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# The Role of Cognitive Factors in the Development of Seasonal Affective Disorder Episodes

Stacy Whitcomb-Smith

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THE ROLE OF COGNITIVE FACTORS IN THE DEVELOPMENT OF  
SEASONAL AFFECTIVE DISORDER EPISODES

By

Stacy Whitcomb-Smith

B.A., Williams College, 1995

A THESIS

Submitted in Partial Fulfillment of the  
Requirements for the Degree of  
Doctor of Philosophy  
(in Psychology)

The Graduate School

The University of Maine

December, 2003

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# THE ROLE OF COGNITIVE FACTORS IN THE DEVELOPMENT OF SEASONAL AFFECTIVE DISORDER EPISODES

By Stacy Whitcomb-Smith

Thesis Advisor: Dr. Sandra T. Sigmon

An Abstract of the Thesis Presented  
in Partial Fulfillment of the Requirements for the  
Degree of Doctor of Philosophy  
(in Psychology)  
December, 2003

Recent literature on Seasonal Affective Disorder (SAD) has begun to focus on diathesis stress models, including Young and colleagues' (1991) Dual Vulnerability Hypothesis (DVH). The DVH posits that individuals must possess both a biological vulnerability to developing vegetative symptoms and a psychological vulnerability to developing mood symptoms in order to develop SAD episodes. Such a model addresses SAD as well as non-seasonal depression, and suggests that there may be an as yet unidentified group suffering from only the biological vulnerability (i.e., winter anergia). However, until very recently few studies have directly tested this model, and most have focused on the possible psychological mechanisms related to mood symptoms (e.g., McCarthy et al., 2002; Young & Azam, 2003). Research has demonstrated a temporal relation between mood and vegetative symptoms, with vegetative symptoms having an earlier onset than mood symptoms (McCarthy et al., 2002; Young et al., 1991) supporting the idea that separate factors related to the two symptom clusters exist. The current study represents a longitudinal assessment of vegetative and mood symptoms, as

well as cognitive factors (i.e., rumination, automatic thoughts, attentional bias) that may represent part of the psychological vulnerability shared by SAD sufferers. Furthermore, the present study represents the first attempt to recruit and classify individuals with winter anergia (i.e., individuals possessing only a biological vulnerability component). Sixty-seven individuals participated in the study across three groups, individuals with a history of SAD (i.e., SAD-HX), history of winter anergia (i.e., WA) and with no history of depression. Findings supported the DVH, with an early vegetative symptom onset than mood symptom for the SAD-HX group. SAD-HX group participants also evidenced more ruminative responses and negative automatic thoughts about the seasons. Findings are generally supportive of Young et al.'s (1991) DVH and directions for future research are suggested.

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## Chapter I

### INTRODUCTION

Although the effects of weather and season on mood have received some attention for centuries (Wehr et al., 1986), Seasonal Affective Disorder (i.e., winter depression) has only been systematically researched over the past few decades. In 1984, Rosenthal and colleagues described and named this syndrome Seasonal Affective Disorder (SAD; Rosenthal et al., 1984). Since the 1980s, investigators have begun to examine the relations between the changing of the seasons and the onset of mood and behavior alterations associated with SAD. Several biological and genetic models have been offered to explain SAD phenomena but only recently have researchers begun to explore the contributions of psychological factors.

Although the vast majority of individuals experience some seasonal mood and behavior changes, (i.e., as much as 92% of the population), a smaller percentage (i.e., 2 - 10%) report experiencing significant changes in mood and behavior that affect their ability to function (Kasper et al., 1989). Because of the widespread experience of seasonal mood and behavior changes (i.e., seasonality), SAD researchers have proposed that seasonality exists on a continuum (Kasper et al., 1989). Examining subsets of the population with symptom profiles that vary along the seasonality continuum from significant to subclinical (i.e., sub-syndromal) to normative may lead to answers about why some individuals develop the disorder. For example, if individuals with SAD share other characteristics besides diagnosis that are not seen in individuals with sub-syndromal or normative degree of seasonal changes, identification of these characteristics may point to additional important etiological factors. In addition, investigating individuals who

experience sub-syndromal levels of seasonality may lead to the identification of protective factors that guard against development of the disorder. Such protective factors may provide answers to questions regarding etiology as well as provide ideas for alternative treatment interventions.

Until recently, much of the empirical focus regarding etiological factors in SAD has been from a biological perspective. Biological theories of SAD have tended to center on the relation between an individual's exposure to light and SAD symptom onset. During the fall and winter, less sunlight becomes available because the days shorten. Due to this observation that less available light coincides with subsequent symptom onset, early research on SAD sought to compensate for the lower levels of light in the environment by exposing SAD sufferers to artificial bright light (e.g., Rosenthal et al., 1984). This type of bright light therapy appears to be an effective treatment for some individuals with SAD (see Terman et al., 1989). However, researchers need to be cautious about inferring causality from treatment studies. For example, a reduction of seasonal symptoms may transpire through pathways separate from etiological mechanisms. In addition, biological models inferring causality related to light exposure have not been able to account for annual differences in the experience of SAD from year to year. Researchers have found that symptom severity in individuals differs from year to year such that some individuals with a history of winter depressive episodes do not meet criteria for a depressive episode every winter (e.g., Rohan, Sigmon, & Dorhofer, 2003; Rosenthal et al., 1984). Although further research may lead to more detailed hypotheses of mechanisms relating light availability to subsequent symptom onset, it is likely that non-physiological factors are also involved.

In an effort to examine the role that psychological factors may play in SAD episode development and symptom severity, researchers have begun to investigate cognitive processing in individuals with SAD (e.g., Bouhuys, Meester, Jansen, & Bloem, 1994; Rohan et al., 2003; Spinks & Dalgleish, 2001). Researchers have compared cognitive processes in individuals experiencing SAD with Controls (e.g., Bouhuys et al., 1994; Rohan et al., 2003) and with individuals suffering from non-seasonal depression (Sigmon, Whitcomb-Smith, Kendrew & Boulard, 2003). These initial studies suggest that cognitive factors (e.g., attentional bias, increased rumination) are related to SAD, similarly to the hypothesized relation of these factors to nonseasonal depression (e.g., Gotlib & McCann, 1984; Nolen-Hoeksema, 1987). Although cognitive processes are less likely to play a major role in the initial onset of the disorder, cognitive factors have been hypothesized to play a role in the onset of subsequent episodes, the maintenance of the disorder and the experience of SAD symptoms (Rohan et al., 2001). However, the precise function of cognitive factors in SAD has yet to be determined. This study is designed to investigate further the role of cognitive factors in the maintenance and experience of SAD symptoms.

By integrating the findings from biological research on SAD and the more recent research on cognitive factors related to SAD, researchers have proposed two possible diathesis-stress models. The earlier model, the dual vulnerability hypothesis, proposes that individuals must possess a biological vulnerability to developing the vegetative symptoms (i.e., physical symptoms such as fatigue, increased appetite, hypersomnia) of SAD *and* a psychological vulnerability to developing depression (Young, Watel, Lahmeyer, & Eastman, 1991) in order to develop SAD. The authors suggest that the



biological vulnerability is a physiological sensitivity to changes in photoperiod related to onset of vegetative symptoms. However, they do not detail what comprises the psychological vulnerability. Although later research suggests that rumination may play a role, the psychological vulnerability component needs to be addressed more thoroughly in the literature. This model predicts that individuals without both vulnerabilities would not develop SAD, but rather subclinical levels of symptoms (i.e., sub-syndromal). Since the dual vulnerability hypothesis was proposed, based upon the findings of one retrospective study, only a few studies directly addressing the model were found in a search of the literature (Lam et al., 2001, McCarthy, Tarrier & Gregg, 2002; Young & Azam, 2003).

A later diathesis-stress model of SAD (Sigmon, Rohan, & Boulard, 2001) expands upon the dual vulnerability hypothesis by integrating Lewinsohn's behavioral theory of depression (1974) and Beck's cognitive theory of depression (e.g., Beck, 1969).

According to the model, reduced positive reinforcement from the environment and rumination by the individual also play a role in the maintenance of depression in SAD. Similarly, this second model is based on preliminary data (Rohan et al., 2003) and also has not been adequately tested. The proposed study seeks to test the hypothesis that there are two separate clusters of SAD symptoms with unique onset, as predicted by the dual vulnerability hypothesis (McCarthy, et al., 2002; Young et al., 1991). In addition, this study will investigate the psychological vulnerability component proposed by both diathesis-stress models through the examination of possible roles for cognitive factors in SAD.

The following review provides a synopsis of findings in the epidemiological literature as well as greater detail regarding research on biological and psychological

factors that have been implicated in SAD. Biological and diathesis-stress models will be covered, as well as psychological models of non-seasonal depression that may have relevance for understanding SAD processes. Lastly, a new study investigating the dual vulnerability hypothesis and cognitive vulnerability related to SAD is proposed. The current study is designed as a test of Young and colleague's (1991) dual vulnerability hypothesis using prospective symptom measurement. In addition, this study investigates the psychological vulnerability proposed in this hypothesis (1991) by the use of self-report measures and weekly diaries of cognitions. Furthermore, this study examines the hypothesized continuity of seasonality by investigating similarities and differences between individuals with SAD, winter anergia (i.e., only experiencing the vegetative symptoms related to SAD), and Controls (i.e., not experiencing significant changes in mood and behavior across the seasons).

#### Seasonal Affective Disorder: Definition of the Disorder

Clinically, SAD is diagnosed as a recurrent depressive mood disorder (i.e., major depression, bipolar I or bipolar II disorders) with a seasonal specifier (APA, 1994). The defining criteria reflect the characteristic onset and remission of symptoms over the course of the year, namely that symptoms appear in late fall or winter and abate in the spring for the winter type. Although the winter type of SAD is more common, recurrent episodes in summer (summer SAD) have also been recognized (APA, 1994). This investigation will target the more prevalent winter type of SAD.

SAD is often distinguished from non-seasonal depression not only by the temporal pattern of recurrent symptom onset and remission, but also by the atypical depression-symptom profile that most individuals with SAD exhibit. In addition to depressed mood

and anhedonia, the symptomatology of seasonally patterned episodes may include anergia, hypersomnia, overeating, weight gain and carbohydrate craving (APA, 1994). However, according to Diagnostic and Statistical Manual –IV (DSM-IV) criteria, the presence of atypical symptoms is not required to meet criteria for SAD (APA, 1994). In order to meet diagnostic criteria for the seasonal specifier, depression symptoms must occur in a seasonal pattern for two consecutive years in the past with no non-seasonal episodes present (APA, 1994). Thus, seasonality of subsequent onset and remission is the sole invariant characteristic of SAD, with depression ranging in severity from mild to severe. In addition to effects typically associated with nonseasonal depression (e.g., depressed mood, anhedonia), the associated symptoms of SAD (e.g., fatigue and hypersomnia) may have secondary effects of decreased job performance, as well as interference with intimate relationships and friendships (Rosenthal et al., 1984).

