalence of SAD in epidemiological studies has ranged from 0.4% (Blazer, Kessler & Swartz, 1998) to 9.7% (Rosen et al., 1990). The discrepancy in prevalence rates can, to a large degree, be explained by the utilization of different criteria in defining SAD. Earlier studies that have relied on screening measures for seasonality, such as the Seasonal Pattern Assessment Questionnaire (SPAQ, Rosenthal, Genhart, Sack, Skwerer & Wehr, 1987), tend to report higher

prevalence rates (e.g., Bartko & Kasper, 1989; Booker & Hellekson, 1992; Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989). In contrast, later studies that use the more restrictive DSM-III-R (American Psychiatric Association, 1987) or DSM-IV (American Psychiatric Association, 1994) diagnostic criteria to ensure the presence of clinical depression in the SAD individuals tend to report lower prevalence rates that range from 0.4% (Blazer et al., 1998) to 6.3% (Haggarty, Cernovsky, & Husni, 2001). Levitt and Boyle (1997) reported that 7.4% of respondents were identified as SAD when the SPAQ was used; however, the lifetime prevalence dropped to 1.7% when the DSM-IV criteria were utilized. This suggests that the use of less restrictive criteria results in the problem of overidentification where false positives are represented. Indeed, research within the last 10 years has recognized the presence of a sub-syndromal SAD (or sub-SAD) group that is thought to exhibit less severe changes in the symptoms and in impairment, compared to the SAD group (Bauer, 1992; Bauer & Dunner, 1993). Sub-SAD individuals may have been misidentified as full SAD with the screening criteria but may have been excluded when the more rigorous diagnostic criteria were employed. A more detailed discussion of the criteria and measures for identifying and diagnosing SAD will be undertaken later under the section of "Assessment of SAD".

A meta-analysis of 40 SAD studies that were conducted in various countries found the mean age of subjects to range from 28.7 to 47.9 years, with a median mean age of 38 (Lee & Chan, 1998). More women than men were found among those with SAD (ratio of 3.45:1). The authors pointed out that this figure could be interpreted as either a gender differential in vulnerability to SAD or a sampling artifact. Given that subjects in the studies were self-referred, it is possible that the women were more willing than the men to admit to having depression or to seek help, thereby artificially inflating the rates for women. A large epidemiological study

(Blazer et al., 1998) found a female to male ratio of 0.46:1 for SAD and 5.01:1 for subsyndromal SAD. Lam and Levitt (1999) report an overall female to male ratio of 1.6:1.

For most patients the seasonal episodes begin in their early twenties (Terman, Terman, Quitkin, McGrath et al., 1989) or late twenties (Winton & Checkley, 1989; Wirz-Justice et al., 1986). Depressive episodes most often begin after the autumnal equinox (Terman, Terman, Quitkin, McGrath et al., 1989). The month of onset of depressive symptoms ranges from August to February, with October being the most common (Winton & Checkley, 1989). The peak of depressive symptoms usually occurs in January and February (Terman, Terman, Quitkin, McGrath et al., 1989). Remission occurs between January and July, most frequently in April (Winton & Checkley, 1989). The duration of the depressive episodes ranges from 3.5 to 5.7 months (Winton & Checkley, 1989; Wirz-Justice et al., 1986, 1989).

Family factors may have role in an individual's predisposition to SAD. A family history of affective disorder is found in 25% to 85% of SAD patients (Terman, Terman, Quitkin, McGrath et al., 1989). A family history of SAD is found in 7 to 37% (Winton & Checkley, 1989). Alcohol abuse is found in the relatives of 7% (Rosenthal et al., 1984) to 41% (Allen et al., 1993) of SAD patients.

Perfectly accurate identification of SAD individuals may not be possible. The degree of seasonality may vary over time such that individuals who meet the criteria for SAD may fail to do so at a future time and vice versa. Rosenthal et al. (1984) noted that since seasonal depression often begins with mild episodes that become more severe with increasing age, it often takes several years for individuals to recognize the pattern. Follow-up studies have found that 37% (Sakamoto et al., 1995) to 44% (Schwartz, Brown, Wehr, & Rosenthal, 1996) of subjects who were originally diagnosed with SAD experienced depressive episodes in other seasons as well, or

exhibited less seasonality 6 to 10 years later. Thompson, Raheja, and King (1995) reassessed 93 SAD subjects 5 to 8 years later and found 30% of them to be definitely nonseasonal. Kripke (1998) suggested that many subjects deemed to be seasonal in some studies might actually be nonseasonal with a coincidental seasonal pattern during 2 or 3 years of recurrent depression. Conversely, some of the nonseasonal subjects in light therapy studies might have seasonal depression; Fleischhauer, Glauser and Hofstetter (1988) had 9 nonseasonally depressed subjects who "had a story of seasonality" (p. 414).

Characteristics of SAD

Atypical depressive symptoms, such as hypersomnia (Garvey, Wesner, & Godes, 1988; Tam et al., 1997), carbohydrate craving (Garvey et al., 1988; Thalen, Kjellman, Morkrid, & Wetterberg, 1995), increased appetite (Tam et al., 1997; Thalen, Kjellam, Morkrid, & Wetterberg, 1995), and hyperphagia (Tam et al., 1997) have been found to be more common in seasonal than in nonseasonal depression. Consequently, SAD is often thought to be characterized by these atypical features, so called because they differ from the pattern of depressive symptoms associated with melancholic features that includes early morning awakening and anorexia or weight loss (American Psychiatric Association, 1994). Increased appetite was found in approximately 67% of SAD patients, while 71% had carbohydrate craving, 75% gained weight, and 79% had hypersomnia (Oren & Rosenthal, 1992). However, Winton and Checkley (1989) found much lower rates of appetite increase, carbohydrate craving, weight gain, and hypersomnia (40%, 47%, 40%, and 60% respectively). Approximately 30% of SAD patients exhibit endogenous or typical symptoms, such as insomnia, decreased appetite and weight loss (Blehar & Lewy, 1990).

Assessment of SAD

According to the Canadian Consensus Guidelines for the Treatment of Seasonal Affective Disorder (Lam & Levitt, 1999), a diagnosis of SAD requires both the presence of a DSM-IV major depressive disorder and a seasonal pattern of the depressive episodes. Although atypical symptoms are often associated with SAD, their presence is not required for the diagnosis of the condition. Furthermore, although the DSM-IV has a seasonal specifier to the major depressive disorders (Bipolar I, Bipolar II, Major Depressive Disorder Recurrent) that explicitly calls for a seasonal pattern in the past 2 consecutive years, the Guidelines suggest that this requirement be used with discretion. A person who has a seasonal pattern for 5 out of the last 6 years but not within the last 2 consecutive years could "reasonably be considered to have a seasonal pattern" (p. 36).

Establishing the presence of depression. In the literature, the assessment of depression is usually conducted via clinical interview to determine whether an individual meets the DSM-IV criteria of major depressive disorder for Major Depressive Disorder Recurrent, Bipolar I and Bipolar II. Earlier studies have relied on DSM-III-R criteria.

A measure that is used extensively in SAD research to assess the severity of depression is the Hamilton Depression Rating Scale (HDRS; Hamilton, 1967) and the various versions of it (e.g., see Avery, Kizer, Bolte, & Hellekson, 2001; Eastman, Young, Fogg, Lui, & Meaden, 1998; Lam, Buchanan, Clark, & Remick, 1991; Putilov, 1998). A study that used the original 17-item HDRS (Schuller, Bagby, Levitt, & Joffe, 1993) did not specify a cut-off score for depression. Studies that used the revised 21-item HDRS have adopted varying cut-off scores of 13 (Oren, Jacobsen, Wehr, Cameron, & Rosenthal, 1992; Wirz-Justice et al., 1993), 14 (Hellekson, Kline, & Rosenthal, 1986; James, Wehr, Sack, Parry, & Rosenthal, 1985; Rosenthal et al., 1984), 15

(Jacobson, Wehr, Skwerer, Sack, & Rosenthal, 1987; Lam, 1994; Rosenthal et al., 1985; Terman, Terman, Quitkin, Stewart et al., 1989; Wehr et al., 1986) and 16 (Jacobson et al., 1987; Lam et al., 1991; Wirz-Justice et al, 1986).

However, the 21-item HDRS was deemed to be inappropriate for use in SAD research because of its focus on the endogenous or typical symptoms, that could lead to an underestimation of the severity of atypical depression (Blehar & Lewy, 1990). In response to this problem, a 7-item addendum (Rosenthal, Genhart, Sack et al., 1987) was adopted to measure fatigability and the atypical symptoms of social withdrawal, increased appetite, increased eating, carbohydrate craving, weight gain, and hypersomnia. This addendum, along with an item measuring reverse diurnal variation (worsening of mood, energy, or both in afternoon or evening), was incorporated into the HDRS to create the Structured Interview Guide for the Hamilton Depression Rating Scale - Seasonal Affective Disorder Version (SIGH-SAD; Williams, Link, Rosenthal, & Terman, 1988) which allows for a more accurate assessment of individuals with SAD (Blehar & Lewy, 1990).

Studies that used the SIGH-SAD have adopted different cut-off scores that range from 18 (Ghadirian, Murphy, & Gendron, 1998) to 20 (Avery, Kizer et al., 2001; Terman & Terman, 1999). In some studies a combination of typical and atypical cut-off scores is used to determine the entry criteria. Postolache et al. (1998) used an overall SIGH-SAD score of at least 20, with a typical score of at least 12, or a typical score of at least 14, whereas Lewy et al. (1998), Terman, Amira, Terman, and Ross (1996), Terman, Terman, and Rafferty (1990), and Terman, Terman, and Ross (1998) used a SIGH-SAD score of at least 20, with a typical score of at least 10 plus an atypical score of at least 5.

Some studies have used the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988) to assess the severity of depression (e.g. Geerts, Kouwert, Bouhuys, Meesters, & Jansen, 2000). Baseline criterion was a BDI score of at least 13 (Geerts et al., 2000; Meesters et al., 1993) or 15 (Dam, Molin, Bolwig, Wildschiodtz, & Mellerup, 1993). A few European studies (Lingjærde & Føreland, 1998, 1999; Reichborn-Kjennerud & Lingjærde, 1996) use the Montgomery-Asberg Depression Rating Scale (MADRS; Montgomery & Asberg, 1979) with a 3- or 4-item atypical scale to assess increased sleep, increased appetite, carbohydrate craving, and fatigability.

Overall, the more extensively used criteria of detecting depression in the current literature is the fulfilment of the DSM-IV major depressive disorder with a seasonal specifier, and the SIGH-SAD or its equivalent (i.e., the HDRS with the special addendum for atypical symptoms).

Establishing the presence of seasonality. In addition to the diagnosis of major depression, the pattern and degree of seasonality of the disorder must be established. The Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Genhart, Sack et al., 1987) is the one measure that is commonly used to screen for SAD (e.g., Bauer, Kurtz, Rubin, & Marcus, 1994; Kasper et al., 1989; Stinson, & Thompson, 1990; Wileman et al., 2001). The SPAQ does not provide diagnostic information but rather evaluates the severity of seasonal mood disturbance (Blazer et al., 1998). It yields three indicators, all of which are needed to determine the degree of seasonality.

First, the degree of seasonality is assessed via the Global Seasonality Score (GSS), which is a numerical rating of seasonal changes in the atypical symptoms of sleep length, social activity, mood, weight, appetite and energy level. Second, the degree of impairment that the individual experiences due to seasonal changes is measured on the levels of mild, moderate,

marked, severe, or disabling. Third, the SPAQ establishes the months during which the individual experiences the symptoms and during which they are absent.

To determine the presence of seasonality, the individual must endorse a seasonal pattern to the depressive symptoms where their presence is evident during the fall and winter months, and their remission is reported during the summer months. However, there are no clear guidelines regarding the number of symptoms that must follow the seasonal pattern or the specific months in which the symptoms are present or absent. Furthermore, for seasonality to be established, the individual must attain a high GSS score that, depending on the studies, has ranged from a minimum of 11 with at least a moderate level of impairment (Kasper et al., 1989; Rosenthal, 1993) to a minimum of 12 (Terman & Terman, 1999). For subsyndromal SAD, the cut-offs range from GSS score greater than 5 (Avery, Kizer et al., 2001) to GSS greater than 10 with mild or no problem (Magnusson, 1996). Rosenthal (1993) defines subsyndromal SAD by a GSS of 8 or 9 with at least mild impairment or a GSS of 10 with no impairment. Nondepressed participants have a GSS less than 7 (Terman & Terman, 1999).

