

Running Head: Seasonal Affective Disorder

Explanatory Style, Coping Style, and Stress in Seasonal Affective Disorder,  
Subsyndromal Seasonal Affective Disorder, and Nonseasonal Depression.

Robert P. Dew

Lakehead University, Thunder Bay, Ontario

M.A. Thesis

September 7, 2002

Submitted in partial fulfillment of the degree of Master of Arts, Clinical Psychology

Supervisor: Dr. J. Tan

Second Reader: Dr. C. Netley

National Library  
of Canada

Bibliothèque nationale  
du Canada

Acquisitions and  
Bibliographic Services

Acquisitons et  
services bibliographiques

395 Wellington Street  
Ottawa ON K1A 0N4  
Canada

395, rue Wellington  
Ottawa ON K1A 0N4  
Canada

*Your file* *Votre référence*

*ISBN: 0-612-83405-0*

*Our file* *Notre référence*

*ISBN: 0-612-83405-0*

The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

L'auteur conserve la propriété du droit d'auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

# Canada

Acknowledgements

This thesis is both a direct and indirect result of many individual's efforts to whom I owe much gratitude. First and foremost, I thank Dr. Tan for her supervision. I have fundamentally grown as a scientist, a student, and as a human being from her careful guidance. Second, I thank my mother and my stepfather for their support and their friendship. Both of your strength is an inspiration to me. Third, I thank both Jeanne Wen and Andrea Wrzeczionek for their friendship and support, without which, this thesis may not have been possible. Finally, I sincerely thank Dr. Netley, Dr. Davis, Dr. Young, Dr. Oinonen, Dr. Wesner, and Jennine Rawana for their time and consideration.

## Table of Contents

Introduction	1
Sad as a Distinct Disorder	3
Distinctions Between SAD and Nonseasonal Depression	5
Seasonality	5
Symptomatology	6
Phototherapeutic Response	8
Pharmacotherapeutic Response	10
Conclusions on Distinctions Between SAD and Nonseasonal Depression	14
Distinctions Between SAD and S-SAD	15
Seasonality	15
Symptomatology	17
Phototherapeutic Response	18
Pharmacotherapeutic Response	19
Conclusions From Research on Distinctions Between SAD, S-SAD, and Nonseasonal Depression	20
Psychological Factors	21
Dual-Vulnerability Hypothesis	22
Explanatory Style	25
Coping Style	27
Psychological Stress	30
The Present Study	33
Method	34
Sample	34
Classification	34
Materials: Research Questionnaire	35
Section A	35
Section B	35
Section C	36
Section D	36
Section E	38
Section F	38
Section G	39
Computerized SCID	40
Recruitment of Subjects	41
Study Procedure	41
Results	42
Research Design	42
Sample Characteristics	43
Overview of the Analyses	44
Pre-Analysis Issues	46
Missing Data	46
Univariate Outliers	47

Multivariate Outliers	47
Normality, Linearity, and Homoscedasticity	48
Multicollinearity and Singularity	48
Main Analysis	48
Response Styles Questionnaire	48
Cognitive Styles Questionnaire	49
Dread Of Winter	49
Stress Associated With Winter Atypical Symptoms	49
Impairment During Winter	50
Ruminative Coping During the Winter	50
Perceived Stress During the Winter	50
Discussion	51
Strengths and Limitations of the Study	59
Summary and Conclusions	62
Directions for Future Research	63
References	66

List of Tables

1. Classification criteria for SAD, S-SAD, Depressed and Control group	84
2. Sample Description and Cell Sizes	85
3. Pooled Correlation Matrix of Dependent Variables	86
4. Means (and Standard Deviations) of the Dependent Variables Within Each Group	87
5. Item Factor Loadings of the Winter Perceived Stress Scale (WPSS) With Varimax Rotation.	88

List of Appendices

Appendix 1: Demographic Information and Assessment of Seasonality and SPAQ	89
Appendix 2: HRDS – 28	94
Appendix 3: Response Style Questionnaire	96
Appendix 4: DSM-IV Diagnostic Questions for Major Depression Episode	99
Appendix 5: Cognitive Styles Questionnaire	102
Appendix 6: Winter Stress Scale	115
Appendix 7: Recruitment Posters	120
Appendix 8: Letters to Health Service Providers	122
Appendix 9: Informed Consent for All Participants	124
Appendix 10: Debriefing	126
Appendix 11: Counselling and Therapy Resource List	128

Abstract

Seasonal Affective Disorder (SAD) is characterized by the regular onset and remission of depressive episodes that follow a seasonal pattern. The present study investigated differences among SAD, S-SAD (subsyndromal SAD), nonseasonally depressed, and nondepressed individuals on the severity of typical and atypical depressive symptoms, and on cognitive variables including explanatory style, coping style and stress appraisal of winter-related stimuli. Results indicated that SAD could be distinguished from other groups in their negative reaction to winter-related stimuli. They experience more severe atypical symptoms, find them to be more impairing, ruminate more about the winter, and perceive themselves as less able to cope. Compared to depressed individuals, the SAD persons had more severe atypical symptoms, higher degree of seasonality, and greater focus on their depression. Compared to S-SAD, the SAD individuals reported more typical and atypical symptoms, greater degree of seasonality, more use of rumination and involvement in dangerous activities to cope with their depression, and more maladaptive explanatory style (more global and stable attributions). The depressed group differed from the S-SAD group in that they had more typical and atypical symptoms, less degree of seasonality, ruminated more on their depression, and had more negative reactions to winter. Except for their greater degree of seasonality, and their more unstable explanatory style, the S-SAD was no different from the nondepressed group.



## Introduction

Although seasonal changes in mood and behaviour have been recognized since the time of Hippocrates (Kane & Lowis, 1999; Rosenthal, Sack, James et al., 1985), the phenomenon has received serious research attention only quite recently within the last two decades. The condition of Seasonal Affective Disorder (SAD) is characterized by the regular onset and remission of depressive episodes that follows a seasonal pattern (Lee et al., 1998; Rosenthal et al., 1984). Two distinct types of SAD have been identified: summer SAD and winter SAD (Rosenthal, 1993). Summer SAD refers to clinical depression that begins during late spring or summer and remits in late summer or early fall. Winter SAD refers to clinical depression that has its onset during late summer or autumn and remits by spring or early summer (Faedda et al., 1993). Most of the research has focussed on winter SAD, and henceforth in this paper, the term SAD refers to winter SAD.

Research shows that SAD tends to onset between the months of September and November (Terman, 1988; Terman, Quitkin, Terman, Mcgrath, & Stewart, 1987). It tends to last approximately 5 months (Terman et al., 1987) with SAD individuals reporting a nadir in depressive moods between December and February (Terman, 1988; Terman et al., 1989). Research also shows that SAD tends to remit between the months of March and April (Terman, 1988; Terman et al., 1987; Winton & Checkly).

Epidemiological studies show the prevalence of SAD to range from .4% to 9.2% (Blazer, Kessler, & Schwartz, 1998; Booker & Hellekson, 1992; Levitt, Boyle, Joffe, & Baumal, 1997; Mersch, Middendorp, Bouhys, Beersma, & Hoofdakker, 1999a). Reasons for such disparity in estimates include different latitude and therefore different climate, and the use of different

diagnostic criteria at different study sites. Research consistently shows the prevalence of SAD to increase with northerly latitude (Booker & Hellekson, 1992; Lee et al., 1998; Mersch, Middendorp, Bouhys, Beersma, & Hoofdakker, 1999b; Sourander, Koskelainen, & Helenius, 1999; Suhail & Cochrane, 1997; Terman, 1988). Rosen and colleagues (1990) assessed SAD rates in four areas within America: Nashua, NH; New York, NY; Montgomery County, MD; and Sarasota, Florida. Results revealed that SAD rates were lower in southerly states such as Florida (1.4%) and higher in northerly states such as New York (4.7%). Researchers suggested that the prevalence of SAD increases with higher latitude areas because these areas have a shorter photoperiod (Rosen et al., 1990; Jacobsen, Wehr, Sack, James & Rosenthal, 1987; Rosen & Rosenthal, 1991; Suhail & Cochrane, 1997). This position was corroborated by evidence of a negative relationship between the prevalence of SAD and the average amount of daily sunshine (Magnusson & Axelsson, 1993; Mersch et al., 1999b; Molin, Mellerup, Bolwig, Scheike, & Dam, 1996; Sakamoto, Nakadaira, Tamura, & Takahashi, 1993), suggesting a possible etiological role of decreased amount of sunlight in the disorder (Lee et al., 1998; Rosenthal & Wehr, 1987; Sakamoto et al., 1993; Terman, Reme, Rafferty, Gallin, & Terman, 1990).

The other possible reason for the vast difference among epidemiological findings is the different inclusion and exclusion criteria used to identify SAD individuals across study sites. Earlier epidemiological studies employed the Seasonal Pattern Assessment Questionnaire (SPAQ, Rosenthal, 1993) as the sole diagnostic device (e.g., Bartko & Kasper, 1990; Booker & Hellekson, 1992; Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989; Mersch et al., 1999a; Rosen et al., 1990). Although the SPAQ shows some desirable psychometric properties including good test-retest reliability (Thompson et al., 1988; Hardin et al., 1991), it is faulted for lacking explicit DSM-IV criteria to identify major depressive episode in SAD candidates (Blazer, 1998; Blazer et

al., 1998; Kane & Lowis, 1999) that is a feature necessary to qualify for a SAD diagnosis (APA, 1994). Relatively recent epidemiological studies that include DSM-IV criteria to identify SAD (e.g., Blazer et al., 1998; Levitt & Boyle, 1997) reveal a substantially different epidemiological picture from that of earlier studies. For example, one study comparing the inclusion criteria employed by both earlier and later studies showed that earlier criteria resulted in four times as many SAD subjects as later criteria (Levitt & Boyle, 1997). This suggests that earlier criteria that are less restrictive may have the problem of overidentification such that subsyndromal individuals are included as positive cases as well. Moreover, the indication from earlier studies that SAD is more prevalent among women than men (Booker & Hellekson, 1992; Jacobsen et al., 1987; Kane & Lowis, 1999; Kasper, Wehr et al., 1989; Lee et al., 1998; Magnusson & Stefansson, 1993; Mersch et al., 1999b; Partonen & Lundquist, 1998; Rosenthal & Wehr, 1987; Terman et al., 1989) is also controverted by more recent epidemiological studies indicating SAD to be more prevalent among men than women (e.g., Blazer et al., 1998).

Thus, research may need to consider a subsyndromal version of SAD called S-SAD that is less impairing than SAD (Kasper, Wehr et al., 1989; Kasper, Rogers et al., 1989) and has a shorter and milder depressive episode (Kasper, Wehr et al., 1989; Kasper, Rogers et al., 1989; Lam et al., 2001). The importance of distinguishing S-SAD from SAD is underscored by the absence of unambiguous and consensual research guidelines to identify SAD versus S-SAD individuals that will increase the validity and reliability of findings and comparisons of results across studies. Comparisons of SAD and S-SAD are undertaken in a later part of this paper under the section titled "Distinctions between SAD and S-SAD".

#### *SAD As A Distinct Disorder*

The concept of SAD as a depressive disorder distinct from other depressive disorders has

been an area of considerable debate (e.g. Bauer, 1992; Bauer & Dunner, 1993; Schuller, Bagby, Levitt, & Joffe, 1993; Spitzer & Williams, 1989). To address this issue, it is important to consider research that is designed to delineate SAD from both its nonseasonal counterpart (i.e., nonseasonal depression) and its subsyndromal counterpart (i.e., S-SAD) as they are very similar to SAD in their symptomatic characteristics (Bauer, 1992; Bauer & Dunner, 1993). Evidence that SAD may be differentiated from nonseasonal depression by its high degree of seasonality (Bauer, 1992; Bauer & Dunner, 1993; Hardin et al., 1991; Rosenthal, 1993; Sher, Goldman, Ozaki, & Rosenthal, 1999; Sher, 2001), intense atypical depressive symptoms (Allen, Lam, Remick & Sadvnick, 1993; Garvey, Wesner, & Godes, 1988; Meesters, Jansen, Bouhys, Beersma., & van den Hoofdakker., 1993; Rosenthal, et al. 1984; Sakamoto, Nakadaira, Kamo, & Takahashi, 1995; Young, Watel, Lahmeyer, & Eastman, 1991), and therapeutic response to light (Thalen, Kjellman, Morkrid, Wibom, & Wetterberg, 1995; Eastman, Young, Fogg, Liu, & Meaden, 1998; Lee & Chan 1999) is controversial as different findings have been reported (e.g., Lee et al., 1998). However, no review has been conducted within the last three years on these apparent differences between SAD and nonseasonal depression. Thus, it is likely that more contemporary research may help to better clarify the status of these differences. Research, though scant, also exists to tentatively comment on whether SAD and S-SAD also differ in terms of seasonality, symptomatology, and therapeutic response to light. However, a review comparing SAD and S-SAD is absent in the literature. Thus, a review of the differences between SAD, S-SAD, and nonseasonal depression will be undertaken in this paper to address the current understanding of SAD's distinctiveness and research necessary to further our understanding of the nature, etiology, and distinctiveness of SAD.

*Distinctions Between SAD and Nonseasonal Depression**Seasonality*

Seasonality has been described as SAD's "sole invariant defining characteristic" (Lee et al., 1998, p. 276). It refers to the seasonal change in mood, energy, sleep, appetite, food preference, and socialization pattern (Bauer & Dunner, 1993; Hardin et al., 1991; Rosenthal, 1993; Sher et al., 1999, Sher, 2001 Spitzer & Williams, 1989) evident in SAD. The operational definition of seasonality involves the consideration of the seasonal changes of both typical depressive symptoms (e.g., low mood, low energy) and atypical depressive symptoms (i.e., increased appetite, carbohydrate craving, hypersomnia,). Although some researchers have suggested that seasonality is unique to SAD (e.g., Rosenthal, 1989), it has been found to be a phenomenon shared by other disorders including nonseasonal bipolar disorder (Thompson et al., 1988), bulimia (Hardin et al., 1991), anorexia nervosa (Wehr, 1989; Hardin et al., 1991), and borderline personality populations (Sakamoto et al., 1995). Moreover, 92% of the general population report some degree of seasonality (Kasper et al., 1989b). Consequently, some researchers have conceptualized seasonality as a single dimension that cuts across diagnostic lines with SAD individuals (extreme seasonal change) constituting one extreme end and completely nonseasonal individuals (i.e., no seasonal change) constituting the other extreme end (Bartko & Kasper, 1989; Bauer, 1992; Bauer & Dunner, 1993; Bauer, Kurtz, Rubin, & Marcus, 1994; Booker & Hellekson, 1992; Kasper, Rogers, et al., 1989; Kasper, Wehr et al., 1989; Jang, Lam, Harris, Vernon, & Livesly, 1998; Lam et al., 2001; Rosen, et al., 1990; Sher, 2001; Young et al., 1991).

One may argue that it is not the presence of seasonality but the severity of seasonal

change that distinguishes SAD individuals from nonseasonal depressed individuals (Hardin et al., 1991; Magnusson, 1996; Thompson et al., 1988). Accordingly, Thompson and colleagues (1988) reported that SAD, nonseasonal bipolar, and nonseasonal nondepressed populations can be effectively distinguished based on the degree of seasonal variation in their depressive symptoms. Likewise, Hardin and colleagues (1991) also reported the separation of SAD from nonseasonal depression based on the severity of seasonal changes in mood and behaviour. Thus, while seasonality per se may not be unique to SAD, research suggests that the intensity of seasonal change in mood and behaviour experienced by SAD individuals is greater than in nonseasonal depressed populations.

### *Symptomatology*

As previously noted, the operational definition of seasonality involves the consideration of the seasonal changes of both typical depressive symptoms (e.g., low mood, low energy) that are commonly associated with nonseasonal depressive episodes (Garvey et al., 1988; Lam et al., 2001; Young et al., 1991), and atypical symptoms (i.e., increased appetite, carbohydrate craving, hypersomnia,) that are rarely associated with nonseasonal depression (Allen et al., 1993; Garvey et al., 1988; Meesters et al., 1993; Rosenthal et al., 1984; Sakamoto et al., 1995; Young et al., 1991). Atypical symptoms (i.e., increased appetite, carbohydrate craving, and hypersomnia) are regarded as atypical because these symptoms are opposite to what nonseasonally depressed individuals would present (i.e., decreased appetite, absence of carbohydrate craving, and insomnia).

Research shows that SAD depressive episodes tend to involve more atypical symptoms than nonseasonal depressive episodes (Allen et al., 1993; Garvey et al., 1988; Lam et al., 2001; Meesters et al., 1993; Rosenthal et al., 1984; Sakamoto et al., 1995; Young et al., 1991).

Surprisingly, although SAD depressive episodes are accompanied by a high degree of atypical symptoms compared to nonseasonal depression, research consistently shows that typical symptoms outnumber atypical symptoms (Kasper, Rogers et al., 1989; Kasper, Wehr et al., 1989; Lam et al., 2001).

Research suggests that atypical symptoms are necessary for understanding SAD. First, atypical symptoms are temporally related to the circannual onset of SAD in that they regularly accompany the depressive episode (Rosenthal et al., 1984; Young et al., 1991), whereas typical symptoms seem to be temporally unrelated to the onset to SAD. For example, Young, Watel, Lahmeyer and Eastmen (1991) report the findings of a study designed to track the onset of both atypical and typical symptoms in SAD individuals during their annual progression from normal to clinical depression. Results showed that whereas atypical symptoms reliably appeared together at the beginning of seasonal depressive episodes, typical symptoms appeared separately as well as randomly throughout the seasonal depressive episode. The researchers speculated that atypical and typical symptoms might be controlled by disparate pathogenic mechanisms. Second, longitudinal research shows that individuals initially diagnosed with SAD who present with atypical symptoms will be more likely to continue to experience SAD in later years than individuals initially diagnosed with SAD who do not present with atypical symptoms (Sakamoto et al., 1995). Third, SAD individuals who present with atypical symptoms are more likely to respond therapeutically to light therapy than SAD individuals who do not present with atypical symptoms (Bauer et al., 1994; Stinson & Thompson, 1990; Terman, Amira, Terman, & Ross, 1996). However, it should be kept in mind that although atypical symptoms may be more frequently associated with SAD, they are not distinct to the disorder because individuals with nonseasonal atypical depression (APA, 1994; Stewart, Quitkin, Terman, & Terman, 1990; Thase, 1989) can in some cases also present with the atypical symptoms.

### *Phototherapeutic Response*

An association between SAD and both artificial and natural environmental light initially seemed distinct to the disorder (Mackert, Volz, Stieglitz, & Orlinghausen, 1990; Stewart et al., 1990; Volz, Mackert, Stieglitz, & Muller-Oerlingerhausen, 1990; Yerevenian, Anderson, Grota, & Bray, 1986). The prevalence of SAD appears to increase with the decrease in both photoperiod (Magnusson & Stefansson, 1993; Mersch et al., 1999a,b; Rosen et al., 1990; Sourander et al., 1999; Suhail & Cochrane, 1997) and the daily amount of sunshine (Mersch et al., 1999b; Molin et al., 1996; Sakamoto et al., 1993). Anecdotal evidence abounds of instances where individuals with SAD experience alleviation of depression during vacation to the tropics and other relatively equatorial areas and paroxysm of depression following return to their northerly habitat (Rosenthal et al., 1984; Rosenthal, 1993; Thase, 1986, 1989). Yet, the question as to whether SAD and nonseasonal depression differ in their response to natural environmental light remains relatively unaddressed.

Light therapy is presently the most efficacious and rapid method of alleviating SAD symptoms (Partonen & Lundquist, 1998; Rosenthal & Wehr, 1987; Thalen et al., 1995). It entails the daily administration of bright artificial light indoors (Terman, 1989). Antidepressant response to light therapy begins within 3 to 4 days of administration (Lee & Chan, 1999; Terman et al., 1990), with the possibility of relapse within the same period of time upon cessation of the therapy (Meesters et al., 1993; Terman et al., 1989). Clinical response to light therapy can be reached within a week (Meesters et al., 1993; Thase, 1989) with optimal gains after 4 weeks (Bauer et al., 1994; Eastman et al., 1998; Terman et al., 1989).

Clinical response to light therapy was initially thought to be distinct to SAD individuals (Mackert et al., 1990; Stewart et al., 1990; Volz et al., 1990; Yerevenian et al., 1986). For



example, Yerevenian, Anderson, Grotta, and Bray (1986) report the nonresponse of 8 nonseasonally depressed individuals to light therapy. Stewart, Quitkin, Terman, and Terman (1990) also report the nonresponse of 10 nonseasonal atypical depressed individuals to light therapy. However, a fairly recent review (Kripke, 1998) suggests that nonseasonally depressed individuals show a rate and magnitude of response to light therapy that is comparable to the rate and magnitude of response demonstrated by SAD patients. Earlier studies were faulted for basing their findings on small sample sizes (e.g., Stewart et al., 1990; Yerevenian et al., 1986). Thus, Thalen, Kjellmanm Morkrid, Wilbom, and Wetterberg (1995) compared the phototherapeutic response of 68 SAD individuals and 22 nonseasonally depressed individuals to 10 consecutive days of light therapy sessions that lasted 2 hours each. Results showed that whereas 53% of SAD individuals evidenced more than 50% reduction in depressive symptoms, only 14% of the nonseasonally depressed individuals reported a 50% reduction in depressive symptoms from baseline. Thus, researchers concluded that SAD responded better to light therapy than nonseasonal depression. However, this study is faulted for not including a convincing placebo for light therapy (Kripke, 1998; Eastman et al., 1998). Moreover, Thalen and colleagues (1995) noted that the SAD group evidenced milder baseline depressive levels than the nonseasonal group. Considering that mild depression levels evidenced by SAD populations (Rosenthal et al., 1984) seem more responsive to light therapy than populations that have more severe baseline depressive symptomatology (e.g., nonseasonal depression, Kripke, 1998; Terman et al., 1989), future research should include study groups that are matched for depression levels.

Interestingly, the general consensus among the few studies that have assessed typical and atypical symptoms separately show that they respond differently to light therapy (e.g., Lam et al., 2001; Lee & Chan, 1999; Terman et al., 1996). For example, Lee and Chan (1999) applied a

meta-analytical methodology to 39 studies on light therapy and SAD. Results showed that whereas typical symptoms evidenced a dose-response (i.e., typical symptoms intensity decreased as light intensity increased), atypical symptoms evidenced a constant phototherapeutic response regardless of light intensity. That is, although both types of symptoms responded to light therapy, they responded differently to light therapy. Researchers concluded that differential response to light therapy evidenced by atypical and typical symptoms reflected independent pathogenic mechanisms.

### *Pharmacotherapeutic Response*

To date, few studies have explicitly compared pharmacological therapy and light therapy on SAD or the response of both SAD and nonseasonal depression to pharmacotherapy (Bauer, 1992; Bauer & Dunner, 1993; Partonen & Lundquist, 1998). Given that atypical symptoms characteristic of SAD are associated with serotonergic functioning (Neumeister, Praschak-Riederm, Hebelmann, Rao et al., 1997; Neumeister, Praschak-Rieder, Hebelmann, Vitouch et al., 1997) most studies relevant to SAD and pharmacotherapy have focussed on drugs that either impede or enhance the production of serotonin (Jacobsen et al., 1994; Lam et al., 1995; McGrath, Buckwald, & Resnick, 1990; Partonen & Lunqvist, 1996; 1998; Van der Does, 2001).

Mcgrath, Buckwald and Resnick (1990) compared the effects of the serotonergic precursor l-tryptophan, placebo, and light therapy on 13 SAD individuals. Participants were assigned to three weeks of treatment alternated with three weeks of no treatment in a counterbalanced order. Results showed that therapeutic response to both l-tryptophan and light therapy were equivalent, yet only therapeutic response to l-tryptophan was significantly greater than that of the placebo. Results also showed a trend towards greater remission of atypical symptoms in l-tryptophan than light therapy. The methodological drawback particular to this

study was the short duration of the treatment. To address this problem, Ghadirian, Murphy and Gendron (1998) randomly assigned 13 patients to both 2 weeks of light therapy or 4 weeks of l-tryptophan alternated by 1 week washout periods. Therapeutic response was defined as 50% decrease in depressive symptoms in comparison to baseline. No significant superiority was found in the efficacy of light therapy over l-tryptophan. Results also showed that whereas eight subjects met the operational criteria of therapeutic response to light therapy alone, only five subjects from l-tryptophan alone met response criteria. Yet, given that all non-responders of tryptophan evidenced some degree of response it is possible that 4 weeks of tryptophan was not the optimal treatment duration. Problems with both of these studies include a small sample size ( $N=13$ ), the possibility of crossover effects, and sub-optimal l-tryptophan treatment durations. Presently, no study has explicitly compared the effect of tryptophan therapy on both SAD and nonseasonal depression.