Previously, Rosenthal and colleagues (1984) had devised more inclusive criteria in order to ensure that SAD researchers would be consistent in their determination of a SAD diagnosis. Research criteria for SAD include a history of depressive episodes that occur during fall-winter with at least two episodes occurring during consecutive years, symptoms remitting during spring-summer, and no seasonally linked psychosocial variable that might be responsible for changes in mood or behavior (Rosenthal et al., 1984). Thus, individuals do not need to be currently depressed, nor must they have experienced a SAD episode the previous winter in order to be diagnosed with the disorder. Although individuals must meet criteria for major depressive episode to be diagnosed with SAD, the disorder is hypothesized to be distinctly different from non-

seasonal major depression. Thus, researchers have treated SAD as a separate entity from major depressive episodes (Rosenthal et al., 1984).

According to the majority of SAD researchers (e.g., Lewy et al., 1988; Rosenthal et al., 1984; Terman, 1988), the atypical symptom profile combined with proposed etiological mechanisms (e.g., amount of light exposure, specific circadian rhythm abnormalities) are not compatible with current models of non-seasonal depression etiology. However, there are few studies directly comparing SAD to non-seasonal depression (for an exception, see Sigmon, Whitcomb, Kendrew, & Boulard, 2003). Therefore, it is difficult to draw conclusions about the relation between SAD and depression. Because there are two alternative, albeit similar, conceptualizations of SAD (i.e., DSM-IV criteria versus research criteria), some inconsistent findings in the literature may be attributed to alternative assessment and diagnostic procedures. In addition, caution is warranted in dismissing the applicability of findings within the non-seasonal depression literature. Even if alternative etiological mechanisms are distinguished for SAD relative to nonseasonal depression, there may be some processes (i.e., psychological factors) in nonseasonal depression that would relate to SAD. In order to allow greater generalizability of the results, this study will include SAD participants that meet both DSM-IV and research sets of criteria.

#### Seasonal Affective Disorder: Epidemiology

Prevalence. The prevalence of SAD has been reported to vary according to the latitude of the population being assessed (e.g., Rosen et al., 1990). In general, higher latitude means increased prevalence of SAD (e.g., Kasper et al., 1989). However, as detailed below, there are many exceptions and this relation appears to follow such a linear

relationship only in the United States (Mersch et al., 1999). As latitude increases, photoperiod (i.e., the length of time the sun is up each day) decreases throughout the fall and although begins to lengthen again, remains shortened throughout the winter months. Evidence from correlational studies suggests that shortening photoperiod represents an important factor in subsequent SAD episode onset (e.g., Potkin et al., 1993; Young et al., 1997).

Initial evidence for the relation between latitude and SAD came from prevalence surveys. Researchers sampled populations meeting criteria for SAD in the United States at varying latitudes and found the following prevalence rates: New York City, NY 4.7%, Nashua, NH 9.7%, Montgomery County, MD 6.3%, and Sarasota, FL 1.4% (Rosen et al., 1990). In an earlier study, the prevalence rate of SAD in Montgomery County, MD was estimated at 4.3% (Kasper et al., 1988). In Alaska, SAD prevalence has been estimated at 9.2% of the population (Booker & Hellekson, 1992). In general, the prevalence of SAD in the US appears to range from about 1% in the south to about 10% in the northern states. Using the more stringent DSM-III-R interview criteria, however, as opposed to self-report surveys, a large scale epidemiological study found that the overall US lifetime prevalence rate of SAD was about 1.0% (Blazer, Kessler & Swartz, 1998). This lower rate may be due to the difference in assessment techniques (i.e., self-report questionnaire versus structured diagnostic interview).

SAD prevalence has also been investigated in other countries, especially in Asia and Europe. For example, prevalence rates in Japan have been estimated at 0.86% (Ozaki et al., 1995), 4.4% in Italy (Muscettola et al., 1995), 1.9% - 2.9% in Britain (Eagles et al., 1996) and 1.5 - 2.0% in Finland (Partonen, Partinen & Lonnqvist, 1993). In separate

reviews of the international prevalence literature, researchers noted that the findings on the relation between SAD prevalence and latitude are discrepant and that the effect of latitude must be small if it exists at all (Mersch, et al., 1999; Murray et al., 2000).

Although some SAD researchers have suggested that the relation between latitude and mood may be mediated by photoperiod and weather (e.g., Young et al., 1997), others hypothesize that cultural (Partonen et al., 1993) and/or cognitive factors (e.g., more negative thoughts about weather or photoperiod; Rohan et al., 2003) may play a mediating role in the relation between latitude and SAD prevalence. Such mediating factors could be responsible for the varying strength of the relation between prevalence and latitude seen across studies.

Gender. Gender differences have been found in prevalence rates. However, there are discrepancies regarding the proportion of women versus men diagnosed with SAD. In a self-report study conducted in Maryland, 71% of individuals self-reporting SAD symptoms were women (Kasper et al., 1989). Similarly, a study of four Eastern United States locales found that 68% of sufferers were women (Rosen et al., 1990). In contrast, 11.1% of women in the population compared to 4.8% of men in Norway experienced winter depression (Hansen, Lund & Smith-Silverstein, 1998). In contrast to these population prevalence studies, one study has found a higher prevalence rate for men when diagnosed using DSM-III-R criteria for Major Depressive Disorder with seasonal pattern (Blazer et al., 1998). However, in that same study, women received significantly more diagnoses of Minor Depressive Disorder (i.e., a proposed new category of depression characterized by the requirement of fewer depression symptoms for diagnosis) with

seasonal specifier. Several review articles indicate that SAD appears to be diagnosed in more women versus men at about a 4:1 ratio (Lee et al., 1998; Lee & Chan, 1998).

In non-seasonal major depression and dysthymic disorders, women generally receive 60-80% of the diagnoses (e.g., Sprock & Yoder, 1997). The relation of gender to SAD prevalence rates is even greater than the relation between gender and non-seasonal depression prevalence rates. This similarity of increased prevalence for women suggests that SAD may resemble major depression more than bipolar disorder (Rosenthal et al., 1984). However, one researcher reports that based on random mailings of the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Bradt & Wehr, 1984) no gender differences were found. According to the authors, females may seek help more than males at SAD research clinics (Terman, 1988). However, it should be noted that the SPAQ is a screening device used to assess the severity of seasonal symptoms, and does not determine a diagnosis of SAD. Although different means of assessing SAD prevalence makes it difficult to draw conclusions about the exact proportion of the gender differences in SAD, the vast majority of studies indicate that there is a higher prevalence of SAD for women.

The SAD literature offers several possible explanations for the differences in prevalence rates across gender. Some researchers suggest that genetic factors may be related to gender differences based on heritability of symptom patterns in twin studies (e.g., Jang et al., 1997). These investigators conducted genotype analyses indicating that the genetic factors related to seasonality in women differ from those genetic factors in men related to seasonality. Others suggest that the gender difference may be due to biochemical responses related to climatic variables or to sampling bias (Lee & Chan,

1998). The DSM-IV (APA, 1994) treats SAD as a specific variant of Major Depressive Disorder (MDD), noting that depressive disorders are twice as frequent in women than men. However, despite acknowledging that women comprise a higher percentage of sufferers with SAD than in MDD, contradictorily the DSM-IV also states that it is unclear whether female gender is associated with a greater risk of developing SAD than is already attributed to developing MDD.

It is possible that the gender differences in SAD may be due to factors implicated in the gender differences seen in nonseasonal depression. For example, Response Styles Theory (Nolen-Hoeksema, 1987) was originally proposed as an explanation for higher rates of MDD in women. However, it has also been suggested that response styles may play a role in recurrent SAD episode onset (Rohan et al., 2003; Young et al., 1991). This theory and its relation to SAD will be discussed in greater detail in a later section. Although several hypotheses have been presented, there is no consensus among researchers regarding the higher proportion of women, compared to men, who suffer from SAD. Additional research investigating gender differences would add to our understanding of the disorder and may shed light on new ideas regarding etiological factors.

Seasonality as Continuum. Some degree of change in energy level, weight gain, increased sleep and lessened social contact across the seasons is commonly experienced in the general population (e.g., Terman, 1988). Epidemiological studies suggest that 92% of the population may experience seasonal changes in mood and behavior in the United States (e.g., Kasper et al., 1989). Individuals, however, differ both in the degree of severity of seasonal symptoms they report and also differ in the degree to which symptom



severity is viewed as a problem. Whereas individuals who experience clinical levels of depression dependent on the season may meet criteria for SAD, others may experience significant, albeit less severe or more transient, changes in mood and behavior with the seasons. Those individuals mildly affected by the seasons have been termed subsyndromal (S-SAD; Kasper et al., 1989) or suffering from “winter blues” (Rosenthal, 1998). Studies have estimated S-SAD prevalence between 13.5% (Terman, 1988) and 18% of the population in the U.S. (Kasper et al., 1989). In support of the seasonality continuity hypothesis, S-SAD appears to be influenced by variables such as age, gender, and latitude in a manner similar to the relation between these variables and SAD.

Within the SAD literature, S-SAD has predominantly been investigated by self-report surveys (e.g., Kasper et al., 1989; Rohan & Sigmon, 1999; Rosen et al., 1990). In addition, it has been described in terms of decreased symptom severity relative to SAD diagnostic and research criteria. Generally, S-SAD has been defined as a score of 11 or greater on the Global Symptom Severity Scale (GSS) of the SPAQ (Kasper, et al., 1989).

The GSS includes ratings of degree of seasonal change of six symptoms: sleep length, social activity, mood, weight, appetite, and energy level. Researchers in the S-SAD literature, however, have not specified the number of symptoms, type of symptoms, or level of severity of each symptom required in the pattern of symptoms in order to be considered S-SAD. Accordingly, some individuals categorized as S-SAD could have more severe psychological symptoms (i.e., mood, social activity) and others could have exclusively vegetative symptoms (i.e., changes in sleep, weight, appetite, and/or energy).

Subtypes of Seasonality. Preliminary evidence suggests that there may be a vegetative symptoms subgroup within the S-SAD population (Boulard & Sigmon, 1999).