The DSM-IV has also established vague criteria for seasonality in its seasonal specifier (see Appendix 1). Accordingly, the depressive episodes must have a particular pattern where their onset and remission occur at specific times each year. Two seasonal depressive episodes must have occurred during the past 2 years, and the seasonal episodes must have substantially outnumbered nonseasonal depressive episodes during the person's lifetime. As well, the seasonal pattern must not be due to seasonal stressors such as the anniversary of the death of a loved one. The DSM-III-R (1987) requires that the depressive episodes have their onset within a particular 60-day period each year, that remission occurs within a particular 60-day period of the year, that there have been at least three seasonal episodes, two of which have been in consecutive

years, and that seasonal episodes outnumber nonseasonal episodes by a ratio of 3 to 1 (see Appendix 2). In the DSM-III-R criteria, the applicability of seasonal pattern to affective disorders Not Otherwise Specified allows for the diagnosis of subsyndromal SAD in subjects who do not meet diagnostic criteria for major depression (Blehar & Lewy, 1990). The Rosenthal criteria (Rosenthal et al., 1984) includes at least 2 consecutive years in which depression developed during fall or winter and remitted by spring or summer, and the absence of any seasonal psychosocial stressors (see Appendix 3).

Summary. According to the Canadian Consensus Guidelines, SAD is established with the presence of a DSM-IV major depressive disorder with a seasonal specifier. Although the seasonal specifier demands that the individual has more depressive episodes that are seasonal than not and that the seasonal episodes occur during the last 2 consecutive years, the Guidelines suggest that this ruling be used with some judgement as long as the individual shows more seasonal depressive episodes than not. As well, the seasonal pattern must not be due to seasonal psychosocial stressors. The SPAQ can be used as a screening measure where seasonality is determined by a GSS of at least 11, a seasonal pattern to the symptoms such that the person feels worst during the fall and winter months, and at least a moderate level of impairment due to the seasonality. The severity of the typical and atypical symptoms is often assessed with the SIGH-SAD or its equivalent HDRS that has the addendum for atypical symptoms.

Etiology

SAD is generally believed to be a reaction to the shorter days of winter in susceptible individuals (Thompson & Silverstone, 1989). Individuals who travel south during their depressed period often report an improvement in mood within a few days (Rosenthal et al., 1984; Wirz-Justice et al., 1986). Many patients relate their depression to the lack of daylight in winter.

This sensitivity to changes in season and latitude was identified by Rosenthal et al. (1984) as the single outstanding clinical feature of SAD. Bright light therapy has been accepted as an effective treatment for this disorder (see reviews by Blehar & Lewy, 1990; Lee & Chan, 1999; Rosenthal, Sack, Skwerer, Jacobsen, & Wehr, 1989; Terman, Terman, Quitkin, McGrath et al., 1989); however, the exact mechanism of therapeutic action is unknown (Lee et al., 1998).

Several hypotheses have been put forth. The photoperiod hypothesis suggests that it is specifically the shorter day length that causes depression and that bright light is necessary in both the morning and the evening to restore normal summer mood and behaviours (Lam & Levitan, 2000). Support for this theory was found in studies that showed increasing prevalence of SAD at more northerly latitudes (Potkin, Zetlin, Stamenkovic, Kripke, & Bunney, 1986; Rosen et al., 1990). However this finding was disputed in a later review (Mersch, Middendorp, Bouhuys, Beersma, & van den Hoofdakker, 1999). Although morning and evening light exposure is an effective treatment for SAD (Postolache et al., 1998; Terman, Terman, Quitkin, McGrath et al., 1996), other studies have found morning light alone to be equally effective (Terman, Terman, Quitkin, McGrath et al., 1989). The photoperiod hypothesis is still being pursued as photoperiod may be implicated in the onset of atypical vegetative symptoms commonly found in SAD (Young, Meaden, Fogg, Cherin, & Eastman, 1997; Young, Watel, Lahmeyer, & Eastman, 1991).

The photon-counting hypothesis suggests that the lower amount of light in winter causes symptoms of depression and that bright light therapy relieves depression by replacing this missing light (Thompson & Silverstone, 1989). Vulnerable individuals may have a decreased capacity to absorb light leading to insufficient light energy to maintain normal mood (Lee et al., 1998). In this case the timing of light therapy is not critical to therapeutic response and phototherapy can be effectively given at any time of day (Dalgleish, Rosen, & Marks, 1996;

Terman, Terman, Quitkin, McGrath et al., 1989). The results of several studies support this theory (e.g. Geerts et al., 2000; Lafer, Sachs, Labbate, Thibault, & Rosenbaum, 1994; Putilov, 1998; Wirz-Justice et al., 1993); however, other studies found a differential response to morning and evening light therapy (e.g. Eastman et al., 1998; Lewy et al., 1998). Further evidence for this theory is the finding of a positive correlation between light intensity and antidepressant effect (Lee & Chan, 1999).

Some researchers suggest that SAD patients have a phase delay of circadian rhythms in winter (Thompson & Silverstone, 1989). This phase delay could produce the hypersomnia with delayed morning awakening and difficulty arising that is common among individuals with SAD (Avery et al., 1990). The onset of melatonin production is an indicator of circadian phase (Lam & Levitt, 1999). Melatonin is normally secreted at night and its production is suppressed by bright light but not by regular artificial light (Rosenthal et al., 1985). The phase-shift hypothesis suggests that the antidepressant effects of bright light are due to the correction of phase-delayed circadian rhythms in SAD patients. Bright light in the morning causes a phase advance while bright light in the evening causes a phase delay (Lewy et al., 1998). If the phase-shift hypothesis is correct, light therapy should be more effective when given in the morning than in the evening (Lewy et al., 1998). Support for this theory is mixed, with some studies finding morning light superior to evening light (see reviews by Blehar & Lewy, 1990; Lam & Levitan, 2000; Lee et al., 1998; Rosenthal, Sack et al., 1989). Several studies failed to find a difference in effectiveness between morning and evening light treatment (see reviews in Blehar & Lewy, 1990; Lam & Levitan, 2000; Rosenthal, Sack et al., 1989; Terman, Terman, Quitkin, McGrath et al., 1989). However, no study has found evening light treatment to be superior to morning light (Lewy et al., 1998). Terman, Terman, Lo, and Cooper (2001) found that morning light exposure resulted

in an earlier onset of melatonin production in the evening, as well as an increase in the time between the onset of melatonin production and sleep. Reductions in SIGH-SAD scores were significantly positively correlated with both the earlier onset of melatonin production and the increase in the interval between the onset of melatonin production and sleep onset.

Neurotransmitters such as serotonin, dopamine and noradrenaline have been implicated in SAD (Dalgleish et al., 1996). Serotonin is of particular interest because it controls appetite and sleep and is a precursor of melatonin. SAD patients may produce too little serotonin in winter. Dietary carbohydrates, which increase the production of serotonin in the brain (Rosenthal, 1993), were found to have an energizing effect on individuals with SAD, while nondepressed individuals reported drowsiness (Rosenthal, Genhart et al., 1989). The carbohydrate craving experienced by many SAD patients may be a way of correcting serotonin deficiency. Light therapy may increase production of serotonin (Rosenthal, 1993).

Some authors support a dual vulnerability hypothesis that posits that SAD is due to two pathogenic processes (Lam, Tam, Yatham, Shiah, & Zis, 2001; Young et al., 1991). The atypical symptoms might be triggered by seasonality (i.e., a combination of a vulnerability trait such as genetics and environmental factors such as decreased exposure to light), whereas the typical symptoms might be triggered by a combination of a vulnerability trait (e.g., genetics) and external effects (e.g., stressful life events). This dual vulnerability hypothesis is supported by observations that atypical symptoms, but not typical symptoms, are temporally associated with the onset of SAD during the fall and winter months (Rosenthal et al., 1984; Young et al., 1991). The various typical symptoms appear randomly throughout the depressive episode. Further supporting evidence lies in the finding that atypical symptoms respond to light therapy regardless

of light intensity whereas the typical symptoms show a dose-response relationship to the light intensity (Lee & Chan, 1999).

In all of the above hypotheses exposure to light is the common theme. However, SAD patients are not a homogeneous group (Blehar & Lewy, 1990) and there is unlikely to be one key to explain the therapeutic action of light therapy (Terman, 1988).

Light Therapy

Given the importance of light in the etiology of SAD, it is not surprising that considerable research efforts have been directed towards the investigation of the effect of light exposure on SAD symptoms. While most of these studies have examined only individuals with SAD (e.g. Avery, Eder et al., 2001; Bauer et al., 1994; Eastman et al., 1998; Geerts et al., 2000; Ghadirian et al., 1998; Lafer et al., 1994; Lam, Tam, Shiah, Yatham, & Zis, 2000; Lewy et al., 1998; Postolache et al., 1998; Putilov, 1998; Terman et al., 1998; Wileman et al., 2001), some have examined only individuals with nonseasonal depression (Deltito, Moline, Pollak, Martin, & Maremmani, 1991; Kripke, Mullaney, Klauber, Risch, & Gillin, 1992; Kripke, Risch, & Janowsky, 1983; Mackert, Volz, Stieglitz, & Müller-Oerlinghausen, 1991, Yamada, Martin-Iverson, Daimon, Tsujimoto, & Takahashi, 1995). Others have compared the relative effectiveness of light therapy with both SAD and nonseasonal depression (Stewart, Quitkin, Terman, & Terman, 1990; Stinson & Thompson, 1990; Thalen, Kjellman, Mørkrid, Wibom et al., 1995; Yerevanian, Anderson, Grota, & Bray, 1986).

The operational definition of therapeutic response used in the literature has relied on either a single response criterion or joint response criteria. The single response criterion usually consists of a 50% reduction in SIGH-SAD score (e.g. Ghardirian, Murphy, & Gendron, 1998; Lafer et al., 1994; Lam et al., 2000; Lam, Tam, Yatham, Shiah, & Zis, 2001), a final SIGH-SAD

score less than 9 (e.g. Terman, Terman, & Ross, 1998), or a final SIGH-SAD score less than 8 (e.g. Lafer et al., 1994). The joint response criteria consists of a 50% reduction in SIGH-SAD score and a final SIGH-SAD score less than 8 (e.g. Postolache et al., 1998) or less than 9 (e.g. Avery, Eder et al., 2001; Eastman et al., 1998; Terman & Terman, 1999; Wileman et al., 2001). Some researchers use joint response criteria consisting of a 50% reduction in SIGH-SAD score with a final score of less than 8 on each of the two measures (HDRS and atypical addendum) resulting in a final score less than 15 (e.g. Lewy et al., 1998) or 16 (e.g. Eastman, Lahmeyer, Watell, Good, & Young, 1992; Putilov, 1998; Terman et al., 1996).

Efficacy of light therapy. Several studies have shown bright light therapy to be an effective treatment for SAD (see reviews by Blehar & Lewy, 1990; Lee et al., 1998; Rosenthal et al., 1989; Terman, Terman, Quitkin, McGrath et al., 1989). Overall response rates in review articles have ranged from 36% to 80% (Lam, Terman, & Wirz-Justice, 1997; Tam et al., 1995; Terman, Terman, Quitkin, McGrath et al., 1989). More recent studies report response rates that range from 30% to 67% (Eastman et al., 1998; Geerts et al., 2000; Ghadirian et al., 1998; Lam et al., 2000, 2001; Lewy et al., 1998; Postolache et al., 1998; Putilov, 1998; Terman & Terman, 1999; Terman et al., 1998; Wileman et al., 2001).

In several studies both typical and atypical symptom scores were significantly reduced following light therapy (e.g. Avery, Kizer et al., 2001; Bauer et al., 1994; Geerts et al., 2000; Lam et al., 1991; Lam, Buchanan, Mador, & Corral, 1992; Lam et al., 2001; Lingjærde & Føreland, 1998, 1999; Postalache, et al., 1998; Putilov, 1998). The beneficial effect of light therapy on atypical symptoms was measured by a decrease in the atypical item score on the SIGH-SAD. Few studies have examined the effect of light therapy on individual atypical symptoms. Rosenthal, Genhart, Jacobsen et al. (1987) found that carbohydrate craving assessed

by the atypical addendum to the HDRS responded most rapidly and dramatically to light treatment. Stewart et al. (1990) found significant reductions in each of the atypical symptoms in SAD patients but no improvement in atypical symptoms in nonseasonal depressed patients. However this study had only 8 nonseasonal patients for analysis.

Assessment of the effects of light therapy on SAD symptoms is often done by retrospective rating of symptoms over the past week on the SIGH-SAD or atypical addendum (Avery, Kizer et al., 2001; Bauer et al., 1994; Geerts et al., 2000; Lam et al., 1991, 1992, 2001; Lingjaerde & Føreland, 1998, 1999; Postalache et al., 1998; Putilov, 1998). An exception was a study by Krauchi, Wirz-Justice, and Graw (1990) that had participants prospectively fill out a daily food diary. They found that improvement in one symptom, carbohydrate intake, was delayed when compared to the other atypical symptoms. Shapiro, Devins, Feldman, and Levitt (1994) found that retrospective reports of sleep duration on the SPAQ did not correlate with information collected on a daily sleep log, suggesting a problem in recall reliability. In essence, retrospective measures of atypical symptoms have low reliability compared to prospective measures (Nayyar & Cochrane, 1996).