Further research concerning the involvement of the serotonergic system in SAD comes from studies using the tryptophan depletion (TD) paradigm which involves the rapid decrease of 5-hydroxytryptophan (Ghadirian, Murphy, & Gendron, 1998; Neumeister, Praschak-Riederm, Hebelmann, Rao et al., 1997; Van der Does, 2001), and therefore a decrease in serotonin. TD studies have been conducted on both remitted (Neumeister, Praschak-Riederm, Hebelmann, Rao et al., 1997) and symptomatic (Neumeister, Praschak-Rieder, Hebelmann, Vitouch et al., 1997) SAD individuals. Yet, evidence for a direct causal role is inconsistent. For example, Neumeister, Praschak-Riederm, Hebelmann, Rao, and colleagues (1997) randomly assigned remitted SAD individuals to receive either TD first and then control (sham TD procedure) or control first and then TD. Results showed that of the 12 SAD subjects in remission from SAD after light therapy, 6 relapsed to depressive levels that were not significantly different from the pre-therapy levels.

Interestingly, proportionately more atypical symptoms appeared after TD than typical symptoms. Nonsignificant results in remitted populations have also been reported elsewhere (e.g., Lam et al., 2000). For example, Neumeister, Praschak-Rieder, Hebelmann, Vitouch and colleagues (1997) randomly assigned 11 drug-free symptomatic SAD subjects to either TD or placebo separated by a 7 to 9 day washout period. Results showed no significant difference between baseline and post-TD depressive levels. While an explicit comparison of TD on both SAD and nonseasonal depression has yet to be conducted, a review on the effects of TD suggests that SAD and nonseasonally depressed individuals tend to respond similarly in both remission and in a symptomatic state (Van der Does, 2001). Nondepressed individuals however tend to respond mildly to TD, if at all (Van der Does, 2001). Researchers speculate that as TD produces inconsistent effects on both remitted and symptomatic SAD individuals it is likely that SAD depression is not directly the result of 5-hydroxytryptophan availability (Neumeister, Praschak-Rieder, Hebelmann, Vitouch et al., 1997; Lam et al., 2000).

Other studies designed to assess therapeutic properties of antidepressants on SAD patients have used fluoxetine, a selective serotonin re-uptake inhibitor (SSRI, Lam et al., 1995; Partonen & Lundqvist, 1996; 1998; Ruhrmann et al., 1998). Lam and colleagues (1995) randomly assigned 68 SAD outpatients to five weeks of either fluoxetine ( $N=36$ ) or placebo ( $N=32$ ). Results showed that whereas clinical response was achieved by 59% of the fluoxetine group, clinical response was only 39% in the placebo group. No statistics were conducted on atypical and typical symptom response to either placebo or treatment. Interestingly, posthoc stratification of the fluoxetine group into mild and severe baseline depressive levels revealed that severely depressed individuals responded best to fluoxetine: a finding opposite that of light therapy (Terman et al., 1989). Ruhrmann and colleagues (1998) randomly assigned 40 SAD

outpatients to either five weeks of fluoxetine ( $N = 20$ ) or five weeks of light therapy ( $N = 20$ ). Results revealed that light therapy and fluoxetine groups did not differ in their clinical response (70% and 65% clinical response, respectively). Results also showed that atypical symptoms responded more rapidly to fluoxetine than to light therapy compared to typical symptoms.

Two criticisms against most studies are that they fail to investigate the comparative efficacy of antidepressants with SAD and nonseasonal depression, and that they have not looked at SAD clinical response to antidepressants that work on non-serotonergic neurotransmitter systems (Partonen, & Lonnqvist, 1998). Thus, Partonen and Lonnqvist (1996) compared SAD and nonseasonal depressed pharmacotherapeutic response to 6 weeks of either fluoxetine ( $N = 18$  and  $N = 55$ , respectively) or moclobemide ( $N = 11$  and  $N = 63$ , respectively): an antidepressant that modulates dopaminergic and catecholaminergic systems in addition to the serotonergic system. Results showed that both SAD and nonseasonal depressed groups showed significant and equivalent clinical response to both moclobemide (64% and 44% response rate, respectively) and fluoxetine (44% and 29% response rate respectively). A 64% response rate to moclobemide by SAD individuals is (nonsignificantly) higher than a 44% response to moclobemide in a 3-week, placebo controlled trial (Lingjaede et al., 1993). Reasons for discrepant results include different treatment durations (three weeks in Lingjaerde et al., 1993 versus six weeks in Partonen & Lonnqvist), different operational definitions of clinical response, and small sample size ( $N=16$  in Lingjaerde, et al. versus  $N=11$  in Partonen & Lonnqvist, 1996).

Preliminary research concerning pharmacotherapy has many implications for future research. First, more studies using larger sample sizes, commensurate criteria of clinical response, optimal treatment durations, and methodologies allowing direct comparison of both SAD and nonseasonal depression to pharmacotherapy, and direct comparison of the effect of

pharmacotherapy and light treatment on SAD are necessary. Second, future research should compare atypical and typical symptomatic response given the possibility that they may reflect disparate pathogenic mechanisms (Lam et al., 2001; Lee & Chan, 1999; Sher, 2001; Terman et al., 1990; Young et al., 1991). Although preliminary evidence indicates that typical and atypical symptoms respond differently to pharmacotherapy, this conclusion is tentative until results can be scrutinized based on rigorous methodology. Preliminary research suggests that antidepressants used for nonseasonal depression (i.e., moclobemide) may work similarly for SAD (Partonen & Lonnqvist, 1996). Yet this conclusion is also tentative until direct comparisons of the paradigm on both SAD and nonseasonal depression are available.

#### *Conclusions on Distinctions Between SAD and Nonseasonal Depression*

Research suggests that a high degree of seasonal change in atypical symptoms and a high degree of phototherapeutic response may be used to differentiate SAD from nonseasonal depression. Research showing that most individuals experience seasonal change in mood and behaviour to some degree have led researchers to posit a single, continuous dimension referred to as seasonality (Bartko & Kasper, 1989; Bauer, 1992; Bauer & Dunner, 1992; Bauer et al., 1994; Booker & Hellekson, 1992; Jang et al., 1998; Kasper, Rogers et al., 1989; Kasper, Wehr et al., 1989; Lam et al., 2001; Rosen et al., 1990; Sher, 2001; Young et al., 1991). As previously written, the dominant concept of seasonality as a single continuum incorporates both typical and atypical symptoms. Extant etiological models reflect this assumption by positing a single pathogenic process in the etiology of SAD (James, Wehr, Sack, Parry, & Rosenthal, 1985; Lee et al., 1998; Lee & Chan, 1999; Meesters et al., 1993; Moller, 1992; Rosenthal et al., 1984; Wurtman, 1990). Yet, findings indicating both a temporal disassociation of typical and atypical symptoms in circannual SAD onset (Young et al., 1991) and their differential response to light

therapy (Lee & Chan, 1999; Terman et al., 1996) may be better explained by a dual rather than a single pathogenic process in the etiology of SAD. Hence, the question as to whether SAD is the extreme manifestation of a single, global dimension (seasonality of typical and atypical symptoms) or of two dimensions, one for typical and the other for atypical symptoms, invites closer examination. Clearly, the importance of studying considering S-SAD is heightened considering that S-SAD is generally considered a milder form of seasonality (Bauer, 1992; Bauer & Dunner, 1993), and hence, represents another vantage from which to scrutinize the continuum paradigm.

#### *Distinctions Between SAD and S-SAD*

It is generally thought that SAD and S-SAD differ from each other on the basis of a severity of seasonal change continuum (Bauer, 1992; Bauer & Dunner, 1993) such that S-SAD individuals are thought to present with milder impairment and milder seasonal changes in mood and behavior than SAD individuals. If S-SAD is a subclinical manifestation of SAD, then it would be logical to assume that S-SAD individuals would experience either lesser number and/or less intense seasonal changes especially with respect to atypical depressive symptoms, report less impairment in functioning due to the seasonality, and would show less response to environmental light and to phototherapy.

#### *Seasonality*

S-SAD has been differentiated from SAD by both degree of impairment and severity of seasonality. Kasper, Wehr, and colleagues (1989) identified S-SAD individuals by using the Seasonality Pattern Assessment Questionnaire (SPAQ, Rosenthal, Bradt, & Wehr, 1987) and considering either the degree of impairment, or the joint contribution of degree of impairment and degree of seasonality. S-SAD individuals either had no significant degree of impairment, or

a mild degree of impairment that would compensate for a higher seasonality score and vice versa (Kasper, Wehr et al., 1989).

Magnusson (1996) attempted to validate the SPAQ classifications of SAD and S-SAD (using Kasper, Wehr et al., 1989 guidelines) against Major Depressive Episode or Major Depressive Disorder: DSM-IV diagnostic criteria necessary for DSM-IV SAD diagnosis (APA, 1994). He identified 32 seasonal individuals as either SAD or S-SAD and followed up on the SPAQ assessment with a diagnostic interview. Results showed that of the 11 participants who were diagnosed with MDE, 5 fulfilled the criteria of SAD and 6 fulfilled the criteria of S-SAD. Results also showed that of the remaining 21 seasonal individuals who were not diagnosed with MDE, 11 fulfilled the SAD criteria whereas 10 fulfilled the S-SAD criteria. Thus, according to the SPAQ, the ratio of S-SAD and SAD in seasonal individuals was similar in those both with MDE and those without MDE. Given that MDE is required for SAD to be diagnosed in accordance with the DSM-IV (APA, 1994), the observation that not more SAD individuals are to be found among those with MDE and less number of SAD individuals among those who did not have MDE is surprising. The researchers concluded that the operational definitions of seasonality used on the SPAQ do not adequately discriminate between SAD and S-SAD. This conclusion must be viewed with the following considerations in mind. First, the study as designed did not allow a comparison of the S-SAD guidelines using severity of impairment versus the joint contribution of impairment and degree of seasonality as the index criterion. It is possible that one may be more useful than the other. Second, this study requires replication to allow greater confidence in the conclusions reached. Thus far, it is the only one that examines the utility of the SPAQ in discriminating between SAD and S-SAD. Nonetheless, Magnusson's finding highlights the possibility (previously discussed) that studies using solely the SPAQ to



identify SAD may include a high number of S-SAD. This finding also questions the utility of SPAQ items including eating, sleep, energy, socialization, mood, and appetite change as discriminators between SAD and S-SAD (Kasper, Wehr et al., 1989; Sher et al., 1999; Sher, 2001). The possibility arises that symptoms useful for discriminating between SAD and nonseasonal depression populations are different from those symptoms that may be useful in discriminating between SAD and S-SAD populations. It is likely that closer examination of both typical and atypical symptomatology evidenced by both SAD and S-SAD populations may indicate which symptoms best discriminate between the two conditions.

### *Symptomatology*

According to the continuum paradigm, a high degree of typical and atypical symptoms are characteristic of SAD (Allen et al., 1993; Garvey et al., 1988; Lam et al., 2001; Thalen et al., 1995). Therefore SAD, individuals should present with more typical and atypical symptoms than S-SAD individuals. Research designed to reveal atypical symptomatological differences between SAD and S-SAD show inconsistent findings. In particular, whereas Kasper, Rogers, and colleagues (1989a) reported that the intensity of atypical symptoms in SAD individuals are two times that of S-SAD individuals, Lam, Tam, Yatham, Shiahand, and Zis (2001) reported no significant differences in atypical symptoms between the two groups. Reasons for the discrepancy may be threefold. First, Kasper, Rogers, and colleagues (1989a) employed a smaller group size (SAD,  $N = 20$ ; S-SAD,  $N = 20$ ) than Lam and colleagues (2001, SAD  $N = 454$ ; S-SAD  $N = 55$ ). Second, whereas Kasper, Rogers, and colleagues drew their sample from the community, Lam and colleagues studied individuals attending a seasonal mood disorders clinic. Thus, it is possible that Lam and colleagues' findings reflect a sample of individuals who tend to experience more severe seasonal change in atypical symptoms than the community subjects

reported by Kasper, Rogers, and colleagues. Finally, whereas Kasper, Rogers, and colleagues (1989a) employed DSM III-R criteria to identify SAD, Lam and colleagues used both DSM-IV and DSM III-R criteria to identify SAD. To date, no study has explicitly been designed to assess how the symptomatological characteristics of those identified as SAD by DSM III-R criteria are different from those identified by DSM-IV criteria. Yet, given that substantial changes have been made to the Seasonal Pattern modifier since its inception in DSM III-R, it is possible that DSM-IV and DSM III-R Seasonal Pattern criteria may identify symptomatologically disparate populations as SAD. Thus, a study with large sample sizes based on DSM III-R and DSM IV criteria should be conducted to address this possibility. Nonetheless, the continuum paradigm is controverted by the finding that S-SAD individuals, regardless of whether the sample is community or clinic based, have similar levels of atypical symptomatology as SAD (Lam et al., 2001). Consistent among both studies are the surprising findings that typical symptoms in SAD significantly outnumber typical symptoms in S-SAD (Kasper et al., 1989a; Lam et al., 2001) suggesting that SAD may be better differentiated from S-SAD using typical symptoms as discriminators rather than atypical symptoms.

#### *Phototherapeutic Response*

A sensitivity to both environmental and artificial light are features presently regarded to be characteristic of SAD (Lam et al., 2001; Mersch et al., 1999a, b; Rosen et al., 1990; Thalen et al., 1995). If S-SAD is a relatively common subclinical form of SAD (i.e., closer to the norm), then specific predictions can be made concerning the sensitivity of S-SAD to environmental and artificial light. First, the prevalence of S-SAD should increase with latitude at a proportionately lower rate than SAD. Yet, Rosen and colleagues (1990) reported that S-SAD prevalence increases with latitude at a proportionately higher rate than SAD. In particular, results showed

that whereas the prevalence of SAD and S-SAD in Florida hovered at 1.7% and 2.6%, respectively, the prevalence of SAD and S-SAD in New York was 4.7% and 12.5%. Thus, S-SAD seems more sensitive to environmental light than SAD: a relationship contrary to that predicted by the continuum paradigm.

A therapeutic response to increased artificial light has also been suggested to be a characteristic feature of SAD (Lundquist & Partonen, 1998; Mackert et al., 1991; Rosenthal & Wehr, 1987; Thalen et al., 1995; Volz et al., 1990; Yerevenian et al., 1986). If S-SAD exists on a continuum between completely nonseasonal individuals who show no response to increased light (Kasper, Rogers et al., 1989; Kasper, Rogers, Madden, Vanderpool, & Rosenthal, 1990) and SAD individuals who show a clinical response response to light (Lam et al., 2001), then individuals with S-SAD should respond less to artificial light than individuals with SAD. Yet, contrary to this prediction, research shows that S-SAD populations show better response to increased light (Lam et al., 2001). The reason for this may lie in the finding that a higher typical to atypical ratio in an individual with winter depression predicts a lower therapeutic response to light therapy (Terman et al., 1996). Given that SAD individuals report more typical symptoms than S-SAD individuals (Kasper Rogers et al., 1989; Kasper, Wehr et al., 1989; Lam et al., 2001), this may explain the better showing of S-SAD individuals in light therapy response.

#### *Pharmacotherapeutic Response*

No research has been published comparing phototherapy and pharmacotherapy on S-SAD or comparing SAD and S-SAD response to pharmacotherapy. Such an area of research may help to differentiate SAD from S-SAD.

*Conclusions from Research on Distinctions Between SAD, S-SAD, and Nonseasonal Depression*

Contrary to the continuum paradigm, research suggests that S-SAD and SAD may be equivalent in their degree of seasonality (according to the SPAQ; Magnusson, 1996), although both conditions invoke a more severe degree of seasonality than nonseasonal depression (Hardin et al., 1991; Magnusson, 1996; Thompson et al., 1988). However, S-SAD might be characterized by milder typical symptoms than SAD (Kasper, Rogers et al., 1989a; Lam et al., 2001) but might not be different from SAD when it comes to atypical symptoms (Lam et al., 2001). Taken together, these findings refute the notion of seasonality as a single dimension (Sher et al., 1999; Sher, 2001). Rather, these findings suggest that whereas both SAD and S-SAD individuals are predisposed to atypical symptoms, only SAD and nonseasonal depression individuals are predisposed to typical symptoms sufficient to warrant a diagnosis of MDE. This suggests that the assessment of SAD and S-SAD has to include two dimensions (typical and atypical symptoms) rather a single dimension (Lam et al., 2001; Sher et al., 1999; Sher, 2001; Young et al., 1991).

As previously discussed, this possibility of two dimensions underlying seasonality is corroborated by research showing that atypical and typical symptoms respond differently to light therapy (Lee & Chan; 1999; Terman et al., 1990; Terman et al., 1996) and a difference in their temporal association with the onset of SAD (Young et al., 1991). Thus, the current conceptualization of seasonality as a single dimension may be masking two active dimensions in SAD: a genuine seasonality dimension associated with atypical symptoms that appears with the onset of SAD (Lam et al., 2001; Young et al., 1991), and a depressive dimension associated with typical symptoms that have no temporal association with the onset of SAD (Allen et al., 1993; Lam et al., 2001; Sakamoto et al., 1995; Sher et al., 1999; Sher, 2001; Young et al., 1991). It is

possible that effective discriminators between SAD and S-SAD would include factors associated with typical symptoms rather than atypical symptoms as used in the SPAQ (Kasper, Rogers et al., 1989; Rogers, Wehr et al., 1989). Finally, given that research suggests that assessing SAD on a single dimension may actually be masking the true nature of SAD (i.e., the coinciding of two dimensions), future research should assess typical and atypical symptoms separately (Lee & Chan, 1999).

Psychological factors that are typically associated with nonseasonal depression have been found to bear a relationship to SAD as well (Geerts et al., 2000; Hodges & Marks, 1998; Jang, Blais, Otto, Hirshfeld, & Sachs, 1998; Levitan, Rector, & Bagby, 1998; Linjaerde, Foreland, A., Regine, & Engvik, 2001; Schuller et al., 1993; Wrzeczionek, 2000). As the initial conceptualization of SAD was driven by the assumption that SAD was the result of an interaction between decreased light and a pathogenic neuromechanism (Lee et al., 1998; Levitan, Rector, & Bagby, 1998; Rosenthal et al., 1984; Terman, Quitkin, Stewart, & McGrath, 1987), most research has focussed on the light and biological aspects of SAD and ignored psychological facets. This neglect is unfortunate considering that psychological distinctions are as important as neurobiological distinctions for a mental disorder to be differentiated from other mental disorders (Bauer & Dunner, 1993). Thus, consideration of psychological factors may have implications for future research delineating the differences between SAD, S-SAD, and nonseasonal depression.

#### *Psychological Factors*

Preliminary research suggests that psychological (Hodges & Marks, 1998; Levitan, Rector, & Bagby, 1998; Wrzeczionek, 2000 ) and personality variables (Geerts et al., 2000; Jang, et al., 1998; Linjaerde et al., 2001; Schuller et al., 1993) that are traditionally associated with nonseasonal depression may have a bearing on SAD. Whether these factors are also related to S-

SAD is unknown (Lam et al., 2001; Young et al., 1991).

Recent studies suggest that like nonseasonally depressed individuals, SAD individuals may employ depressogenic cognitive styles as evidenced in their dysfunctional cognitions (Hodges and Marks, 1998), negative explanatory style (Levitan et al., 1998), and depressogenic coping style (Wrzecionek, 2000). Although S-SAD populations report experiencing depression at times (Lam, et al., 2001), their depressive episodes are less severe and shorter in duration (Lam et al., 2001). This difference may be a function of how SAD and S-SAD populations infer causality for, and cope with their depressive moods. Furthermore, understanding the similarities and differences in the psychological aspects of SAD, S-SAD and nonseasonal depression has bearing on the dual-vulnerability hypothesis that attempts to account for the etiology of both typical and atypical symptoms in seasonal depression and their differential response to phototherapy.

#### *The Dual-Vulnerability Hypothesis*

Almost all of the etiological models of SAD derive from a biological perspective. They include the melatonin hypothesis (Rosenthal et al., 1985), the phase shift hypothesis (Lee et al., 1998; Lewy, Miller, & Hoban, 1987; Meesters et al., 1993;), the photon counting hypothesis (James et al., 1985; Lee et al., 1998; Lee & Chan, 1999; Rosenthal et al., 1985), and the serotonin hypothesis (Lee et al., 1998; Moller, 1992; Rosenthal et al., 1984; Schwartz et al., 1999; Wurtman, 1990). Equivocal evidence exists for all of these explanations and a review of them is beyond the scope of this paper. Interested readers are referred to the aforementioned works for more information (Lee et al., 1998; Toru, 1997).

The commonality among all the biological models is their consideration that the combined interaction between decreased exposure to sunlight and an underlying pathogenic

mechanism is necessary and sufficient to induce the typical and atypical symptoms of SAD (Kane & Lowis, 1999; Lee et al., 1998; Levitan et al., 1997; Young et al., 1991). Yet, these models cannot account for research showing that typical and atypical symptoms might be manifested by two separate processes (i.e., not merely an interaction between biology and environment; Lee & Chan, 1999; Terman et al., 1990; Young et al., 1991). Logically, if a decrease in light is sufficient to induce both typical and atypical symptoms, then both symptoms should show some relation in temporal onset but such is not the case. Typical and atypical symptoms are temporally unrelated in both onset and remission (Partonen & Lonnquist, 1998; Rosenthal et al., 1984; Young et al., 1991), show differential response to light therapy (Lee & Chan, 1999), and are uncorrelated in magnitude (Terman et al., 1990). Such findings suggest that typical and atypical symptoms may be served by different mechanisms (Lam et al., 2001; Lee & Chan, 1999; Young et al., 1991; Young, 1999) rather than one as posited by traditional biological models.

Traditional biological models are also unable to account for the equivalent rate of atypical symptoms reported by individuals with SAD and S-SAD. One remembers that atypical symptoms are apparently characteristic of SAD (Rosenthal, 1984; Sakamoto et al., 1995; Thompson & Isaacs, 1988; Young et al., 1991). Logically, if SAD is a more severe form of S-SAD, then atypical symptoms in individuals with SAD should be more intense than atypical symptoms in individuals with S-SAD. Yet, research shows that both SAD and S-SAD individuals can experience similar rates of depressive atypical symptoms (Lam et al., 2001). Rather, research suggests that SAD is associated with relatively more typical symptoms than S-SAD (Lam et al., 2001; Kasper, Rogers et al., 1989). Taken together, traditional biological models cannot account for disassociation in magnitude and temporal onset of typical and atypical

symptoms in individuals with SAD and S-SAD (Mersch et al., 1999a; Lee et al., 1998; Lee & Chan, 1999).

The dual-vulnerability hypothesis (DVH), as developed by Young, Watel, Lahmeyer, and Eastman (1991) is unlike traditional etiological models in that it views SAD to be the result of two pathogenic processes, rather than a single pathogenic process (Lam et al., 2001; Young et al., 1991; Young, 1999). More specifically, the DVH posits that SAD is the result of a vulnerability to both seasonality (i.e., decreased exposure to light and a pathogenic mechanism) and depression (e.g., depressogenic cognitive patterns). The typical symptoms of SAD are hypothesized to develop from psychological vulnerabilities coupled with stress arising from either the anticipation or the experience of the atypical symptoms (i.e., energy, weight, and sleep disturbance) as stressful (Lam et al., 2001; Young, et al., 1991; Young, 1999). In contrast, the atypical symptoms are thought to arise from seasonality or decreased exposure to light (Lam et al., 2001; Young et al., 1991). Thus, the DVH anticipates typical depressive symptoms to appear either before (in anticipation of) or after (in reaction to) atypical symptoms. This temporal relationship is one that cannot be accounted for by biological models.