Other researchers have noted the usefulness of studying such a population (Young, 1999a), yet this research has not been conducted in any systematic fashion. Thus far, winter anergia has mostly been described in clinical reports (e.g., Ibatoullina, Praschak-Reider, & Kasper, 1997; Mueller & Davies, 1986), in part because it does not fit any diagnostic category or published research criteria (Boulard & Sigmon, 1999).

In a pilot study, individuals with S-SAD winter anergia were compared to individuals with S-SAD with depressed mood (Boulard & Sigmon, 1999). Individuals that scored at least 12 on GSS somatic items and included significant ratings of depressed mood (total score  $\geq 8$  on mood items) as a symptom were placed in the winter dysphoria group (WD). Individuals who scored at least 12 on GSS somatic symptoms and scored less than six on the mood items (i.e., slight or no changes in mood across seasons) were categorized as the winter anergia (WA) group. As expected, depressed mood and negative cognitive styles scores (i.e., automatic thoughts and dysfunctional cognitions) were significantly higher in the WD group than the WA group. As indicated on the SPAQ, fewer participants in the WA group than those in the WD group perceived seasonal changes to be a problem. Individuals in the WA group also tended to report feeling comparatively better than the WD group when affected by winter weather (e.g., cold, cloudy, snowy). The researchers also found that the WD participants reported more changes in food preference than the WA group.

This study provides initial support for differentiating subtypes of S-SAD. However, the investigators used the SPAQ, which measures symptoms retrospectively and assesses one's *general* experience across seasons. In addition, the studies used only college students and the sample sizes were relatively small (i.e., In study 1,  $n_{WA} = 25$ ,  $n_{WD}$

= 21; In study 2,  $n_{WA} = 15$ ,  $n_{WD} = 11$ ). Also, the WA group was compared to what is essentially a nonclinical, S-SAD group. It may be important to investigate the validity of different symptom profiles (i.e., different types) of SAD and S-SAD. Subtypes of SAD and S-SAD may have different factors that maintain the symptoms, which may have implications for differential treatment methods.

There have been no comparisons between a winter anergia subtype and the SAD population. Nor have there been published studies using WA groups with prospective symptom measurement. Prospective symptom measurement is important for corroborating retrospective symptom reporting. The overall lack of research on the WA subtype within the SAD literature may be in part due to the fact that SAD has been described as a disorder of depression (Young et al., 1991) and SAD research criteria require the diagnosis of major depressive disorder (MDD). Therefore, the concentration has included mood symptoms (i.e., depressed mood, anhedonia) rather than focusing exclusively on patterns of vegetative symptoms. Vegetative symptoms are generally given less attention than depressed mood in the nonseasonal depression literature. This is likely due to the DSM-IV criteria for depressive episodes setting depressed mood and anhedonia as primary (i.e., one or both are necessary for diagnosis; APA, 1994). Furthermore other symptoms, including vegetative symptoms, are necessary for diagnosis but are not seen as defining of the disorder as are the mood symptoms. Clearly, these distinctions need to be further investigated in SAD and S-SAD populations.

There are several important reasons to examine winter anergia and its relation to SAD. The dual vulnerability hypothesis suggests that SAD may be a depressive episode that occurs *in reaction to* or *in relation to* a core set of anergic symptoms (Young et al.,

1991). Although individuals with an annual pattern of WA display primarily vegetative seasonal changes at the time of assessment, they may be at risk for developing SAD at a later point in the life span. Alternatively, studying individuals who report significant vegetative symptoms with little or no mood changes may hold clues to factors that buffer the effects of the changing seasons on the development of depression. In addition, studying this group may lead to answers about why individuals with a history of SAD do not necessarily meet diagnostic criteria for MDD every year, although they may exhibit sub-clinical levels of symptoms (e.g., Rohan et al., 2003).

Similarly, it is important to examine why some individuals with significant seasonal changes develop clinical levels of depression and others do not. Investigating both WA and SAD may lead to the identification of etiological factors that contribute to the development of initial episodes of SAD. In addition, research on WA may lead to the development of alternative treatment strategies for SAD. Individuals with WA may exhibit behavior or cognitive patterns that act as protective factors against developing SAD. If these behavioral or cognitive protective factors can be identified, it is possible that teaching these strategies to individuals with SAD may diminish their affective symptoms. On the other hand, research may find that the two populations are sufficiently unrelated and changes in seasonal symptoms can be viewed as distinct from the disordered mood and behavior of SAD. Clearly, more research is needed to investigate similarities and differences between WA and SAD.

### Current Models and Therapies

Genetic Relations. In one of the first studies investigating seasonal depression, Rosenthal and colleagues (1984) noted that the majority of participants (69%) reported a

first degree relative with a major affective disorder. Interestingly, only 17% of participants reported a family history of SAD. However, the researchers noted that reliable diagnoses and information about the total number of first degree relatives of the participants were absent (Rosenthal et al., 1984). Twin studies have also been used to investigate the role of genetic factors in the etiology of SAD (e.g., Jang et al., 1998). Several studies suggest that there may be a genetic component for seasonality (e.g., Jang et al., 1997; Madden et al., 1996). For example, in an Australian sample, genetic influences have been reported to account for 29% of the variance in the risk onset of seasonal symptoms in both genders (Madden et al., 1996). In another study investigating gender differences in seasonality, heritability of seasonal changes was estimated to be 69% for men and 45% for women (Jang et al., 1997). To account for these different heritability estimates, the researchers suggest that males may inherit genetic protective factors that make it less likely that a male would develop SAD. In addition, the heritable factors affecting seasonality in females appear to be different than the factors affecting males. For example, the genetic influences measured for SPAQ symptoms indicating heritability are greater for male monozygotic twins than for female monozygotic twins.

It is important to note that genetic studies have investigated *seasonality*, not SAD per se, using the SPAQ. As noted previously, the SPAQ assesses mood and physiological changes across the seasons but lacks the precision and depth to distinguish SAD from subclinical levels of seasonal changes (Magnusson, 1996). Therefore, findings regarding genetic contributions to SAD are limited. Although results from genetic studies generally indicate that a significant amount of variance is accounted for by familial transmission, clearly there are other influences as well. More research is needed in order to determine

exactly what genetic information is transmitted that translates into SAD or S-SAD vulnerability. In addition, other researchers have failed to find strong relations between seasonal changes in sleep, appetite and weight among biological relatives in the general population (Sasaki et al., 1998). Yet, investigators have found significant correlations between spouses for these changes (Sasaki et al., 1998). These recent findings suggest a prominent role for environmental influences (Jang et al., 1997), especially for seasonal behavioral changes in SAD episode development. Taken together, research suggests that genetic factors, the environment, and likely the interaction of the two, represent possible factors in the etiology of initial SAD episodes and need to be accounted for in a comprehensive model of the disorder.

Neurotransmitter Dysregulation. Dopamine, noradrenaline, and serotonin dysregulation have been implicated in the development of SAD episodes (e.g., Rosenthal et al., 1984, Skwerer et al., 1988; Wirz-Justice et al., 1992). In particular, serotonin is hypothesized to play a role in the general regulation of sleep, appetite, activity, and mood. Some investigators conceptualize that carbohydrate craving and the need for increased sleep observed in SAD represent compensatory mechanisms that strive for homeostasis in the serotonergic system (e.g., Thase & Howland, 1995). In part due to the observation that these behaviors are commonly disrupted in SAD, serotonin models have increasingly received more attention than other neurotransmitter models (Dalglish et al., 1996).

Serotonin depletion also has been hypothesized to play a role in the development and maintenance of non-seasonal depressive episodes. Studies investigating the possibility of abnormalities in serotonergic receptor functioning in SAD, however, have been inconclusive (e.g., Schwarz et al., 1999). For example, using a drug challenge with

a partial agonist of serotonin in individuals diagnosed with SAD, researchers found that post-synaptic serotonergic receptors appeared to function normally (Schwartz et al., 1999). However, there is some support for the role of serotonin in that drugs hypothesized to act on the serotonergic system have resulted in reports of reduced levels of SAD symptoms (e.g., Lam et al., 1995).

Serotonin selective-reuptake inhibitor (SSRI) antidepressant drugs have been studied in the treatment of SAD (e.g., Hesselmann et al., 1999; Lam et al., 1995; Ruhrmann et al., 1998) as well as non-seasonal depression (e.g., Beasley, Nilsson, Koke, & Gonzalez, 2000). One SAD study, using a multi-center, placebo-controlled design, found a 59% clinical response rate (had a 50% or greater reduction in scores on SIGH-SAD or BDI) for fluoxetine compared to 34% for pill placebo (Lam et al., 1995). However, the researchers reached these conclusions despite the lack of significant difference between fluoxetine and placebo groups on the SIGH-SAD and BDI at the end of the study. Another SAD study used a randomized, parallel design to investigate the effects of fluoxetine versus light therapy (Ruhrmann et al., 1998). The drug group received one week of placebo, followed by 5 weeks of fluoxetine treatment (i.e., 20 mg per day). The light therapy group received one week of dim “placebo light” followed by 5 weeks of light therapy (i.e., 3000 lux for 2 hours per day). Both treatments improved depression scores compared to the baseline; light improved overall depression scores faster and medication improved atypical (e.g., social withdrawal, weight gain, increased appetite, carbohydrate craving, hypersomnia, fatigue, late day energy or mood slump) depression symptoms faster (Ruhrmann et al., 1998). No wait-list or office visit support controls were included in this study, so it is difficult to conclude whether either treatment

contained interventions that improved depression or if passage of time played a major role in symptom reduction. Although treatment studies utilizing serotonergic drugs lend support to theories of neurotransmitter dysregulation in SAD, it is important to recognize that SSRIs have effects on other neurotransmitters as well. In general, antidepressant therapy is considered less effective in the treatment of SAD and light therapy is still considered to be the treatment of choice (Dalglish et al., 1996; Lee & Chan, 1999; Terman & Terman, 1999).

Light-dependency Models. There are several proposed mechanisms that specifically relate to the action of light on symptoms of SAD. However, the majority of scientific support for the biological models of SAD comes from correlational findings of decreased photoperiod (especially at higher latitudes) during winter (i.e., when SAD symptoms occur) and that light therapy represents an effective treatment for SAD symptoms. Some of the biological models of SAD that account for light therapy efficacy are detailed below. None of the theories, as of yet, details fully articulated mechanisms of how sub-threshold levels of light would translate to depression and atypical vegetative symptoms or why only a small portion of the population is affected by clinically significant seasonal changes. Nor do light-dependency models account for the variability of experience of SAD episodes from year to year for many sufferers (i.e., why individuals with a history of SAD do not meet criteria for depressive episode every winter). Furthermore, the current biological models cannot fully account for the cultural and gender differences found in prevalence rates.