Several studies on light therapy with nonseasonal depression have reported significant reduction in the severity of depressive symptoms (e.g., Fleischhauer, et al., 1988; Kripke, 1998; Wirz-Justice, Graw, Röösli, Glauser, & Fleischhauer, 1999; Yamada et al., 1995). Deltito et al. (1991) found significant results for bipolar patients but not for unipolar patients. Mackert et al. (1991) reported a statistically significant reduction in the severity of depressive symptoms after light therapy; however the reduction was too small to be considered clinically significant.

In contrast, four studies that compared seasonal and nonseasonal patients reported that the nonseasonal group either showed no response (Stinson & Thompson, 1990; Yerevanian et al.,

1986) or showed a much lower response than the SAD group (Stewart et al., 1990; Thalen, Kjellman, Morkrid, Wibom et al., 1995). Three of these studies had very few nonseasonal patients (Stewart et al. 1990, $n = 8$; Stinson & Thompson, 1990, $n = 3$; Yerevanian et al., 1986, $n = 8$). Brighter light sources, such as 10,000 lux commonly used in recent studies have not been used in nonseasonal depression (Lam et al., 1997).

Side effects of light therapy. The most frequently reported side effects following 10,000 lux light therapy were mild eye and vision problems (e.g. eye strain, excessive glare, seeing spots, etc.), headaches, (Avery, Eder et al., 2001; Kogan & Guilford, 1998; Labbate, Lafer, Thibault, & Sachs, 1994) and nausea (Avery, Eder et al., 2001; Kogan & Guilford, 1998). Sleep disturbance was found in individuals exposed to evening light (Avery, Eder et al., 2001; Labbate et al., 1994). Mild hypomania (Avery, Eder et al., 2001) and agitation occurred in a few individuals and remitted with decreased light exposure (Bauer et al., 1994; Labbate et al., 1994). In most cases side effects ceased by the third day of light therapy. Terman and Terman (1999) found an overall improvement in 88 physical, psychological, and behavioural symptoms in SAD patients receiving 30 minutes of 10,000 lux light therapy for 10 to 14 days.

Light characteristics and treatment response. Researchers who examine the specific characteristics of light therapy that are necessary for optimal response have focussed on the intensity of light, timing of sessions, length of sessions, and duration of treatment. Most studies used bright light of 2,000 to 2,500 lux (Avery, Kizer et al., 2001; Bauer et al., 1994; Lafer et al., 1994; Lam et al., 2000; Lewy et al., 1998; Lingjærde & Føreland, 1998, 1999; Putilov, 1998) with brighter light (10,000 lux) becoming more widely used in recent studies (e.g., Geerts et al., 2000; Ghadirian et al., 1998; Lam et al., 2001; Postolache et al., 1998; Terman & Terman, 1999; Terman et al., 1998; Wileman et al., 2001).

Research that attempts to find a link between the intensity of light and improvement in symptoms has produced equivocal results. While some studies have noted that light intensity has no bearing on symptom improvement (see review by Dalgleish et al., 1996; Wileman et al., 2001;Wirz-Justice et al., 1986), others report that bright light is superior to dim light (see reviews by Dalgleish et al., 1996; Lee et al., 1998; Rosenthal, Sack et al., 1989; Sato, 1997; Terman, Terman, Quitkin, McGrath et al., 1989). When typical and atypical symptoms were examined separately, research suggested a dose-response for typical symptoms such that strong light was superior to medium light which, in turn, was superior to dim light in its effect (Lee & Chan, 1999). Atypical symptom improvement appears to be unrelated to light intensity.

Studies on light treatment have mostly used morning exposure while evening exposure is less common. The time of morning exposure ranges from 5:00 a.m. (Fleischhauer et al., 1988; Kripke et al., 1992) to noon (Avery, Kizer et al., 2001). Some studies specify the time as upon awakening (Eastman et al., 1992, 1998). The most common time for morning sessions is between 6:00 a.m. and 8:00 a.m. (e.g. Bauer et al., 1994; Ghardirian et al., 1998; Lafer et al., 1994; Lam et al., 1991, 2000; Lewy et al., 1998; Thalen, Kjellman, Morkrid, Wibom et al., 1995; Yamada et al., 1995). Evening light therapy session times range from 4:30 p.m. to 6:30 p.m. (Meesters et al., 1993) to 8:00 p.m. to 11:00 p.m. (Kripke et al., 1992). The most common time has been 6:00 p.m. to 8:00 p.m. (e.g. Putilov, 1998; Stewart et al., 1990; Thalen, Kjellman, Morkrid, Wibom et al., 1995; Yamada et al., 1995).

Several studies have demonstrated the superiority of morning light compared to evening light (Eastman et al., 1998; Lewy et al., 1998; Terman et al., 1998; see also reviews by Blehar & Lewy, 1990; Lam & Levitan, 2000; Lee et al., 1998; Rosenthal, Sack et al., 1989; Tam, Lam, & Levitt, 1995; Terman, Terman, Quitkin, McGrath et al., 1989). Other studies failed to find a

difference in effectiveness between morning and evening treatment (Avery, Kizer et al., 2001; Geerts et al., 2000; Putilov, 1998; see also reviews by Blehar & Lewy, 1990; Dalgleish et al., 1996; Lam & Levitan, 2000; Lee et al., 1998; Rosenthal, Sack et al., 1989). However no study has reported evening treatment to be more effective than morning treatment (Lam & Levitt, 1999; Sato, 1997) and morning light is recommended as the first choice for treatment (Lam & Levitt, 1999).

The length of treatment sessions has been found to influence outcome (Terman, Quitkin, Terman, Stewart, & McGrath, 1987). In seven studies reviewed by Sato (1997), patients who had received the shortest duration of treatment were least likely to improve. However, 30 minutes (Lewy, Sack, Singer, & White, 1987) or 1 hour (Lingjærde & Føreland, 1999) were found to be equivalent to 2 hours of light treatment. Brief light treatment (30 minutes exposure) resulted in a response rate of 31% (Terman, Terman, Quitkin, McGrath et al., 1989). The relationship between length of treatment session and outcome has not been precisely determined and an individual patient may respond best with either short or long treatment sessions (Lewy et al., 1987). Although results have been reported with treatments that lasted only 1 to 2 weeks (Lam. 1994; Lam et al., 1992; Oren et al., 1992; Reichborn-Kjennerud & Lingjaerde, 1996; Stinson & Thompson, 1990), superior results have been found with longer treatments of 3 or 4 weeks (Bauer et al., 1994; Eastman et al., 1998; Labbate, Lafer, Thibault, Rosenbaum, & Sachs, 1995). Six weeks of bright light therapy resulted in a response rate of 48% (Avery, Eder et al., 2001), but this was less than the response to a simulated dawn condition of increasing white light and no different from the placebo dawn condition with dim red light.

Response usually occurs within 2 to 4 days (Lam & Levitt, 1999) with measurable improvement within 1 week and with a similar time period for relapse after completion of light

therapy. Clinical efficacy varies with time of exposure, intensity of light, and baseline severity. The Canadian Consensus Guidelines (Lam & Levitt, 1999) recommends 10,000 lux for 30 minutes per day in the early morning, on awakening.

Patient characteristics and treatment response. Several researchers have attempted to determine the types of patient factors that predict response to light therapy. Some studies found that patients with more severe atypical symptoms have better response to light therapy than patients with less severe atypical symptoms (Lam, 1994; Lam et al., 1992; Nagayama et al., 1991; Oren et al., 1992; Stinson & Thompson, 1990; Terman et al., 1996). Baseline atypical scores were significantly positively correlated with the degree of improvement in atypical scores (Lam, 1994; Lam et al., 1992; Oren et al., 1992; Stinson & Thompson, 1990) and with the degree of improvement in total (typical plus atypical) scores (Lam et al., 1992; Nagayama et al., 1991; Oren et al., 1992). The ratio of atypical score to total SIGH-SAD score was found to be a significant predictor of improvement in total SIGH-SAD score (Terman et al., 1996, 1998). Individuals with a higher percentage of atypical symptoms had a greater reduction in total SIGH-SAD scores. Specific atypical symptoms that positively correlated with improved response to light therapy include hypersomnia (Lam, 1994; Lam et al, 1992; Oren et al., 1992; Terman et al., 1996), increased eating (Lam, 1994), afternoon/evening slump, and carbohydrate craving (Terman et al., 1996). Terman et al. (1996) found reverse diurnal variation (worsening of mood in the afternoon) to be a positive predictor of response. However, Meesters et al. (1993) found that individuals with greater variability in mood throughout the day were less likely to respond to light therapy.

Other studies found that the severity of atypical symptoms at baseline was not correlated with light treatment response (Dam et al., 1993; Lam et al., 2001; Meesters et al., 1993;

Reichborn-Kjennerud & Lingjaerde, 1996; Thalen, Kjellman, Morkrid, Wibom et al., 1995). Eastman et al. (1992) deliberately chose SAD patients with atypical symptoms in order to obtain a more homogeneous group for their placebo-controlled trial of light therapy. This group had a response rate of only 28% after 6 days of 7,000 lux for 1 hour per day, but this rate was no different from the placebo response. However, Stewart et al. (1990) found a response rate of 13% in 8 nonseasonal depressed patients with atypical symptoms, while SAD patients had a response rate of 92%. The small number of nonseasonal patients in this study gave it limited power.

Two studies examined the relationship between GSS and improvement in symptoms following light therapy. Avery, Kizer et al. (2001) found a significant correlation between GSS and screening SIGH-SAD score in individuals with subsyndromal SAD. However GSS was not correlated with improvement in SIGH-SAD scores. Reichborn-Kjennerud and Lingjærde (1996) found that GSS was not a significant predictor of outcome among SAD patients in that GSS did not differ between responders and non-responders to light therapy.

Placebo control. Several controlled trials have been conducted using various placebo conditions. Eastman et al. (1998) used a deactivated negative ion generator that resulted in fewer remissions (32%) than the active treatment (61%) after 3 weeks. However Terman, Terman, and Ross (1998) found unexpected clinical improvement with high-density negative ion treatment. Seventy-one per cent of patients responded to morning light, 67% to evening light, and 50% to high-density negative ions. Dim white light (500 lux) showed no significant effect on SIGH-SAD scores (Lam et al., 1991), but dim red light (500 lux) resulted in a 33% remission rate using Terman's (1989) strict joint response criteria (Wileman et al., 2001). Although most controlled studies in nonseasonal depression found no significant response to dim light (e.g. Kripke et al.,

1992; 1983; Mackert et al., 1991; Yamada et al., 1995), Deltito et al. (1991) found that bipolar nonseasonal patients showed significant improvement with either bright (2500 lux) or dim (400 lux) light.

Expectancy effects. An expectation of improvement can have an effect on the response to any treatment (Brown, 1990). Several studies used an expectancy scale to measure participants' expectations of light therapy. Some studies used visual analogue scales (VAS; Eastman et al., 1992; Kripke et al., 1992; Wirz-Justice et al., 1993). In other studies participants were asked how they felt the light treatment would make them feel and were given a range of possible responses from "much worse", through "no change", to "much better" with a 5- (Lam et al., 1991), 7- (Bauer et al., 1994; Eastman et al., 1998; Sack et al., 1990; Wileman et al., 2001) or 9- (Jacobsen et al., 1987) point scale for the response. Most studies found no correlation between expectancy scores and treatment response (Bauer et al., 1994; Eastman et al., 1992; Kripke et al, 1992; Sack et al., 1990; Wileman et al., 2001; Wirz-Justice et al., 1993). Terman et al. (1996) found that responders to bright light therapy had significantly higher expectations than non-responders. Eastman et al. (1998) found a small negative correlation ($r = -0.18, p = .04$) at Week 3 of treatment, but a nonsignificant correlation at Week 4. In the study by Eastman et al. (1998), patient expectations were not significantly different between light therapy and placebo (de-activated negative ion generator) groups. However, Terman et al. (1998) found that expectations were higher for the light group than for the ion group.