The DVH also offers explanations for research showing equivalent rate of atypical symptoms reported by individuals with SAD and S-SAD. That is, the DVH may better account for research showing equivalent atypical symptoms between SAD and S-SAD populations (Lam et al., 2001; Young, 1999) rather than atypical symptoms being relatively more pronounced in SAD than S-SAD populations as predicted by the continuum paradigm (Bauer, 1992; Bauer & Dunner, 1993). Lam and colleagues (2001) extended the DVH by suggesting that an individual's degree of vulnerability to both the seasonality dimension and depression dimension will determine one's symptomatological intensity and clinical status. For example, whereas an



individual with high seasonality and low depressogenic tendencies will generally experience S-SAD, an individual with high seasonality and high depressogenic tendencies will likely experience SAD (Lam et al., 2001; Thase, 1986; Young et al., 1991). The DVH also posits that individuals with nonseasonal depression are low on seasonality, yet highly vulnerable to depression. One remembers that SAD is associated with milder typical symptoms necessary for diagnosis of MDE (and therefore a diagnosis of SAD) than nonseasonal depression (Garvey et al., 1988; Lam et al., 2001; Rosenthal et al., 1984; Thalen, Kjellman, Morkrid, & Wetterberg, 1995). Thus, the DVH posits that individuals with nonseasonal depression are more vulnerable to MDE than SAD individuals (Lam et al., 2001; Young et al., 1991). The DVH posits cognitive vulnerabilities as possible mechanisms creating typical symptoms in SAD, and therefore nonseasonal depression should be associated with significantly more cognitive vulnerabilities (e.g., negative explanatory style, maladaptive coping style). Yet, research shows that SAD is associated with significantly more typical symptoms than S-SAD (Kasper, Rogers, et al., 1989; Lam et al., 2001) and therefore SAD individuals should demonstrate more cognitive vulnerability associated with MDD than would S-SAD individuals. These predictions remain unaddressed.

### *Explanatory Style*

One psychological vulnerability to depression that may differ among individuals with SAD, S-SAD, and nonseasonal populations is explanatory style (Allen et al., 1993; Schuller et al., 1993). Explanatory style refers to a dispositional (Abramson, Metalsky, & Alloy, 1989; Peterson & Seligman, 1984) method of causally explaining negative life events (e.g., Abramson et al., 1989; Abramson et al., 1984; Billings & Moos, 1985; Hilsman & Garber, 1995; Joiner, 2001; Peterson & Seligman, 1984; Ralph & Mineka, 1998; Raps, Peterson, Reinhard, Abramson,

& Seligman, 1982). It is conceptualized as consisting of three attributional (causal) dimensions: internal/external, global/specific, and stable/unstable (Abramson, Seligman, & Teasdale, 1979; Abramson, Garber, & Seligman, 1980, Abramson et al., 1989; Abramson & Martin, 1981; Brewin, 1985; Coyne & Gotlib, 1983; Hilsman & Garber, 1995; Joiner, 2001; Ralph & Mineka, 1998; Raps et al., 1982; Seligman et al., 1982). A depressed person tends to attribute negative outcomes to internal, stable and global causes which in turn can maintain or even exacerbate the depression.

In the case of seasonal depression (SAD and S-SAD), it is not clear whether these individuals might also present the same pattern of attributional style as nonseasonally depressed people. Given that the depressive episodes in SAD and S-SAD are time-limited and related to the weather, it is possible that these individuals might ascribe their depression to external (e.g., decreased exposure to light), unstable (e.g., temporary) and specific (e.g., it happens only during the winter but not in the summer) causes (Allen et al., 1993; Schuller et al., 1993). Furthermore, if one adopts the DVH perspective that SAD and S-SAD individuals are different in their vulnerability to depression, then SAD individuals should have a more depressogenic explanatory style when compared to a S-SAD individual (Lam et al., 2001; Young et al., 1991).

Contrary to logic, Levitan, Rector and Bagby (1998) reported that individuals with SAD and nonseasonal depression do not differ in their explanatory style. However, their findings have to be viewed within the following considerations. First, Levitan and colleagues (1998) failed to assess all three attributional dimensions separately. Instead, they used a composite dimension of stability and generality that may mask differences among the different attributional dimensions. Second, the dimension of internality was not measured. This may be a critical oversight given that SAD is related to an external factor (change in seasons) whereas nonseasonal depression is

often attributed by the depressed individuals themselves to an internal reason (e.g, defective self).

According to the DVH, individuals with SAD and S-SAD differ in their vulnerability to developing typical symptoms of depression (Lam et al., 2001; Young et al., 1991). One remembers that explanatory style is not only affected by the nature of an event (Abramson et al., 1989; Abramson et al., 1994; Peterson & Seligman, 1984), but also by the disposition of an individual to causally explain an event (Abramson, Metalsky, & Alloy, 1989; Peterson & Seligman, 1984). In turn, explanatory style affects the intensity and duration of a depressive episode. More specifically, individuals who tend to employ an internal, stable and global explanatory style are vulnerable to experiencing a lengthy and severe depressive episode (Abramson, Seligman, & Teasdale, 1979; Abramson, Garber, & Seligman, 1980, Abramson et al., 1989; Abramson & Martin, 1981; Seligman et al., 1982). Although S-SAD individuals report experiencing feelings of depression (Lam et al., 2001), S-SAD depressive episodes are significantly less intense and shorter in duration than SAD depressive episodes (Kasper, Rogers et al., 1989; Lam et al., 2001). Research suggests that SAD individuals tend to employ a pessimistic attributional style similar to that of nonseasonally depressed individuals (Levitan et al., 1998). The relatively short and transient depressive episodes of S-SAD (Lam et al., 2001) as compared to SAD may be associated with S-SAD individuals' disposition to use a more optimistic explanatory style. This proposition needs to be addressed.

### *Coping Style*

Another psychological variable that may differ among individuals with SAD, S-SAD, and nonseasonal populations is coping style (Young et al., 1991; Lam et al., 2001). Coping style may be described as a dispositional and stable type of response to stressful events, or in this case,

to depressive moods (Butler & Hoeksema, 1994; Nolen-Hoeksema, 1991; Nolen-Hoeksema, Morrow, & Frederickson 1993; Nolen-Hoeksema & Davis, 1999). Research shows that SAD, nonseasonally depressed and even S-SAD individuals experience depressed moods (Lam et al., 2001). Research generally shows that how one copes with a depressive mood has different implications for the intensity and duration of that depressive mood (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksoma, 1987; 1991; Nolen-Hoeksoma et al., 1993)

Nolen-Hoeksema's (1987; 1991) response styles theory contends that two styles of coping have specific implications for both the duration and the severity of a depressive episode: ruminative and distractive coping (Lyubormirsky & Nolen-Hoeksoma, 1999; Nolen-Hoeksoma, 1987, 1991; Rusting & Nolen-Hoeksoma, 1998). Ruminative coping refers to the use of cognitions and behavior that focus one's attention on possible causes and consequences of one's depression and on one's depressive symptomatology (Nolen-Hoeksema, 1987; 1991; Nolen-Hoeksoma et al., 1993). It may serve to extend and intensify depressive episodes in two ways. First, it may interfere with instrumental behavior (Lyubormirsky et al., 1998; Lyubormisky & Morrow, 1993; Nolen-Hoeksema, 1991; Nolen-Hoeksoma et al., 1993), thereby amplifying feelings of helplessness and depression. For example, Morrow and Nolen-Hoeksema (1990) reports that depressed individuals who were instructed to engage in rumination were less likely to generate solutions to life problems than were individuals who were instructed to engage in distractive coping. Second, it may extend feelings of depression by biasing depressed individuals to remember other negative memories and to generate negative inferences (e.g., negative explanatory style; Lyubormisky & Morrow, 1993; Lyubormirsky et al., 1998; Nolen-Hoeksoma & Lyubormirsky, 1999 Nolen-Hoeksoma et al., 1993; Nolen-Hoeksema, 1991;).

The second coping style, distractive coping, refers to the use of cognitions and behaviors

that distracts one's focus either from possible causes and consequences of one's depression or one's depressive symptomatology by pleasant or neutral activities (Nolen-Hoeksema, 1987; 1991; Nolen-Hoeksoma et al., 1993). Distractive coping may serve to lift depressed moods through allowing an individual to engage in active problem solving (Nolen-Hoeksoma & Morrow, 1991) and through distraction from self-perpetuating negative cognitions and inferences until the depressed mood has subsided (Morrow, 1990; Nolen-Hoeksoma et al., 1993).

Response styles theory recognizes other coping styles including problem solving and dangerous activities. Problem solving refers to the tendency to generate and engage in possible solutions to a given problem (Nolen-Hoeksema, 1987; 1991). Dangerous activities coping style refers to the tendency to engage in activities that are maladaptive and may result in negative consequences for the individual's health and functioning (Nolen-Hoeksema, 1987; 1991). Although problem solving and dangerous activity coping styles may have implications for depression, they are not central to this study and is not discussed further.

The different nature of seasonal and nonseasonal depression may result in different degrees of ruminative coping. Given that SAD is associated with the winter and has a predictable onset and remission, the cause of seasonal depression is readily understandable and may give less reason for the SAD individual to spend time and effort in ruminating about its causes and consequences. In contrast, nonseasonally depressed persons may ruminate more as the reason for their depression is less circumscribed. However, a study by Wrzecionek (2000) found no differences between SAD and nonseasonally depressed individuals in their ruminative or distractive coping style. The findings are unfortunately limited by the small sample size ( $N = 25$ ). The dispositional tendency to engage in distraction and rumination may differ between SAD and S-SAD individuals (Lam et al., 2001). One remembers that rumination is not only

affected by the nature of an event, but also by the disposition of an individual. Furthermore, rumination affects the intensity and duration of a depressive episode. In particular, individuals who tend to ruminate are vulnerable to experiencing a lengthy and severe depressive episode. Although S-SAD individuals report experiencing feelings of depression (Lam et al., 2001; Schlager, 1995), S-SAD depressive episodes are significantly less intense and shorter in duration than SAD depressive episodes. Research suggests that SAD individuals tend to ruminate similarly to seasonal depression individuals. The relatively short and transient depressive episodes of S-SAD, as compared to SAD, may be associated with S-SAD individuals' disposition to use a less ruminative and more distractive coping style in response to their feelings of depression. (Jang et al., 1998; Lam et al., 2001; Young et al., 1991). This possibility remains unexplored.

### *Psychological Stress*

According to the dual-vulnerability hypothesis (Lam et al., 2001; Young et al., 1991), SAD individuals may develop typical depressive symptoms in the presence of stress associated with either the anticipation of winter, or stress associated with atypical symptoms (i.e., sleep, appetite, and energy disturbance). Rosenthal (1993) also stated that SAD individuals both dread and are distressed by the anticipation of winter, and the experience of atypical symptoms associated with SAD. Yet, these observations have yet to be explicitly addressed in the SAD literature.

Psychological stress involves the subjective evaluation of both an event's valence and of one's ability to cope with that event (Cohen, Kessler, & Gordon, 1995; Monroe & Kelley, 1995). Psychological stress results when one determines that a given event is negative and unmanageable (Cohen, Kamarck, & Mermelstein, 1983; Cohen, 1986; Fava et al., 1992; Kuiper, Olinger, & Lyons, 1986; Martin, Kazarian, & Breiter, 1995; Monroe & Kelley, 1995; Otto et al.

1990). Research shows that psychological stress is strongly associated with nonseasonal depression such that greater psychological stress is associated with greater severity of depression (Cohen, Kamarck, & Mermelstein, 1983; Cohen, 1986; Fava et al., 1992; Folkman, Lazarus, Gruen, & DeLongis, 1986; Kuiper et al., 1986) yet such a relation has yet to be fully addressed between SAD and stress.

The perceived stress associated with an event can be affected by both the nature of the event and the dispositional characteristics of the appraiser (Chang, 1998; Cohen et al., 1983; Cohen, 1986). Potentially stressful atypical symptoms such as hypersomnia, weight gain and carbohydrate craving are found in SAD and S-SAD. Hypersomnia has the paradoxical effect of one spending more time in bed yet ultimately experiencing less refreshing sleep, and less energy (Jouvet, 1969). Energy loss and fatigue may effectively handicap an individual in dealing with normal life events that may have been little trouble during the summer: a time when SAD individuals report having abundant energy (Terman, 1988). Weight gain and the craving of carbohydrate saturated foods associated with weight gain may also be perceived as undesirable and negative.

The stress associated with an event is also affected by dispositional characteristics in the individual appraising it. The DVH posits that SAD individuals may be predisposed to appraising both winter and atypical symptoms as stressful. For example, an individual who possesses a negative explanatory style will tend to infer negative consequences and causes for a given event (Levitan, et al., 1998), and hence, be disposed to regarding ambiguous events as negative. Moreover, an individual who tends to employ an ineffectual coping style (i.e., rumination) in the presence of negative events may tend to regard negative events as more unmanageable. Given that preliminary research suggests that SAD individuals evidence these stable cognitive characteristics (i.e, negative explanatory style, Levitan et al., 1998; rumination, Wrzeczionek,

2000) it is likely that SAD individuals are predisposed to evaluating atypical symptoms (or the thought of winter) as stressful. This stress in turn might be associated with typical symptoms evident in SAD individuals.

Extant research designed to explore the possible association between psychological stress and SAD indicate that the two phenomena are unrelated to each other (Nayyars & Cochrane, 1996; Suhail & Cochrane, 1997). For example, Suhail and Cochrane (1997) attempted to track stress levels associated with specific events of individuals with SAD through 1 year using a “Hassles” scale. “Hassles” scales can be characterized as life event inventories designed to render a cumulative stress score based on either the total amount of recently occurred life events and/or the subject’s perception of these events as stressful (e.g., Life Experiences Scale, Sarason, Johnson, and Siegel, 1978, or The Impact of Event Scale, Horowitz, Wilner, & Alvarez, 1979). Results showed that whereas depression levels varied with the seasons, events regarded as stressful on the “Hassles” scale did not. Thus, researchers concluded that individuals with SAD generally become depressed in the absence of stress. Yet this conclusion must be considered with the idea that “Hassles” scales are limited to the specific type of events and ignore the subjective experience of stress (Chang, 1998; Cohen, et al., 1983; Cohen, 1986; Kuiper & Olinger, 1986; Monroe & Kelley, 1995). Given that Suhail and Cochrane (1997) developed their scale from a pre-existing scale that was not designed to explicitly assess potentially stressful events associated with SAD, and that their scale does not consider the *subjective* experience of stress or include *depressive symptoms as stressors in themselves*, the validity of the use of Suhail and Cochrane’s measure with seasonal depression is questionable. The development of a measure appropriate to assess the psychological stress in seasonal depression is warranted.

According to the DVH, individuals with S-SAD have lower depressogenic tendencies that buffer them from experiencing stress from potentially negative events (Lam et al., 2001;



Young et al., 1991). Thus, the relatively less intense depressive episodes experienced by individuals with S-SAD may be a reflection of S-SAD individuals' dispositions to appraise life as less stressful than individuals with SAD. This proposition remains unaddressed.

### *The Present Study*

The present study examined individuals with SAD, S-SAD and nonseasonal depression (depression) to establish similarities and differences among the three groups. A control group consisting of nondepressed individuals (nondepressed) was also included for comparison. Specifically, the four groups were examined for their differences in their typical and atypical symptoms, attributional style, styles of coping with depression, and stress associated with winter-related stimuli. Findings from the present study have implications for distinguishing among SAD, S-SAD and depression, for determining how the different psychological variables may be related to the severity of typical and atypical symptoms, and furthering investigations into the dual vulnerability hypothesis.

The following hypotheses were made:

1. SAD and depressed participants would present with more typical symptoms than S-SAD participants.
2. SAD and depressed participants would have a more depressogenic explanatory style (i.e., more internal, stable, and global) than S-SAD participants, while depressed participants would have a more depressogenic attributional style compared to SAD participants.
3. Depressed participants would use a more ruminative coping style than SAD participants, while S-SAD participants would rely the least on the same coping style.
4. Both S-SAD and SAD participants would use a more distracting strategy to cope with their depression compared to depressed.

5. SAD participants would report perceiving winter-related stimuli as more stressful than other groups.

## Method

### *Participants*

Participants consisted of 208 individuals recruited from the Lakehead University's Psychology classes and the general community of Thunder Bay. One hundred and eight of them fulfilled classification criteria presented in Table 1 and were allotted to either the SAD ( $n = 32$ ), the S-SAD ( $n = 31$ ), the nonseasonally depressed (depressed,  $n = 20$ ) or the nondepressed group (nondepressed,  $n = 25$ ).

### *Classification*

The assessment and criteria for classification of the participants (see Table 1) were guided by research in the literature and the Canadian Consensus Guidelines for the Treatment of Seasonal Affective Disorder, (Lam & Levitt, 1999). The SAD and depressed group had to meet the depression criteria that consisted of a high score of 19 or greater on the 28-item Hamilton Depression Rating Scale (HDRS-28 – see Appendix 1 Section C below for more information) plus a positive identification on MDE indicators consisting of a minimum of five symptoms including depressed mood and/or loss of interest or pleasure, and impairment. The S-SAD and the nondepressed groups did not meet the MDE depression criteria. The SAD and S-SAD groups had to meet the seasonality criteria that consisted of five characteristics: a seasonal pattern to their symptoms, a seasonal pattern in the last two years, a high GSS, impairment, and the absence of a seasonal stressor as presented in Table 1. In order to meet the seasonality criteria, all of these criteria had to be present. In the S-SAD group, the GSS and the impairment were considered jointly. The joint criteria included a GSS score of 9 to 11 with at least a mild impairment score, or a GSS of 11 or higher with an impairment that was

either absent or mild. For depressed and nondepressed groups, they must not have a seasonal pattern to their symptoms, and therefore their GSS scores were not counted as a selection criterion.

### *Materials*

#### *Research Questionnaire (Appendix 1)*

Information was collected with a research questionnaire that was used in a larger project on SAD. It has several sections; however, only the sections that are relevant to the present study are discussed below while those that are pertinent to the other studies in the larger project are excluded from the discussion.

*Section A (see Appendix 1).* This section queried about the subject's demographic information, residential pattern (i.e., duration of residence at permanent address and areas of annual dwelling that may contribute to seasonal changes), and substance use pattern (including alcohol, drugs, and medication) that may account for some of the depressive symptoms.

*Section B (see Appendix 1).* Items in this section were geared towards the assessment of seasonality. Some items were derived from Rosenthal's (1993) SPAQ while other items were developed specifically to address DSM-IV-TR seasonal specifier criteria and to obtain more information. Those that are derived from the SPAQ are identified as such below.

Question 1 was derived from the SPAQ to ascertain the pattern of seasonality with respect to typical and atypical depressive symptoms. Whereas SAD and S-SAD individuals endorsed symptom exacerbation during winter and fall months and symptom alleviation during spring and fall months, depressed and nondepressed individuals endorsed no characteristic seasonal pattern.

Question 4 was derived from the original Rosenthal's SPAQ. It assessed the degree of seasonality by summing the degree of change scores over six symptoms. The degree of change

was rated on a 5-point scale where 0 indicated no change and 4 indicated extremely marked change. The sum of the ratings yielded a Global Seasonality Score where a score of 0 to 7 suggested no seasonality and a score of 12 or more suggests high seasonality.

Question 5 was derived from the SPAQ to assess degree of impairment associated with the seasonal changes.

Question 6 and 7 were developed by Wesner and Tan (1999) to assess for the presence of seasonal stressors as the DSM-IV criteria specifies that the seasonal changes could not be accounted for by seasonal stressors.

*Section C (see Appendix 2).* The 28-item Hamilton Depression Rating Scale (HDRS-28) was used to assess severity of both atypical and typical depressive symptoms. The HDRS-28 is technically the Self-Assessment Mood Scale for SAD (SAM-SAD, Rosenthal, 1993) that is adapted from the Structured Interview Guide for the Hamilton Depression Rating Scale - Seasonal Affective Disorder version (Williams et al., 1988). The HDRS-28 is based on the 21-item Hamilton Depression Rating Scale (Hamilton, 1960, 1967) that was deemed to be insufficient for assessment of SAD (Rosenthal, 1989; Wirz-Justice & Anderson, 1990) because it did not address atypical symptoms. Consequently, an addendum of 7 more items to address the atypical symptoms was developed to expand the HDRS-21 to HDRS-28. Each item on the HDRS-28 is rated on a 5-point scale that ranges from 0 (not at all) to 4 (marked or severely).

The HDRS-28 was scored in three ways. First, a typical symptom score (HDRS-A) was obtained by summing the responses to items 1-21. Second, an atypical symptoms score (HDRS-B) was computed by summing items 22-28. Finally, an overall score (HDRS-T) consisting of the summing across items 1-28 was obtained. Higher HDRS scores indicate greater depressive severity.

*Section D (see Appendix 3).* The Response Styles Questionnaire (RSQ) contained 41

items designed to assess dispositional coping styles to depressive episodes (Nolen-Hoeksema, 1990). More specifically, the RSQ was composed of four scales that assessed ruminative coping, distractive coping, problem-solving coping, and dangerous activities coping. The Ruminative coping scale included items designed to assess the tendency to focus on self, symptoms, and possible causes and consequences of depressive mood. The Distracting Responses Scale included items designed to assess the tendency to engage in pleasant and benign distractive activities in response to a depressed mood. The Ruminative Response Scale and the Distracting Response Scale possess acceptable internal consistency at .89 and .80, respectively (Nolen-Hoeksema & Morrow, 1993). The RSQ shows acceptable test-retest reliability (Just & Alloy, 1997), and acceptable predictive validity (Butler & Nolen-Hoeksema, 1994; Butler & Nolen-Hoeksema, 1990 in Nolen-Hoeksema & Morrow, 1991; Just & Alloy, 1997; Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991).

Other scales included on the Response Styles Questionnaire included the Dangerous Activities Scale and the Problem Solving Scale. The Dangerous Activities Scale assessed the tendency to engage in reckless or dangerous activities in response to the participant's depressive symptoms. The Problem Solving Scale assessed responses that served to actively solve some problems that were related to the participant's depressive symptoms. Convergent and predictive validity for these scales have been found to be acceptable (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema & Morrow, 1991).

Each item on the RSQ was a statement that encapsulated one of the four coping responses to a depressed mood. Each item contained a response scale ranging from 1 (never) to 4 (always). One's response indicated the degree to which one tends to employ the (item's) coping style in response to a depressive mood. To score each scale on the RSQ one merely sums each scale. A higher score on a particular scale indicated a greater tendency to employ that particular coping

style in response to a depressive mood.

*Section E (see Appendix 4).* This section consisted of diagnostic questions designed to screen for the presence of Major Depressive Episode (MDE) in accordance with criteria stipulated in DSM-IV (APA, 1994). Questions were extrapolated from the computerized version of the NIMH Quick Diagnostic Interview Schedule-III-R (Robins, Heltzer, Croughan, & Ratcliff, 1981). These questions had been cross-checked from the current Structure Clinical Interview for DSM-IV (First, Gibbon, Williams, & Spitzer, 1995). Additional questions had been included to assess impairment, the exclusionary criteria of depressive symptoms arising from medical conditions, bereavement, and substance use, previous occurrence of MDE, and whether the participant believed that he/she was experiencing the symptoms of MDE at the time of assessment.

*Section F (see Appendix 5).* The Cognitive Styles Questionnaire (Abramson, Metalsky, & Alloy, 1990) is a measure of cognitive vulnerability to depression. It is a revised version of the Attributional Style Questionnaire (ASQ; Seligman, et al., 1979) that has good psychometric properties (Peterson, 1991; Peterson & Seligman, 1984 for a review). Modifications to the ASQ that were incorporated into the CSQ include the addition of extra hypothetical events to make a total of twelve positive and twelve negative achievement and interpersonal events, and the addition of three inferences to each item concerning the consequences, self-worth implications, importance of each hypothetical event. The CSQ yields attributional dimensions of internality, stability, globality, consequences, and self-worth implications. Both convergent and predictive validity of the CSQ is acceptable (Just & Alloy, 1997). Predictive validity for the CSQ is also discussed in Alloy, Abramson, Murray, Whitehouse, and Hogan (1997). Internal consistency of both the twelve positive and the twelve negative events is acceptable at alphas of .86 and .88, respectively (Just & Alloy, 1997).