Photoperiod. According to the photoperiod model of SAD, symptoms of SAD are similar to photoperiod-dependent seasonal physiological changes seen in animals (e.g.,



Lewy et al., 1988). Thus, researchers have investigated the decreasing photoperiod as an etiological factor in the development of SAD episodes (i.e., as the length of daylight shortens, depressive symptoms begin their onset). Light therapy is generally administered in the morning or evening, rather than mid-day, and bright light exposure is hypothesized to extend the number of hours of exposure to daylight (e.g., Lewy et al., 1988). The photoperiod model emphasizes that the length of daylight represents a cue for the emergence of certain abnormal physiological changes (e.g., fatigue, hypersomnia). In essence, this model posits that SAD is set in motion by an insufficient amount of time that an individual is exposed to natural light (i.e., inadequate photoperiod).

However, light therapy does not improve all of the symptoms associated with SAD for many individuals (Terman et al., 1996). In addition, the photoperiod model does not address the mechanism by which decreased hours of daylight would cause depressive symptoms. Even in short photoperiods (e.g., Alaska, Iceland), the prevalence rate of SAD is less than 10% of the general population (Booker & Hellekson, 1992; Magnusson & Stefansson, 1993). Although the entire population is exposed to shortened periods of daylight during the winter season, only a small percentage reports being affected. In addition, researchers have found that the amount of time spent outdoors in natural sunlight is not predictive of onset of recurrent SAD episodes (Graw et al., 1999). In this particular study, women with SAD did not spend less time out of doors in the winter than Controls. In general, the research findings for the photoperiod model are contradictory, small samples are utilized, and results are not seen as conclusive in confirming the role of photoperiod in SAD (Lee et al., 1998). Furthermore, investigators have also found that using light therapy to alter the length of the photoperiod is not necessary (i.e., midday

bright light exposure) for the therapeutic effects of light therapy (e.g., see Terman et al., 1989). The empirical findings on light exposure suggest that sensitivity to light rather than length of exposure may be the more important factor.

Circadian phase-shift. Biological rhythms in cycles of about 24 hours (i.e., circadian rhythms) represent internally regulated cycles of bodily function. These rhythms include cycles of wake and sleep, body temperature, and hormonal and neurochemical levels among others. In the phase-shift model, individuals with SAD display abnormally phase-advanced or phase-delayed biological cycles during the winter (Lewy et al., 1988). In other words, daily patterns of wake/sleep, hormonal secretions, and others, do not match the daylight (i.e., photoperiod) and daily routine of SAD individuals.

According to this model, light therapy serves to “reset” the biological clock that controls the biological rhythms, therefore, individuals whose circadian rhythms are phase-delayed should respond best to bright light administered in the morning (Lewy et al., 1988). However, comparisons of administration times of bright light with individuals with SAD have been inconclusive (Lee et al., 1998; Sato, 1997; Terman, et al., 1989). There is some evidence that morning administration of light therapy is more effective than evening administrations (e.g., Lewy et al., 1987). However, other researchers have found that evening light therapy is also efficacious (e.g., Terman et al., 1989; Wehr et al., 1986), and suggested that more research is needed regarding this theory. Recently, researchers using larger samples and measuring circadian phase from onset of melatonin secretion, have found morning light to advance circadian phase and to be more efficacious in remitting SAD symptoms (Terman et al., 2001). Researchers, however,

have still not been able to identify why some individuals might be more susceptible to developing phase-delays than other individuals. Furthermore, the mechanism by which phase-delays of melatonin levels produce SAD symptomatology have not been specified.

Photon-counting. The photon-counting model posits that the quantity of light energy sufficient for normal mood maintenance is not available during the shorter photoperiod (Rosenthal & Wehr, 1992). In examining the validity of the hypothesis, dose-response relationships of light therapy have been investigated. According to this hypothesis, a threshold amount of light would be needed to produce antidepressant effects (Rosenthal & Wehr, 1992). In general, a dose-response relationship can be seen with bright light therapy, although contradictory results have been observed across studies when researchers have varied intensity and duration of bright light (e.g., Lee & Chan, 1999; Lee et al., 1998; Tam, Lam, & Levitt, 1995). In addition, initial studies suggest that individuals with SAD have differences in retinal sensitivity to light relative to Controls in the winter (Terman & Terman, 1999).

However, researchers who examined the relation between the amount of natural light (i.e., daily hours of sunshine and total daily radiation) and mood have failed to find an effect over seven years of collecting data on recurrent SAD symptoms' onset (e.g., Young et al., 1997). Because support for this theory comes primarily from findings with light therapy, the shortcomings in the light therapy literature detailed below also apply to the photon counting model. The neural mechanism underlying this theory has not been proposed, and more research is needed to establish support for this model (Lee et al., 1998). Furthermore, investigators need to be able to explain why some individuals need more light than other individuals.

Light therapy. Light therapy generally consists of exposure to a bright high intensity light source of 2,000 - 10,000 lux (i.e., units of illumination). In comparison, the average household light bulb provides approximately 500 lux. Typically, an individual sits in front of a light box for about 30 minutes to 2 hours daily following the subsequent onset of SAD symptoms. For individuals who respond to light therapy, symptom remission typically begins within a few days of commencing treatment and relapse generally occurs within a few days after terminating exposure to bright light (Lee et al., 1998).

Due to methodological differences and small sample sizes, bright light exposure treatment outcome research has led to discrepant findings (Lee et al., 1998; Terman et al., 1998). However, bright light therapy is currently considered the “gold standard” for treatment of SAD. Several doses of light therapy, measured in light intensity, length of time, as well as time of day (e.g., morning versus evening, or both) have been studied (e.g., Rosenthal et al, 1984; Terman et al., 1998; Terman et al., 2001). According to one meta-analysis, the best protocol for light therapy is an intensity of greater than 2500 lux for two hours per day (Tam, Lam, & Levitt, 1995). This protocol produced response rates of 36 - 75% SAD symptom reduction across the reviewed studies. The same review, however, noted that protocols with greater light intensity (e.g., 10,000 lux) for shorter periods of time (e.g., 30 minutes per day) have also been investigated, with remission rates similar to the less intense light treatments.

Although light therapy is currently the most effective treatment for SAD, even the best light dose protocols do not alleviate all SAD symptoms. Furthermore, there are some side effects of light therapy such as eye strain, headaches, feeling wired, nausea, and

dizziness as well as induction of manic phases (Levitt et al., 1993; Terman & Terman, 1999). Individuals with eye disorders are cautioned against using light therapy, and the long-term effects of light therapy are not known (Tam, Lam & Levitt, 1995). It is important to investigate other factors (e.g., cognitive factors) in search of alternative treatment strategies for use with individuals who cannot use or benefit from light therapy. Furthermore, it is important to continue to investigate other etiological factors that may account for incomplete remission of symptoms with light therapy and may contribute to a more comprehensive model of SAD.

One of the criticisms of light therapy outcome research has been the lack of a valid placebo for comparison (e.g., Brown, 1990). Due to the nature of the treatment, the majority of patients can obviously determine if they are being exposed to light. Meta-analyses also support findings that even dim light therapy provides significant symptom remediation (Lee et al., 1998; Tam et al., 1995; Terman, 1988; Terman et al., 1989). In fact, some researchers have argued that light therapy is simply a placebo (e.g., Brown, 1990). Others have found that patient response to light therapy may be related to the observed behaviors of both patients and interviews during assessment, suggesting interpersonal factors may play a role in treatment response (Geertz, Bouhuys, Meesters, & Jansen, 1995). However, a dose response based on light intensity has been demonstrated by combining findings across several investigations in a meta-analysis (Lee & Chan, 1999). Research has shown that variance in remission rates may be dependent on the timing of light administration and strength of light used (e.g., Dangleish et al., 1996). The literature analyzed suggested that light therapy dose was positively correlated with degree of improvement in typical depressive symptoms; however, this relation was not

found for the atypical symptoms of SAD (Lee & Chan, 1999). Overall, the findings indicate that greater intensities of light treatment are correlated with more improvement on measures of depressive symptomatology, suggesting that light therapy is not just a placebo (Terman, 1988; Terman et al., 1989).

The outcome literature for light therapy is also hampered by studies using small sample sizes (Terman et al., 1989). Small sample sizes are likely to account for part of the variability of efficacy in light therapy findings. Small sample sizes also decrease the power of a study to find differences in efficacy across dose conditions. Power analyses do suggest that most light therapy studies employ sample sizes too small to find statistical differences between light therapy protocols, if they exist (Terman et al., 1989). Future research investigating the promising treatment of light therapy needs to include adequate sample sizes relative to the expected effect size so that findings may be interpreted with confidence, and so that they will be useful to researchers planning future studies.

In addition to the problem of small samples, it has been suggested that SAD research literature contains samples composed of heterogeneous symptom profiles (Boulard & Sigmon, 1998). Heterogeneity of symptom profiles within samples may hinder the ability to find statistical significance in some studies. Similarly, researchers have suggested that diagnostic criteria are not reliable enough and that individuals who do not demonstrate a treatment response to light therapy should not be diagnosed with SAD (Terman et al., 1996). Although researchers did find that light therapy responders were characterized by suffering from more of the atypical symptoms and non-responders reported more of the melancholic symptoms, all measured symptoms were observed in each group (Terman et al., 1996). The symptom clusters in responders versus non-

responders were not distinct or unique. In addition, more evidence for possible etiological mechanisms for SAD related to insufficient light are necessary before drastically altering diagnostic criteria. Although some research suggests that less than half of individuals with SAD respond to light therapy, it would seem more prudent to label light therapy responders as a sub-set of SAD rather than redefine the disorder based on treatment response.

Light therapy, however, is not a cure for SAD; sufferers must treat their symptoms throughout every winter. Fortunately, light therapy has a good treatment response rate. The average response rate in research is about 50% (Hodges & Marks, 1998) ranging from less than 30% to about 70% across studies of varying doses (e.g., Lee, 1995; Terman et al., 1996). However, a significant proportion of individuals with SAD (i.e., more than 30%) do not meet criteria for treatment response following light therapy. Moreover, individuals using light therapy who do respond continue to experience significantly greater symptomatology during light therapy (i.e., as assessed by the Hamilton Depression Rating Scale, Seasonal Affective Disorder version) than they experience during the summer months (Postolache et al., 1998). The majority of those who do respond to light therapy continue to experience significant symptoms and physiological differences (e.g., Lee et al., 2001; Michalon, Eskes & Mate-Kole, 1997). Individuals who reported increased numbers of atypical symptoms on the Hamilton Depression Scale, however, tended to predict improved response to light therapy (Oren et al., 1992). Although light therapy has received empirical support, the evidence also suggests that there is certainly room for improvement.