Conclusion. Light therapy is an effective treatment for SAD with many studies showing response rates between 50% and 67% using Terman's (1989) strict joint criteria (Bauer et al., 1994; Eastman et al., 1998; Terman & Terman, 1999; see also review by Terman, Terman, Quitkin, McGrath et al., 1989). Light therapy also appears to be somewhat effective for

nonseasonal depression with several studies reporting significant decrease in depression scores after light treatment (Fleischhauer et al., 1988; Kripke et al., 1983, 1992; Wirz-Justice et al., 1999;Yamada et al., 1995). Light therapy was effective in reducing both typical and atypical symptoms in SAD patients (Avery, Kizer et al., 2001; Bauer et al., 1994; Geerts et al., 2000; Lam et al., 1991; Lingjærde & Føreland, 1998, 1999; Postolache et al., 1998; Putilov, 1998; Wileman et al., 2001) but was ineffective for atypical symptoms in nonseasonally depressed patients (Stewart et al., 1990). A dose-response relationship was found for typical symptoms whereas atypical symptoms appear to improve regardless of light intensity (Lee & Chan, 1999). Attempts to determine which patient characteristics predict response to light therapy have had mixed results. Some studies found that individuals with more severe atypical symptoms have better response to light therapy than individuals with less severe atypical symptoms (Lam, 1994; Lam et al., 1992; Nagayama et al., 1991; Oren et al., 1992; Stinson & Thompson, 1990; Terman et al., 1996). Other studies found no relationship between severity of atypical symptoms and treatment response (Dam et al., 1993; Lam et al., 2001; Meesters et al., 1993; Reichborn-Kjennerud & Lingjaerde, 1996; Thalen, Kjellman, Morkrid, Wibom et al., 1995). A relationship between GSS and treatment outcome was not found in two studies (Avery, Kizer et al., 2001; Reichborn-Kjennerud & Lingjærde 1996).

The Present Study

Previous attempts to determine which patient characteristics predict response to light therapy have produced mixed results. Some studies reported that individuals with more severe atypical symptoms have better response to light therapy than individuals with less severe atypical symptoms (Lam, 1994; Lam et al., 1992; Nagayama et al., 1991; Oren et al., 1992; Stinson & Thompson, 1990; Terman et al., 1996), while other studies have found no relationship between

severity of atypical symptoms and treatment response (Dam et al., 1993; Lam et al., 2001; Meesters et al., 1993; Reichborn-Kjennerud & Lingjaerde, 1996; Thalen, Kjellman, Morkrid, Wibom et al., 1995). It is possible that therapy changes may be predicted by the degree of seasonality, and the severity of both typical and atypical depressive symptoms.

The objective of the present study was to investigate within a prospective design the seasonality and depression predictors of light therapy response after controlling for treatment expectancy and pre-therapy functioning. Seasonality was indicated by the GSS on the SPAQ, and the depression predictors were measured with the SIGH-SAD that yielded severity levels for the typical and atypical depressive symptoms. Individuals with elevated SIGH-SAD total depression scores were administered 14 days of light therapy. Their pre- and post-therapy (Time 1 and Time 2, respectively) functioning on several variables was measured: typical and atypical depressive symptoms, sleep duration, sleep quality, carbohydrate craving, carbohydrate intake, and level of energy.

It was expected that GSS and Time 1 atypical symptom score would be positively correlated and that both would predict response to light therapy. More specifically, higher GSS would predict lower Time 2 total depression (typical and atypical combined) measured by the SIGH-SAD. As well, higher Time 1 atypical symptom score would predict lower total depression, lower atypical symptom scores, shorter sleep duration, decreased carbohydrate craving, and increased energy at Time 2.

More detailed examination of the data was carried out with the assessment of two other predictors: Atypical balance and reverse diurnal variation (Rdv). Atypical balance was defined as the percentage of the Time 1 atypical score to the Time 1 total SIGH-SAD score. Higher atypical balance score therefore indicates a greater proportion of atypical symptoms to total

symptoms (Terman et al., 1996). Reverse diurnal variation (Rdv) represents the extent to which individuals experience lower mood in the afternoon as opposed to in the morning. It was calculated by summing three SIGH-SAD items that related to the construct. Afternoon mood slumps are more common in SAD (Terman et al., 1996) than in typical depression that is characterized by low mood in the morning with improvement during the day. Higher Rdv indicates more severe afternoon mood slumps.

Method

Participants

Participants were recruited from the undergraduate student, faculty and staff population at Lakehead University and from the clinical and general community of Thunder Bay. They had a total depressive symptom score of at least 22 on the SIGH-SAD at initial screening. The highest cut-off depressive score found in the literature to classify depressed or SAD individuals is 20 (Lewy et al., 1998; Postolache et al., 1998; Terman et al., 1990, 1996, 1998; Terman & Terman, 1999). A cut-off of 22 in the present study was selected to avoid a floor effect and allow measurement of changes in total depression score after light therapy. Typical symptom scores, atypical symptom scores and GSS were free to vary in order to examine the relationship of these predictors to the responses to light therapy.

An upper age limit of 55 years was imposed because of age-related macular degeneration that may be asymptomatic. Individuals with this condition may be more sensitive to light (Lam & Levitt, 1999). The prevalence of age-related macular degeneration is approximately 1% of those individuals aged 55 years in the United States (Oneill, Jamison, McCulloch, & Smith, 2001).

Participants who had a history of antidepressant use were free of the antidepressants for at least 4 weeks (8 weeks for fluoxetine) before the commencement of their light therapy sessions (Deltito et al., 1991; Lewy et al., 1998; Meesters et al., 1993). None of the participants had previous experience with light therapy (Eastman et al., 1992, 1998; Lam et al., 1991). The Canadian Consensus Guidelines (Lam & Levitt, 1999) noted that there are no absolute contraindications to light therapy and recommended that light therapy for individuals with eye diseases can be carried out with close ophthalmological monitoring. However participants in the present study were free of pre-existing retinal or eye diseases such as retinal detachment, glaucoma, cataracts, macular degeneration (Lam & Levitt, 1999; Terman et al., 1996), systemic illnesses that affect the retina such as diabetes mellitus and lupus, and use of photosensitising medications or supplements including tetracycline (Kripke et al., 1992), melatonin, hypericum (St. John's Wort), and beta blockers (Lam & Levitt, 1999) within the last 4 weeks of the commencement of light therapy.

Apparatus

Three light boxes (Brite Lite IV by Apollo Light Systems) were used for light therapy. Each box provides exposure to 10,000 lux of full spectrum light without ultraviolet rays at the recommended distance of 26 inches.

Materials

Assessment of seasonality and depressive symptoms (see Appendix 4). These measures are part of a Research Questionnaire that was used in a program of research on SAD at Lakehead University that incorporated several studies. Only the sections of the Research Questionnaire that were relevant to the present study are covered below.

Section A was used to collect demographic and medical information. Noteworthy are the

specific questions that ask the participant about eye diseases, systemic illnesses, and use of prescribed and over-the-counter drugs.

Section B is primarily based on the SPAQ (Rosenthal, Genhart, Sack et al., 1987) with several extra questions to assess the DSM-IV seasonal specifier criteria and to obtain additional information on the respondent's history and experience with seasonality. Question 1, which is based on the SPAQ, is used determine whether or not the participant exhibits a seasonal pattern to typical and atypical depressive symptoms. Questions 2 and 3 were developed for the SAD program of study to assess the extent of the seasonal pattern over the last 6 years. Question 4 is derived from the SPAQ. It calls for the participant to rate on a 5-point scale the degree of seasonal change associated with each of six symptoms. Summation of the ratings yields the Global Seasonality Score (GSS) that can range from 0 to 24. Question 5 is also derived from the SPAQ to measure the degree of impairment associated with the seasonal changes. In keeping with the DSM-IV seasonal specifier criteria, questions 6 and 7 were designed for the SAD program of research to discount the role of seasonal stressors in the activation of depressive episodes.

Section C contains the 28-item SIGH-SAD (Structured Interview Guide for the Hamilton Depression Rating Scale – Seasonal Affective Disorder version, Williams, Link, Rosenthal, & Terman, 1988). It is based on the Hamilton Depression Rating Scale (Hamilton, 1967) and has 21 typical depressive symptom items, plus 7 additional items for atypical depressive symptoms that are associated with SAD. Each item can be rated on a 5-point scale that ranges in severity from 0 (not at all) to 4 (marked or severely).

The SIGH-SAD yields three types of scores. The first 21 items when summed produce the typical depressive symptom score. The last 7 items when summed produce the atypical

depressive symptom score. Adding up all the items in the SIGH-SAD gives the total depressive symptom score. Higher scores reflect greater severity of depressive symptoms. All participants had a total score of 22 or more on initial screening.

The SIGH-SAD was also used to calculate two additional variables. First, Atypical Balance, defined as the percentage of the Time 1 atypical score to the Time 1 total SIGH-SAD score, was calculated as (Time 1 Atypical Score /Time 1 Total SIGH-SAD) X 100. Second, reverse diurnal variation (Rdv) that represented the extent to which individuals experience lower mood in the afternoon as opposed to the morning, was computed by summing responses to questions 16 (reverse-scored), 17, and 28 on the pre-therapy SIGH-SAD.

Assessment measures for therapy response (see Appendix 5). A variety of measures were developed for the present study to allow the participants to report average daily functioning before and after light therapy. Section A consists of two visual analogue scales (VAS) to measure average energy level and carbohydrate craving during the past week. The VAS is a 10-centimetre line where one end represents the complete absence of the dimension under assessment while the other end represents the maximum quantity. The participant was instructed to make a mark on the line that represented their current state (Lingjærde & Føreland, 1998). The distance from the left end of the line was measured in millimetres, giving a range of 0 to 100. VAS ratings have been found to correlate significantly with depression ratings (Luria, 1975) and showed high test-retest reliability (Lingjærde & Føreland, 1998).

Section B assesses sleep quality and duration. A VAS was used to measure sleep quality by asking participants to report the degree of restfulness they felt upon awakening. Participants were also asked to report their estimated average nightly sleep onset time, number and length of nocturnal awakenings, and the duration of naps (Eastman et al., 1992, 1998). The total amount

of sleep per day (in minutes) was calculated based on the information obtained. In Section C participants documented any unusual stress that they had experienced during the past week that might account for significant observations in sleep, energy, and mood. Section D assesses the participant's expectations of the effectiveness of light treatment. A 7-point scale was used where 1 = much worse, 4 = no change, and 7 = much improvement.

Section E is a food diary that is adapted from the Food/Drink Frequency Questionnaire (Krauchi et al., 1990). Participants were asked to record the average daily frequency with which they ate various types of foods over the last week. Carbohydrate intake was calculated by counting the number of servings of sweet foods (jam, honey, sugar, chocolate, ice cream, cake, cookies, pastry, pop, and candies) and the number of servings of starchy foods (breads, bagels, rolls, potatoes, yams, corn, rice, pasta, cereal, potato chips, and tortilla chips) eaten each day (Krauchi et al., 1990).

Procedure

Recruitment. Recruitment of participants for this study was carried out together with recruitment of participants for another study on Seasonal Affective Disorder. Posters about the research (see Appendix 6) were put up around the university, in public areas in the community (e.g. malls, health food stores), and in hospitals and clinics. Notices were placed in the local newspaper, The Chronicle Journal, Helping Hands column, the Lakehead University student newspaper, The Argus, and on the local community cable television channel. Leaflets with recruitment information were enclosed in 5,000 issues of The Chronicle Journal that was delivered in targeted areas. Recruitment information based on the recruitment poster was featured in the "Communications Bulletin" that was sent electronically to Lakehead University faculty, staff, and students. The researchers for both studies gave brief presentations in

undergraduate Psychology classes to explain their studies and recruit interested students. As well, letters (see Appendix 7) were sent to health service providers working at Lakehead Psychiatric Hospital, St. Joseph's Care Group, Thunder Bay Regional Hospital, medical clinics, and private psychological and counseling services in the city to engage their assistance in reaching the clinical population.

Individuals who responded to the recruitment efforts were contacted and informed of the study, including its objectives, the procedures, the duration of the study, and all details regarding the ethical considerations of running the project (e.g., informed consent, right to withdraw, confidentiality of data, risks and benefits of participation, data storage, opportunity to receive summary of results). Interested individuals were informed that they would need to be assessed to determine whether they met the inclusionary research criteria for the study before they could participate. Those who fulfilled the research criteria and chose to participate were given an individual appointment to meet with the researcher. The study was presented to potential participants as a research project that examines how people respond physically and psychologically to bright light intervention.

Light therapy. During their initial meeting with the researcher, participants were reminded of the details of the study, given their personal identity code number for use in the study to protect their anonymity, and given an informed consent form (see Appendix 8) to complete. They also filled out the pre-therapy (Time 1) measures relating to their energy level, carbohydrate craving, restfulness after sleep, time spent sleeping, unusual stress, food intake, expectancy regarding changes in themselves from the light intervention (see Appendix 5), and the SIGH-SAD (see Appendix 4). Possible side effects were discussed with them. They were also given appointments for bright light therapy for 14 consecutive days. All participants were

entered into three $100 random prize draws that were held at the end of the project. Introductory Psychology students additionally received 1 bonus point for participating in each week of light treatment (for a total of 2 bonus points) toward their course grade.