For the purpose of the present study, only the 12 negative events and the dimensions of Internality, Stability, and Globality were of interest. The Internality dimension was assessed with items (b) in each hypothetical situation, Stability dimension with items (c), and Globality with items (d). Each item was scored on a 1-7 rating point scale. Scores of each attributional dimension was obtained by summing the ratings of the relevant items across all hypothetical situations. Higher scores on each dimension indicated greater ascription to that particular dimension of explaining an event outcome.

*Section G (see Appendix 6).* The Winter Stress Scale (WSS) was specifically developed for the present study to investigate the respondent's dread of winter (item 1), stressfulness of atypical symptoms during the winter (item 2), degree of impairment caused by the atypical symptoms experienced during the winter (item 3), rumination strategy used to cope with the winter atypical symptoms (item 4), and psychological stress in the winter/fall (item 6). These items are discussed in greater detail below.

- Item 1 measured dread of winter on a scale of 1 (not at all) to 5 (extremely). Higher scores indicated greater level of dread.
- Item 2 first assessed whether the respondent experienced the atypical symptoms during the winter, and if s/he did, the degree of stress (on a rating scale of 1 to 5) associated with each symptom. Higher scores indicated higher stress. Absence of a particular atypical symptom was indicated by a score of "0".
- Item 3 tapped into the degree of impairment caused by the winter atypical symptoms. This construct was measured on a scale of 1 to 5. Higher scores indicated greater impairment.
- Item 4 had six questions that tapped into the rumination coping strategy during

fall/winter. Each question was rated on a 5-point rating scale. Higher summed scores showed greater use of rumination. The conceptual underpinning of these questions were based on Nolen-Hoeksema's (1987) theory that depressed people's tendency to ruminate on the negative and engage in wishful thinking maintain and/or exacerbate their depression.

- Item 6, which was based on the Perceived Stress Scale (PSS, Cohen et al., 1983), measured the extent to which the respondent felt life to have been more unpredictable, uncontrollable, and unmanageable in the fall/winter than usual. The questions in item 6 were almost identical to the PSS with two differences. The PSS used a frequency scale of measurement ("how often") while Item 6 measured the degree of agreement with the statement on a 5-point scale. The timeframe assessed by the PSS was within the last month whereas item 6 compared the fall/winter functioning of the respondent against his/her usual functioning. The PSS has been found to predict psychological outcomes independently of psychopathology (Cohen, 1986; Cohen et al., 1983, 1993), and its predictive validity has been supported (Cohen, 1993; Fava et al., 1992; Pbert et al., 1992). Congruent with the PSS, item 6 had seven questions that were negatively worded (question a, b, c, h, k, l, and n) and seven questions that were positively worded (question d, e, f, i, k, and m) and needed to be reverse-scored. Ratings across the fourteen items were then summed, after the appropriate reverse scoring had been performed. Higher summed scores reflected greater perceived stress in fall/winter.

### *Computerized SCID*

The computerized version of SCID Patient Questionnaire (First, Gibbon, Williams, & Spitzer, 1999) was used to screen for DSM-IV Axis I symptoms to determine whether or not the



respondent met the Major Depressive Episode diagnostic criteria.

### *Recruitment of Participants*

Recruitment efforts of the present study were combined with the recruitment efforts of another study on seasonal affective disorder. The populations recruited included the Lakehead University undergraduate students and the general population of Thunder Bay. To reach the general community, recruitment posters (see Appendix 7) were posted around the campus and in public places in the community (e.g., supermarkets, grocery stores, malls). Advertisements (see Appendix 7) were also taken out on the community channel of cable television, and the SAD projects were featured on Thunder Bay Post and the Chronicle Journal. For the campus populations, recruitment efforts were also carried out via emails or the daily "Communications Bulletin" that were sent out electronically. Psychology students were approached during their classes and were informed of the present study. Research questionnaires were provided in the classes for students to fill out on their own time. Students were also instructed on where and how to return completed questionnaires. In all of the aforementioned recruitment efforts, a contact name and telephone number were given.

All individuals who responded to the recruitment efforts were informed that the present study investigated the winter emotional, cognitive and behavioural experiences of people who are depressed, seasonally depressed, subsyndromal seasonally depressed, and nondepressed, and that it required individuals whose symptoms and seasonality ranged from none to severe. Introductory Psychology student participants received two bonus points towards their course marks. The remaining participants were entered into three random prize draws of \$100 each.

### *Study Procedure*

Individuals who participated in the present study filled out a consent form (see Appendix

9), and completed the Research Questionnaire that contained all the measures in this study (Appendices 1 to 6). They also were given a page of debriefing (Appendix 10) and a list of counseling/therapy resources (Appendix 11) in the event they wished to access professional help. Those who met the criteria for depression on the questionnaire were asked to return, ostensibly to follow up on their responses to the questionnaire. They were administered the computerized SCID to determine whether they met the DSM-IV major depressive episode criteria. No diagnosis was given out to participants. Those who wished to see a clinician for further information or referral had the opportunity to talk to Dr. Tan, the project supervisor and a clinical psychologist. A summary of the findings from this present study were mailed out to interested participants upon the completion of the study.

## Results

### *Research Design*

The research design in this study consisted of one independent variable, clinical group (group) with four levels: seasonal affective disorder (SAD), subsyndromal seasonal affective disorder (S-SAD), nonseasonally depressed (depressed) and the control nondepressed group (nondepressed). The dependent variables are presented below:

- i. The four subscales on the Response Styles Questionnaire consisting of Ruminative Coping (Rumination), Distractive Coping (Distract), Problem-Solving Coping (Problem-Solve), and Dangerous Activities Coping (Danger).
- ii. Three subscales on the Cognitive Styles Questionnaire that measure, respectively, the dimensions of Internality, Stability, and Globality in explanatory style.
- iii. Dread of winter (W-Dread) measured by item 1 in the Winter Stress Scale
- iv. Atypical depressive symptoms specifically associated with winter (W-Atypical) as

- measured by item 2 in the Winter Stress Scale
- v. Impairment associated with the experience of atypical symptoms during the winter time (W-Impair) as measure by item 3 in the Winter Stress Scale
  - vi. Ruminative coping style specifically associated with winter (W-Ruminate) as measured by item 4 in the Winter Stress Scale
  - vii. Perceived stress during the winter (Perceived Winter Stress) as measured by item 6 in the Winter Stress Scale.

### *Sample Characteristics*

A total of 203 individuals responded to the recruitment efforts in this study. Of these, 104 met the inclusion criteria of the four clinical groups as specified previously in Table 1. Description of the groups and the cell sizes are presented in Table 2.

An ANOVA conducted on Age as a function of group in variables revealed a significant effect,  $F(3, 104) = 4.25, p < .01$ , effect size  $\eta^2 = .11$ . Post-hoc Scheffe test revealed that SAD was significantly higher than S-SAD. All other comparisons were non-significant.

An ANOVA conducted on HRDS-T as a function of Group revealed a significant effect,  $F(3, 104) = 117.96, p < .001, \eta^2 = .77$ . Post- hoc Scheffe test revealed that SAD scored the highest among all groups, followed by Depressed who were significantly higher than S-SAD and Nondepressed. The S-SAD and Nondepressed were not different from each other. No other means comparisons were significant.

An ANOVA conducted on HRDS-A as a function of Group revealed a significant effect,  $F(3, 104) = 87.80, p < .001, \eta^2 = .72$ . Post-hoc Scheffe test revealed that both the SAD and Depressed scored higher than S-SAD and Nondepressed. No other significant means comparisons were found.

An ANOVA performed on the HRDS-B showed a significant group effect,  $F(3, 104) = 39.79, p < .001, \eta^2 = .54$ . Scheffe test indicated that the SAD was the highest among all the groups, followed by Depressed who were significantly higher than S-SAD and Nondepressed. No other means comparisons were significant.

An ANOVA performed on the GSS showed a significant group effect,  $F(3, 104) = 79.22, p < .001, \eta^2 = .70$ . Scheffe test indicated that the SAD was the highest among all the groups, followed by S-SAD, which was higher than Depressed, which was higher than Nondepressed. The remaining means comparisons were nonsignificant.

Multicollinearity and singularity of the HDRS scales were examined through correlation matrices according to Tabachnick and Fidell (2001, pp. 82-83). A correlation in excess of .90 indicates multicollinearity and singularity, meaning that the two variables essentially provide the same information. Thus, only one of the two correlated variables need be used in the analysis. An inspection of the correlations involving the HDRS scales showed that HDRS-A and HDRS-T were significantly and highly correlated ( $r = .97, p < .001$ ) with each other. Therefore, as the HDRS-T (total score of the HRDS) was less relevant than the HDRS-A (typical symptom score of the HRDS) as the theoretical interest in the study was a comparison between the typical (HRDS-A) and atypical (HRDS-B) depressive symptoms, HRDS-T was dropped from subsequent analysis. The correlation between HRDS-A and HRDS-B was significant but not high enough ( $r = .65, p < .001$ ) for concerns to be raised about multicollinearity and singularity.

#### *Overview of the Analyses*

Three types of analytic strategy were used to analyse the data. First, separate multivariate analysis of variance (MANOVA) with Group as the independent variable was used to analyze the data for coping styles (Rumination, Distract, Prob-Solve, and Danger) and for cognitive

styles (Internality, Stability, and Globality). Pillai's trace was used to determine multivariate significant effects as it the most robust criterion for use with unequal group sizes (Tabachnick & Fidell, 2001, p. 348). The strength of association between the independent variable and set of dependent variables was denoted by  $\eta^2$ , the proportion of variance accounted for in the linear combination of dependent variables (Tabachnick & Fidell, 2001, p. 338-339).

When a significant multivariate effect was found, the results were followed up with Discriminant Function Analysis to determine the relative contribution of the individual dependent variables to the group differentiation (Tabachnick & Fidell, 2001, pp. 483-488). Although there are two other alternate strategies, Univariate  $F$  with a Bonferroni type adjustment and Roy-Bargmann Stepdown Analysis, for assessing the dependent variables, they were not utilized here for the following reasons. The Univariate  $F$  test is typically problematic for use with correlated dependent variables because of overlapping variance that can led to the mistaken interpretation that the independent variable affects different dependent variables. As well, there is a danger of inflated Type I error because the error rate cannot be adjusted directly with correlated univariate  $F$ 's (Tabachnick & Fidell, 2001, p. 349). The Roy-Bargman Stepdown Analysis would be suitable for correlated dependent variables if there were to be a logic to the prioritizing of the dependent variables in the sequential multiple regression (Tabachnick & Fidell, 2001, p. 350). However, given that none of the dependent variables in the multivariate analysis in this study have a theoretical or temporal priority, the Roy-Bargman Stepdown Analysis was not used. Instead, Discriminant Function Analysis was the choice of analytic strategy because it takes into account the correlated dependent variables and does not require a priority ordering. The loading matrices were examined where the dependent variables with loadings of .33 or greater were interpreted (Tabachnick & Fidell, 2001, p. 485).

To determine how the Groups differed from each other on the dependent variables that were deemed to contribute to group discrimination, as assessed by the Discriminant Function Analysis, post-hoc Scheffe comparison tests were used. Scheffe test was chosen over other post-hoc tests because it is most conservative (Tabachnick & Fidell, 2001, p. 51).

The second type of analytic technique used was the univariate analysis of variance (ANOVA) with Group as the independent variable to assess the data associated with W-Dread, W-Atypical, W-Impair and W-Ruminate. The strength of association between the independent variable and dependent variable was indicated by  $\eta^2$  (Tabachnick & Fidell, 2001, p. 52). Post-hoc Scheffe comparison test was used to follow up on significant univariate results to determine how the groups differ from each other.

Finally, a Principal Components Analysis was performed on the Perceived Winter Stress data to determine the number of underlying components. A MANOVA was then performed on the principal components with the aforementioned tests to follow up on a significant multivariate effect.

#### *Pre-analysis issues*

Prior to statistical analyses, all dependent variables were examined for accuracy of data entry, missing values, and fit between their distributions and the assumptions of multivariate and univariate analysis.

#### *Missing Data*

The data set was examined for missing values. It was noted that one case from the S-SAD group had more than 50% of responses missing. Hence this individual was excluded from the analyses, resulting in the cell size of 32 for the S-SAD group. The rest of the participants, for the most part, had complete set of responses. In some cases where there were missing values on

any questionnaire item, the group mean for that item was substituted for that missing value. This mean substitution strategy serves to preserve the participant in the data without changing the mean of the distribution for that variable of that particular group (Tabachnick & Fidell, 2001, p. 62).

#### *Univariate Outliers*

Within-group univariate outliers, defined as cases with standardized scores greater than  $z = 3.29$  (Tabachnick & Fidell, 2001, p. 67) were identified and their raw scores recoded to one unit higher than the next most extreme score in their distribution (Tabachnick & Fidell, 2001, p. 71). This strategy serves to reduce the influence of these cases while still preserving their deviancy with respect to the other cases within the group (Tabachnick & Fidell, 2001, p. 71). In the present study, one nondepressed and three S-SAD cases on the HRDS-B variable, and one S-SAD on the Internality variable were identified as univariate outliers and were handled accordingly.

#### *Multivariate Outliers*

Within-group multivariate outliers were looked for using two criteria: the Mahalanobis distance and Cook's D. A multivariate outlier, as assessed by the Mahalanobis distance strategy, is a case whose distance from the centroid of all remaining cases within a group is greater than a critical value defined by a  $\chi^2$  critical value (Tabachnick & Fidell, 2001, p. 68). This critical value is dependent upon the group size, number of dependent variables involved, and the alpha level and can be looked up in a table provided by Stevens (1986, p. 93). In Cook's D strategy, a multivariate outlier is defined as a case that has a Cook's D greater than 1.00. These cases are considered outliers by virtue of their influence and deviancy when compared against the other cases within the group (Tabachnick & Fidell, 2001, p. 69). For the purpose of the present study,

a multivariate outlier was considered to be any case that either exceeded the critical value for the Mahalanobis distance or Cook's D, and would be deleted if found. However, no multivariate outliers were found, and hence no cases were deleted.

#### *Normality, Linearity, and Homoscedasticity*

Normality was assessed via a within group detrended normality plot for each dependent variable, while linearity and homoscedasticity were examined via within-group bivariate scatterplots involving dependent variables that were analyzed simultaneously in multivariate analyses (Tabachnick & Fidell, 2001, pp. 72-80). Analyses revealed that these assumptions were met.

#### *Multicollinearity and Singularity*

Multicollinearity and singularity were examined through correlation matrices of dependent variables for correlations in excess of .90 (Tabachnick & Fidell, 2001, pp. 82-83). As can be seen from Table 3, no cases involving multicollinearity and singularity were found.

#### *Main Analyses*

The descriptive statistics of the dependent variables as a function of Group are presented in Table 4. The results of the statistical analyses are discussed below.

#### *Response Styles Questionnaire*

A MANOVA performed on the four coping styles (Rumination, Distract, Prob-Solve, and Danger) as a function of Group showed a significant effect,  $F(12, 309) = 6.12, p < .001, \eta^2 = .192$ . Discriminant Function Analysis showed that there was one discriminant function that significantly contributed to group discrimination,  $\chi^2(12) = 81.10, p < .001$ , and that accounted for 94.50% of the between-group variability. The loading matrix showed that the dependent variables that contributed to group discrimination were Rumination (loading = .99) and Danger



(loading = .36). Scheffe tests revealed that on Rumination, the SAD group scored higher than the depressed group, that in turn was higher than both the S-SAD group and the nondepressed group (see Table 4). There was no difference between the S-SAD and nondepressed group. The Scheffe posthoc means comparison also showed that the SAD group was higher on Danger than both S-SAD and nondepressed groups (see Table 4). No other means comparisons were significant.

#### *Cognitive Styles Questionnaire*

A MANOVA carried out on the three explanatory styles, Internality, Stability, and Globality, showed a significant Groups effect,  $F(9, 312) = 3.01, p < .01, \eta^2 = .08$ . Discriminant Function Analysis showed one significant discriminant function,  $\chi^2(9) = 26.75, p < .01$ , that accounted for 72.40% of the between-group variability. The loading matrix indicated that Globality (loading = .98) and Stability (loading = .69) significantly contributed to the linear combination separating the groups. Scheffe post-hoc comparisons revealed that on Globality, SAD group scored higher than the S-SAD and nondepressed groups. No other means comparisons were significant. On Stability, the S-SAD scored lower than the SAD and nondepressed groups. No other significant means comparisons were found.

#### *Dread of Winter (W-Dread)*

An ANOVA performed on W-Dread as a function of Group revealed a significant effect,  $F(3, 104) = 12.91, p < .001, \eta^2 = .27$ . Post-hoc Scheffe test showed that the SAD group scored higher than the depressed, S-SAD, and nondepressed groups (see Table 4). All other means comparisons were nonsignificant.

#### *Stress Associated with Winter Atypical Symptoms*

An ANOVA on W-Atypical as a function of Group revealed a significant effect,  $F(3,$

104) = 42.55,  $p < .001$ ,  $\eta^2 = .55$ . Post-hoc Scheffe tests indicated that the SAD group scored significantly higher than the depressed group, which in turn was significantly higher than both the S-SAD and nondepressed groups (see Table 4). No other means comparisons were significant.

#### *Impairment During Winter (W-Impair)*

An ANOVA performed on W-Impair as a function of Group showed a significant effect,  $F(3, 104) = 55.00$ ,  $p < .001$ ,  $\eta^2 = .61$ . Scheffe tests indicated that the SAD group scored highest, followed by the depressed group that in turn was higher than both the S-SAD and nondepressed groups (see Table 4). No other means comparisons were significant.

#### *Ruminative Coping Style in Wwinter (W-Ruminate)*

An ANOVA on W-Rumination showed a significant Group effect,  $F(3, 104) = 48.97$ ,  $p < .001$ ,  $\eta^2 = .59$ . Post hoc Scheffe revealed that the SAD group scored higher than the depressed group, that in turn was higher than both the S-SAD and nondepressed group (see Table 4). No other significant means differences were found.

#### *Perceived Stress During the Winter (Perceived Winter Stress)*

A Principal Components Analysis with varimax rotation was performed on the Winter Perceived Stress Scale (WPSS) that measured perceived winter stress. This was done to determine whether the modified items constituting the WPSS demonstrated similar factor properties to those evidenced in the original Perceived Stress Scale (PSS; Hewitt et al., 1992; Pbert et al., 1992). A demonstrated factorial similarity between the two scales would suggest that the interpretation offered by previous researchers would apply to both scales.

The results from the WPSS (see Table 5) bore striking similarity to the PSS reported by Pbert et al. (1992) and Hewitt et al. (1992). There were two factors, each accounting for 38%

and 27% of the variance, respectively. As in the PSS, Factor 1 was composed of items related to negative affect items and the Factor 2 consisted mainly of perceived ability to cope with stressors. Thus, Factor 1 could be interpreted as negative affective reactions to winter stimuli and Factor 2 could be interpreted as perceived ability to cope with extant winter stressors. The WPSS was subsequently analyzed in the present study as two factors.

A MANOVA was performed on Factor 1 and Factor 2 as a function of Group. Pillais Trace indicated a significant multivariate effect,  $F(6, 208) = 14.54, p > .001, \eta^2 = .30$ . Discriminant Function Analysis showed that there were two discriminant functions that contributed to group discrimination,  $\chi^2(6) = 83.58, p < .001$ . After removal of the first discriminant function, there was still a significant association between Group and the dependent variables,  $\chi^2(2) = 8.09, p < .05$ . The two discriminant functions accounted for 93% and 7%, respectively, of the between-group variability. An examination of the loading matrix showed that the only factor that significantly loads on the first discriminant function is Factor 1 (loading = .96), whereas the only factor that significantly loads on the second discriminant function is Factor 2 (loading = .96). In short, the Discriminant Function Analysis showed that both Factor 1 and Factor 2 were responsible for group discrimination.

Post-hoc Scheffe test revealed that on Factor 1, the SAD group scored higher than the depressed group, that in turn scored higher than both the S-SAD and nondepressed groups (see Table 4). S-SAD and nondepressed groups did not differ significantly from each other, and no other significant means comparisons were found. On Factor 2, the S-SAD group was lower than the SAD group. All other comparisons were nonsignificant.

### Discussion

This study investigated differences among individuals with seasonal affective disorder

(SAD), subsyndromal seasonal affective disorder (S-SAD), nonseasonal depression and no depression, in their depressogenic explanatory style, coping styles in response to depressed moods, and stress reactions to winter.

Hypothesis 1, which stated that SAD and depressed persons would present with more typical depressive symptoms than S-SAD persons was supported. The findings are consistent with the Lam et al.'s (2001) study. As well, the SAD group reported more severe atypical symptoms than did the depressed group, who in turn reported more severe atypical symptoms than the S-SAD group. For both typical and atypical symptoms, the S-SAD group did not differ from the nondepressed group. These findings are not entirely unexpected given that the SAD and depressed groups were selected on the basis of their more severe (typical and atypical combined) depressive scores on the Hamilton than the S-SAD and nondepressed. However, when one examines the typical and atypical symptoms separately to determine how the groups differ, it would seem that the S-SAD group can be distinguished from the SAD and depressed groups by their less severe typical and atypical scores, and that the SAD group can be distinguished from the depressed group by their greater atypical scores. However, no distinction between the S-SAD and nondepressed individuals can be made on the basis of either the typical or atypical scores.

The results obtained with the typical and atypical symptoms have implications for the dual vulnerability hypothesis (DVH). The DVH posits that atypical symptoms are associated with seasonality (Lam et al., 2001; Young et al., 1991). Accordingly, one would expect that those high in seasonality (i.e., SAD and S-SAD) would report more severe atypical symptoms than those low on seasonality (i.e., depressed and nondepressed). Findings in this study revealed a mixed picture. Indeed that the SAD group had more severe atypical symptoms than the

depressed and nondepressed groups, but the depressed group had *more* severe atypical symptoms than the S-SAD group despite having a *lower* seasonality score (as measured by the Global Seasonality Score or GSS). Why this is so is puzzling. A possible explanation may lie with activity level during the fall/winter period. Research shows that physical activity decreases the number of the atypical symptoms represented on the Hamilton and the severity of the GSS (Pinchasov, Shirguja, Grischin, & Putilova 1998). If the S-SAD individuals were more physically active than the depressed, they may consequently report less atypical symptoms at the time of the study, even though their self-reported GSS was higher. It should be noted that the GSS measured with the Seasonal Pattern Assessment Questionnaire (SPAQ) gives a *general*, and *not current*, measure of the seasonal change experienced by the respondent, whereas the atypical symptoms assessed were *current* and within the last two weeks. Hence, the timeframe assessed for the atypical symptoms and for the GSS are different. To clarify this issue, future research would need to examine the effect of physical activity on the current GSS and current atypical symptoms of seasonal and nonseasonal individuals.

Hypothesis 2 stated that the depressed individuals would adopt a more depressogenic explanatory style (more internal, stable, and global) than the SAD individuals, who in turn would adopt a more depressogenic explanatory style than the S-SAD individuals. This hypothesis was partially supported in the study.

First, only the stable and global dimensions played a part in discriminating among the groups, while the internal dimension did not. This is a surprising finding given that mood and behavioural changes experienced by both S-SAD and SAD groups are associated with an obviously external cause (i.e., the change of the seasons). In comparison, depressed persons whose depressive episodes do not follow the seasonal changes have no comparable external

reasons to which they can attribute their disorder. The measure that was used to assess cognitive vulnerability in the present study may account for the findings. The internal/external dimension on the Cognitive Styles Questionnaire does not distinguish between characterological and behavioural internal explanatory styles (Anderson, Miller, Riger, Dill, & Sedikides, 1994). The depressed individuals might make internal attributions that are related to a characterological and uncontrollable flaw, leading to guilt and feelings of one deserving punishment that are often seen in nonseasonal depression (Lewinsohn & Gotlib, 1995). In comparison, the SAD and S-SAD individuals might make internal attributions that are related to a behavioural and controllable deficit, such as not engaging in sufficient physical activity or getting out more to be exposed to sunlight. Hence while the depressed, SAD, and S-SAD individuals may all adopt an internal attributional style, the type of internal attributions that they make (characterological versus behavioural) may differ on the dimension of controllability. Possibly, controllability may be a more relevant dimension than internality to explore for differences in explanatory style between seasonal and nonseasonal depression.