In summary, none of the etiological models involving biological explanations for SAD has gained overwhelmingly conclusive support (Lee et al., 1998). Most of the biological models are based on the observation that SAD symptoms tend to respond to bright light therapy, and work backward to explain why light alleviates symptoms (Lee et al., 1998). However, recent research has suggested that symptoms of non-seasonal depression also respond to light therapy (Kripke, 1998) and light therapy has also reduced symptoms associated with bulimia nervosa and premenstrual dysphoria (Lam, 1998). Findings like these reiterate the premise that just because a treatment works, it does not mean that it works for the reasons that are hypothesized.

Even though the photoperiod shortens and lengthens predictably every year, individual SAD sufferers do not necessarily experience a depressive episode every winter (e.g., Rohan et al., 2003; Rosenthal et al., 1984). In addition, although children have been diagnosed with the disorder (e.g., Giedd, Swedo, Lowe, & Rosenthal, 1998; Glod, et al., 1997; Glod & Blaisden, 1999; Sweedo, et al., 1995), many individuals with SAD appear to develop symptoms during adolescence through the early 20's (Rosenthal et al., 1984). The light-based biological models cannot account for why would an individual suddenly react differently to light. Research is sorely needed to further investigate the timing and onset of initial SAD episode development. Furthermore, not all SAD sufferers experience symptom remission with bright light therapy. Of those that do experience a response to light treatment, not all symptoms may remit (Terman et al., 1996). It would seem that there are other factors that require investigation regarding the etiology and development of initial SAD episodes as well as the factors involved in subsequent SAD episodes. Investigation into the role of cognitive processes may provide some answers.



In addition, ignoring cognitive factors in the investigation and development of models of SAD disregards the established findings regarding the process of depression (Sigmon et al., 2003).

### Psychological Models of SAD

In SAD, negative cognitions or thinking patterns may be particularly relevant to the cyclical and recurrent nature of the disorder. The changing of the seasons, or shorter periods of daylight, in the autumn months could be seen as a cue or prime for negative cognitions. These cues may activate depressive schemas and induce the systematic use of cognitive errors. The exact role that cognitions might play in the etiology, maintenance, or recurrence of the disorder is not known (O'Brien et al., 1993), but several studies found that negative cognitions are, at the very least, correlated with the disorder (e.g., Bouhuys et al., 1994; Rohan et al., 2003).

Cognitive behavioral therapy (CBT) has already been advocated for treating SAD (Jang et al., 1998; Rosenthal, 1993), however, no data have been published on treatment outcomes for CBT for SAD (Tam et al., 1995). SAD researchers have acknowledged that cognitions are probably not the primary etiological factor in the development of the first SAD episode but do support seasonality models integrating biological and psychological mechanisms (Lacoste & Wirz-Justice, 1989). Two models integrating biological and psychological findings have been proposed, the Diathesis-Stress Model of SAD (Sigmon, Rohan & Boulard, 2001) and the Dual Vulnerability Hypothesis (Young et al., 1991). These similar theories will be covered in detail in a later section. First, however, it may be useful to explore cognitive theories of non-seasonal depression (e.g., Beck, 1979) and how they may relate to SAD.

Beck's Cognitive Model of Depression. The negative cognitions that have been explored in research on psychological factors of SAD are congruent with Beck's Cognitive Theory of Depression (Beck et al., 1979). His theory postulates three cognitive components important to the construct of depression: cognitive errors, negative schemas, and the cognitive triad (Beck et al., 1979). The components of Beck's Cognitive Theory can be synthesized to summarize his proposed mechanism. First, the negative schemas are activated (e.g., by low, sad mood), which influences cognitive processing of environmental perceptions in a distorted, negative way (i.e., cognitive errors). The distortion of the perceptions of the environment leads to production of negative cognitions regarding topics within the cognitive triad, which leads to clinical levels of depression (Kwon & Oei, 1994). Each of these concepts will be explored in more detail and data from SAD research will be examined that is congruent with Beck's model.

Depressive schema may be viewed as pervasive cognitive patterns or structures that color the way information is attended to and processed within an individual. They have also been referred to as "core beliefs." A depressive schema can also be thought of as a deeper level of cognition, "set[s] of rigid and unrealistic beliefs and expectations in life (Kwon & Oei, 1994, p.335)." Alternatively, they can be conceptualized as dysfunctional attitudes that refer to the cognitive triad.

According to Beck (1979), the cognitive triad consists of three negative thought patterns that an individual endorses regarding the self, the future and the interpretation of experiences in the environment (i.e., the world around). Additional depressive symptomatology is then manifested as a consequence of the negative thoughts about material within these three domains. These negative thoughts may be based in reality

(e.g., I don't have many friends) or they may take the form of cognitive distortions (e.g., Nobody will ever like me). Pathological processes would include negative thoughts in the form of cognitive distortions.

Cognitive errors are ways of thinking that serve to maintain negative cognitions despite contradictory information. A few examples of this type of thinking are: All or none thinking (e.g., I am a complete failure or I am perfect, there is no in-between), using "shoulds" to evaluate self or event (e.g., I should have done better), emotional reasoning (e.g., I feel like a failure, so I must be a failure), and overgeneralizing (e.g., I'll never do it right because I did it wrong once). Cognitive distortions may be situation or domain specific, in that a person may use this type of thinking to evaluate one area of life, but not others. Similarly, negative cognitions are hypothesized to be stable within an individual but only accessible to the individual during periods of depressed mood (Persons & Miranda, 1992). This mood-state hypothesis proposes a solution to the problem in research in which negative cognitions are not consistently found in those vulnerable to developing depression (Persons & Miranda, 1992). According to this model, an individual must experience depressive symptoms (e.g., sad mood) before the negative cognitions can be accessed.

Cognitive Findings in the SAD Literature. Many symptoms (e.g., depressed mood, anhedonia, fatigue, and negative cognitions) are shared between SAD and non-seasonal depression (e.g., Rohan et al., 2003). Similarly, psychological factors are hypothesized to play an important role in the development and maintenance of non-seasonal depression (Beck, 1979), this has not yet been thoroughly investigated in SAD. Negative patterns of cognitions similar to those found in non-seasonal depression can be

considered as possible mediators or moderators of SAD. Researchers have proposed that deep cognitions (i.e., dysfunctional attitudes) may function as cognitive moderators between negative events and depression (Kwon & Oei, 1994). In addition, they suggest that automatic thoughts (i.e., surface level cognitions) may function as cognitive mediators. Investigators describe a moderator as a variable that affects the relation between the independent and dependent variable by interacting with the independent variable (Kwon & Oei, 1994). For example, when a negative life event occurs, dysfunctional cognitions moderate the effect of the negative event through cognitive processing (i.e., the perception of the event).

Preliminary correlational support for faulty cognitive processing has been found in studies of SAD and personality dimensions (e.g., Jang et al., 1998). In a study of monozygotic and dizygotic twins, researchers using the SPAQ to categorize individuals with SAD found a positive correlation between seasonality and cognitive dysregulation as measured by a personality pathology scale (Jang et al., 1998). Those same researchers found a positive correlation between seasonality and high trait anxiety. Researchers hypothesize that high trait anxiety may increase rumination about seasonal changes by sensitizing individuals to the seasonal changes (Jang et al., 1998). However, these initial studies are correlational in nature and do not use clinical samples, so their contributions regarding deficiencies in cognitive processing are limited.

Distorted perceptions of the environment are thought to play a role in the development of depressive episodes in SAD (Levitan, Rector & Bagby, 1997). In comparing individuals with seasonal depression and individuals with nonseasonal depression, researchers found that when depressed, SAD patients report negative

attributions at levels similar to nonseasonal depressed patients (Levitan, et al., 1997). Nonetheless, the researchers concluded that dysfunctional cognitions may play a lesser role in SAD than in nonseasonal depression because attributional style did not predict response to light therapy. However, only 50% of the SAD participants responded to the light therapy, providing a small sample size for predictive analyses. In addition, it should be noted that light therapy does not target negative attributions or cognitions and it is not clear what role light exposure plays in the etiology or maintenance of SAD. It is not relevant to judge the importance of the influence of negative attributions based on treatment outcomes (e.g., light therapy), especially when the etiology is unknown. In contrast to the researchers' discussion of the results, these findings lend support for the role of negative attributions in the maintenance of recurrent SAD symptoms or episodes.

Dysfunctional cognitions regarding light and dark (i.e., schemas) appear to be related to SAD episodes (Bouhuys et al., 1994). To investigate the role that symbolic light plays in the development of SAD, researchers gave pictures of line-drawn, cartoon faces of varying expressions embedded in a darkly shaded or white background to non-depressed individuals with a history of SAD episodes. Participants rated on a five point scale their perception of the degree of emotion (e.g., elation, sadness, invitation, rejection, activation, or sleepiness) shown by the faces. The greater the difference in invitation perceived by the participants due to light or dark background, the earlier the participants relapsed in the fall (Bouhuys et al., 1994). Using this technique of measuring symbolic light, Bouhuys and colleagues (1994) concluded that SAD patients have a higher sensitivity to negative cognitions about decreased light conditions than controls. It can be argued that this technique measures negative cognitions about light in relation to social

situations, as facial expressions were utilized as stimuli. Critics have pointed out that the results of the study on symbolic light could also be viewed as a consequence of recurrent episodes rather than as a cause of initial SAD episode onset (Sigmon et al., 2001).

Based in part on Bouhuys and colleagues' findings (1994), researchers have proposed a light-specific cognitive component in SAD (Rohan et al., 2003; Rohan, Sigmon, Dorhofer, & Boulard, 2001). These researchers hypothesize that individuals vulnerable to developing SAD episodes possess light-relevant schema. The light schema are believed to contain aspects of positive beliefs related to perceived exposure to environments with greater light intensity and negative beliefs regarding perceived exposure to low light environments. In a test of this hypothesis, Rohan and colleagues (2003) found that women with a history of SAD reported exacerbated depressed mood following presentation of low light stimuli, compared to bright and ambiguous light condition stimuli. Using color slides of scenery with varying levels of natural lighting as the stimuli, these results were found regardless of whether the SAD participants were tested in the fall, winter, or summer. In addition, Rohan and colleagues (2003) found that women with a history of SAD reported improved mood following presentation of bright light stimuli when compared to controls. These findings supported preliminary work with S-SAD individuals that found similar mood effects for light stimuli of varying intensity (Rohan et al., 2001).