Participants were exposed daily for 14 days to bright light therapy via light boxes of 10,000 lux at a distance of 26 inches. A string attached to the light box ensured that the participants were seated at the correct distance. Each exposure session lasted 30 minutes and was scheduled between 8:15 a.m. and 12:45 p.m. Given that three light boxes were used simultaneously, the light therapy sessions were staggered to allow the researcher to supervise each participant individually. As much as possible each participant's sessions were spread out over the morning so that each participant had a range of early-morning and late-morning sessions. A timer was set for 30 minutes. When the timer went off, the researcher or volunteer turned off the light, thanked the participant for coming, and confirmed the next treatment session.

Participants were requested to report any side effects to the researcher. Three of them experienced side effects such as eyestrain or headache that was consistent with the literature (Kogan & Guilford, 1998). They were reminded not to look directly at the light. In all cases these side effects ceased following a minor adjustment to the position of the light box. No participant withdrew from the study because of side effects.

During the last treatment session, participants provided post-therapy (Time 2) information on their functioning by completing the same measures (see Appendix 5), excluding the expectancy scale, that they had completed prior to the commencement of their light exposure. They also completed the SIGH-SAD to assess their depressive symptoms during the previous week. On the last day of their involvement in the study, each participant was debriefed (see

Appendix 9) and given a listing of therapy/counseling resources in town to keep for their own information (see Appendix 10). Those who wished to have the results of the study were mailed an information letter.

At all times throughout the study, the project supervisor/clinical psychologist was available for follow up in the event that any participant wished to have clinical information, or required clinical intervention or referral. No follow up was necessary.

Results

Sample

Twenty-three individuals who met the research criteria agreed to take part in the study. Of these, 17 were included in the final sample. Four potential participants were excluded because their SIGH-SAD score had decreased to below 22 when they turned up for their first light therapy session. Two participants decided they were unable to commit beyond 3 days in this 14-day study. Of the final sample of 17 participants, 11 completed 14 sessions. The remaining completed 13 sessions ($n = 2$), 12 sessions ($n = 3$), or 9 sessions ($n = 1$) only because of family or study commitments and travel plans. Nevertheless, the 6 participants who did not follow the protocol through to a full 14 days were included in the final sample because previous research indicates that light therapy effects are seen within 2 to 4 days (Lam & Levitt, 1999) and benefits have been reported with treatments that last only 1 week (Lafer et al., 1994; Lam et al., 1991; Lingjaerde & Foreland, 1998). Participant responses on the stress measure pre- and post-therapy revealed that no participant experienced a major crisis during the therapy period that could have influenced their responses on the other measures.

Participants were 16 females and 1 male, ranging in age from 18 to 45 years with a mean age of 23 years ($SD = 7.33$). Their pre-therapy (Time 1) mean scores (SD) were 30.29 (8.61) on

the SIGH-SAD, 13.24 (3.90) on the GSS and 5.00 (0.90) on the expectancy scale. Mean scores (*SD*) for all other measures at pre-therapy (Time 1) and post-therapy (time 2) are presented in Table 1.

Overview of Analyses

The primary statistical technique used in the data analyses was sequential multiple regression. It allowed the examination of the relationships between predictors and post-therapy (Time 2) functioning after partialling out the expectancy score (Expect) and Time 1 functioning. Two sets of predictors were used. The first set consisted of GSS, Time 1 atypical symptom score on the SIGH-SAD (Atypical1), and Time 1 typical symptom score on the SIGH-SAD (Typical1). The second set of predictors consisted of Atypical Balance and reverse diurnal variation (Rdv). As mentioned previously, Atypical Balance = (Atypical1/ Time 1 total SIGH-SAD) X 100%. Rdv is a measure of the severity of afternoon mood slumps. It is calculated by Rdv = Time 1 SIGH-SAD Items (16 reverse-scored + 17 + 28). In both sets of regression, the criterion variables were Time 2 scores on sleep duration (SlpDuration2), sleep quality (SlpQuality2), carbohydrate craving (CarbCrave2), carbohydrate intake (CarbIntake2), energy level (Energy2), typical symptom score on the SIGH-SAD (Typical2), atypical symptom score on the SIGH-SAD (Atypical2), and total symptom score on the SIGH-SAD (TotalDep2). The effects of the treatment expectancy scores and Time 1 score of the criterion variable were partialled out at Step 1 of the sequential multiple regression before determining whether the predictors were associated with the criterion variable that was measured at Time 2.

Pre-analysis Issues

There were no missing values in the data set. Using the guideline of a *z*-score greater than $\pm$3.29 standard deviations (Tabachnick & Fidell, 2001) to identify univariate outliers, no

outliers were found on any of the predictor or criterion variables. The two sets of predictors were examined separately for multivariate outliers. None were identified through the Mahalanobis distance at $p < .001$ (Tabachnick & Fidell, 2001). No influential data points were identified either using a standard of Cook's distance > 1 (Stevens, 2002).

Predictor variables that would be involved together in the any one run of the multiple regression were checked for multicollinearity and singularity. Multicollinearity and singularity refer to situations within multiple regression in which the correlation between the variables is very high; greater than .90 for multicollinearity, and near 1.00 for singularity (Tabachnick & Fidell, 2001). In both cases the variables contain redundant information. If two such variables are used in the same analysis, they tend to weaken that analysis by inflating the size of the errors terms (Tabachnick & Fidell, 2001). Bivariate correlations are checked for multicollinearity or singularity through examination of the correlation matrix. In the present study the correlation matrix revealed a very high correlation between Typical2 and TotalDep2 ($r = .98, p < .01$; see Table 2). Therefore Typical2 was excluded as a criterion variable.

A multivariate multicollinearity or singularity is identified through a tolerance statistic that is produced by the SPSS regression program. If the tolerance level is too low (between .01 and .0001) the variable is excluded from the analysis (Tabachnick & Fidell, 2001). Tolerance levels were examined in all multiple regression analyses and no multicollinearity or singularity was found.

The data were examined for skewness and kurtosis using SPSS Version 11.0. A moderate negative skewness was found for CarbCrave1. A solution would be to perform a square root transformation on the variable (Tabachnick & Fidell, 2001). However, this was decided against for three reasons. First, transformation of the original data would result in

difficulty in interpreting the results and relating it back to the original measure. Second, CarbCrave2 did not need any transformation. Finally, the focus of the regression analysis was not on CarbCrave1. Instead it was on CarbCrave2 after partialling out CarbCrave1.

Finally, the data were examined for violations of assumptions of linearity and homoscedasticity (Tabachnick & Fidell, 2001) through the examination of the scatterplots of the residuals. The scatterplots revealed no clear patterns that would indicate nonlinearity or heteroscedasticity.

Analyses

Correlations. Bivariate correlations among all the variables in the study were carried out. Correlation results for Time 1 variables (see Table 3) reveal no significant correlation between GSS and any other Time 1 variable. Contrary to expectations, participants who had a higher GSS reported no greater severity of Atypical1 than those with a lower GSS. A negative correlation ($r = -.561, p < .05$) was found between Energy1and Expect. Individuals who felt less energetic before light therapy had higher expectations of the effects of light therapy than individuals who felt more energetic. CarbCrave1 correlated significantly with Atypical Balance ($r = .538, p < .05$). Individuals with greater carbohydrate craving had a greater percentage of atypical symptoms to total symptoms than individuals with less carbohydrate craving.

Time 2 correlation results (see Table 2) show significant correlations between SlpQuality2 on one hand, and three variables on the other: Energy2 ($r = .78, p < .01$), Typical2 ($r = -.67, p < .01$), TotalDep2 ($r = -.63, p < .01$). Following light therapy, individuals who felt more rested had more energy, lower typical depressive symptoms, and lower total depressive symptoms than individuals who felt less rested.

Correlations between predictor variables and Time 2 criterion variables (see Table 4) reveal that Expect correlated significantly with CarbCraving2 ($r = -.64, p < .01$). Individuals with lower expectations for improvement had higher carbohydrate craving following light therapy. No significant correlation was found between Atypical Balance and any Time 2 criterion variable. Rdv was significantly correlated with CarbCrave2. Typical1 correlated significantly with Atypical2 and TotalDep2. Atypical1 correlated significantly with Atypical2. None of the predictors correlated significantly with SlpQuality2 or CarbIntake2.

Implications of the correlations. Multiple regression analysis allows the researcher to determine whether an independent variable is a predictor of the criterion variable after controlling for the effect of other variables. If there is no significant correlation between a predictor and a criterion variable, the best estimate of the value of a specific criterion variable is the variable mean (Runyon & Haber, 1991). In light of this, only those predictor variables that had significant correlations with criterion variables were entered into multiple regression analyses (Reichborn-Kjennerud & Lingjaerde, 1996). Since there was no significant correlation was found between Atypical Balance and any Time 2 criterion variable (see Table 4), Atypical Balance was dropped as a predictor. None of the remaining predictors correlated significantly with SlpQuality2 or CarbIntake2. Therefore these two variables were excluded as criterion variables in subsequent analyses. Finally, it is noteworthy that GSS bore no relationship to any of the Time 1 or Time 2 variables in the study (see Tables 3 and 4). It was therefore excluded as a predictor.

Time 1 – Time 2 Differences. To determine whether there were significant differences between pre- and post-therapy measures, paired samples *t*-tests were performed on each variable at Time 1 and Time 2 (see Table 1). Given that no significant difference was found for Energy

or for SlpDuration, indicating that the research participants showed no change in either variable from pre- to post-therapy, these two variables were excluded as criterion variables from subsequent analyses. All other therapy response variables showed significant changes in the expected direction between Time 1 and Time 2. Significant improvement was found in sleep quality as well as significant reductions in carbohydrate craving, carbohydrate intake, typical symptom score, atypical symptom score, and total symptom score. These variables were therefore entered into the multiple regression analyses as criterion variables.

Multiple Regression. The criterion variables that were assessed in the final multiple regression analyses were CarbCrave2, Atypical2, and TotalDep2 (see Table 4). The predictor for CarbCrave2 was Rdv. Typical1 served as predictor for Atypical2. Typical1 and Atypical1 both acted as predictors for TotalDep2.

For the regressions on CarbCrave2 and Atypical2, treatment expectancy scores and the corresponding Time 1 score of the criterion variable were entered at step 1. For the regression on TotalDep2, only Expect was entered at Step 1. The corresponding pre-therapy TotalDep1 was not partialled out at step 1 because of its very high association with the predictors, Typical1 and Atypical1, in the same regression. Note that TotalDep1 is derived by summing Typical1 and Atypical1.

Before the regression analyses were conducted, the three criterion variables (CarbCrave2, Atypical2, and TotalDep2) were examined for their interrelationships. It was found that Atypical2 and TotalDep2 were significantly correlated with each other (see Table 2). To keep Type 1 error rate at .05, the Bonferroni-type split approach was adopted: the regression involving these two criterion variables were each assessed at $\alpha = .025$. Given the absence of significant

correlation between CarbCrave2 and the other two criterion variables (see Table 2), the regression involving CarbCrave2 was assessed at $\alpha = .05$.

A sequential multiple regression analysis was run on CarbCrave2 as the criterion variable. Expect and CarbCrave1 were entered at Step 1. The result was significant, $F(2, 14) = 6.57, p < .01$, with Expect as a significant predictor. Participants with higher expectations for improvement reported less carbohydrate craving following light therapy. Rdv was entered at Step 2. No further predictive value was found, $\Delta R^2 = .13$, $\Delta F(1, 13) = 4.51$, n.s. (see Table 5).

A sequential multiple regression analysis was run on Atypical2 as the criterion variable. Expect and Atypical1 were entered at Step 1. The result was significant, $F(2, 14) = 8.08, p < .01$, with Atypical1 as a significant predictor. Participants with higher atypical symptom score before light therapy had higher atypical symptom score following light therapy. Typical1 was entered at Step 2. Results were significant, $\Delta R^2 = .18$, $\Delta F(1, 13) = 7.95, p < .025$, with Typical1 as a significant predictor (see Table 6). Atypical1 was no longer a significant predictor ($\beta = .45$, n.s.). Higher typical symptom score before light therapy predicted higher atypical symptom score following light therapy.

A sequential multiple regression analysis was run on TotalDep2 as the criterion variable. Expect was entered at Step 1. Results were not significant. Typical1 and Atypical1 were entered at Step 2. Results were significant, $\Delta R^2 = .59$, $\Delta F(2, 13) = 11.10, p < .01$, with Typical1 as a significant predictor (see Table 7). Higher typical symptom score before light therapy predicted higher total symptom score following light therapy.