Second, it was expected that the most depressogenic explanatory style would be adopted by the depressed individuals, followed by the SAD group, and the S-SAD group the least. The rationale for this prediction lies with previous reports that SAD is associated with milder typical symptoms that are necessary for a diagnosis of major depression when compared to nonseasonal depression (e.g., Lam et al., 2001; Thalen et al., 1995), but is associated with more typical symptoms than S-SAD (Kasper, Rogers et al., 1989; Lam et al., 2001). A logical extension of this observation would be that nonseasonally depressed individuals would be more vulnerable to major depression and to the associated cognitive vulnerabilities (e.g., negative explanatory style, maladaptive coping style), followed by the SAD group and finally the S-SAD group. What the

present study revealed was that the SAD group held more stable and global explanations than did the S-SAD and nondepressed groups, which is expected. The depressed individuals did not differ from either the SAD or S-SAD.

The above pattern of findings is difficult to explain. Perhaps the expectation of repeated depressive episodes is more salient for the SAD group than for the S-SAD group because the symptoms for the SAD group are more severe and impairing. Therefore, the SAD group might hold a more stable and global view of their depressive episodes. The degree of the SAD stable and global belief is no different from that of a nonseasonally depressed individual whose symptoms are equally severe and who might assume stability of the depression because s/he is unable to predict its remission. More specifically, a SAD person may hold a stable belief that the depression would come back every year whereas a nonseasonally depressed person may hold an equally stable belief that there is no foreseeable relief from the current depressive episode. Additionally, both the SAD and depressed persons might hold equally global attributions in that the effect of the depressive symptoms on their functioning is wide-ranging in various spheres of their lives given the equivalent severity of their symptoms.

The type of explanatory style (less global and less stable) held by the S-SAD compared to the SAD and depressed individuals may act as a protective factor against depression. Research shows that although both S-SAD and SAD persons do experience negative mood and behavioural changes associated with the onset of winter (Lam et al., 2001; Schlager et al., 1995), the changes for the S-SAD person do not last long enough to warrant the diagnosis of Major Depressive Episode. As well, it has been shown that a stable and global explanatory style predicts a lengthier depressive episode than an unstable and more specific explanatory style (Abramson et al., 1989; Abramson et al., 1994; Peterson & Seligman, 1984). Thus, the more

adaptive explanatory style by the S-SAD person may play a part in protecting them from major depression. If this were true, the findings in this study hold implications for the use of cognitive therapy with seasonal individuals.

Hypothesis 3 predicted that depressed individuals would engage in rumination as a coping response to their depression more so than SAD, who in turn would engage in this coping style more so than S-SAD. This hypothesis was partially supported in that the SAD used rumination more so than the S-SAD. However, the unexpected finding was that the SAD also ruminated more than the depressed persons. Research shows that ruminative coping both lengthens and amplifies an otherwise mild depressive episode (Nolen-Hoeksema, 1987). Indeed, the SAD individuals in the present study had higher depressive scores (typical and atypical combined) than did the depressed or S-SAD individuals. This may be associated with the greater number of women in the SAD group ( $n = 25$ ) than in the depressed group ( $n = 13$ ). In nonclinical samples, women tend to have higher depression scores than men and they also tend to ruminate more (Nolen-Hoeksema, 1987).

The above findings have implications from an etiological perspective. It has been thought that SAD may develop from an interaction between decreased light and a neuropathogenic mechanism which together precipitate mood and behavioural change (Kane & Lewis, 1999; Levitan et al., 1998). Therefore, advocates of this paradigm have predicted that the SAD depressive episode will occur regardless of the SAD individual's cognitive variables such as coping strategy. Such may not be the case when one considers that S-SAD individuals, who have been found in the present study to ruminate less than the SAD individuals, tend not to develop major depression (Lam et al., 2001), despite the fact that both SAD and S-SAD suffer from seasonal changes and report increased negative affect upon the onset of fall/winter (Lam et



al., 2001; Schlager et al., 1995). Possibly because they ruminate less, the S-SAD persons may be less likely to amplify their mild and transient depressive state into major depression. Hence, it would be informative to determine whether the rumination in SAD covaries with the depressive episode. If it were to be demonstrated that the rumination occurs only during the fall/winter time in the presence of a depressive episode and not any other time during the year, this would suggest that coping strategy might be related to the maintenance or exacerbation of SAD depressive episodes.

Hypothesis 4 that predicted S-SAD and SAD groups to use a more distracting strategy than the depressed group to cope with their depression was not supported. Specifically, there was no significant difference among the three groups in the degree to which they used distraction as a coping strategy. This lack of finding is in agreement with other research that shows distraction coping strategy does not differentiate between pathological and normal groups (Nolen-Hoeksema, 1991).

However, one coping strategy that distinguishes the SAD from the S-SAD group was the greater indulgence by the SAD individuals in dangerous and risky activities (e.g, use of alcohol, take feelings out on someone else). These behavioural responses are maladaptive and may serve to maintain or exacerbate the depressive symptoms. Whether the S-SAD individuals engage in activities, such as physical exercise, that are more beneficial to their health remains to be seen.

Hypothesis 5 that posited the SAD group would perceive winter as more stressful than other groups was confirmed in various ways. SAD people dreaded winter more than did S-SAD, depressed, and nondepressed individuals who did not differ among themselves. This corroborates anecdotal reports that SAD individuals dread the winter (e.g., Rosenthal, 1993). Furthermore, the present study also found that SAD persons reported more stress and greater

impairment associated with the experience of atypical symptoms, more rumination regarding winter situations and atypical symptoms, and greater negative affective reaction to winter stimuli than the other three groups. The depressed group was next highest on all these characteristics (stress, impairment, rumination, and negative affective reaction), followed by the S-SAD and nondepressed groups who did not differ from each other. Finally, SAD individuals perceived themselves as being less able to cope with winter than did the S-SAD.

The above findings are interesting when viewed within the context of the psychological distinctiveness of SAD. Although it was found in the present study that SAD and depressed individuals are no different from each other in their maladaptive explanatory style, and that SAD is more maladaptive than S-SAD, it would seem that the one characteristic that sets SAD individuals apart from the other groups is their reaction to winter-related stimuli. The gestalt of the findings suggests that SAD individuals dread the winter, find the atypical symptoms (carbohydrate craving, weight gain, energy loss, and increased sleep) to be stressful and interfering with their daily life functioning in various domains (work, academic, social, family, and personal), ruminate on the limitations imposed on them by the winter, react to the winter with negative affect, and believe that they are unable to cope with the winter.

An intriguing observation is that although the S-SAD individuals in this present study were *more* seasonal than the depressed individuals in that they had a seasonal pattern to their symptoms and found their seasonal changes to be more marked, they nevertheless reported winter to be *less* aversive. One would expect seasonality to be positively associated with aversion to winter because of the relationship between winter and the onset of depressive symptoms. Perhaps cognitive vulnerability may play a role. If one is prone to depressive thinking, engaging in maladaptive explanatory style, and ruminating on the negative, then

negative experiences associated with winter may be perceived as even more negative and winter may be viewed as highly aversive. On the other hand, if one's thinking, explanatory and cognitive coping styles are more adaptive, then whatever seasonal changes one experiences might be viewed in a less catastrophic fashion. Winter then might not be deemed as aversive.

One final observation regarding the entire set of findings in the present study deals with the lack of differences between the S-SAD and nondepressed individuals in different areas: severity of typical and atypical symptoms, cognitive coping style (rumination and dangerous activities), global style of explanation, and reactions to winter stimuli (dread of winter, severity of atypical symptoms during the winter, rumination over winter, negative affect towards winter, and perceived ability to cope with winter). The only differences that were found between S-SAD and nondepressed groups lie in S-SAD's higher seasonality score and less use of stable explanatory style. It would appear that despite having higher degree of seasonality and impairment (one of the selection criteria for S-SAD), S-SAD individuals are not at a disadvantage compared to the nondepressed individuals. It is wondered whether cognitive factors may play a protective role, especially when one compares S-SAD with SAD persons who have a more maladaptive coping and explanatory style.

#### *Strengths and Limitations of the Present Study*

The present study attempted to adopt strict clinical criteria to classify the different groups that were more in keeping with the Canadian Consensus Guidelines for the Treatment of Seasonal Affective Disorder, (Lam & Levitt, 1999), that in turned relied heavily on the DSM-IV recommendations for major depression. To this end, the paper version of the SCID was administered followed by the computer version to increase the validity of the clinical classification. Those who had previous and/or current major depressive episode (MDE) were

considered for inclusion into either the SAD or depressed group, but only if their current depressive symptoms as assessed on the Hamilton were severe. This approach was adopted to circumvent the difficulty of getting sufficient participants who could meet the pure DSM-IV-TR criteria of current MDE. Unfortunately, it also entailed an inherent weakness in that some of those who had previous MDE may not have current MDE. A partial solution to this drawback was to ensure that those who had previous MDE were currently suffering from severe depressive symptoms.

The seasonality criteria adopted in the present study were also more stringent than those typically used in previous works. Other studies typically rely on the use of the Global Seasonality Score to identify seasonality in SAD individuals (e.g., Bartko & Kasper, 1990; Mersch et al, 1999a). However, this may not be sufficient as it has to be established that the onset and remission of symptoms follow a seasonal pattern, that the pattern has to be evident at least within the last two years, that the seasonal pattern has to be impairing to the individual, and that there were no seasonal stressors that could account for the annual onset of depression. These criteria were adopted in the present study. Hence individuals who reported a high seasonality score but showed no seasonal pattern to their symptoms, did not have a seasonal pattern at least within the last two years, and who did not experience impairment were excluded.

The inclusion of two other comparison groups, S-SAD and nondepressed that are typically not represented in previous research, allowed a more complete examination of the varying effects of seasonality and depression. Previous investigations concerning the differences between seasonal and nonseasonal disorders have generally included only a nondepressed group, a nonseasonal depressed group, and a SAD group (e.g., Allen et al., 1993; Garvey et al., 1988). Such comparisons, however neglect the S-SAD group. As can be seen from the present study,

the S-SAD individuals differ from the SAD and depressed groups in several significant ways.

Unfortunately, the number of participants examined in the four groups were unequal. This may pose a potential statistical problem because uneven cell sizes can compromise the power of the statistical analysis (Tabachnick & Fidell, 2001). However, given that several significant effects were obtained with effect sizes ranging from low (.11) to high (.77), this problem may not be an important issue.

The sampling and distribution of the participants across the groups need to be considered. Most of the participants were derived from an undergraduate student population, with a minority (approximately 10%) of them from the general community. It can be argued that the reliance on an undergraduate student population limits the generalizability of the findings of the study. As well, given that the academic year begins in the fall and continues through the winter, it is very possible that school is a stressor that brings about the depressive episodes in the SAD and S-SAD groups. This issue was kept in mind when assessing participants for seasonality. It was confirmed with the participants that school did not present a stressor for them and their responses were cross-checked with their pattern of symptoms over the 12 months on the SPAQ. Specifically, the participants in the SAD and S-SAD groups reported exam times during December and April to be stressful for them. However, the onset of their symptoms was before December and the remission in April and May. As well, despite reporting that December was stressful for them, several of the participants indicated an improvement of symptoms in that month. When asked, they responded that December meant Christmas, family times and party fun. Hence, there was no correspondence between their stated stressful months and the onset and remission of their typical and atypical symptoms, suggesting that school may not be a seasonal stressor for these people. As well, several of the participants reported summer, rather than fall

and winter, to be a stressful time for them because they worried about summer employment and their financial situation. Determining that the occurrence of depressive symptoms did not covary temporally with situational stresses helped to address the criteria that no seasonal stressor account for the depression.

It is wondered while the use of a largely undergraduate sample limits the generalizability of findings to the seasonal and nonseasonal population as a whole. It could be argued that if the cause of SAD is light-related, then light deprivation should affect everyone in a similar fashion, regardless whether they are students or otherwise. However, research suggests that the prevalence of SAD drops in individuals between the ages of 40 and 50 (Kasper, Wehr et al., 1989). Therefore it is possible that older individuals have greater experience of coping with their problems, and hence, may cope with their problems in a more adaptive manner. Furthermore, the demographics of the current sample were highly homogenous where most of the participants were students, Caucasian, and not recruited from a clinical setting. The present study needs to be replicated with other demographic groups to ensure the stability of the results.

Another issue that arises here is the small proportion of people recruited from the community. Most of them had been previously diagnosed as depressed by their physicians and were on antidepressants, and were assigned to either the SAD or depressed group. Whether the use of antidepressants presents a confound in the findings is unclear. Future research needs to examine whether the use of antidepressants serve to modify cognitive, behavioural, and affective reactions in depressed and seasonal individuals.

### *Summary and Conclusions*

Findings from the present study indicate that the characteristic that distinguishes SAD individuals from others is their negative reaction to winter. They experience more severe

atypical symptoms, find them to be more impairing, ruminate more about winter, and perceive themselves as less able to cope. The differences between SAD and depressed individuals lie in the SAD having more severe atypical symptoms, higher degree of seasonality, and greater focus about their depression. Compared to S-SAD individuals, the SAD individuals reported more typical and atypical symptoms, greater degree of seasonality, more use of rumination and involvement in dangerous activities to cope with depression, and more maladaptive explanatory style (more global and stable attributions). The depressed group differed from the S-SAD group in that they had more typical and atypical symptoms, less degree of seasonality, ruminate more on their depression. They also had more negative reactions to winter in that the atypical symptoms they experienced during winter were more severe and more impairing; they ruminated and had more negative affective reactions more about the winter. Except for their greater degree of seasonality, the S-SAD was no different from the nondepressed group. It is noted that the results reported here have to be interpreted with the limitations of the study in mind.

#### *Directions for Future Research*

Findings from the present study raise several questions. The generalizability of the current findings needs to be investigated. Replication of the results with non-student samples would enhance the validity of the findings. It would be interesting to repeat the study with the older population and with clinical populations to see how they differ from the present sample. In particular, the role of antidepressants needs to be examined. Given the cross-sectional nature of the present study, it is impossible to establish whether the pattern of results reported would hold across the seasons. Would coping and explanatory styles remain the same when the depression is in remission or are these characteristics stable? A longitudinal study would help to answer these questions as well as enable one to investigate the predictive validity of the SAD

classification. Previous longitudinal research shows that only 35-67% of individuals who were categorized as SAD remained so over time (Lam et al., 2001). Some of these individuals became sub-syndromal (Sakamoto et al., 1995). Concomitant changes in the cognitive and coping styles could be tracked.

The relationship of physical activity to typical and atypical symptoms, current seasonal changes (measured as within the current year), and to explanatory and coping style could be investigated in seasonal and nonseasonal individuals. This would help to determine any beneficial effects that physical activities may have in alleviating the depressive symptoms, and the psychological moderators or mediators involved in the relationship.

The sensitivity of the SAD individuals to winter-related stimuli found in the present study could be followed up with other tests to ensure the validity of the current findings. One such test is the Stroop effect cognitive processing task that was conducted on SAD individuals by Dagleish and Spinks (2001). However, comparisons with nonseasonally depressed and subsyndromal SAD individuals were not undertaken.

The dread of winter has been postulated to relate to the onset of atypical symptoms in the dual vulnerability hypothesis (Young et al., 2001). It would be informative to prospectively follow seasonal and nonseasonal individuals throughout the seasons to track their aversion to winter dread and any associated changes in their symptomatic functioning and in their cognitive, behavioural and affective reactions. This would control for the possibility of retrospective memory bias in self-reports.

This research suggests that SAD individuals possess heightened rumination, high psychological stress, and a global explanatory style during their depressive episode: factors that can negatively impact a mild depressive episode (Abramson et al., 1989; Abramson et al., 1994;



atypical symptoms respond best to light therapy (Lam et al., 2001; Sakamoto et al., 1995; Terman et al., 1996; Young et al., 1991), and that typical symptoms are associated with cognitive variable (Lam et al., 2001; Young et al., 1991), the combination of light therapy and a SAD-relevant cognitive therapy may possess additive effects affecting more therapeutic change than either cognitive and light therapy alone.

## References

- Abramson, L., Seligman, M., & Teasdale (1978). Learned Helplessness in Humans: Critique and reformulation. *Journal of Abnormal Psychology, 87(1)*, 49 - 74.
- Abramson, L., Garber, J., Edwards, N.B., & Seligman, M. (1978). Expectancy changes in depression and schizophrenia. *Journal of Abnormal Psychology, 87(1)*, 102-109
- Abramson, L., Garber, J., & Seligman, M. (1980). *Learned Helplessness in Humans: An attributional Analysis*. Garber, J. & Seligman, M.(Eds.).New York: Academic Press, Inc.
- Abramson, L., & Martin, D. (1981). Depression and the causal process. *New Directions in Attribution Research, Volume 3*. Harvey, J., Ickes W., & Kidd, R. Lawrence Erlbaum Associates, Inc; Hillsdale, New Jersey
- Abramson, L., Metalsky, G., & Alloy, L. (1989). Hopelessness Depression: A Theory-based subtype of depression. *Psychological Review, 96(2)*, 358-372.
- Abramson, L.Y., Metalsky, G.I., & Alloy, L.B. (1999). *The Cognitive Style Questionnaire: A measure of the diathesis featured in the hopelessness theory of depression*. Unpublished manuscript, University of Wisconsin-Madison.
- Allen, J., Lam, R., Remick, R., & Sadovnick, A.(1993). Depressive symptoms and family history in seasonal and nonseasonal mood disorders. *American Journal of Psychiatry, 150(3)*, 443-448.
- Alloy, L.B., Abramson, L.Y., Murray, L.A., Whitehouse, W.G., & Hogan, M.E. (1997). Self-referent information processing in individuals at high and low cognitive risk for depression. *Cognition and Emotion, 11*, 539-568.
- Anderson, C.A., Miller, R.S., Riger, A. L., Dill, J.C., & Sedikides, C. (1994). Behavioral and characterological attributional styles as predictors of depression and loneliness: Review,

- refinement, and test. *Journal of Personality and Social Psychology*, 66(3), 549-558
- Avery, D., Khan, A., Dager, S., Cox, G., & Dunner, B. (1990) Bright light of winter depression: morning versus evening light. *Acta Psychiatrica Scandinavica*, 82(5), 335-338
- Bartko, J., & Kasper, S. (1989). Seasonal changes in mood and behavior: A cluster-analytic approach. *Psychiatry Research*, 28(2), 227-239.
- Bauer, M. (1992). Defining seasonal affective disorder. *Biological Psychiatry*, 31(12), 1185-1189.
- Bauer, M., & Dunner, D. (1993) Validity of Seasonal Pattern as a modifier for mood disorders for DSM-IV. *Comprehensive Psychiatry*, 34(3), 159-170.
- Bauer, M., Kurtz, J., Rubin, L., & Marcus, J. (1994). Mood and behavioral effects of four-week light treatment in winter depressives and controls, *Journal of Psychiatric Research*, 28(2), 135-145.
- Blazer, D., Kessler, R., & Schwartz, M. (1998). Epidemiology of recurrent major and minor depression with seasonal pattern: The national comorbidity survey. *British Journal of Psychiatry*, 172, 164-167.
- Bielski, R., Mayor, J., & Rice, J. (1992). Phototherapy with broad spectrum white fluorescent light: A comparative study. *Psychiatry Research*, 43(2), 167-175.
- Billings, A., & Moos, R. (1985). Psychosocial stressors, coping, and depression. *Handbook of Depression, Treatment, Assessment and Research*. Beckham, E., & Lelievre, W. (Eds.). The Dorsey Press: Illinois.
- Blehar, M., & Rosenthal, N. (1989). Seasonal affective disorders and phototherapy. *Archives of General Psychiatry*, 46 (5), 469-474.
- Booker, J., & Hellekson, C. (1992). Prevalence of seasonal affective disorder in Alaska.

- American Journal of Psychiatry*, 149 (9), 1176-1182.
- Brainard, G., Sherry, R., Skwerer, M., Waxler, M. Kelly, K., & Rosenthal, N. (1990) Effects of different wavelengths in seasonal affective disorder. *Journal of Affective Disorders*, 20 (4), 209-216.
- Brewin, R. (1985). Depression and causal attributions: What is their relation? *Psychological Bulletin*, 98(2), 297-309.
- Buckwald, B., & Resnick, et al., (1990). The effect of L-tryptophan on seasonal affective disorder. *Journal of Clinical Psychiatry*, 51(4), 162-163.
- Butler, L., & Nolen-Hoeksema, S. (1994). Gender differences in responses to depressed mood in a college sample. *Sex Roles*, 30 (5-6), 331-346.
- Cafarri, G., Davolio, M., & Chiodera, P. (1993). Abnormal serotonergic control of prolactin and cortisol secretion in patients with seasonal affective disorder. *Psychoneuroendocrinology*, 18, 551-556
- Carlsson, A. (1980). Seasonal and circadian and monoamine variations in human brains examined post-mortem. *Acta Psychiatrica Scandinavica*, 61, 75-83.
- Chang, E. (1998). Does dispositional optimism moderate the relation between perceived stress and psychological well-being?: A preliminary investigation. *Personality & Individual Differences*, 25 (2), 233-240.
- Cohen, S (1986). Contrasting the hassle scale and the perceived stress scale: Who's really measuring appraised stress? *American Psychologist*, 716-718.
- Cohen, S., Kamarck, T., & Mermelstein, R. (1983) A global measure of perceived stress. *Journal of health and social behavior*, 24 (December), 385-396.
- Cohen, J., Kessler, R. & Gorden (1995). *Measuring stress: A guide for health and social scientists*. London, Oxford University Press.