One possible problem with this research is that the type of stimuli utilized may present demand characteristics. The stimuli used in both studies involved the presentation of artistic landscape photography slides with varying levels of light. For participants volunteering to participate in a study on SAD, there may be subtle cues for

the participants that there should be mood shifts in the hypothesized directions (i.e., dark equals sad and light equals happy). This may cause conscious alterations in responses leading to confounds of social desirability in responses. In addition, the recent increased exposure of SAD and light therapy in the local media would suggest that many participants may have been aware of the hypothesized link between light and seasonal symptoms. Investigation cognitive processes in a way that would preclude demand characteristics in responses would be best.

A recent study investigated the role of negative cognitions in recurrent SAD episodes and found that women with a history of SAD reported more automatic negative thoughts in fall and winter (Rohan et al., 2003) compared to controls. The researchers measured negative cognitions using the Automatic Thoughts Questionnaire across fall, winter, and summer assessment sessions. Women who met criteria for SAD in the previous winter reported more negative thoughts than women who had never been depressed. At the summer assessment, however, the two groups did not differ in frequency of negative thoughts (Rohan et al., 2003). These findings are in agreement with the mood-state hypothesis that posits negative cognitions related to depressive episodes are not accessible when the person is not experiencing depressed mood (Persons & Miranda, 1992). Although Rohan and colleagues (2003) did not analyze for depressed mood per se, there were no differences between groups in depressive symptoms at the summer assessment, whereas women with a history of SAD did report more overall depressive symptoms than controls at the fall and winter assessments.

Similar findings regarding the self-report of negative cognitions in SAD have been observed in other studies (e.g., Hodges & Marks, 1998). Comparing SAD,

nonseasonal depressed, and control groups of small sample sizes during the winter, the researchers found that individuals in the SAD and nonseasonal depressed groups reported more negative thoughts and attitudes than controls, but did not differ from each other (Hodges & Marks, 1998). The small sample size, however, may have hindered finding differences between the two groups. In addition, the instruments used were measures of general negative thoughts and cognitions. It may be that in addition to the general depressotypic thoughts and attitudes, SAD individuals also have negative thoughts and attitudes that are more specific to the changing seasons.

Much of the published research to date on negative-content cognitions has been cross-sectional in design (e.g., Levitan et al., 1997; Hodges & Marks, 1998). Although Rohan and colleagues' (2003) study represented the first longitudinal design in measuring cognitive variables across the seasons, this study still cannot answer the question of how negative thought patterns might develop, how they change across the seasons, and what factors might be related to changes in thought patterns. In addition, Rohan and colleagues (2003) only investigated the cognitive patterns in women with SAD. Although women suffer disproportionately from the disorder, studying a disorder in only one gender can lead to general conclusions with gender bias. It is important to investigate whether these results are consistent across gender or specific to women with SAD. It is possible that Rohan and colleagues' (2003) findings regarding frequency of negative cognitions are related to an interaction between SAD and gender.

Other cognitive impairments in SAD have been noted by researchers as well (e.g., Bouhuys et al., 1994). Depressed patients with SAD have been found to exhibit impairment on computerized cognitive tasks, such as spatial memory (e.g., forced choice



recognition of location of the previous position of a square) and learning tasks (i.e., paired associates learning; Michalon, Eskes & Mate-Kole, 1996; O'Brien, Sahakian & Checkley, 1993). The researchers found that women who were currently depressed and diagnosed with SAD exhibited deficits in spatial memory and learning, as well as increased latency to respond. The results were interpreted as representing slowed information processing rather than sensory or motor function dampening. Upon remission of their SAD episode, these women continued to exhibit increased latency to respond to spatial memory tests (O'Brien, Sahakian, & Checkley, 1993). However, one of the studies used a small sample consisting only of female individuals with SAD ( $n = 11$ ) and controls ( $n = 10$ ; O'Brien, Sahakian, & Checkley, 1993).

Other researchers have found increases in cognitive failures (i.e., everyday errors, or the ability to monitor and modify goal directed intentions), and deficits in visual memory and visual reconstruction (i.e., drawing and remembering abstract designs and faces) related to SAD (Michalon, Eskes, & Mate-Kole, 1996). Utilizing a sample of 30 individuals with SAD and 29 age- and education-matched controls, researchers investigated cognitive functioning prior to and following 2 weeks of active and placebo light therapy with repeated testing across seasons (Michalon et al., 1996). As expected, cognitive failures in the SAD participants were not affected by light therapy. However, only about 60% (9 of 15) of participants responded to light therapy, leaving the researchers with a small sample and low statistical power (Michalon, et al., 1996). The researchers also found that visual memory deficits were also apparent in the summer in the remitted SAD group, when other cognitive functions were at normal levels. These findings suggest that cognitive deficits related to SAD may be present even when an

individual is not experiencing SAD symptoms and that SAD may be related to long-term deficits (Michalon, Eskes & Mate-Kole, 1996). Both of these studies, however, fail to elaborate on the possible relations between cognitive deficits and SAD. Does SAD cause cognitive deficits, and do the cognitive deficits play a role in the development, maintenance or experience of the disorder? Although these studies documenting cognitive deficits in individuals with SAD are interesting, more research is needed to elucidate how these findings are related to the disorder.

Cognitive Interference and the Stroop Task. The Stroop test was designed as a method for examining cognitive interference phenomenon (Stroop, 1935). The original paper presented three separate experiments (Stroop, 1935). The first test consisted of stimulus words and symbols written in incompatible colors of ink and the task was to read the word aloud. The control condition had color words written in black ink only. Participants were significantly faster at reading black ink words. The second test was identical, but task was to identify the color of the ink and the control condition consisted of color squares rather than words. Participants were significantly faster at naming the colors. The third experiment examined practice effects over 8 days. Stroop (1935) found a significant reduction in interference from incompatible words with practice. However, because Stroop did not collect daily color square naming baselines each day, it is not clear if the results may have been due to a general practice effect (MacLeod, 1991).

Since the original paper, more than 700 articles have addressed various versions of the Stroop task with many different populations (see MacLeod, 1991 for a review of recent studies). One alternate version, the emotional Stroop task, has been designed to measure the latency to respond to words with affective content as an indication of

attentional bias (Gotlib & McCann, 1984; Williams, Mathews, & MacLeod, 1996).

Cognitive models assume that attentional bias plays a role in the development and maintenance of depression, rather than being a result of a depressive disorder (e.g., Beck, 1979). Furthermore, Beck (1979) hypothesized that the organizational structures (i.e., schemas) within the brain designed for encoding, storage, and retrieval functions are negatively biased in depressed individuals. In general, it is to be expected that individuals who are more distracted by the word meaning would evidence a longer latency to name the color of the ink. It would also be expected that individuals with depression would be primed for negative content and therefore more distracted by negative content words. Thus, individuals with depression would be expected to produce longer latencies to respond on the color naming task.

The modified, emotional Stroop task has been used in non-seasonal depression literature to assess cognitive interference in response to depressotypic and neutral words (e.g., Bradley, Mogg, Millar, & White, 1995; Gotlib & McCann, 1984; McNeil et al., 1999). For example, one study investigated attentional bias related to depression and anxiety relevant Stroop stimuli across several anxiety disorders and non-seasonal depression (McNeil et al., 1999). The researchers created a Depression Stroop task using 20 depression-relevant words with 20 matched neutral words and each word was presented randomly five times. Similar anxiety and standard color-word Stroop tasks were also devised. The non-seasonal depressed participants ( $n = 18$ ) showed a trend toward increased latency to respond to depressotypic versus neutral words, however, the small sample size may have hindered tests of significance.

Other studies have also used the emotional Stroop task to investigate attentional bias in individuals with some symptoms of depression and with nonseasonal depression. One study found that patients with Parkinson's Disease who had more depression symptoms showed deficits on an Emotional (i.e., depression) Stroop task as compared to patients with Parkinson's disease who were less depressed (Serra-Mestres & Ring, 1999). Another study demonstrated that individuals diagnosed with depression were significantly slower at naming colors of negative words compared to neutral or negative words in another study using the modified Stroop task (Gotlib & McCann, 1984). These researchers used 15 mildly depressed and 15 low depression symptom participants to look at response time in naming the color of 50 neutral, 50 negative, and 50 positive words. They followed up with a second study that used mood induction in non-depressed participants by having them read depressing, neutral or elated self-statements. The researchers found that there were no differences on Stroop performance, suggesting that interference is less related to transient mood states than to stable cognitive processes related to depression (Gotlib & McCann, 1984).

In an effort to replicate these results and clarify stable versus transient mood differences, researchers recruited 42 women with no depression, mild depression, moderate depression, or severe depression as assessed by the BDI short form (Williams & Nulty, 1986). Depression scores were assessed one year prior to the experiment and at the time of the emotional Stroop task administration. The researchers found that the interference effect was strongest for participants who had been depressed at both assessments and the interference effect was absent for participants who were not depressed at either assessment. In addition, they replicated Gotlib and McCann's (1984)

findings of increased interference in a chronically mildly depressed group (i.e., mildly depressed at both assessments). Furthermore, they found that degree of interference on the emotional Stroop task for those participants whose depression remitted between first and second assessments appeared to be more a function of previous depression severity than of current depression level. These findings suggest a relation between lifetime severity of depression and degree of attentional bias. Although some reviews suggest inconsistencies in the literature using the modified Stroop in depression (i.e., Williams et al., 1996), there have been concordant findings using the emotional Stroop methodology consistent with the procedure proposed in this study (i.e., blocked presentation of empirically chosen stimuli and greater than 20 participants; e.g., Gotlib & McCann, 1984; Williams & Nulty, 1986 ).

Several researchers (e.g., Williams et al., 1996) agree with Cohen et al.'s connectionist theory and parallel processing explanation of the interference effects of the Stroop task. A general overview of the model can illustrate how emotionally laden words can interfere with the speed of response to a color-naming task. In Cohen and colleagues' model (1990), attention is viewed as a modulator that acts as an additional source of input within a processing pathway. The researchers hypothesize that two processing pathways are involved: one pathway for color naming, one for word reading. It has been suggested that cognitive units for emotionally-valenced and mood-congruent words within these processing pathways may require more activation for the unit to respond (Williams et al., 1996). According to this hypothesis, input units for relevant emotion words may have higher resting levels of activation or that input units associated with threat or loss may have additional neuromodulatory controls governing their

responsivity (Williams et al., 1996). In addition, certain words may gain neuromodulatory control after being associated with threat or loss. This complex model offers an explanation for why increased latency to respond occurs with depressotypic stimuli as well as a mechanism for how individuals may develop cognitive interference for some words. This model can help explain how words that might be neutral to the controls (i.e., autumn, season) can create attentional bias in individuals with SAD.