Discussion

Study Objective

The objective of the present study was to investigate the seasonality and depression predictors of response to 2 weeks of bright light therapy at 10,000 lux after controlling for treatment expectancy and pre-therapy functioning. Predictors were GSS, atypical symptom score on the SIGH-SAD, typical symptom score on the SIGH-SAD, atypical balance, and reverse diurnal variation. Response to light therapy was measured by the participants' functioning in the following domain after light therapy – atypical depressive symptom score on the SIGH-SAD, typical depressive symptom score on the SIGH-SAD, total depressive symptom score on the SIGH-SAD, sleep duration, sleep quality, carbohydrate craving, carbohydrate intake, and energy level.

Findings

Participants in the present study reported significant changes in their functioning on several indices after light therapy. Specifically, they experienced improvement in sleep quality, decrease in carbohydrate craving and carbohydrate intake, and lower total depression, atypical depressive symptoms, and typical depressive symptoms. However, they experienced no difference in their energy level or sleep duration. This contradicts previous findings from Avery, Kizer et al. (2001) who reported significant improvement in energy levels in individuals with subsyndronomal SAD following both morning and evening light treatment. In that study mean energy scores (*SD*) at baseline were 17.0 (33) for the morning light group and 32.0 (29) for the evening light group. These scores are substantially lower than the baseline energy scores in the present study, where the mean (*SD*) was 44.65 (19.54). The study by Avery, Kizer et al. (2001) used a VAS with the typical winter level of the item at one end of the line and the typical

summer level at the other end (p. 269). In the present study participants were asked to rate how energetic they felt with one end of the line representing "not at all energetic" and the other end "extremely energetic". The difference in baseline energy level and the different method of recording energy levels could account for the discrepancy in findings between the two studies.

The first hypothesis in the study which stated that GSS (measure of seasonality) and pre-therapy atypical depressive symptoms would be positively correlated was not borne out by the findings. Neither was the hypothesis that the GSS would predict lower total depression scores corroborated. In fact, the results showed that GSS was not correlated with any other variable, including time 1 measures or time 2 measures. This finding supports the results of Reichborn-Kjennerud and Lingjaerde (1996) who found no statistical significant correlation between GSS and treatment outcome as a continuous variable following 6 days of light therapy, and no difference in GSS between responders and non-responders. Avery, Kizer et al. (2001) found a significant correlation between GSS and screening SIGH-SAD score, but like other researchers, reported that the GSS did not correlate with response to light therapy. The results of the present study support the general conclusion in the literature that GSS is not predictive of response to light therapy.

The hypothesis that higher atypical symptom score at baseline would predict lower SIGH-SAD scores, lower atypical symptom scores, shorter sleep duration, decreased carbohydrate craving, and increased energy was not supported. This is congruent with two previous studies. Lam et al. (2001) found that atypical symptoms did not predict response rates. As well, Reichborn-Kjennerud and Lingjaerde (1996) found no correlation between baseline atypical score and percentage reduction in total score following light therapy. However, other studies have found that individuals with more severe atypical symptoms showed greater

improvement in atypical symptom score (Stinson & Thompson, 1990), total symptom score (Nagayama et al., 1991), or both atypical and total symptom score (Oren et al., 1992).

The present study found typical symptom score and expectancy score to be specific predictors of response to light therapy. Higher typical symptom score before light therapy predicted higher atypical symptom score and higher total symptom score following light therapy. Participants with higher expectations for improvement reported less carbohydrate craving following light therapy.

Other studies have found that typical symptom score predicted poorer response to light therapy. Terman et al. (1996) found that nonresponders had more melancholic symptoms than responders. Stinson and Thompson (1990) found that greater severity of typical symptoms predicted worse response to treatment, while patients with more severe atypical symptoms were more likely to respond. Yerevanian et al. (1986) found that none of the 8 nonseasonal patients with higher baseline depression scores met any of the criteria for recovery while all 9 seasonally depressed patients, with lower baseline depression scores, met three criteria for recovery.

In the present study the ratio of atypical symptoms to total symptoms, atypical balance, was not a significant predictor of response to light therapy. Terman et al. (1996) found higher atypical balance to be the sole significant predictor of greater percentage of improvement in total SIGH-SAD score following light therapy. Meesters et al. (1993) found that greater atypical balance predicted less favourable response on typical symptoms, but not total symptoms or atypical symptoms. Reichborn-Kjennerud and Lingjaerde (1996) found no correlation between atypical balance and total symptom response. Geerts et al. (2000) found no relation between change in BDI score and baseline typical/atypical ratio.

Reverse diurnal variation was not found to predict response to light therapy. Reverse diurnal variation represents the extent to which individuals experience lower mood in the afternoon as opposed to in the morning. Afternoon mood slumps are more common in SAD (Terman et al., 1996) than in typical depression that is characterized by positive diurnal variation (i.e. low mood in the morning with improvement during the day). Terman et al. (1996) found reverse diurnal variation to be predominate among responders to light therapy with a moderate effect size ($d = 0.51$), while nonresponders felt worse in the morning ($d = -0.67$). Meesters et al (1993) found that SAD patients with greater daily variability of mood were less likely to respond to light therapy. That study did not distinguish between patients with positive diurnal variation and reverse diurnal variation.

Placebo effect

The unexpected finding of expectancy score as a significant predictor of carbohydrate craving following light therapy demands a closer look at placebo effects and expectancy response in seasonal depression studies. The phrase "placebo effect" has been used to describe "phenomena as different as patients' improvement after a placebo intervention, the effect of a placebo intervention, psychologically mediated effects in general, the effect of the patient-provider interaction, the effect of suggestion, the effect of expectancies, and the effect of patients' experience of meaning, etc." (Hrobjartsson & Gotzsche, 2001, p. 312).

Placebo effect varies throughout studies depending on a variety of factors including the disorder that is being treated, the type of placebo used, and the strength of the intervention that is being tested. Placebo injections are more effective than placebo pills; placebo morphine is more effective than placebo aspirin. Kirsch and Scoboria (2001) suggest that the fact that placebo response varies is in itself proof that a placebo response actually exists. "One placebo cannot be

more effective than another unless placebos are capable to producing an effect" (p. 307). Kirsch and Sapirstein (1999) found that on average only 25% of the response in studies of antidepressants was due to the active components of the drugs, 50% was due to placebo effect and 25% was due to other nonspecific factors.

In light therapy studies it is difficult to find a credible placebo that is similar to the light therapy and that is perceived to be as effective as the light therapy. Many bright light therapy studies use dim light as a placebo control and in most of these studies patients have higher expectations for the treatment condition than for the control condition (Eastman, 2001).

Terman, Terman, and Ross (1998) found that patients in the bright light therapy group had higher expectations than those in the control group who were treated with negative ions. However in that study 50% of patients treated with high-density negative ions met the response criteria compared to 71% for morning light and 67% for evening light. Eastman et al (1992, 1998) used deactivated negative ion generators as the placebo control. In the first study (Eastman et al., 1992) patients had higher expectations for the treatment condition than for the placebo condition. For the later study the researchers successfully increased expectations for the negative ion generators by making them more impressive and by ensuring that the staff were equally enthusiastic about both treatments (Eastman, 2001). In that study a significant difference between light treatment and placebo did not emerge until the third week of treatment (Eastman et al., 1998).

Expectancy response

Because of the difficulty in finding a plausible placebo for light therapy, many studies measure expectations for treatment (Bauer et al., 1994; Kripke et al, 1992; Sack et al., 1990; Terman et al., 1996; Terman et al., 1998; Wileman et al., 2001; Wirz-Justice et al., 1993).

Measures used included questionnaires asking how logical, useful, successful participants thought the light therapy would be and whether they would recommend it to others (Wileman et al., 2001). Likert scales are commonly used to measure response to expectation questions (e.g. Bauer et al., 1994). In most of these studies no correlation was found between expectations and response (Bauer et al., 1994; Eastman et al., 1992; Kripke et al, 1992; Sack et al., 1990; Terman et al., 1998; Wileman et al., 2001; Wirz-Justice et al., 1993). Terman et al. (1996), however, found that responders to light therapy had higher expectations than non-responders.

Expectancy response is one aspect of placebo effect. Expectations of mood have been found to predict mood (Catanzaro & Mearns, 1999) and to have a causal effect on mood (Catanzaro, 1989). Individuals who predicted that they would enjoy an experimental task set lower goals for measuring their own success than those who predicted that they would find the task difficult. These more optimistic individuals changed their behaviour in such a way that their prediction came true. It is possible that participants in the present study who felt their mood would improve following light therapy changed their behaviour in a similar manner so that they did indeed feel better.

This self-confirming aspect of response expectancies is examined by Kirsch (1999). Expectations affect a person's perception of a stimulus, especially if that stimulus is ambiguous one, such as an internal state. Carbohydrate craving could be one of the internal states to which Kirsch (1999) refers. Individuals who anticipate that bright light therapy will reduce their craving for carbohydrates may change their eating behavior so that their prediction will come true. The expectation may increase a participant's motivation and enable her to change her eating behavior. This behavioral change may then be interpreted as a decrease in carbohydrate craving. Rosenthal, Genhart, Jacobsen et al. (1987) found that carbohydrate craving assessed by

the atypical addendum to the HDRS responded most rapidly and dramatically to light treatment. However a study by Krauchi et al. (1990), that had participants prospectively fill out a daily food diary, found that improvement in carbohydrate intake was delayed when compared to the other atypical symptoms. The relationship between carbohydrate craving, carbohydrate intake, and expectations for light therapy may be quite complex and is deserving of further research.

Strengths and Limitations

The major limitation of the present study is the small number of participants and the composition of the sample. The participants in the present study were not formally assessed for clinical depression. The objective of the study was to examine the severity of depressive symptoms as a predictor of response to light therapy. Since clinical status was not evaluated, participants were mainly students who were experiencing a subclinical episode rather than patients with major depressive disorder. Attempts were made to recruit individuals from the community who were experiencing seasonal depression. Flyers were placed in the local paper, letters were sent to health service providers, and notices were posted in health food stores, malls, and other high volume locations. These notices generated a great deal of interest but very few participants, because most interested community residents were taking antidepressants and were therefore ineligible to participate in the study. Community residents and students who met the diagnosis for major depressive episode were usually directed into another ongoing research program in the Psychology department. Several of these individuals were asked if they would like to participate in a second study, but all refused.

Several potential participants who obtained a depression score of over 22 during the initial screening procedure, scored much lower when they were brought in for the first light therapy session. These individuals were turned away and did not take part in the study. Those

who participated in the study maintained their elevated depression scores from screening to the first light therapy session, and were not having a mere transient negative mood. This increases the validity of the study.

Seventeen participants is not an unusually small number in these types of studies. Several published studies of light therapy have had 17 or fewer participants per group (Bauer et al., 1994; Deltito et al., 1991; Ghadirian, et al., 1998; Kripke et al., 1992; Lam et al., 1991; Postolache et al., 1998; Stewart et al. 1990; Stinson & Thompson, 1990; Yerevanian et al., 1986). However a larger number of participants would have given the present study more power in its data analyses.

The entry criteria for the present study were similar to those for a study of subsyndromal SAD (Avery, Kizer et al., 2001). Those participants were required to have a minimum SIGH-SAD score of 12, a minimum GSS of 6 and no major depression. Participants in that study were similar in many respects to participants in the present study. In that study GSS ranged from 6 to 17 with a mean of 11.0 (SD = 2.85) and mean total SIGH-SAD score at baseline was 23.5 (SD = 6.4). In the present study GSS ranged from 6 to 19 with a mean of 13.24 (SD = 3.90). Participants in the present study had more severe depressive symptoms with mean total SIGH-SAD score at baseline of 30.29 (SD = 8.61). In contrast Levitt, Lam, and Levitan (2002) used DSM-IV criteria for minor depression when recruiting their subsyndromal participants.

Another strength of the present study was the number of sessions of light therapy that participants received. Many previous studies used a shorter treatment protocol, often 5 to 7 days (e.g. Lafer et al., 1994; Lingjaerde & Foreland, 1998). Longer treatment of 2 or more weeks has provided superior results (Bauer et al., 1994; Lam et al., 2000). The 14 sessions in the present

study made recruitment of participants more difficult, but allowed a better opportunity for treatment response.

A further strength of the present study was the multiple criterion variables that were measured. Most previous studies only looked at responses on depression measures – typical, atypical, and total symptoms – following light therapy. The present study also assessed the effects of bright light therapy on energy level, carbohydrate craving and intake, and sleep duration and quality. These items reflect the atypical symptoms that are characteristic of SAD. By assessing changes in these individual atypical symptoms, rather than looking only at an atypical symptom score, we can determine more precisely the effects of light therapy. Previous studies have found that improvement in carbohydrate craving was delayed compared to improvement in other atypical symptoms (Krauchi et al., 1990), occurred earlier than improvement in other symptoms (Rosenthal, Genhart, Jacobsen et al., 1987), and was more dramatic than improvement in other symptoms (Rosenthal, Genhart, Jacobsen et al., 1987). The present study found that improvement in carbohydrate craving was predicted by greater expectations of improvement. Clearly more research in this area is needed.