- Coyne, J., & Gotlib, I. (1983). The Role of Cognition: A critical appraisal. *Psychological Bulletin*, 94 (3), 472-505.
- Eastman, C., Lahmeyer, H., Watell, L., Good, G., Young, M. (1992). A placebo-controlled trial of light treatment for winter depression. *Journal of Affective Disorders*, 26, 211-222.
- Eastman, C., Young, M., Fogg, L., Liu, L., & Meaden, P. (1998). Bright light treatment of winter depression. *Archives of General Psychiatry*, 55 (10), 883-889.
- Faedda, G., Tondo, L., Teicher, M., Baldessarini, R., Gelbard, H., & Gianfranco, F. (1993). Seasonal mood disorders: Patterns of recurrence in mania and depression. *Archives of General Psychiatry*, 50 (1), 17-23.
- Fava, M., Rosenbaum, J., McCarthy, M., Fava, J., Steingard, R., & Fox, R. (1992). Correlation between perceived stress and depressive symptoms among depressive outpatients. *Stress Medicine*, 8 (2), 73-76.
- First, M., Gibbon, M., Williams, J., & Spitzer, R. (1999). *Computer Program for Windows: SCID Screen Screen Patient Questionnaire*. Mental-Health System, Inc., & American Psychiatric Press, Inc.
- Folkman, S., Lazarus, R., Gruen, R., & DeLongis, A. (1986). Appraisal, coping, health status, and psychological symptoms. *Journal of Personality & Social Psychology*, 50(3), 571-579.
- Garvey, M., Wesner, R., & Godes, M. (1988). Comparison of seasonal and nonseasonal affective disorders. *American Journal of Psychiatry*, 145 (1), 100-102.
- Geerts, E., Kouwert, E., Bouhuys, N., Meesters, Y., & Jansen, J. (2000). Nonverbal interpersonal attunement and extravert personality predict outcome of light treatment in seasonal affect in seasonal affective disorder. *Journal of Affective Disorder*, 59 (3), 193-204.
- Ghadirian, A., Murphy, B., & Gendron, M. (1998). Efficacy of light versus tryptophan therapy in

- seasonal affective disorder. *Journal of Affective Disorders*, 50 (1), 23-27.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry*, 23, 56-62.
- Hamilton, H. (1967). Development of a rating scale for primary depressive illness, *British Journal of Social and Clinical Psychology*, 6 (4), 278-296. *Journal of Psychiatric Research*, 25 (3), 75-87.
- Hardin, t., Wehr, T., Brewerton, T., & Kasper, S. (1991). Evaluation of seasonality in six clinical populations and two normal populations.
- Hellekson, C., Kline, J., Rosenthal, N. (1986). Phototherapy for seasonal affective disorder in Alaska. *American Journal of Psychiatry*, 143 (8), 1035-1037.
- Hewitt, P., Flett, G., & Mosher, S. (1992). The Perceived Stress Scale: Factor structure and relation to depression symptoms in a psychiatric sample. *Journal of Psychopathology and Behavioral Assessment*, 14 (3), 247-257.
- Hilsman, R., & Garber, J. (1995). A test of the cognitive diathesis-stress model of depression in children: Academic stressors, attributional style, perceived competence, and control. *Journal of Personality and Social Psychology*, 69 (2), 310-380.
- Hodges, S., & Marks, M. (1998). Cognitive characteristics of seasonal affective disorder: A preliminary investigation. *Journal of Affective Disorders*, 50 (1), 59-64.
- Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of event scale: A measure of subjective distress. *Psychosomatic Medicine*, 41, 209-218.
- Ibatoullina, E., Praschak-Rieder, N., & Kasper, S. (1997). Severe atypical symptoms without depression in SAD: Effects of bright light therapy. *Journal of Clinical Psychiatry*, 58(11), 495.
- Jacobsen, F., Wehr, T., Sack, D., James, S., & Rosenthal, N. (1987). Seasonal affective disorder:

- A review of the syndrome and its public health implications. *American Journal of Public Health*, 77 (1), 57-60.
- Jacobsen, F., Wehr, T., Skrewer, Sack, D., & Rosenthal, N. (1987) Morning versus midday phototherapy of seasonal affective disorder. *American Journal of Psychiatry*, 144(10), 1301-1305
- Jacobsen, F., Mueller, E., Rosenthal, N., Rogers, S., Hill, J., & Dennis, M. (1994). Behavioral responses to intravenous meta-chlorophenylpiperazine in patients with seasonal affective disorder and control subjects before and after phototherapy. *Psychiatry Research*, 52 (2), 181-197.
- Jacobsen, F., Sack, D., Wehr, T., Rogers, S., Rosenthal., N. (1987). Neuroendocrine response to 5-hydroxytryptophan in seasonal affective disorders. *Archives of General Psychiatry*, 44(12), 1086-1091.
- James, S., Wehr, T., Sack, D., Parry, B., Rosenthal, N. (1985). Treatment of seasonal affective disorder with light in the evening. *British Journal of Psychiatry*, 147, 424-428.
- Jang, K., Lam, R., Harris, J., Vernon, P., & Livesly, J.(1998). Seasonal mood change and personality: An investigation of genetic co-morbidity. *Psychiatry Research*, 78(1-2), 1-7.
- Joffe, R., Moul, D., Lam, R., Levitt, A., Teicher, M., Lebeque, B., Oren, D., Buchanan, A., Glod, C., Murray, M., Brown, J., & Schwartz, P. (1993). Light visor treatment for seasonal affective disorder: A multicenter study. *Psychiatry Research*, 46 (1), 29-39.
- Joiner, T. (2001). Negative attributional style, hopelessness depression and endogenous depression. *Behavior Research and Therapy*, 39, 139-149.
- Jouvet, M. (1969). Biogenic amines and the states of sleep. *Science*, 163 (3862), 32-41.
- Just, N., & Alloy, L.B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology*, 106, 221-229.

Kane, A., & Lowis, M. (1999), Seasonal affective Disorder and personality, age, and gender.

*South African Journal of Psychology, 26 (9), 124-127.*

Kasper, S., Roger, S., Yancey, A., Schulz, P., Skwerer, R., & Rosenthal, N. (1989b).

Phototherapy in individuals with and without Subsyndromal Seasonal Affective Disorder.

*Archives of General Psychiatry, 46 (9), 837-844.*

Kasper, S., Wehr, T., Bartko, J., Gaist, P., & Rosenthal, N. (1989b). Epidemiological findings of

seasonal changes in mood and behavior. *Archives of General Psychiatry, 46 (9), 823-833.*

Kasper, S., Rogers, S., Madden, P., Vanderpool, R., & Rosenthal, N. (1990). The effects of

phototherapy in the general population. *Archives of General Psychiatry, 18(3), 217-219.*

Kripke, D.(1998). Light treatment for nonseasonal depression: speed, efficacy, and combined

treatment. *Journal of Affective Disorders, 49 (2), 109-117*

Kuiper, N. A., Olinger, J., & Lyons, L. (1986) Global perceived stress levels as a moderator of

the relationship between negative life events and depression. *Journal of Human Stress,*

*12(4), 149-153.*

Lam, R., Bowering, T., Tam, A., Grewal, L., Yatham, I., & Zis, A. (2000). Effects of rapid

tryptophan depletion in patients with seasonal affective disorder in natural summer

remission. *Psychological Medicine, 30 (1), 79-87.*

Lam, R., Tam, E., Yatham, L., Shiah, I., & Zis, A. (2001). Seasonal depression. The dual

vulnerability hypothesis revisited. *Journal of Affective Disorders, 63 (1-3), 123-132.*

Lam, R., Buchanan, A., Clark, C., & Remick, R. (1991). Ultraviolet versus non-ultraviolet light

therapy for seasonal affective disorder. *Journal of Clinical Psychiatry, 52(5), 213-216.*

Lam, R., Gorman, C., Michalon, M., Steiner, M., Levitt, A., Corral, M., Watson, G., Morehouse,

R., Tan, W., Joffe, R. (1995). Multi-center, placebo-controlled study of fluoxetine in



- seasonal affective disorder. *American Journal of Psychiatry*, 152(12), 1765-1770.
- Lam, R. W., & Levitt, A. J. (1999). *Canadian consensus guidelines for the treatment of Seasonal Affective Disorder*. Canada: Clinical & Academic Publishing.
- Lee, T., & Chan, C. (1998). Vulnerability by sex to seasonal affective disorder. *Perceptual and Motor Skills*, 87 (3), 1120-1122.
- Lee, T., & Chan, C. (1999). Dose-response relationship of phototherapy for seasonal affective disorder: a meta-analysis. *Acta Psychiatrica Scandinavica*, 99, 315-323.
- Lee, T., Chen, E., Chan, C., Paterson, J., Janzen, H., & Blashko, C. (1998). Seasonal affective disorder. *Clinical Psychology: Science and Practice*, 5(3), 275-290.
- Lenzinger, E., Neumeister, A., Praschak-Reddner, N., Fuchs, K., Gerhard, E., Willeit, M., Sieghart, W., Kasper, S., Hornik, K., & Aschauer, H. (1999). Behavioral effects of tryptophan depletion in seasonal affective disorder associated with the serotonin transporter gene. *Psychiatry Research*, 85 (3), 241-246.
- Levitan, R., Rector, N., & Bagby, R. (1998). Negative attributional style in seasonal and nonseasonal depression. *American Journal of Psychiatry*, 155 (3), 428-430.
- Levitt, A., Boyle, M., Joffe, R., & Zillah, B. (2000). Estimated prevalence of the seasonal subtype of major depression in a Canadian community sample. *Canadian Journal of Psychiatry*, 45 (7), 650-654.
- Lewinsohn, P., & Gotlib, I. (1995). The behavioral theory and treatment of depression. In W. Leber & E. Beckham(Eds.), *Handbook of Depression: Second Edition* (pp. 340-363). Guilford Press: New York.
- Lewy, J., Miller, S., & Hoban, T. (1987). Antidepressant and circadian phase-shifting effects of light. *Science*, 235 (4786), 352-354.

- Lingjaerde, O., Foreland, A., & Engvik, H. (2001). Personality structure in patients with winter depression, as assessed in a depression-free state according to the five-factor model of personality. *Journal of Affective Disorders, 62* (3),
- Lingjaerde, O., Reichborn-Kjennerud, T., Haggag, A., Gartner, L., Narud, K., & Berg, E. (1993). Treatment of depression in Norway: A comparison of the selective monoamine oxidase A<sub>1</sub> inhibitor moclobemide and placebo. *American Journal of Psychiatry 88*(5), 372-380.
- Lucas, C. (1991). Seasonal Affective Disorder in adolescence. *British Journal of Psychiatry, 159*, 863-865.
- Lyubormisky, S., & Nolen-Hoeksema, S. (1999). Self-perpetuating properties of dysphoric rumination. *Journal of Personality & Social Psychology, 65*(2), 339-349.
- Mackert, A., Volz, H., Stieglitz, R., & Orlinghausen, B. (1990). Effect of bright, white light on non-seasonal depression. *Biological Psychiatry, 23* (3), 257-268.
- Magnusson, A. (1996). Validation of the Seasonal Pattern Assessment Questionnaire (SPAQ). *Journal of Affective Disorders, 40* (3), 121-129.
- Magnusson, A. & Axelsson, J. (1993). The prevalence of seasonal affective disorder is low among descendants of Icelandic immigrants in Canada. *Archives of General Psychiatry, 50* (12), 947-951.
- Martin, R. A., Kazarian, S., & Breiter, H. J. (1995). Perceived stress, life events, dysfunctional attitudes, and depression in adolescent psychiatric inpatients. *Journal of Psychopathology and Behavioral Assessment, 17* (1), 81-95.
- Mcgrath, R., Buckwald, B., & Resnick, E. (1990). The effect of l-tryptophan on seasonal affective disorder. *Journal of Clinical Psychiatry, 51*(4), 162-163.
- Meesters, J., Jansen, L., Bouhys, D., Beersma, D., & van den Hoofdakker. (1993) Morning and

- evening light treatment of seasonal affective disorder: Response, relapse, and prediction. *Journal of Affective Disorders*, 28 (3), 165-177.
- Mersch, P., Middendorp, H., Bouhys, Beersma, D., Hoofdakker, R. (1999a). The prevalence of seasonal affective disorder in the disorder: A prospective and retrospective study of seasonal mood variation in the general population. *Society of Biological Psychiatry*, 45(8), 1013-1022.
- Mersch, P., Middendorp, H., Bouhys, Beersma, D., Hoofdakker, R. (1999a). Seasonal affective disorder and latitude: A review of the literature. *Journal of Affective Disorders*, 53 (1), 35-48.
- Moller, S. (1992). Serotonin, Carbohydrates, and Atypical Depression. *Pharmacology and Toxicology*, 71, 61-71.
- Moller, H. (1998). Effects of fluoxetine versus bright light in the treatment of seasonal affective disorder. *Psychological Medicine*, 23, 923-933.
- Molin, J., Mellerup, E., Bolwig, T., Scheike, T., & Dam, H. (1996). The influence of climate on development of winter depression. *Journal of Affective Disorders*, 37(2-3), 151-155.
- Monroe, S., & Kelley, J. (1995). Measuring stress appraisal: A guide for health and social scientists (pp 122-147). In Cohen, S., Kessler, R. & Gordon (Eds.). *Measurement of Stress Appraisal*. London: Oxford University Press.
- Morrow, J., & Nolen Hoeksoma, S. (1990). Effects of responses to depression on the remediation of depressive affect. *Journal of Personality & Social Psychology*, 58 (3), 519-527.
- Nayyar, K., & Cochrane, R. (1996). Seasonal changes in affective state measured prospectively and retrospectively. *British Journal of Psychiatry*, 168 (5), 627-632.
- Neumeister, A., Praschak-Riederm N., Hebelmann, B., Rao, M., Gluck, J., & Kasper, S. (1997).

- Effects of tryptophan depletion on drug-free patients with seasonal affective disorder during a stable response to bright light therapy, *Archives of General Psychiatry*, 54(2), 133-138.
- Neumeister, A., Praschak- Rieder, N., Hebelmann, B., Vitouch, O., Rauh, M., Barocka, A., & Kasper, S. (1997). *American Journal of Psychiatry*, 154(8), 1153-1155.
- Neumeister, A., Habeler, A., Praschak-Reider, N., Willeit, M., & Kasper, S. (1999). Tryptophan depletion: A predictor of future depressive episodes in seasonal affective disorder? *International Clinical Psychopharmacology*, 14, 313-315.
- Nolen-Hoeksema, S. (1987). Sex differences in unipolar depression: Evidence and theory. *Psychological Bulletin*, 101 (2), 259-282.
- Nolen-Hoeksema, S.(1991).Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100(4), 369-382.
- Nolen-Hoeksema, S & Morrow, J. (1991). A prospective study of depression and posttraumaticstress symptoms after a natural disaster: The 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology*, 61 (1), 115-121.
- Nolen-Hoeksema, S., Morrow, J., & Frederickson, B. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, 102(01), 20-28.
- Nolen-Hoeksema, S., & Davis, C. (1999). "Thanks for sharing that": Ruminators and their social support networks. *Journal of Personality & Social Psychology*, 77 (4), 801-814.
- Otto, M., Fava, M., Penava, S., Bless, E., Muller, R., & Rosenbaum, J. Life event, mood, and cognitive predictors of perceived stress before and after treatment for major depression. *Cognitive Therapy and Research*, 21(4), 409-420.
- Partonen, T., & Lonnqvist, J. (1996). Moclobemide and fluoxetine in treatment of seasonal

- affective disorder. *Journal of Affective Disorders*, 41(2), 93-99
- Partonen, T., & Lonnquist, J. (1998). Seasonal affective disorder: A guide to diagnosis and management. *Central Nervous System Drugs*, 9(3), 203-212
- Pbert, L., Doerfler, L., & Decosimo, D. (1992). An evaluation of the received Stress Scale in two clinical populations. *Journal of Psychopathology & Behavioral Assessment*, 14 (4), 363-375.
- Peterson, C. (1991). The meaning and measurement of explanatory style. Psychological Inquiry, 2, 1-10.
- Peterson, C., & Seligman, M. (1984). Causal explanations as a risk factor for depression: Theory and evidence. *Psychological Review*, 91 (3), 347-374.
- Pinchasov, B., Shirguja, A., Grischin, O., Putilova, A. (2000). Mood and energy regulation in seasonal depression and nonseasonal depression before and after midday treatment with physical exercise or bright light. *Psychiatry Research*, 94(1), 29-42.
- Ralph, J., & Mineka, S. (1998). Attributional style and self-esteem; The prediction of emotional distress following a midterm exam. *Journal of Abnormal Psychology*, 107(2), 203-215.
- Raps, C., Peterson, C., Reinhard, Abramson, L., & Seligman, E. (1982). Attributional style among depressed patients. *Journal of Abnormal Psychology*, 91(2), 102-108.
- Reilly-Harrington, N. & Alloy, B. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology*, 108(4), 567-578.
- Rosen, L., & Rosenthal, N. (1991). Seasonal variations in mood and behavior in the general population: A factor-analytic approach. *Psychiatry Research*, 38 (3), 271-283.
- Rosen, L., Targum, S., Terman, M., Bryant, M., Hoffman, H., Kasper, S., Hamovit, J., Docherty,

- J., Welch, B., Rosenthal, N. (1990). Prevalence of Seasonal Affective Disorder at four latitudes. *Psychiatry Research*, 31 (2), 131-144.
- Rosenthal, N., Sack, D., Gillin, C., Lewy, A., Goodwin, A., Davenport, Y., Mueller, P., Newsome, D., Wehr, T. (1984). Seasonal affective disorder: A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry*, 41, 72-80.
- Rosenthal, N., Sack, D., Carpenter, C., Parry, B., Mendelson, W., & Wehr, T. (1985). Antidepressant effects of light in seasonal affective disorder. *American Journal of Psychiatry*, 142(2), 1985
- Rosenthal, N., Sack, D., James, S., Parry, B., Mendelson, W., Tamarkin, L., & Wehr, T. (1985). Seasonal affective disorder. *Annals of the New York Academy of Sciences*, 453, 260-269.
- Rosenthal, N., Carpenter, C., James, S., Parry, B., Rogers, S., Wehr, T. (1986). Seasonal affective disorder in children and adolescents. *American Journal of Psychiatry*, 143(3), 356-358.
- Rosenthal, N., Rotter, A., Jacobsen, F., & Skwerer, R. (1987). No mood-altering effects found after bright light treatment in normal subjects. *Psychiatry Research*, 22 (1), 1-9.
- Rosenthal, N., Skwerer, R., Sack, D., Duncan, C., Jacobsen, F., Tamarkin, L., & Wehr, T. (1987). Biological effects of morning-plus-evening bright light treatment of seasonal affective disorder. *Psychopharmacology Bulletin*, 23, 364-369.
- Rosenthal, N., & Wehr, T. (1987). Seasonal affective disorders. *Psychiatric Annals*, 17(10), 670-674.
- Rosenthal, N. (1993). *Winter Blues*. New York: The Guilford Press.
- Ruhrmann, S., Kasper, S., Hawellek, B., Martinez, B., Hoflich, G., Nickelson, T., & Moller, H. (1998). Effects of fluoxetine versus bright light in the treatment of seasonal affective

- disorder. *Psychological Medicine*, 28(4), 923-933.
- Rusting, C., & Nolen-Hoeksema, S. (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. *Journal of Personality and Social Psychology*, 74(3), 790-803
- Sack, R., Lewy, A., White, D., Singer, C., Fireman, M., & Vandiver, R. (1990) Morning vs evening light treatment for winter depression : Evidence that the therapeutic effects of light are mediated by circadian phase shifts. *Archives of General Psychiatry*, 47 (4), 343-351.
- Sakamoto, K., Nakadaira, S., Tamura, A., Takahashi., K. (1993). A nationwide survey of seasonal affective disorder at 53 university clinics Japan. *Acta Psychiatrica Scandinavica*, 87 (4), 258-265.
- Sakamoto, K., Nakadaira, S., Kamo, K., Kamo, T., & Takahashi, K. (1995). A longitudinal follow-up study of seasonal affective disorder. *American Journal of Psychiatry*, 152(6), 862-868.
- Sakamoto, S., & Kambara, M. (1998). A longitudinal study of the relationship between attributional style, life events, and depression in Japanese undergraduates. *The Journal of Social Psychology*, 138(2), 229-240.
- Sarason, I., Johnson, J., & Siegel, J. (1978). Assessing the impact of life changes: Development of the Life Experiences Survey. *Journal of Consulting & Clinical Psychology*, 46, 932-946.
- Schlager, D., Froom, J., & Jaffe, A. (1995). Winter depression and functional impairment among ambulatory primary care patients. *Comprehensive Psychiatry*, 36 (1), 18-24.
- Seligman, M., Abramson, L., Semmel., & von Baeyer (1979). Depressive attributional style.

*Journal of Abnormal Psychology*, 88(3), 242-247.

- Sher, L. (2001). Genetics of seasonal affective disorder and seasonality. *Comprehensive Psychiatry*, 42 (2), 105-110.
- Sher, L., Goldman, D., Ozaki, N., & Rosenthal, N. (1999). The role of genetic factors in the etiology of seasonal affective disorder and seasonality. *Journal of Affective Disorders*, 53 (3), 203-210.
- Sourander, A., Koskelainen, M., & Helenius, H. (1999). Mood, latitude, and seasonality among adolescents. *Journal of American Academic Child and Adolescent Psychiatry*, 38(10), 1271-1276.
- Spitzer, R., & Williams, J. (1989). The validity of Seasonal Affective Disorder. From: *Seasonal Affective Disorders and Phototherapy*. Rosenthal, N., & Blehar, C. (Eds.), New York: Guilford Press.
- Stevens, J. (1986). *Applied multivariate statistics for the social sciences*. Mahwah, New Jersey: Lawrence Erlbaum Associates.
- Stewart, J., Quitkin, F., Terman, M., & Terman J. (1990). Is seasonal affective disorder a variant of atypical depression? Differential response to light therapy. *Psychiatry Research*, 33 (2), 121-128
- Stewart, K., Gaddy, J., Byrne, B., Miller, S., & Brainard. Effects of green or white light for treatment of seasonal affective disorder. *Psychiatry Research*, 38 (3), 261-270.
- Stinson, D., & Thompson, C. (1990). Clinical experience with phototherapy. *Journal of Affective Disorders*, 18 (2), 129-135.
- Magnusson, A., & Stefansson, J. (1993). Prevalence of seasonal affective disorder in Iceland. *Archives of General Psychiatry*, 50 (12), 941-946.



- Suhail, K., & Cochrane, R. (1997). Seasonal changes in affective state in samples of Asian and white. *Social Psychiatry Psychiatric Epidemiological*, 32 (3), 149-157.
- Tabachnick, B. & Fidell, L. (2001). *Using Multivariate Statistics*. Toronto: Allyn and Bacon.
- Terman, M. (1988). On the question of mechanism in phototherapy for seasonal affective disorder. *Journal of Biological Rhythms*, 3(2), 155-172
- Terman, M., Quitkin, F., Terman, J., Mcgrath, P., & Stewart, J. (1987). The timing of phototherapy: effects on clinical response and the melatonin cycle. *Psychopharmacological Bulletin*, 23, 354-357.
- Terman, M., Terman, J., Quitkin, M., & Mcgrath, P. (1989). Light therapy for seasonal affective disorder: A review of efficacy. *Neuropsychopharmacology*, 2(1), 1-22.
- Terman, M., Reme, C., Rafferty, B., Gallin, P., & Terman, J.(1990). Bright light therapy for winter depression: potential ocular effects and theoretical implications. *Photochemistry and photobiology*, 51(6), 781-792
- Terman, M., Terman, J., & Rafferty, B. (1990). Experimental design in the treatment of winter depression by bright light. *Psychopharmacology Bulletin*, 26(4), 505-510.
- Terman, M., Amira, L., Terman, J., & Ross, D. (1996). Predictors of response and nonresponse to light treatment for winter depression. *American Journal of Psychiatry*, 153(11), 1423-1429.
- Terman, M., Terman, J., & Ross, D.(1998). A controlled trial of timed bright light and negative air ionization for treatment of winter depression. *Archives of General Psychiatry*, 55 (10), 875-882.
- Thalen, B., Kjellman, B., Morkrid, L., Wibom, R., & Wetterberg. (1995). Light treatment in seasonal and nonseasonal depression. *Acta Psychiatrica Scandinavica*. 91 (5), 352-360.

Thase, M. (1986). Defining and treating seasonal affective disorder. *Psychiatric Annals*, *16*(12), 733-737.

Thase, M. (1989). Comparison between seasonal affective disorder and other forms of recurrent depression. In N. E. Rosenthal & M. C. Blehar (Eds.). *Seasonal Affective Disorder and Phototherapy* (pp. 64-78). New York: Guilford Press.

Thompson, C., & Isaacs, I (1988). Seasonal affective disorder - A British Sample: Symptomatology in relation to mode of referral and diagnostic subtype. *Journal of Affective Disorders*, *14* (1), 1-11.

Thompson, C., Stinson, D., Fernandez, M., Fine, J., & Isaacs, G. (1988). A comparison of normal, bipolar and seasonal affective disorder using the Seasonal Pattern Assessment Questionnaire. *Journal of Affective Disorders*, *14*, 257-264.

Thompson, C., & Silverstone, T. London: CNS Publishers. Rosenthal, N. (1993). *Winter Blues*. New York: The Guilford Press.

Thompson, C, Suhil, R., & King, E. (1995). A follow-up of seasonal affective disorder. *British Journal of Psychiatry*, *167*, 380-384.

Toru, S. (1997) Seasonal affective disorder and phototherapy: A critical review. *Professional Psychology: Research and Practice*, *28*(2), 164-168.

McCann, C., & Endler, N (Eds.). (1990) *Depression: New Direction in theory, research, and practice*. Toronto: Wall & Emerson

Wirz-Justice, A., & Anderson, J. (1990). Morning light exposure for the treatment of winter depression: The one true light therapy? *Psychopharmacology Bulletin*, *26* (4), 511-519.

Wirz-Justice, A., Graw, P., Krauchi, K., Gisin, B., Jochum, A., Arendt, J., Fisch, H., Buddeburg, G., & Poldinger, W. (1993). Light therapy in seasonal affective disorder is independent

of time of day or circadian phase. *Archives of General Psychiatry*, 50 (12), 929-937.

Van der Does, A. (2001). The effect of tryptophan depletion on mood and psychiatric symptoms.

*Journal of Affective Disorders*, 64(2-3), 107-119.

Volz, H., Mackert, R., Stirglitz, R., & Muller-Oerlingerhausen, B. (1990). Effect of bright white

light therapy on non-seasonal depressive disorder: Preliminary results. *Journal of*

*Affective Disorders*, 19 (1), 15-21.