Two studies have been published utilizing the Stroop task in evaluating cognitive processing in SAD. One study utilized the original Stroop task, designed to assess attentional interference (Drake et al., 1996). SAD participants were recruited and assessed during the winter and were assessed again during the summer. Participants read names of colors printed in various colors as fast as possible (Drake et al., 1996). The investigators did not find significant differences in response times between individuals in the SAD and control conditions. They did find, however, that both groups had shorter response latencies in the summer than in the winter, suggesting a seasonal effect regardless of an individual's level of seasonality. Although mean differences were found in the expected direction (i.e., SAD participants had longer response latencies than controls at both sessions), this study used a small sample size (i.e., 10 SAD and 9 controls) that likely limited the study's power.

A recent preliminary study used a modified, emotional Stroop task to evaluate cognitive interference in SAD (Spinks & Dalgleish, 2001). Investigators recruited only SAD participants and presented the task during the winter first and then in the summer. The task consisted of naming the type-face color of zeros (i.e., 00000), neutral words (e.g., statue), negative words (e.g., immature), and season related words (e.g., seasonal).

The stimulus words, matched for frequency and length, were presented on cards each containing 50 words. The time it took to name the colors of all the words on a card was recorded. Overall, the findings indicated that individuals with SAD were slower to name colors of all words in the winter than in the summer. In addition, individuals with SAD were slower to name the colors of negative words and seasonal words than for the zeros or neutral words regardless of season of assessment. The researchers also found that the interference for negative, but not seasonal, material (i.e., lonely) in the winter predicted depression scores and state anxiety in the summer (Spinks & Dalgleish, 2001).

However, there were some problems with this study. For example, the methodology of hand timing participants allows for the possibility of experimenter error and bias (i.e., the experimenters could not be blind to response set). In addition, the methodology of using large blocks of words for each category has some problems. The seasonal words and negative words could have more similarity than neutral words, because the neutral words were not of a given category. This difference in the degree of relatedness between words in a group may cause increased cognitive interference and longer response latencies (MacLeod, 1991). The finding that the participants took longer to name the colors of the zeros than to name the neutral words supports this hypothesis (Spinks & Dalgleish, 2001).

In addition, the Spinks and Dalgleish (2001) study considered the participants with SAD as their own control in this longitudinal design. However, other researchers have found that previous experience of depression affects subsequent performance on the Stroop regardless of current state (Williams & Nulty, 1996). Furthermore, the lack of a non-depressed control group prohibited comparison of response latencies across season

and word types to further investigate seasonality as a continuum. It would also be interesting to investigate how Stroop response latency measured in the summer predicts severity of SAD the following winter, instead of the symptoms during the following summer. Furthermore, predicting winter depression scores may be more useful in terms of understanding factors that may exacerbate or moderate recurrent SAD episodes. The addition of a counterbalanced group would have allowed for these comparisons and the evaluation of order effects. The proposed study does include a non-depressed control group, as well as a S-SAD group for comparison. In addition, the design of the study includes administering the Stroop task in the summer with follow up during the winter so that it will be possible to examine the predictive value of Stroop task results for onset of depression.

Response Styles Theory. Investigators have suggested that Nolen-Hoeksema's (1987) Response Styles theory of depression may have implications for SAD (Rohan et al., 2003; Young, 1999a; Young, 1999b). According to Response Styles theory, rumination, (i.e., focusing on the roots and consequences of depressive symptoms) can serve to exacerbate and lengthen periods of depressed mood (e.g., Nolen-Hoeksema & Morrow, 1993). Rumination has been shown to intensify depressed mood in both clinically depressed (Nolen-Hoeksema & Morrow, 1993) and analogue populations (i.e., undergraduate students in which depressed mood was induced; Nolen-Hoeksema & Morrow, 1993). Although Response Styles theory has been studied primarily in non-seasonal depression, the predictable, recurring nature of SAD may lend itself to ruminative processes.



Response Styles theory was originally advanced in part to account for the gender differences in prevalence rates found in MDD. About twice as many women are diagnosed with nonseasonal depression than men (APA, 1994), and in SAD the ratio has been estimated to be at least twice that (e.g., Kasper et al., 1989; Rosen et al., 1990). In questionnaire studies, gender differences have been found, suggesting that women use more ruminative and less active responses to depressed mood, whereas men are more likely to actively distract themselves from the depressed mood (Nolen-Hoeksema, 1987). Gender differences have also been found in non-clinical types of depression (i.e., following loss of a loved one) in that women reported using more rumination than men (Nolen-Hoeksema, Parker, & Larson, 1994). In contrast, studies that utilized distraction versus rumination tasks found no gender differences (e.g., Katz & Bertelson, 1993; Morrow & Nolen-Hoeksema, 1990). These studies suggest that the two styles of responses (i.e., distracting and ruminating) have similar effects when they are used as coping responses, regardless of gender.

Although the mechanism by which rumination affects levels of depression is not known, Nolen-Hoeksema (1987) details three possible mechanisms through which rumination may serve to exacerbate depressed mood and distraction may act to alleviate depressed mood. One possible mechanism is based on the effects of depression on cognitive processing. For example, negative symptoms of depression seem more anxiety-provoking and salient to the individual (Nolen-Hoeksema & Morrow, 1993). The cognitive difficulties related to rumination (i.e., decreased concentration and attention) may lead to increased failures and foster a sense of helplessness and lack of control over one's environment (Nolen-Hoeksema, 1987). Alternatively, the use of distraction

interferes with the effects that depressed mood has on cognitive processing and may also serve to reward the individual through negative reinforcement.

Secondly, Nolen-Hoeksema (1987) suggests that rumination may serve to exacerbate depressed mood by exposing the person to greater negative memories and cognitive content. As reported by Teasdale (1985), mood state influences recall and learning. For example, individuals with depressed mood who ruminate would tend to recall past events more negatively as well as recall more negative past events. In a study with individuals who had previously been dysphoric, researchers induced both positive and negative moods and had participants complete an emotional Stroop task (Gilboa & Gotlib, 1997). The researchers later assessed memory for the Stroop stimulus words and found that individuals who had previously been dysphoric remembered significantly more negative words than controls. It may be that through repeatedly retrieving biased memory recall, rumination works to activate latent negative schema, thus setting the depressive spiral in motion or serving to perpetuate it.

Similarly, Nolen-Hoeksema's (1987) third proposed mechanism hypothesizes that rumination may exert its effects by increasing the likelihood that current events will be evaluated more negatively. By interpreting events more negatively, it is hypothesized that individuals increase negative expectations of the future, leading to feelings of hopelessness and helplessness (Abramson, et al., 1978). If individuals interpret current situations in a negative way, they are more likely to predict that the future will not improve regardless of their efforts and therefore, they are likely to become more depressed (Miller & Seligman, 1976). In other words, individuals who ruminate are more likely to find depressing reasons for their depressed mood.

It also may be that ruminating about depressed mood can have the effect of reducing positive reinforcement from the environment (Lewinsohn, 1974). For example, if an individual is focusing on feelings and their source, she or he may discount or overlook positive or neutral current events in searching for the root of her or his depressotypic feelings. Negative cognitions have been hypothesized to moderate the effects of the environment (Lewinsohn, et al., 1985). For example, the meaning an individual gives to a situation will determine the strength of the reinforcer. Individuals ruminating about depressed mood interpret current situations negatively, thus depriving them of the benefit of a positive reinforcement. Furthermore, individuals with depressed mood are less willing to engage in pleasant activities (e.g., distractions), which would provide an additional source of positive reinforcement, even when they believe it could relieve their depressed mood (Lyubomirsky & Nolen-Hoeksema, 1993). Although individuals report that a motive for using rumination is the goal of gaining insight into emotional responses and problem situations, high-rumination individuals are actually less effective at problem solving tasks (Lyubomirsky & Nolen-Hoeksema, 1993).

Although research has indicated some reasons why individuals might utilize rumination as a strategy for coping with depressed mood, it is unclear exactly why more women than men use the strategy. For example, it might be useful to study children and adolescents to determine when these gender differences appear and to investigate the factors related to developing a ruminative cognitive style. Although correlational and analogue studies support Response Styles theory, more research is necessary to determine what the mechanism of action is for rumination and why more women than men ruminate in response to depressed mood.

SAD represents a disorder with a predictable, cyclic pattern. Rumination may play a role in the development of recurrent SAD episodes and may play a key role in the maintenance and exacerbation of the disorder. Individuals who have experienced a winter depression may be particularly vulnerable to rumination because the repetitive nature of the disorder suggests a distinct cause (i.e., wintertime, shorter periods of daylight) for depression. Individuals may ruminate about their feelings and mood in anticipation of winter's approach. They may also focus their thoughts on feelings of having little control over the aversive symptoms of SAD.

In the first study examining the role of rumination in SAD, researchers have found that degree of rumination in general (i.e., not season-specific) in September predicted the severity of a SAD episode in February (Young, 1999b). In a second study, researchers found that women with SAD who had elevated rumination scores in the fall developed a depressive episode, whereas those with low rumination scores did not meet criteria for a depressive episode later that winter (Rohan et al., 2003).

However, these studies employed a trait measure that assessed how an individual responds to depressed mood in general, as opposed to how they respond to depressed mood related to the changing of the seasons or across specific situations. For example, these studies used a measure that asks what an individual usually does in response to depressed mood. It may be that individuals have different responses depending on the time of year or depending on the attribution the individual makes about the depressed mood. Although preliminary evidence suggests that a ruminative style may be related to SAD, it may be important to examine the frequency and intensity of rumination across seasons in order to elucidate how rumination plays a role in SAD. The pattern of

rumination used by individuals can best be addressed by use of prospective measurement of symptoms that would be able to detect changes in mood state and changes in rumination intensity that have a temporal relation. In addition, cognitions should also be monitored at the same time, such that a measure of attribution can be compared to the intensity patterns of rumination.

### Diathesis-Stress Models

Diathesis-stress models in general reflect an important development in the psychopathology literature. Typically, biological and psychological models focus only on one aspect of a disorder and thereby ignore a potential role for other factors. In contrast, diathesis-stress models recognize the etiological complexity of the disorder by often positing an interaction of biological and psychological factors. Clearly, SAD is not just a simple, heritable, biological deficiency. Although genetic and biological deficiencies may play a large role, preliminary research has demonstrated that varied types of psychological factors affect the experience of SAD as well. The biological view, in general, focuses on an underlying etiology that can account for the initial episode of SAD, but not variations in experience of symptoms from year to year. The psychological approaches, in general, address the experience of SAD episodes and factors that may serve to exacerbate or maintain the disorder. However, psychological models do not provide much information about the possible factors involved in the onset of the initial episode of the disorder.