Summary and Conclusions

After completion of 14 days of bright light therapy participants reported a mean significant increase in restfulness and significant decrease in carbohydrate craving, carbohydrate consumption, typical symptom score, atypical symptom score, and total depression score. GSS was not found to significantly predict any change in any symptom following light therapy. The present study found typical symptom score and expectations to be specific predictors of response to light therapy. Higher typical symptom score before light therapy predicted higher atypical

symptom score and higher total symptom score following light therapy. Participants with higher expectations for improvement reported less carbohydrate craving following light therapy.

The present study provided support for previous studies that found that individuals with more severe typical symptoms have a poorer response to bright light therapy. Findings of the present study underscore the need for placebo controls as greater expectations for improvement were found to predict less carbohydrate craving following light therapy.

Recommendations

Further research is needed to determine which patient characteristics predict response to light therapy. This issue is not yet resolved, as various studies have had equivocal results. Some studies have found that individuals with more severe typical symptoms have a poorer response to light therapy. Several studies have found that some aspect of atypical symptoms – either severity of atypical symptoms or atypical balance – is predictive of response to light therapy. Future studies could examine which atypical symptoms are predictive of response to light therapy.

Future studies need to have credible placebo controls and to ensure that expectations are equivalent in both treatment and control groups. Only one study achieved the goal of equivalent expectations for treatment and placebo and found a superior result for bright light therapy only after three weeks of treatment (Eastman 2001).

Future researchers may choose to look at specific symptoms and the effect of expectations on those symptoms. Findings of the present study suggest that the relationship between expectations for improvement and carbohydrate craving and consumption may be quite complex. The question about expectations in the present study was very general and could have referred to any symptom the participants were experiencing. By asking questions about

expectations for specific symptom improvement researchers may be able to determine whether certain symptoms are more responsive to expectation effects.

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Table 1

Time 1 and Time 2 Symptom Scores and t-test Results

Variable	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)	*t*(16)	Time 1 – Time 2 *M* (*SD*)
Energy (mm)	44.65 (19.54)	56.94 (18.63)	-1.72	-12.29 (29.48)
CarbCrave (mm)	69.82 (24.06)	49.59 (20.68)	2.97**	20.23 (28.14)
SlpQuality (mm)	34.29 (18.51)	53.59 (19.95)	-2.96**	-19.29 (25.43)
SlpDuration (mm)	486.35 (121.17)	487.65 (82.41)	-0.05	-1.29 (109.44)
CarbIntake (number of servings)	10.18 (5.46)	6.24 (2.99)	4.07**	3.94 (3.99)
Typical score	20.47 (5.86)	15.65 (12.19)	2.27*	4.82 (8.76)
Atypical score	9.82 (4.17)	5.35 (4.36)	4.97**	4.47 (3.71)
TotalDep score	30.29 (8.61)	21.00 (15.76)	3.52*	9.29 (10.88)

* $p < .05$, ** $p < .01$.

Table 2

Correlations for Time 2 Variables

Time 2 Variable	1	2	3	4	5	6	7	8
1. Energy2	-	.16	.78*	.01	-.10	-.41	-.16	-.36
2. CarbCrave2		-	-.23	-.23	.31	.23	.34	.27
3. SlpQuality2			-	.18	-.20	-.67*	-.39	-.63*
4. SlpDuration2				-	-.16	-.22	.25	-.10
5. CarbIntake2					-	.21	.32	.25
6. Typical2						-	.76	.98*
7. Atypical2							-	.86*
8. TotalDep2								-

* $p < .01$.

Table 3

Correlations for Time 1 Variables

Time 1 Variable	1	2	3	4	5	6	7	8	9	10	11
1. GSS	-	.18	.21	.22	.00	.38	.05	.17	.40	.44	.26
2. Expect		-	-.56*	.08	-.28	.24	.00	-.11	.14	.21	.21
3. Energy1			-	.26	.16	-.14	.04	-.25	.02	.24	-.20
4. CarbCrave1				-	-.30	-.14	.44	-.23	.36	.54*	-.12
5. SlpQuality1					-	.09	-.01	.03	-.28	-.29	.08
6. SlpDuration1						-	-.22	.32	.32	.05	.00
7. CarbIntake1							-	.00	.44	.48	.06
8. Typical1								-	.46	-.20	-.19
9. Atypical1									-	.76**	-.08
10. Atypical Balance										-	.11
11. Rdv											-

* $p < .05$, ** $p < .01$.

Table 4

Correlations for Predictor Variables and Time 2 Criterion Variables

	Predictor Variable					
Criterion Variable	GSS	Expect	Typical1	Atypical1	Atypical Balance	Rdv
CarbCrave2	-.28	-.64**	-.02	-.18	-.24	-.52*
SlpQuality2	-.05	.26	-.30	-.20	-.01	.34
CarbIntake2	.15	-.27	.00	.42	.44	-.01
Atypical2	.19	-.29	.72**	.62**	.11	-.48
TotalDep2	.14	-.25	.78**	.46	-.07	-.34

* $p < .05$, ** $p < .01$.

Table 5

Sequential Multiple Regression Analysis Predicting Carbohydrate Craving at Time 2 (CarbCrave2)

Step	Predictor variable	*B*	*SEB*	*β*	R^2
Step 1					.41
	Expect	-14.67	4.26	-.66*	
	CarbCrave1	.23	.17	.27	
Step 2					.53
	Rdv	-3.43	1.61	-.38	

* $p < .01$.

Table 6

Sequential Multiple Regression Analysis Predicting Atypical Score at Time 2 (Atypical2)

Step	Predictor variable	*B*	*SEB*	β	R^2
Step 1					.47
	Expect	-1.81	.86	-.39	
	Atypical1	.71	.19	.68**	
Step 2					.65
	Typical1	.36	.13	.48*	

* $p < .025$, ** $p < .01$.

Table 7

Sequential Multiple Regression Analysis Predicting Total Depression Score at Time 2 (TotalDep2)

Step	Predictor variable	*B*	*SEB*	β	R^2
Step 1					.00
	Expect	-4.29	4.21	-.25	
Step 2					.58
	Typical1	1.79	.50	.67*	
	Atypical1	.71	.72	.19	

* $p < .01$.

Appendix 1

DSM-IV Seasonal Pattern Specifier

Specify if:

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

A. There has been a regular temporal relationship between the onset of Major Depressive Episodes in Bipolar I or Bipolar II Disorder or Major Depressive Disorder, Recurrent, and a particular time of the year (e.g., regular appearance of the Major Depressive Episode in the fall or winter).

   Note: Do not include cases in which there is an obvious effect of seasonal- related psychosocial stressors (e.g., regularly being unemployed every winter).

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

C. In the last 2 years, two Major Depressive Episodes have occurred that demonstrated the temporal seasonal relationships defined in Criteria A and B, and no nonseasonal Major Depressive Episodes have occurred during that same period.

D. Seasonal Major Depressive Episodes (as described above) substantially outnumber the nonseasonal Major Depressive Episodes that may have occurred over the individual's lifetime.

Appendix 2

DSM-III-R Criteria for Seasonal Pattern

A. There has been a regular temporal relationship between the onset of an episode of Bipolar Disorder (including Bipolar Disorder NOS) or Recurrent Major Depression (including Depressive Disorder NOS) and a particular 60-day period of the year (e.g., regular appearance of depression between the beginning of October and the end of November).

Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors, e.g., regularly being unemployed every winter.

B. Full remissions (or a change from depression to mania or hypomania) also occurred within a particular 60-day period of the year (e.g., depression disappears from mid-February to mid-April).

C. There have been at least three episodes of mood disturbance in three separate years that demonstrated the temporal seasonal relationship defined in A and B; at least two of the years were consecutive.

D. Seasonal episodes of mood disturbance, as described above, outnumbered any nonseasonal episodes of such disturbance that may have occurred by more than three to one.

Appendix 3

Rosenthal Criteria for Seasonal Affective Disorder

1) A history of major affective disorder according to the Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978);

2) At least two consecutive years in which the depressions have developed during fall or winter and remitted by the following spring or summer (a history of this pattern changing with changes in latitude or climate would strengthen the diagnosis);

3) Absence of any other axis I psychiatric diagnosis;

4) Absence of any clear-cut seasonally changing psychosocial variables that would account for the seasonal variability in mood and behavior, eg., work stresses.

Appendix 4

RESEARCH QUESTIONNAIRE (SAD 2001-2002)

Section A: ***This section asks for your demographic information. This is for statistical purposes so that we may know the composition of the people in the project.***

Age: _____ Sex: Male / Female Program Year: _____

Marital Status: Single / Common-law / Married / Divorced / Separated / Widowed

Ethnicity, check one:

___ Aboriginal
___ White, not of Hispanic origin (origins in Europe, North Africa, Middle East)
___ Black, not of Hispanic origin (origins in Africa)
___ Asian/Pacific Islander (origins in Far East, Southeast Asia, India Subcontinent, Pacific Islands)
___ Latino or Hispanic (Mexican, Puerto Rican, Cuban, Central or South America, or other Spanish culture or origin)
___ Other, please specify _____

Place of birth (city, country): _____

Place of permanent residence: _____

How long have you lived at your permanent address: ___ years and ___ months

Where do you spend your summer? _____

Are you currently using prescribed medication and/or over-the-counter drugs and supplements (e.g., St. John's Wort)? Yes / No

- if yes, what are they and for what condition?

Do you use alcohol on a regular basis? Yes / No

- if yes, how often do you use alcohol? _____

Do you use mood-altering drugs on a regular basis? Yes / No

- if yes, *what drug* and how *often*? _____

Please list all prescribed medication, over-the-counter drugs, and *supplements (e.g., St. John's Wort)* that you have had in the last 8 weeks:

If you are taking antidepressant medication, we are interested in knowing whether or not you experience any changes in your vision after you started taking your medication. Please circle the number on the rating scales below that best describes your visual experience:

Colour appearance

1	2	3	4	5
Faded colour Washed out, Dim		No change		Deeper colour Brighter, Richer

Light/dark contrast

1	2	3	4	5
Low contrast, Low acuity, Less detail, Hazy		No change		High contrast High acuity, Greater detail, Sharp

Do you have any eye diseases such as optic neuritis, retinitis pigmentosa, macular degeneration, glaucoma, detached retina, amblyopia (lazy eye), tunnel vision, cataracts, keratitis, uveitis (eye inflammation)? Yes / No

Do you have any systemic illnesses that affect the retina such as diabetes mellitus or system lupus erythematosis? Yes / No

Do you have any illnesses for which exposure to bright light is contraindicated such as skin cancer? Yes / No

Have you ever had bright light therapy before? Yes / No

- if yes, for how long?
- If yes,when was the last time you had the light therapy?

Do you need corrective visual aids? Yes / No

When was the last time you had an eye examination?

Section B: ***The purpose of this form is to find out if and how your mood and behavior change over time. Please fill in all the relevant circles. Note: We are interested in <u>your</u> experience, <u>not</u> <u>others</u> you may have observed.***

1. In the following questions, fill in circles for all applicable months. This may be a single month ●, a cluster of months, e.g., ● ● ●, or any other grouping. At what time of the year do you...