Yerevian, B., Anderson, J., Grota, L., & Bray. (1986). Effects of bright incandescent light on

seasonal and nonseasonal major depressive disorder. *Psychiatry Research*, 18, 355-364

Young, M., Watel, L., Lahmeyer, H., & Eastmen, C. (1991). The temporal onset of individual

symptoms in winter depression: Differentiating underlying mechanisms. *Journal of*

*Affective Disorders*, 22 (4), 191-197

Table 1

*Classification criteria for SAD, S-SAD, Depressed and Nondepressed Groups*

Criteria	SAD	S-SAD	Depressed	Nondepressed
<b>Seasonality<sup>A</sup></b>				
Seasonal pattern <sup>b</sup>	Yes	Yes	No	No
Seasonal pattern last 2 years	Yes	Yes	Irrelevant	Irrelevant
GSS <sup>c</sup>	>11	9-11 / >11	Irrelevant	Irrelevant
Impairment <sup>d</sup>	At least moderate	At least mild / No or mild	No or mild	No or mild
Seasonal stressor	No	No	Irrelevant	Irrelevant
<b>Depression</b>				
HRDS-28 score	≥19	<14	≥19	<14
MDE <sup>e</sup>	Positive	Negative	Positive	Negative

<sup>A</sup>Seasonality criteria were assessed with the Seasonal Pattern Assessment Questionnaire (Rosenthal, et al., 1987).

<sup>b</sup>Seasonal pattern refers to the presence of symptoms during the fall and winter months (September to April) and the absence of symptoms during the spring and summer months.

<sup>c</sup>GSS = Global Seasonality Score. The GSS score for the S-SAD is based on the criteria in (Kasper, Wehr, et al., 1989), whereas that for SAD is from Rosenthal and Colleagues (1987). For Depressed and Nondepressed, GSS did not matter as their was no seasonal pattern to their symptoms

<sup>d</sup>Impairment = degree of impairment associated with the seasonal changes. The impairment guideline for S-SAD is based on that proposed by Kasper, Wehr, et al., 1989, whereas that for SAD is proposed by Rosenthal and colleagues (1987).

<sup>e</sup>MDE = major depression episode as assessed with the questions based on the QDIS-III-R and the DSM-IV SCID. Positive = minimal of five depressive symptoms including depressed mood and/or loss of interest/pleasure, plus impairment.

Table 2

Sample Description and Cell Sizes

Characteristics	Group			
	SAD ( <i>n</i> = 31)	S-SAD ( <i>n</i> = 32)	Depressed ( <i>n</i> = 20)	Nondepressed ( <i>n</i> = 25)
Type of participants				
Students	22	32	19	25
Community	9	0	1	0
Sex of participants				
Male	5	3	7	12
Female	26	29	13	13
Mean age of participants (standard deviation)	29.23 (13.56)	21.44 (4.32)	26.75 (13.17)	22.00 (5.51)
Mean HRDS-T <sup>a</sup> (standard deviation)	44.42 (13.88)	13.00 9.09	34.00 (11.52)	5.52 (4.03)
Mean HRDS-A <sup>b</sup> (standard deviation)	32.03 (11.54)	6.17 (2.70)	25.65 (10.26)	4.00 (3.33)
Mean HRDS-B <sup>c</sup> (standard deviation)	12.4 (6.35)	2.84 (2.46)	8.35 (5.21)	1.24 (1.33)
Mean GSS <sup>d</sup> (standard deviation)	17.30 (3.04)	11.47 (2.76)	8.48 (5.09)	3.92 (2.22)

<sup>a</sup>HRDS-T = total score on the HRDS-28

<sup>b</sup>HRDS-A = typical score on the HRDS-28

<sup>c</sup>HRDS-B = atypical score on the HRDS-28

<sup>d</sup>GSS = Global Seasonality Score on the Seasonal Pattern Assessment Questionnaire

Table 3

## Pooled Correlation Matrix of Dependent Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Ruminare	1												
2. Distract	0.08	1											
3. Danger	0.43***	-0.05	1										
4. Problem	>0.01	0.64***	-0.13	1									
5. Internal	0.13	-0.10	0.09	-0.11	1								
6. Stable	0.22*	-0.12	0.15	-0.27**	0.42**	1							
7. Global	0.38***	0.04	0.35***	-0.03	0.23*	0.58***	1						
8 W-Dread	0.39***	0.07	0.26**	0.05	0.09	0.09	0.28**	1					
9. W-Atypical	0.56***	0.15	0.31***	0.17	0.13	.20*	0.42***	0.50***	1				
10. W-Impair	0.70***	0.02	0.39***	-0.03	0.08	.22*	0.50***	0.51***	0.68***	1			
11. WINTERUM	0.67***	0.12	0.40***	0.04	0.05	0.15	0.43***	0.57***	0.72***	0.80***	1		
12. Factor 1	0.61***	0.01	0.48***	-0.03	-0.07	0.14	0.50***	0.47***	0.65***	0.74***	0.75***	1	
13 Factor 2	0.34***	-0.31	0.20*	-0.37***	0.22***	0.35***	0.16	0.12	0.19	0.34***	0.26**	0.18	1

Note. Factor 1 and Factor 2, respectively, are the Negative Affect and Perceived Ability to Cope factors that were derived from the Principal Component Analyses of the Winter Perceived Stress Scale.

\* $p < .05$ . \*\* $p < .01$ . \*\*\* $p < .001$

Table 4

Means (and Standard Deviations) of the Dependent Variables Within Each Group

Dependent Variable	Group			
	SAD	S-SAD	Depressed	Nondepressed
<b>Coping Styles</b>				
Rumination	55.51 ( 9.37 )	36.83 ( 8.23 )	45.93 ( 8.06 )	34.80 ( 8.04 )
Danger	6.93 ( 1.86 )	5.75 ( 1.44 )	6.70 ( 1.49 )	5.68 ( 0.95 )
Prob-Solve	8.33 ( 2.06 )	8.91 ( 2.01 )	8.55 ( 4.70 )	7.92 ( 2.99 )
Distract	24.07 ( 5.78 )	24.76 ( 3.97 )	26.05 ( 4.70 )	23.48 ( 6.95 )
<b>Cognitive Styles</b>				
Internal	60.48 ( 9.74 )	57.53 ( 9.71 )	58.96 ( 9.34 )	60.40 ( 7.51 )
Stability	50.23 ( 15.12 )	39.63 ( 9.90 )	46.10 ( 10.93 )	49.00 ( 11.39 )
Globality	50.90 ( 15.44 )	36.44 ( 12.39 )	43.55 ( 12.16 )	39.48 ( 12.17 )
<b>Winter Stress</b>				
W-Dread	3.52 ( 1.15 )	2.18 ( 0.93 )	2.30 ( 1.13 )	2.04 ( 0.89 )
W-Atypical	11.74 ( 3.93 )	5.27 ( 3.21 )	7.10 ( 3.51 )	2.12 ( 2.13 )
W-Impair	3.71 ( 0.82 )	1.77 ( 0.75 )	2.85 ( 0.99 )	1.29 ( 0.59 )
W-Ruminate	22.20 ( 3.56 )	12.59 ( 3.87 )	16.40 ( 5.34 )	10.20 ( 3.52 )
Factor 1	27.48 ( 4.35 )	17.28 ( 5.68 )	22.20 ( 6.27 )	13.60 ( 5.77 )
Factor 2	18.00 ( 2.00 )	15.82 ( 1.82 )	17.40 ( 2.14 )	17.12 ( 2.79 )

*Note.* Factor 1 and Factor 2, respectively, are the Negative Affect and Perceived Ability to Cope factors that were derived from the Principal Component Analyses of the Winter Perceived Stress Scale.

Table 5

Item Factor Loadings of the Winter Perceived Stress Scale (WPSS) With Varimax Rotation

WPSS Item Number	Factor One	Factor Two
1	0.82	
2	0.77	
3	0.85	
8	0.87	
9	0.87	
11	0.82	
12	0.66	
14	0.81	
4		0.84
5		-0.82
6		0.82
7		0.76
10		0.83
13		0.63

*Note.* Factor 1 and Factor 2, respectively, are the Negative Affect and Perceived Ability to Cope factors that were derived from the Principal Component Analyses of the Winter Perceived Stress Scale.



**Appendix 1**

**Section A of Research Questionnaire: Demographic Information**

**Section B of Research Questionnaire: Assessment of Seasonality and SPAQ**

**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 erythematosus? 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 your experience, not others 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



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? \_\_\_\_\_

**Appendix 2**

**Section C of Research Questionnaire: HRDS-28**

**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 3**

**Section D of Research Questionnaire: Response Style Questionnaire**



**Section D: People think and do many different things when they feel sad, blue, or depressed.**

*Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each one when you feel sad, down, or depressed. Please indicate what you generally do, not what you think you should do.*

Items	Rating Scale			
	Never	Sometimes	Often	Always
1. Ask someone to help you overcome a problem.	1	2	3	4
2. Think about how alone you feel.	1	2	3	4
3. Think "I won't be able to do my job/work because I feel so badly."	1	2	3	4
4. Think about your feelings of fatigue and achiness.	1	2	3	4
5. Think about how hard it is to concentrate.	1	2	3	4
6. Try to find something positive in the situation or something you learned.	1	2	3	4
7. Take recreational drugs or drink alcohol.	1	2	3	4
8. Think "I'm going to do something to make myself feel better".	1	2	3	4
9. Help someone else with something in order to distract yourself.	1	2	3	4
10. Think about how passive and unmotivated you feel.	1	2	3	4
11. Remind yourself that these feelings won't last.	1	2	3	4
12. Analyze recent events to try to understand why you are depressed.	1	2	3	4
13. Think about how you don't seem to feel anything any more.	1	2	3	4
14. Think "Why can't I get going?"	1	2	3	4
15. Think "Why do I always react this way?"	1	2	3	4
16. Go to a favourite place to get your mind off your feelings.	1	2	3	4
17. Go away by yourself and think about why you feel this way.	1	2	3	4
18. Talk it out with someone whose opinions you respect (friend/family/ clergy).	1	2	3	4
19. Think "I'll concentrate on something other than how I feel."	1	2	3	4
20. Write down what you are thinking about and analyze it.	1	2	3	4
21. Do something that has made you feel better in the past.	1	2	3	4
22. Think about a recent situation, wishing it had gone better.	1	2	3	4
23. Think "I'm going to go out and have some fun."	1	2	3	4
24. Make a plan to overcome a problem.	1	2	3	4
25. Stay around people.	1	2	3	4
26. Concentrate on your work.	1	2	3	4
27. Think "Why do I have problems other people don't have?"	1	2	3	4
28. Do something reckless or dangerous.	1	2	3	4

Items	Rating Scale			
	Never	Sometimes	Often	Always
29. Think about how sad you feel.	1	2	3	4
30. Think about all your shortcomings, failings, faults, mistakes.	1	2	3	4
31. Do something you enjoy.	1	2	3	4
32. Think about how you don't feel up to doing anything.	1	2	3	4
33. Do something fun with a friend.	1	2	3	4
34. Analyze your personality to try to understand why you are depressed.	1	2	3	4
35. Take your feelings out on someone else.	1	2	3	4
36. Go someplace alone to think about your feelings.	1	2	3	4
37. Deliberately do something to make yourself feel worse.	1	2	3	4
38. Think about how angry you are with yourself.	1	2	3	4
39. Listen to sad music.	1	2	3	4
40. Isolate yourself and think about the reasons why you feel sad.	1	2	3	4
41. Try to understand yourself by focusing on your depressed feelings.	1	2	3	4

**Appendix 4**

**Section E of Research Questionnaire: DSM-IV Diagnostic Questions for  
Major Depression Episode**

**Section E:** *The questions below ask about your thoughts, feelings, and behaviours. Please answer them by circling either "YES" or "NO", or writing in your response.*

1. In your lifetime, have you ever had 2 weeks or more when nearly every day you felt sad, blue, or depressed?	YES	NO
2. Has there ever been 2 weeks or longer when you lost all interest in things like work or hobbies or things you usually liked to do for fun?	YES	NO
3a. Has there ever been a period of 2 weeks or longer when you lost your appetite?	YES	NO
b. Have you ever lost weight <u>without</u> trying to – as much as 2 pounds a week for several weeks or as much as 10 pounds altogether?	YES YES	NO NO
c. Has there ever been at least 2 weeks when you had an increase in appetite?	YES	NO
d. Have you ever had a period when your eating increased so much that you gained as much as 2 pounds a week for several weeks or 10 pounds altogether?		
4a. Have you ever had 2 weeks or more when nearly every night you had trouble falling asleep, staying asleep, or waking up too early?	YES	NO
b. Have you ever had 2 weeks or longer when nearly every day you were sleeping too much?	YES	NO
5a. Has there ever been 2 weeks or more when nearly every day you talked or moved more slowly than is normal for you?	YES	NO
b. Has there ever been two weeks or more when nearly every day you had to be moving all the time – that is, you couldn't sit still and paced up and down?	YES	NO
6. Has there ever been a period of 2 weeks or more when you lacked energy or felt tired out all the time even when you had not been working very hard?	YES	NO
7. Has there ever been 2 weeks or more when nearly every day you felt worthless, sinful, or guilty?	YES	NO
8a. Has there ever been 2 weeks or longer when nearly every day you had a lot more trouble concentrating than is normal for you?	YES	NO
b. Has there ever been 2 weeks or more when nearly every day your thoughts came much slower than usual or seemed mixed up?	YES	NO
c. Have you ever had 2 weeks or more when nearly every day you were unable to make up your mind about things you ordinarily have no trouble deciding about?	YES	NO
9a. Has there ever been a period of 2 weeks or more when you thought a lot about death – your own, someone else's, or death in general?	YES	NO
b. Has there ever been a period of 2 weeks or more when you felt like you wanted to die?	YES	NO
c. Have you ever felt so low you thought about committing suicide?	YES	NO
d. Have you ever attempted suicide?		

If you answered "yes" to any of the items 1-9, complete the rest of the questionnaire:

<p>10. Was any spell so bad that it influenced your life such as your work, your social life, personal or family life? If yes, explain how:</p>	<p>YES</p>	<p>NO</p>
<p>11a. Did any of those spells occur just after someone close to you or a beloved pet died? b. Did any of those spells occur during a period when you had a medical condition? c. Did any of those spells occur during a period when you were using drugs or alcohol? d. Did any of those spells occur when you were <u>NOT</u> grieving, <u>NOT</u> having a medical condition, and <u>NOT</u> using drugs or alcohol?</p>	<p>YES YES YES YES</p>	<p>NO NO NO NO</p>

If you answered "yes" to question 11, please answer the questions below:

- Approximately how many of such spells have you had in your lifetime? \_\_\_\_\_
- When was the last time you had such a spell? \_\_\_\_\_
- Do you think you are currently having such a spell? *YES / NO*  
→ If yes, approximately how long ago did the current spell start  
\_\_\_\_\_

**Appendix 5**

**Section J of Research Questionnaire: Cognitive Styles Questionnaire**

**Section J: CSQ**

**Directions:** Please try to vividly imagine yourself in each of the situations or sequences of events that follow. Picture each situation as clearly as you can and as if the events were happening to you right now. Place yourself in each situation and decide what you feel would have caused it if it actually happened to you. Although events may have many causes, we want you to choose only one—the major cause if the event actually happened to you. For each situation, you will write down this cause in the blank provided. Then we will ask you some questions about the cause. After you have answered the questions about the cause of the event, think about how you'd react if the situation actually occurred in your life and what the occurrence of the situation would mean to you. Then we will ask you some questions about your views of and reactions to the situation.

It is important to remember that there are no right or wrong answers to the questions. The important thing is to answer the questions in a way that corresponds to what you would think and feel if the situations actually were occurring in your life.

\*\*\*\*\*

1. Imagine that the following sequence of events actually happens to you:

**You take an exam and receive a low grade on it.**

Questions 1 a-d ask about the cause of your low grade on the exam.

- a. Write down the one major cause of your low grade on the exam.

- b. Is it something about you or something about other people or circumstances that caused your low grade on the exam: [Circle one number.]

	1	2	3	4	5	6	7	
Totally caused by other people or circumstances								Totally caused by me

- c. In the future when taking exams, will the cause of the low grade on this exam also cause other exam grades of yours to be low? [Circle one number.]

	1	2	3	4	5	6	7	
Will never again cause my exam grades to be low								Will always cause my exam grades to be low

- d. Is the cause of your low grade on the exam something that just causes problems in your exam grades, or does it also cause problems in other areas of your life? [Circle one number.]

	1	2	3	4	5	6	7	
Causes problems just in my exam grades								Causes problems in all areas of my life

Questions 1e-g ask for your views of and reactions to your low grade on the exam and not about the cause of your low grade on the exam.

- e. How likely is it that your receiving a low grade on the exam will lead to other negative things happening to you? [Circle one number.]

	1	2	3	4	5	6	7	
Not at all likely to lead to other negative things happening to me								Extremely likely to lead to other negative things happening to me.

f. *To what degree does your low grade on the exam mean to you that you are flawed in some way? [Circle one number.]*  
 Definitely does not mean I am flawed in some way 1 2 3 4 5 6 7 Definitely does mean I am flawed in some way

g. *How important is it to you that your grade on the exam is low? [Circle one number.]*  
 Not at all important 1 2 3 4 5 6 7 Extremely important

\*\*\*\*\*

2. **Imagine that the following sequence of events actually happens to you:**

**You don't have a boy/girlfriend (or spouse) although you want one.**

Questions 2a-d ask about the cause of your not having a boy/girlfriend (or spouse) although you want one.

a. *Write down the one major cause of your not having a boy/girlfriend (or spouse) although you want one.*

b. *Is it something about you or something about other people or circumstances that caused your not having a boy/girlfriend (or spouse) although you wanted one? [Circle one number.]*  
 Totally caused by other people or circumstances 1 2 3 4 5 6 7 Totally caused by me

c. *In the future when you want a boy/girlfriend (or spouse), will the cause of your not having a boy/girlfriend (or spouse) now also cause you to not have a boy/girlfriend (or spouse) then? [Circle one number.]*  
 Will never again cause me to not have a boy/girlfriend (or spouse) 1 2 3 4 5 6 7 Will always cause me to not have a boy/girlfriend (or spouse)

d. *Is the cause of your not having a boy/girlfriend (or spouse) something that just causes problems in whether or not you have a boy/girlfriend (or spouse), or does it also cause problems in other areas of your life? [Circle one number.]*  
 Causes problems just in whether or not I have a boy/girlfriend (or spouse) 1 2 3 4 5 6 7 Causes problems in all areas of my life

Questions 2e-g ask for your views of and reactions to your not having a boy/girlfriend (or spouse) and not about the cause of your not having a boy/girlfriend (or spouse).

e. *How likely is it that your not having a boy/girlfriend (or spouse) will lead to other negative things happening to you? [Circle one number.]*  
 Not at all likely to lead to other negative things happening to me 1 2 3 4 5 6 7 Extremely likely to lead to other negative things happening to me.



- f. *To what degree does your not having a boy/girlfriend (or spouse) mean to you that you are flawed in some way? [Circle one number.]*  
 Definitely does not mean I am flawed in some way      1      2      3      4      5      6      7      Definitely does mean I am flawed in some way
- g. *How important is it to you that you don't have a boy/girlfriend (or spouse)? [Circle one number].*  
 Not at all important      1      2      3      4      5      6      7      Extremely important

\*\*\*\*\*

3. **Imagine that the following sequence of events actually happens to you:**

**A friend comes to you with a problem, and you are not as helpful as you would like to be.**

Questions 3a-d ask about the cause of your not being as helpful as you would like to be to your friend.

a. *Write down the one major cause of your not being as helpful as you would like to be to your friend.*

b. *Is it something about you or something about other people or circumstances that caused your not being as helpful as you would like to be to your friend? [Circle one number.]*  
 Totally caused by other people or circumstances      1      2      3      4      5      6      7      Totally caused by me

c. *In the future when a friend comes to you with a problem, will the cause of your not being as helpful as you would like to be to your friend now also cause you to not be as helpful as you would like to be to your friend then? [Circle one number.]*  
 Will never again cause me to not be as helpful as I would like to be      1      2      3      4      5      6      7      Will always cause me to not be as helpful as I would like to be

d. *Is the cause of your not being as helpful as you would like to be to your friend something that just causes problems in your helping friends, or does it also cause problems in other areas of your life? [Circle one number.]*  
 Causes problems just in my helping friends      1      2      3      4      5      6      7      Causes problems in all areas of my life

Questions 3e-g ask for your views of and reactions to your not being as helpful as you would like to be to your friend and not about the cause of your not being as helpful as you would like to be to your friend.

e. *How likely is it that your not being as helpful as you would like to be to your friend will lead to other negative things happening to you? [Circle one number.]*  
 Not at all likely to lead to other negative things happening to me      1      2      3      4      5      6      7      Extremely likely to lead to other negative things happening to me.

f. To what degree does your not being as helpful as you would like to be to your friend mean to you that you are flawed in some way? [Circle one number.]

Definitely does not mean I am flawed in some way	1	2	3	4	5	6	7	Definitely does mean I am flawed in some way
--	---	---	---	---	---	---	---	--

g. How important is it to you that you are not being as helpful as you would like to be to your friend? [Circle one number.]

Not at all important	1	2	3	4	5	6	7	Extremely important
----------------------	---	---	---	---	---	---	---	---------------------

\*\*\*\*\*

4. Imagine that the following sequence of events actually happens to you:

**As an assignment, you give an important talk in class, and the class reacts negatively.**

Questions 4a-d ask about the cause of the class reacting negatively to your talk.

a. Write down the one major cause of the class reacting negatively to your talk.

b. Is it something about you or something about other people or circumstances that caused the class to react negatively to your talk? [Circle one number.]

Totally caused by other people or circumstances	1	2	3	4	5	6	7	Totally caused by me
---	---	---	---	---	---	---	---	----------------------

c. In the future when you give important talks in class, will the cause of the class reacting negatively to your talk now also cause the class to react negatively to your talk then? [Circle one number.]

Will never again cause the class to react negatively to my talks	1	2	3	4	5	6	7	Will always cause the class to react negatively to my talks
--	---	---	---	---	---	---	---	---

d. Is the cause of the class reacting negatively to your talk something that just causes problems when you give talks, or does it also cause problems in other areas of your life? [Circle one number.]

Causes problems just when I give talks	1	2	3	4	5	6	7	Causes problems in all areas of my life
--	---	---	---	---	---	---	---	---

Questions 4e-g ask for your views of and reactions to the class reacting negatively to your talk and not about the cause of the class reacting negatively to your talk.

e. How likely is it that the class reacting negatively to your talk will lead to other negative things happening to you? [Circle one number.]

Not at all likely to lead to other negative things happening to me	1	2	3	4	5	6	7	Extremely likely to lead to other negative things happening to me.
--	---	---	---	---	---	---	---	--

f. To what degree does the class reacting negatively to your talk mean to you that you are flawed in some way? [Circle one number.]

Definitely does not mean I am flawed in some way	1	2	3	4	5	6	7	Definitely does mean I am flawed in some way
--	---	---	---	---	---	---	---	--

g. How important is it to you that the class reacted negatively to your talk? [Circle one number].  
 Not at all important 1 2 3 4 5 6 7 Extremely important

\*\*\*\*\*

5. Imagine that the following sequence of events actually happens to you:

**Your parents have been treating you in a negative way.**

Questions 5a-d ask about the cause of your parents treating you in a negative way.

a. Write down the one major cause of your parents treating you in a negative way



b. Is it something about you or something about other people or circumstances that caused your parents to treat you in a negative way? [Circle one number.]  
 Totally caused by other people or circumstances 1 2 3 4 5 6 7 Totally caused by me

c. In the future when interacting with your parents, will the cause of your parents treating you in a negative way now also cause your parents to treat you in a negative way then? [Circle one number.]  
 Will never again cause my parents to treat me in a negative way 1 2 3 4 5 6 7 Will always cause my parents to treat me in a negative way

d. Is the cause of your parents treating you in a negative way something that just causes problems when you interact with them, or does it also cause problems in other areas of your life? [Circle one number.]  
 Causes problems just when I interact with my parents 1 2 3 4 5 6 7 Causes problems in all areas of my life

Questions 5e-g ask for your views of and reactions to your parents treating you in a negative way and not about the cause of your parents treating you in a negative way.

e. How likely is it that your parents treating you in a negative way will lead to other negative things happening to you? [Circle one number.]  
 Not at all likely to lead to other negative things happening to me 1 2 3 4 5 6 7 Extremely likely to lead to other negative things happening to me.

f. To what degree does your parents treating you in a negative way mean to you that you are flawed in some way? [Circle one number.]  
 Definitely does not mean I am flawed in some way 1 2 3 4 5 6 7 Definitely does mean I am flawed in some way

g. How important is it to you that your parents treat you in a negative way? [Circle one number].  
 Not at all important 1 2 3 4 5 6 7 Extremely important

\*\*\*\*\*

6. Imagine that the following sequence of events actually happens to you:

Your gradepoint average (GPA) for the semester is low.