	J	F	M	A	M	JN	JL	A	S	O	N	D	No particular month stands out as extreme
A. Feel best	0	0	0	0	0	0	0	0	0	0	0	0	0
B. Tend to gain most weight	0	0	0	0	0	0	0	0	0	0	0	0	0
C. Eat most	0	0	0	0	0	0	0	0	0	0	0	0	0
D. Sleep least	0	0	0	0	0	0	0	0	0	0	0	0	0
E. Feel most energetic	0	0	0	0	0	0	0	0	0	0	0	0	0
F. Socialize least	0	0	0	0	0	0	0	0	0	0	0	0	0
G. Crave carbohydrates most	0	0	0	0	0	0	0	0	0	0	0	0	0
H. Feel worst	0	0	0	0	0	0	0	0	0	0	0	0	0
I. Eat least	0	0	0	0	0	0	0	0	0	0	0	0	0
J. Sleep most	0	0	0	0	0	0	0	0	0	0	0	0	0
K. Lose most weight	0	0	0	0	0	0	0	0	0	0	0	0	0
L. Crave carbohydrates least	0	0	0	0	0	0	0	0	0	0	0	0	0
M. Feel least energetic	0	0	0	0	0	0	0	0	0	0	0	0	0
N. Socialize the most	0	0	0	0	0	0	0	0	0	0	0	0	0

2. Please check the year(s) in the past 6 years which had the same pattern as above:
   _ Sept.99/Aug.00 _ Sept.98/Aug.99 _ Sept.97/Aug.98
   _ Sept.96/Aug.97 _ Sept.95/Aug.96 _ Sept.94/Aug.95

3. (a) Please check the year(s) in the past 6 years which DID NOT have the same pattern as above:
   _ Sept.99/Aug.00 _ Sept.98/Aug.99 _ Sept.97/Aug.98
   _ Sept.96/Aug.97 _ Sept.95/Aug.96 _ Sept.94/Aug.95

   (b) Please specify how these years marked in 3(a) above differed:

4. To what degree do you change with the seasons on the following? (Circle only one answer per item)

	No Change	Slight Change	Moderate Change	Marked Change	Extremely Marked Change
A. Sleep length	0	1	2	3	4
B. Social activity	0	1	2	3	4
C. Mood (overall feeling of well being)	0	1	2	3	4
D. Weight	0	1	2	3	4
E. Appetite	0	1	2	3	4
F. Energy level	0	1	2	3	4

5. If your experiences in question 4 changes with the seasons, do you feel that they are a problem for you? Yes / No
   - If yes, is this problem:
   __ mild __ moderate __ marked __ severe __ disabling

6. Do you experience any regular occurring, seasonally linked stressors in your life, for example, seasonal unemployment, anniversary of the death of a loved one, etc.? Yes / No
   If yes, please specify what the stressor is and the months you experience it: _____

   _____

7. Is starting school a seasonal stressor for you? Yes / No
   If yes, when does it become a stressor for you? (specify the months): _____

8. By how much does your weight fluctuate during the course of the year?
   _ 0-3 lbs _ 4-7 lbs
   _ 8-11 lbs _ 12-15 lbs
   _ 16-20 lbs _ over 20 lbs

9. Approximately how many hours of each 24-hour day do you sleep during each season, including naps? (Circle only one answer per question)
   WINTER (Dec 21-Mar 20)
   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 18+
   SPRING (Mar 21-June 20)
   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 18+
   SUMMER (June 21-Sept 20)
   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 18+
   FALL (Sept 21-Dec 20)
   1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 18+

10. Using the scale below, indicate how the following weather changes make you feel (fill in only one circle per question):

-3 = in very low spirits or markedly slowed down
-2= moderately low/slowed down
-1= mildly/slowed down
0= no effect
+1= slightly improves your mood or energy level
+2= moderately improves your mood or energy level
+3= markedly improves your mood or energy level

	-3	-2	-1	0	+1	+2	+3	Don't know
A. Cold weather	0	0	0	0	0	0	0	0
B. Hot weather	0	0	0	0	0	0	0	0
C. Humid weather	0	0	0	0	0	0	0	0
D. Sunny weather	0	0	0	0	0	0	0	0
E. Dry weather	0	0	0	0	0	0	0	0
F. Grey and cloudy	0	0	0	0	0	0	0	0
G. Long days	0	0	0	0	0	0	0	0
H. High pollen	0	0	0	0	0	0	0	0
I. Foggy and smoggy	0	0	0	0	0	0	0	0
J. Short days	0	0	0	0	0	0	0	0

11. Do you notice a change in food preference during the different seasons, for example a preference for salts, sweets, fats, or carbohydrates? Yes / No

- If yes, please specify the type of craving and the months they typically occur in:

_____

_____

12. Do you believe you have the seasonal blues (ie. periods of feeling down, or blue, that are linked to specific seasons)? Yes / No

13. If you answered "yes" to question 12, please continue with the items below:

- Please specify the months you are typically blue in: _____
- How old were you when you started having the seasonal blues? _____
- Counting only the years from when you started having the seasonal blues until now, what proportion of the years would you say you have the seasonal blues? _____
- How do you know that you have the seasonal blues? What changes, if any, do you notice occurring in yourself, emotionally, psychologically, mentally, and physically?

_____

_____

_____

- Do you think you are having the seasonal blues NOW? Yes / No
- If you are not having the seasonal blues now, when do you think it will start this year? _____

Section C: ***Compared to how you feel when you are in an even or normal mood state, how would you rate yourself on the following items during the past 2 weeks?***

I have been feeling	Not at all 0	Just a little 1	More than just a little 2	Quite a bit, moderately 3	Marked or severely 4
1. down and depressed	0	1	2	3	4
2. less interested in doing things	0	1	2	3	4
3. less interested in sex	0	1	2	3	4
4. less interested in eating	0	1	2	3	4
5. that I've lost some weight	0	1	2	3	4
6. that I can't fall asleep at night	0	1	2	3	4
7. that my sleep is restless	0	1	2	3	4
8. that I wake up too early	0	1	2	3	4
9. heavy in my limbs or aches in back, muscles, or head, more tired than usual	0	1	2	3	4
10. guilty or like a failure	0	1	2	3	4
11. wishing for death or suicidal	0	1	2	3	4
12. tense, irritable, or worried	0	1	2	3	4
13. sure I'm ill or have a disease	0	1	2	3	4
14. that my speech and thought are slow	0	1	2	3	4
15. fidgety, restless, or antsy	0	1	2	3	4
16. that morning is worse than evening	0	1	2	3	4
17. that evening is worse than morning	0	1	2	3	4
18. unreal or in a dream state	0	1	2	3	4
19. suspicious of people/paranoid	0	1	2	3	4
20. preoccupied/obsessed that I must check things a lot	0	1	2	3	4
21. physical symptoms when worried	0	1	2	3	4
22. like socializing less	0	1	2	3	4
23. that I have gained weight	0	1	2	3	4
24. that I WANT to eat more than usual	0	1	2	3	4
25. that I HAVE eaten more than usual	0	1	2	3	4
26. that I crave sweets and starches	0	1	2	3	4
27. that I sleep more than usual	0	1	2	3	4
28. that my mood slumps in the afternoons or evenings	0	1	2	3	4

Please do not write below this line

Score (1-21)
Supplemental Score (22-28)

Appendix 5

Code #: _____ **Date:** _____

Pre- and Post-Light Intervention Measures

Section A: Below are two questions about your energy level and carbohydrate craving. Each question is followed by a horizontal line. Anchored at the left end of the line is the total absence of the feature we are measuring. The right end of the line denotes an extreme amount of the feature. Please read each question carefully and put a cross (i.e., "X") on the horizontal line following the question, specifically at the point that you think best reflects your answer.

On the average, how energetic have you felt **during the past week?**

not at all energetic	extremely energetic

On the average, how much have you craved carbohydrates (candy, chocolate, bread, pasta, etc.) **in the past week?**

no carbohydrate craving	extreme craving

****

Section B: Below is a question about your assessment of restfulness after sleep. Please put a cross (i.e., "X") on the horizontal line following the question, specifically at the point that you think best reflects your answer.

On the average, how rested have you felt when you woke up in the morning **during the past week?**

not at all rested	extremely well rested

Please tell us how you have been sleeping during the past week.

What time did you usually get into bed? _____

Approximately at what time did you fall asleep? _____

How many times did you wake up on average? _____

If you did get wake up at night, approximately how long were you up each time? _____

What time did you usually get up in the morning? _____

How many daytime naps did you take each day? _____

How long was each nap? _____

Section C: Have you experienced any unusual stress in the past week (e.g. failed exam, illness in the family, broke up with boyfriend/girlfriend, etc.)? If yes, please specify.

Section D: How do you think treatment with bright light will make you feel? (Circle one answer only)

1	2	3	4	5	6	7
Much worse	Worse	Slightly worse	No change	Slightly better	Better	Much better

Section D: We would like to know what kinds of foods you ate LAST WEEK. Please tick off in the appropriate column to indicate <u>how often per day on average</u> you have consumed the following foods in the last week::

Types of Foods	Not at all	On the average, once (1) per day	On the average, twice (2) per day	On the average, three (3) times per day	On the average, four or more (4+) times per day
1. Breads, bagels, rolls, etc					
2. Milk					
3. Coffee, tea, cola					
4. Fruit					
5. Fruit juice					
6. Cheese, yogurt					
7 . Jam, honey, sugar					
8. Butter, margarine					
9. Peanut butter					
10. Potatoes, yams, corn					
11. Rice, pasta, cereal					
12. Beef, pork					
13. Salad dressing, oil					
14. Beans, eggs, tofu					
15. Chicken, fish					
16. Cooked vegetables					
17. Raw vegetables, salad					
18. Chocolate					
19. Ice cream					
20. Cake, cookies, pastry					
21. Beer or wine					
22. Liquor (rye, vodka, etc.)					
23. Pop (not diet, not cola)					
24. Candies					
25. Potato chips, tortilla chips					
Other (please specify and rate individually):					

Appendix 6

Note: This poster will carry the Lakehead University logo.

LU LOGO Lakehead University

DEPRESSION AND WINTER BLUES STUDIES

The Department of Psychology, Lakehead University is looking for volunteers (ages 18-55) with depression and winter blues to participate in one or more of 3 studies on the psychological and behavioural characteristics, and light treatment for depression and winter blues. Three prizes of $100 within each project will be awarded to participants selected in random draws.

For more information, call Rob in the Vision Lab at

(807) 346-7756

Appendix 8

INFORMED CONSENT FORM

I, _____, the undersigned, hereby consent to participate in the research project entitled "Effects of Bright Light Intervention" conducted at Lakehead University by Miriam Ketonen under the supervision of Dr. Josephine Tan. The procedure for this project has been described to me and is outlined below:

1. For 14 consecutive days I will come in for bright light intervention for 30 minutes per session. On the first day I will rate myself on my average daily sleep, energy, carbohydrate craving, and food intake during the previous week. I will also complete a scale concerning my expectation of light treatment. On the last day I will again complete the average daily ratings of sleep, energy, carbohydrate craving and food intake. I will also complete a self-report mood scale.

2. My responses to the measures will be kept confidential. I am free to withdraw from the study at any time without explanation or penalty.

3. I understand that there may be no direct benefit to me as a result of participating this study. There is no psychological risk as a result of participating in this study. Some but not all individuals may experience some minor physical discomfort. These feelings will be monitored by the researcher. Should the discomfort prove to be distressing to me, I am able to terminate cease my participation at any time I choose to do so.

4. The data will be stored in secure and confidential storage in Dr. Tan's lab at Lakehead University for a period of 7 years. After that, the data will be destroyed.

5. In return for my participation, I will be given 2 bonus points where 1 point will be given for each week of my participation in the study. I will also be entered into 3 random draws of $100 each.

6. If I am an Introductory Psychology student, I will receive 2 bonus points for participation.

7. A summary of results will be available from the researcher upon my request.

By signing below, I indicate that I have read, understood and agreed to the details about the study above, and that I agree to participate in this study out of my own free will.

_____	_____	_____
Name of participant (Print)	Signature	Date
_____	_____	_____
Name of witness (Print)	Signature	Date

Do you want a copy of the results of the study to be mailed to you? Yes / No

Please give us your permanent mailing address and telephone number in case you are selected as one of the winners in our random cash prize draws:

Name: _____

Postal address, including postal code:

_____

_____

_____

Telephone number: _____

Appendix 9

Debriefing

Before you leave the study, we would like to give you more information on this study and see whether you have any questions or comments for us. This study is on depression and seasonal depression. Seasonal depression, also known as Seasonal Affective Disorder or SAD, is like the regular nonseasonal depression except that the depressive episodes follow a seasonal pattern. Often, the depression comes during the fall or winter, and remits during the spring or summer. As well, research has found that SAD tends to have some symptoms, that are called atypical symptoms, including carbohydrate craving, oversleeping, overeating, and weight gain.

Bright light therapy has been found to be very effective with SAD but the degree to which it is effective in nonseasonal depression is unclear. We don't know what predicts therapeutic response to bright light therapy. Is it the severity of the depressive symptoms, of the atypical symptoms, or is it the degree of seasonality? What we mean by seasonality is the degree to which the person feels adversely affected by seasonal changes associated with the depressive symptoms. This study is designed to find out what predicts therapeutic response to bright light.

Therefore, we had you rate your physical and psychological functioning before light treatment began, and again at the end of light treatment. We will compare your answere from these two time periods to see whether any changes have occur as a result of your being exposed to light treatment.

We don't know what the results will show us. However, if you wish to know the outcome of the study and have indicated to us your interest, we will be pleased to mail you a copy of our findings once the study is over. It should be completed by the fall of 2002. We will also inform you should you win the random cash prize draws.

Do you have any questions or comments for us? Thank you so much for your participation in this study. Without the generous involvement of research volunteers like yourself, research projects would not be possible.

Appendix 10

Resources for Counselling/Therapy

- Lakehead University Health and Counselling Services (for LU students only): 343-8361
- Family Services Thunder Bay: 626-1880
- Catholic Family Development Centre: 345-7323
- Emergency services are available at the Thunder Bay Regional Hospital (McKellar site)
- Lakehead Psychiatric Hospital has an Urgent Care for walk-ins in the Outpatient Department.

Please keep this page for your own information.