Questions 6a-d ask about the cause of your low gradepoint average (GPA) for the semester.

a. Write down the one major cause of your low gradepoint average (GPA) for the semester.

b. Is it something about you or something about other people or circumstances that caused your low gradepoint average (GPA) for the semester? [Circle one number.]

Totally caused by other people or circumstances	1	2	3	4	5	6	7	Totally caused by me
---	---	---	---	---	---	---	---	-------------------------

c. In the future when you receive your grades for a semester will the cause of this semester's low gradepoint average (GPA) also cause other semesters' gradepoint averages (GPA's) of yours to be low? [Circle one number.]

Will never again cause my semester gradepoint averages (GPA's) to be low	1	2	3	4	5	6	7	Will always cause my semester gradepoint averages (GPA's) to be low
---	---	---	---	---	---	---	---	--

d. Is the cause of your low gradepoint average (GPA) for the semester something that just causes problems in your grades, or does it also cause problems in other areas of your life? [Circle one number.]

Causes problems just in my grades	1	2	3	4	5	6	7	Causes problems in all areas of my life
---	---	---	---	---	---	---	---	---

Questions 6e-g ask for your views of and reactions to your low gradepoint average (GPA) for the semester and not about the cause of your low gradepoint average (GPA) for the semester.

e. How likely is it that your low gradepoint average (GPA) for the semester will lead to other negative things happening to you? [Circle one number.]

Not at all likely to lead to other negative things happening to me	1	2	3	4	5	6	7	Extremely likely to lead to other negative things happening to me.
---	---	---	---	---	---	---	---	---

f. To what degree does your low gradepoint average (GPA) for the semester mean to you that you are flawed in some way? [Circle one number.]

Definitely does not mean I am flawed in some some way	1	2	3	4	5	6	7	Definitely does mean I am flawed in some way
--	---	---	---	---	---	---	---	---

g. How important is it to you that your gradepoint average (GPA) for the semester is low? [Circle one number].

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

\*\*\*\*\*

7. Imagine that the following sequence of events actually happens to you:

At a party, people don't act interested in you.

Questions 7a-d ask about the cause of people not acting interested in you at the party.

a. Write down the one major cause of people not acting interested in you at the party.

b. Is it something about you or something about other people or circumstances that caused people to not act interested in you at the party? [Circle one number.]

	1	2	3	4	5	6	7	
Totally caused by other people or circumstances								Totally caused by me

c. In the future when at parties, will the cause of people not acting interested in you at this party also cause people to not act interested in you at other parties? [Circle one number.]

	1	2	3	4	5	6	7	
Will never again cause people to not act interested in me at parties								Will always cause people to not act interested in me at parties

d. Is the cause of people not acting interested in you at the party something that just causes problems at parties, or does it also cause problems in other areas of your life? [Circle one number.]

	1	2	3	4	5	6	7	
Causes problems just in my interactions at parties								Causes problems in all areas of my life

Questions 7e-g ask for your views of and reactions to your low gradepoint average (GPA) for the semester and not about the cause of your low gradepoint average (GPA) for the semester.

e. How likely is it that people not acting interested in you at the party will lead to other negative things happening to you? [Circle one number.]

	1	2	3	4	5	6	7	
Not at all likely to lead to other negative things happening to me								Extremely likely to lead to other negative things happening to me.

f. To what degree does people not acting interested in you at the party mean to you that you are flawed in some way? [Circle one number.]

	1	2	3	4	5	6	7	
Definitely does not mean I am flawed in some way								Definitely does mean I am flawed in some way

h. How important is it to you that at a party, people don't act interested in you? [Circle one number].

	1	2	3	4	5	6	7	
Not at all important								Extremely important

\*\*\*\*\*

8. Imagine that the following sequence of events actually happens to you:

**You can't get all the work done that others expect of you.**

Questions 8a-d ask about the cause of your not getting all the work done that others expect of you.

a. Write down the one major cause of your not getting all the work done that others expect of you.

b. Is it something about you or something about other people or circumstances that caused your not getting all the work done that others expect of you? [Circle one number.]

Totally caused by other people or circumstances	1	2	3	4	5	6	7	Totally caused by me
---	---	---	---	---	---	---	---	-------------------------

c. In the future when doing the work that others expect, will the cause your not getting all the work done that others expect of you now also cause you to not get all the work done then? [Circle one number.]

Will never again cause me to not get all the work done	1	2	3	4	5	6	7	Will always cause me to not get all the work done
---	---	---	---	---	---	---	---	--

d. Is the cause of your not getting all the work done that others expect of you something that just causes problems in your getting the work done that others expect, or does it also cause problems in other areas of your life? [Circle one number.]

Causes problems just in getting the work done that others expect	1	2	3	4	5	6	7	Causes problems in all areas of my life
---	---	---	---	---	---	---	---	---

Questions 8e-g ask for your views on and reactions to your not getting all the work done that others expect of you and not about the cause of your not getting all the work done that others expect of you.

e. How likely is it that your not getting all the work done that others expect of you will lead to other negative things happening to you? [Circle one number.]

Not at all likely to lead to other negative things happening to me	1	2	3	4	5	6	7	Extremely likely to lead to other negative things happening to me.
---	---	---	---	---	---	---	---	---

f. To what degree does your not getting all the work done that others expect of you mean to you that you are flawed in some way? [Circle one number.]

Definitely does not mean I am flawed in some some way	1	2	3	4	5	6	7	Definitely does mean I am flawed in some way
--	---	---	---	---	---	---	---	---

g. How important is it to you that you can't get all the work done that others expect of you? [Circle one number].

Not at all important	1	2	3	4	5	6	7	Extremely important
-------------------------	---	---	---	---	---	---	---	------------------------

\*\*\*\*\*

9. Imagine that the following sequence of events actually happens to you:

**You apply for admission into graduate or professional schools but don't get accepted at any you want to attend.**

Questions 9a-d ask you about the cause of your not getting accepted at any of the graduate or professional schools you want to attend.

a. Write down the one major cause of your not getting accepted at any of the graduate or professional schools you want to attend.

b. Is it something about you or something about other people or circumstances that caused your not getting accepted at any of the graduate or professional schools you want to attend? [Circle one number.]

Totally caused by other people or circumstances	1	2	3	4	5	6	7	Totally caused by me
---	---	---	---	---	---	---	---	----------------------

c. In the future when applying for admission into graduate or professional schools, will the cause of your not getting accepted at any of the graduate or professional schools you want to attend now also cause you to not get accepted at any of the graduate or professional schools you want to attend then? [Circle one number.]

Will never again cause me to not get accepted at the graduate or professional schools I want to attend	1	2	3	4	5	6	7	Will always cause me to not get accepted at the graduate or professional schools I want to attend
--	---	---	---	---	---	---	---	---

d. Is the cause of your not getting accepted at any of the graduate or professional schools you want to attend something that just causes problems in your getting accepted at graduate or professional schools you want to attend, or does it also cause problems in other areas of your life? [Circle one number.]

Causes problems just in getting accepted at graduate or professional schools I want to attend	1	2	3	4	5	6	7	Causes problems in all areas of my life
---	---	---	---	---	---	---	---	---

Questions 9e-g ask for your views of and reactions to your not getting accepted at any of the graduate or professional schools you want to attend and not about the cause of your not being accepted.

e. How likely is it that your not getting accepted at any of the graduate or professional schools you want to attend will lead to other negative things happening to you? [Circle one number.]

Not at all likely to lead to other negative things happening to me	1	2	3	4	5	6	7	Extremely likely to lead to other negative things happening to me.
--	---	---	---	---	---	---	---	--

f. To what degree does your not getting accepted at any of the graduate or professional schools you want to attend mean to you that you are flawed in some way? [Circle one number.]

Definitely does not mean I am flawed in some way	1	2	3	4	5	6	7	Definitely does mean I am flawed in some way
--	---	---	---	---	---	---	---	--

g. How important is it to you that you don't get accepted at any of the graduate or professional schools you want to attend? [Circle one number].

Not at all	1	2	3	4	5	6	7	Extremely
important								important

\*\*\*\*\*

10. Imagine that the following sequence of events actually happens to you:

During the first year of working in the career of your choice, you receive a negative evaluation of your job performance from your employer.

Questions 10a-d ask about the cause of the negative evaluation of your job performance from your employer.

a. Write down the one major cause of the negative evaluation of your job performance from your employer.

b. Is it something about you or something about other people or circumstances that caused the negative evaluation of your job performance from your employer? [Circle one number.]

Totally caused	1	2	3	4	5	6	7	Totally caused
by other people								by me
or circumstances								

c. In the future when your job performance in the career of your choice is evaluated, will the cause of this negative job evaluation also cause other job evaluations to be negative? [Circle one number.]

Will never again	1	2	3	4	5	6	7	Will always
cause my job								cause my job
evaluations to								evaluations to
be negative								be negative

d. Is the cause of the negative evaluation of your job performance from your employer something that just causes problems in your job evaluations in the career of your choice, or does it also cause problems in other areas of your life? [Circle one number.]

Causes problems	1	2	3	4	5	6	7	Causes problems
just in my job								in all areas
performance in								of my life
the career of my choice								

Questions 10e-g ask for your views of and reactions to the negative evaluation of your job performance from your employer and not about the cause of the negative evaluation of your job performance from your employer.

e. How likely is it that the negative evaluation of your job performance from your employer will lead to other negative things happening to you? [Circle one number.]

Not at all likely	1	2	3	4	5	6	7	Extremely likely
to lead to other								to lead to other
negative things								negative things
happening to me								happening to me.

f. To what degree does the negative evaluation of your job performance from your employer mean to you that you are flawed in some way? [Circle one number.]

Definitely does	1	2	3	4	5	6	7	Definitely does
not mean I am								mean I am
flawed in some								flawed in
some way								some way



- g. How important is it to you that during the first year of working in the career of your choice, you receive a negative evaluation of your job performance from your employer? [Circle one number].
- |                      |   |   |   |   |   |   |   |                     |
|----------------------|---|---|---|---|---|---|---|---------------------|
| Not at all important | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Extremely important |
|----------------------|---|---|---|---|---|---|---|---------------------|

11. Imagine that the following sequence of events actually happens to you:

**Your relationship with your boy/girlfriend (or spouse) ends even though you would like it to continue.**

Questions 11a-d ask about the cause of your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue.

- a. Write down the one major cause of your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue

- b. Is it something about you or something about other people or circumstances that caused your relationship with your boy/girlfriend (or spouse) to end even though you would like it to continue? [Circle one number.]
- |   |   |   |   |   |   |   |   |                      |
|---|---|---|---|---|---|---|---|----------------------|
| Totally caused by other people or circumstances | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Totally caused by me |
|---|---|---|---|---|---|---|---|----------------------|

- c. In the future when you are involved in a relationship, will the cause your relationship with your boy/girlfriend (or spouse) ending now also cause other relationships with boy/girlfriends (or spouses) to end even though you would like them to continue? [Circle one number.]
- |  |   |   |   |   |   |   |   |   |
|--|---|---|---|---|---|---|---|---|
| Will never again cause my relationships with boy/girlfriends (or spouses) to end | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Will always cause my relationships with boy/girlfriends (or spouses) to end |
|--|---|---|---|---|---|---|---|---|

- d. Is the cause of your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue something that just causes problems in your relationships, or does it also cause problems in other areas of your life? [Circle one number.]
- |  |   |   |   |   |   |   |   |   |
|--|---|---|---|---|---|---|---|---|
| Causes problems just in my relationships | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Causes problems in all areas of my life |
|--|---|---|---|---|---|---|---|---|

Questions 11e-g ask for your views of and reactions to your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue and not about the cause of your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue.

- e. How likely is it that the ending of your relationship with your boy/girlfriend (or spouse) will lead to other negative things happening to you? [Circle one number.]
- |  |   |   |   |   |   |   |   |  |
|--|---|---|---|---|---|---|---|--|
| Not at all likely to lead to other negative things happening to me | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Extremely likely to lead to other negative things happening to me. |
|--|---|---|---|---|---|---|---|--|
- f. To what degree does your relationship with your boy/girlfriend (or spouse) ending even though you would like it to continue mean to you that you are flawed in some way? [Circle one number.]
- |  |   |   |   |   |   |   |   |  |
|--|---|---|---|---|---|---|---|--|
| Definitely does not mean I am flawed in some way | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Definitely does mean I am flawed in some way |
|--|---|---|---|---|---|---|---|--|

g. How important is it to you that your relationship with your boy/girlfriend (or spouse) ends even though you would like it to continue? [Circle one number].  
 Not at all      1      2      3      4      5      6      7      Extremely important  
 important

\*\*\*\*\*

12. Imagine that the following sequence of events actually happens to you:

A person with whom you really want to be friends does not want to be friends with you.

Questions 12a-d ask about the cause of the person not wanting to be friends with you.

a. Write down the one major cause of the person not wanting to be friends with you

b. Is it something about you or something about other people or circumstances that caused the person to not want to be friends with you? [Circle one number.]  
 Totally caused by other people or circumstances      1      2      3      4      5      6      7      Totally caused by me

c. In the future when you want to be friends with someone, will the cause of this person not wanting to be friends with you also cause other people to not want to be friends with you? [Circle one number.]  
 Will never again cause other people to not want to be friends with me      1      2      3      4      5      6      7      Will always cause other people to not want to be friends with me

d. Is the cause of the person not wanting to be friends with you something that just causes problems in your making friends, or does it also cause problems in other areas of your life? [Circle one number.]  
 Causes problems just in my making friends      1      2      3      4      5      6      7      Causes problems in all areas of my life

Questions 12e-g ask for your views on and reactions to the person not wanting to be friends with you and not about the cause of the person not wanting to be friends with you.

e. How likely is it that the person not wanting to be friends with you will lead to other negative things happening to you? [Circle one number.]  
 Not at all likely to lead to other negative things happening to me      1      2      3      4      5      6      7      Extremely likely to lead to other negative things happening to me.

f. To what degree does the person not wanting to be friends with you mean to you that you are flawed in some way? [Circle one number.]  
 Definitely does not mean I am flawed in some way      1      2      3      4      5      6      7      Definitely does mean I am flawed in some way

g. How important is it to you that person with whom you really want to be friends does not want to be friends with you? [Circle one number].  
 Not at all important      1      2      3      4      5      6      7      Extremely important

**Appendix 6**

**Section K of Research Questionnaire: Winter Stress Scale**

**Section K : Please respond to the questions below and write clearly.**

1. How much do you dread the winter?

- 1                      2                      3                      4                      5  
 not at all          a little              moderately          very                  extremely

• Please explain your answer:

2. Some people experience certain changes in themselves during the winter. If you experience these changes as well, please rate how stressful they are for you.

- 0            1            2            3            4            5  
 does not   not stressful   quite   moderately   very   extremely  
 apply      at all      stressful   stressful   stressful   stressful

CHANGES	0	1	2	3	4	5
Carbohydrate craving	0	1	2	3	4	5
Weight gain	0	1	2	3	4	5
Energy loss	0	1	2	3	4	5
Increased sleep	0	1	2	3	4	5
Other (please specify below):	0	1	2	3	4	5

Please provide additional information below to help us understand better your experience with the changes (where applicable):

3. *To what degree do the winter changes (the ones that you endorsed in question 2) interfere with your ability to cope with daily life difficulties in your life (i.e., work, academic, social, family, and personal)?*

1                      2                      3                      4                      5  
 not at all          a little              moderately          very                  extremely

*Please explain your answer:*

- 
4. *How often do you engage in the following behaviour? Please use the rating scale below to answer the questions.*

1                      2                      3                      4                      5  
 never                  rarely                  sometimes          often                  always

- (a) During the winter I find myself thinking about how any changes in mood and behavior that I experience may (or actually do) affect my life. \_\_\_\_\_
  - (b) During the winter/fall I find myself wishing that I could go outside more. \_\_\_\_\_
  - (c) During the winter/fall I think about how I never seem to have enough energy to do what I can during the summer. \_\_\_\_\_
  - (d) During the winter/fall I think things like, "I won't be able to do job/work because I feel so bad". \_\_\_\_\_
  - (e) During the winter/fall I find myself thinking about how any changes in mood in behavior that I am experiencing affect my life. \_\_\_\_\_
  - (f) During the winter/fall I wish that I felt more like I do during the summer. \_\_\_\_\_
-

5. Do you experience any negative mood or behavioural changes (e.g, feelings, sleep, appetite, social interactions, weight, etc.) from summer to fall/winter? ..... Yes / No

*If yes, please answer the questions below:*

(I) Please list the reason(s) why you think you experience the negative mood or behavioural changes.

(S1) Do you think that the changes you experience during the fall/winter will disappear when the weather gets better?.....Yes / No

Please explain why:

(S2) Do you think that you will continue to experience these changes year after year?.....Yes/No

Please explain why:

(G) Do these changes affect you in your:

- Personal life.....Yes / No
- Family life.....Yes / No
- Social life.....Yes / No
- Academic life (if applicable).....Yes / No
- Career life (if applicable).....Yes / No

6. Please answer the questions using the rating scale given below:

1	2	3	4	5
disagree strongly	disagree	neutral	agree	agree strongly

**During the fall and/or winter:**

- (a) I get more upset than usual because of something that happened unexpectedly \_\_\_\_\_
- (b) I am less able to control the important things in my life \_\_\_\_\_
- (c) I feel more nervous and “stressed” than usual \_\_\_\_\_
- (d) I am more successful than usual at dealing with irritating life hassles \_\_\_\_\_
- (e) I cope more effectively than usual with important changes that are occurring in my life \_\_\_\_\_
- (f) I am more confident than usual about my ability to handle my personal problems \_\_\_\_\_
- (g) I feel more than usual that things go my way \_\_\_\_\_
- (h) I am less able to cope than usual with things that I have to do \_\_\_\_\_
- (i) I am less able than usual to control irritations in my life \_\_\_\_\_
- (j) I feel more on top of things than usual \_\_\_\_\_
- (k) I get more angry than usual because of things that happen that (l) are outside of my control \_\_\_\_\_
- (m) I think more than usual of things that I have to accomplish \_\_\_\_\_
- (n) I am more able to control the way I spend my time \_\_\_\_\_
- (o) I feel more so than usual that difficulties are piling up so high that I can not overcome them \_\_\_\_\_

**Appendix 7**

**Recruitment Posters**



*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 research projects investigating 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**

**Letters of Health Service Providers**

Date of letter  
Name of Health Service Provider  
Address  
Address

Dear Dr. X,

The Psychology Department of Lakehead University is conducting research on depression and Seasonal Affective Disorder (winter blues). Our projects investigate the psychological characteristics, coping strategies, and winter stress experiences of seasonally depressed, sub-syndromal SAD and nonseasonally depressed individuals. As well, we are examining the effects of light intervention on the symptoms.

As part of our recruitment efforts, we would like to contact individuals who have these disorders and present information on our studies to them. Given that you have extensive contact with patients, we hope that you will consider assisting us by informing the appropriate patients about our efforts to contact people in the community who have depression and the winter blues. This can be done simply by posting our recruitment poster in your waiting room or leaving some posters for pick-up by patients in your reception area.

Another more direct way would be to inform the appropriate patients of our recruitment efforts and giving them a copy of or recruitment poster. Any patient who is interested more information can call our Recruitment Coordinator, Mr. Rob Dew, at 346-7756. The name and telephone number is listed on the poster.

We hope that you will assist us in reaching out to your patients. If you have any questions or concerns, please do not hesitate to contact either of us below. Thank you very much.

Sincerely,

Josephine Tan, Ph.D., C. Psych.  
Associate Professor / Clinical Psychologist  
346-7761

**Appendix 9**

**Informed Consent Form for All Participants**

Informed Consent Form (for ALL Participants)

1. Title of research: Winter Experiences and Functioning
  2. This study serves two purposes: a) it investigates the emotional, cognitive and behavioral experiences of people during the winter and b) it serves as an assessment to determine whether you meet the research criteria for another study on the winter blues
  3. You will be asked to complete a questionnaire that asks you questions about what you think, feel, and do during the winter. It will take you approximately 1.5 to 2 hours.
  4. Your participation is strictly voluntary and you are free to withdraw from the study at any time. You may also refrain from answering any question that you do not wish to answer.
  5. All of your responses are strictly confidential.
  6. We will keep your name solely for the purpose of contacting you so that we can inform you about the outcome of this assessment. Your name will not be attached to your responses when we examine the research questionnaires from all our participants. That way nothing can be traced back to you.
  7. There is no risk or benefit to you by participating in this study.
  8. If you are not an introductory psychology student, you will be entered into three \$100 draws for your participation. If you are an Introductory Psychology student, you will receive 2 bonus points towards your course marks, or three \$100 draws if you have already maximized the number of points you can get in your course.
  9. All data will remain in a secure and confidential storage with Dr. Tan at Lakehead University for seven years. Upon the completion of this seven year period the questionnaires will be destroyed.
- If you have reward the above, understand it, and wish to participate in this study, please sign below to indicate your informed consent for participation.

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

**Appendix 10**

**Debriefing**

### Debriefing

Thank you for your participation in this study. We'd like to tell you more about this research project. We are conducting this study to see how individuals with seasonal affective disorder (SAD) and depression are different from each other. SAD is also known as "winter blues" and is the kind of depression that comes on during the fall and/or winter. By summer, the person feels better and is not depressed anymore. When the SAD person is depressed, generally the symptoms are similar to the regular nonseasonal form of depression except for some atypical symptoms such as craving for high carbohydrate foods, weight gain, sleeping more than usual, and feelings of fatigue. Not all SAD people will experience these symptoms though.

It is assumed that although SAD and nonseasonal depression are similar, they are caused by different events. For example, whereas depression may be caused by a tendency to see the world and the self in a negative and critical way or by stress, SAD is precipitated by the winter. Research has indicated that insufficient exposure to light in the winter is a factor.

However, because people with SAD show similar depressive symptoms as people with nonseasonal depression, we wanted to find out whether the way SAD people think when they are depressed, how they behave, how they cope with their depression, and their reactions to winter are similar or different from people who are nonseasonally depressed.

For instance, people who are depressed tend to blame their depression on themselves. Do SAD people do the same, or do they recognize that their depression is related to the winter and therefore, react differently? Depressed people also tend to react to their depression by ruminating or thinking of negative things over and over again. Do SAD people do the same thing or do they try to distract themselves by being active?

We also wanted to look at people who are subsyndromal SAD, meaning that their winter blues is not as serious as the full-blown SAD. Subsyndromal SAD is something that research has not paid much attention. We wanted to find out how similar or different the subsyndromal SAD people are from the SAD people, and from the nonseasonally depressed people.

Your participation has been invaluable in helping us to understand winter blues better. If you have any questions about this study, we would be very happy to answer them. You can contact either Rob Dew at 346-7756 or Dr. Josephine Tan (project supervisor) at 346-7751. If you asked for a summary of the results, we will be mailing it to you sometime in August after all the data has been collected and analyzed.

We would like to ask a favour of you. We hope that you will not discuss what you know of this study openly with others until after the end of April. This is because until then, we will still be running the project, and we don't want people to know in full details exactly what we are investigating until they have completed the study themselves. Having such information ahead of time might influence the answers they give us.

Thank you again for your help in our project. Your participation has been most invaluable!

Rob Dew

MA Clinical Psychology Student

346-7756

Dr. Josephine Tan

Associate Professor/Supervisor

346-7751



**Appendix 11**

**Counselling and Therapy Resource List**

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.*