Diathesis-stress models seek to integrate the important findings that relate to the initial onset, maintenance, recurrence, exacerbation and experience of SAD episodes from different disciplines (i.e., biology, physiology, and psychology). Most importantly,

diathesis-stress models suggest that both the initial onset and recurrence of SAD episodes is dependent on a combination of factors that may have additive and/or interactive effects necessary for a SAD episode to occur. These types of models provide a heuristic for continuing to study which variables are necessary, which variables interact, and which variables are only correlated with SAD. In addition, by investigating the contribution of psychological factors, we may discover findings for unanswered questions and resolve conflicting findings within the existing literature (Sigmon et al., 2001).

Diathesis-stress models may also help to explain why individuals do not necessarily experience a SAD episode every year. In some winters, the symptoms do not reach clinical levels for many individuals (e.g., Rohan et al., 2003). An integration of biological and psychological findings in recent studies may help explain this phenomenon. For example, a person may be exposed to more natural light some years, therefore experiencing fewer vegetative symptoms and having a less frequent or less intense depressed mood. Alternatively, some years individuals may be more active (i.e., distracting) such that they do not ruminate as often about depressed mood and other symptoms, preventing the development of SAD.

Two diathesis-stress models of SAD have been posited. Sigmon and colleagues (2001) proposed a model that incorporates physiological, cognitive, and behavioral factors in accounting for the recurrence and severity of SAD episodes. In their model, a biological sensitivity to the effects of decreased photoperiod represents a diathesis, in spite of the lack of consensus or clear support for any one biological model regarding the etiology of SAD. In addition, the researchers suggest that the core physiological symptoms of SAD (i.e., fatigue, hypersomnia, increased appetite) may act as the stressor

for the model. They note that the risk of onset for these “core symptoms” is related to the onset of a subsequent episode of SAD (Young et al., 1991). Either way, this diathesis-stress model of SAD allows for the role of biological mechanisms as the stress that sets a subsequent SAD episode in motion.

The diathesis-stress model (Sigmon, et al., 2001) also recognizes the role of cognitive and behavioral factors in the severity of SAD symptoms. In particular, their model implicates rumination (i.e., Response Styles Theory; Nolen-Hoeksema, 1991) as playing a significant part in the maintenance of the disorder. Environmental cues (i.e., cooler temperatures, shorter days) associated with past depression and physiological symptoms may trigger monitoring of and rumination about mood and symptoms. These cues and expectations may lead to negative changes in cognitive products (i.e., automatic thoughts) that contribute to increased SAD severity and occurrence. In these ways, individuals’ cognitive responses (e.g., perception, memory, thoughts) to the changing seasons, mood and vegetative symptoms play a role in future SAD episode recurrence. These cognitive responses may form schema that serve as part of the diathesis within the model.

In addition to cognitive factors, behavioral factors are implicated in this diathesis-stress model as well (Sigmon et al., 2001). Drawing from Lewinsohn’s (1974) integrated model of depression, the authors posit that decreases in positive reinforcements within the environment (e.g., social, emotional), in combination with pre-existing vulnerabilities (i.e., biological sensitivity to decreased photoperiod, development of SAD schema) may exacerbate recurrences of depressive episodes. Currently depressed women with SAD have demonstrated elevated psychophysiological responses to low-light stimuli,

suggesting a possible conditioned emotional response to certain environmental cues (Rohan et al., 2001). Furthermore, in the same study, researchers found that scores indicating lower levels of enjoyment of pleasant events could predict the severity of SAD symptoms the following winter. Although some initial studies provided support for this model, more research needs to be conducted to assess the role that both cognitive and behavioral factors play in recurrent SAD episodes.

Because Sigmon et al.'s (2001) diathesis-stress model draws from Young et al.'s dual vulnerability hypothesis (1991) and posits involvement of similar processes, it is important to further investigate the dual vulnerability hypothesis. The major difference between the two models is that Young's dual vulnerability hypothesis (1991) does not include behavioral factors, whereas Sigmon et al.'s model (2001) does address these factors as playing a role in the development and maintenance of SAD episodes. Furthermore, Sigmon and colleagues draw on additional principles from Beck's (1967) cognitive theory of depression and suggest a cognitive diathesis related to onset of subsequent SAD episodes. Young and colleagues propose that individuals vulnerable to developing depression (i.e., diathesis) develop depressive symptoms secondarily to a set of core physical (i.e., vegetative) symptoms. In this model, depressive symptoms develop in the presence of fatigue, hypersomnia, and increased appetite, which collectively serve as a stressor. Thus, in order to develop the syndrome of SAD, individuals must possess a vulnerability to experiencing significant seasonal changes as well as a vulnerability to developing depression.



However, Young (1999) only argues that a vulnerability to developing seasonal vegetative symptoms may exist. He does not posit what it is that may comprise the vulnerability other than to note that there are some studies in the literature suggesting a heritable factor may be related. He does address the psychological vulnerability to developing the depression secondary to the vegetative symptoms, and hypothesizes that ruminative processes may be involved (Young, 1999a; Young, 1999b; Young & Azam, 2003). He has explored Response Styles Theory (Nolen-Hoeksema, 1981) in relation to SAD in preliminary studies and found that early fall rumination scores predicted SAD severity the subsequent winter (Young, 1999a; Young 1999b; Young & Azam, 2003).

The dual vulnerability hypothesis, however, is based on the findings of a single study (Young et al., 1991). Young and colleagues found that individuals with SAD exhibited two separate groups of symptoms based on temporal onset. The researchers recruited individuals with SAD who were currently experiencing an episode of winter depression. Participants selected and then temporally ordered cards that contained relevant symptoms. Participants were then asked to retrospectively determine the week of symptom onset for each of the symptoms experienced. Using a survival analysis to calculate hazard rate functions (i.e., risk of onset), the onset of each symptom was calculated. The results indicated that the risk of onset for the vegetative symptoms (i.e., fatigue, hypersomnia, increased appetite/weight) was related to the onset of an episode of SAD that fall or winter. The risk of onset for other symptoms characteristic of SAD (i.e., depressed mood, social withdrawal, difficulty concentrating, self-reproach, etc.) did not appear to be related to the subsequent onset or course of the SAD episode.

Unfortunately, Young et al.'s (1991) study did not measure the severity of the symptoms experienced during the current episode. Thus, the hazard rates may have been calculated based on vegetative symptoms that were very mild or within normal limits. Furthermore, the study did not include a control group to determine if the experience of symptoms and temporal onset was different from what might be seen in the general population. Most importantly, Young et al.'s (1991) study is based on retrospective self-reports of currently depressed individuals who received help from researchers in determining when each symptom began. The assistance from the researcher may have inadvertently influenced the choice of onset dates. In addition, the selected onset dates may be inaccurate due to the use of retrospective self-reports of individuals who were currently depressed.

Previous research has found that depression serves to have detrimental as well as biasing effect on memory, especially retrieval of information (e.g., Ellis & Moore, 1999; Murray, Whitehouse, & Alloy, 1999). For example, a more distressing symptom could be perceived as having persisted for a longer time to an individual experiencing a current episode of depression. In addition, individuals with SAD may not recall when a symptom began and either guess or respond to cues from investigators. Individuals with nonseasonal depression have demonstrated deficits in recall (e.g., Ilsley, Moffoot, & O'Carroll, 1995). In a study with 15 individuals with depression and matched controls, researchers found that those with depression were significantly impaired at recalling details of everyday memory function, such as stories and messages (Ilsley et al., 1995). Together, these findings suggest that individuals with depression do not have trouble encoding information, but instead have difficulty with the search process. This memory

impairment could be due to the cognitive effects of depression, such as difficulty with concentration. Regardless of the reason, there is ample evidence to support the possibility that participants in Young and colleagues' (1991) study may not have had accurate recall for the symptom presence and onset.

Accuracy of retrospective report of symptoms could also be affected by the degree of self-monitoring an individual engages in (e.g., Williams, Lees-Haley, & Price, 1998). Some individuals may be more sensitive to the effects of mild vegetative symptoms than to mild mood symptoms, which may affect recall. Similarly, the results (Young et al., 1991) could also be biased if there are some symptoms that an individual monitors more than others (e.g., sleep difficulties versus energy level). Bias could also result from the attributions an individual makes about the symptoms he or she experiences. If an individual did not attribute a symptom to part of the experience of SAD, he or she may not pay as much attention to the symptom, therefore resulting in a poorer memory of onset. There are many possible confounds in the use of retrospective reports of symptoms from participants diagnosed with MDD and without control groups for comparison.

Lastly, regardless of mood state, it is unclear how accurate the retrospective report of symptom onset was. Although the researchers sought to anchor symptom onset to a timeline with personally relevant information, a significant amount of time elapsed from symptom onset to participatory recall. The mean amount of time between reported onset and recall was 12.5 weeks, or more than 3 months, and the range was 3 weeks to over 2 years. This duration between reported onset and recall suggests possible variability in accuracy and precision of reports of symptom onset.

Although other researchers have looked at depression longitudinally, they have tended to use the Beck Depression Inventory (BDI; Beck, Steer & Brown, 1993) and other self-report surveys of mood (e.g., Harmatz et al., 2000; Molin et al., 1996). However, the BDI does not assess many of the vegetative symptoms or atypical symptoms associated with SAD (i.e., fatigue). Therefore, it is unclear how these atypical symptoms vary across the seasons in the general population as compared to the experience of individuals with SAD. For example, one study using the BDI found that there was a seasonal component to mood variation in the general population (Harmatz et al., 2000). Using a longitudinal design with a large sample, and excluding individuals with possible SAD, the researchers measured mood at four points (i.e., winter, spring, summer, and fall) across one year. Negative mood valence was highest in the fall and winter months (Harmatz et al., 2000), yet symptoms common to SAD, such as fatigue and increased food consumption were not measured. However, these results do highlight the importance of including a control group when assessing symptom onset and severity for SAD groups as there appears to be a normative level of change in mood.

In investigating the relation between depressed mood in individuals with SAD (i.e., BDI scores) and climactic variables one study found correlations with minutes of sunshine, global radiation, length of daylight, and temperature (Molin et al., 1996). This study followed 126 participants with SAD over four years from September to May. Another study investigated changes in SAD symptoms longitudinally, but only gathered data at three points throughout one year, (i.e., fall, winter, and spring; Rohan, et al., 2003). Due to the limited data points, these types of studies cannot answer questions regarding the pattern of symptom onset. No other published study has systematically

investigated the onset of SAD symptom profile prospectively. It is important to determine whether there is additional support for the validity of Young et al.'s (1991) dual vulnerability hypothesis. In particular, it is important to investigate whether the same pattern of clustered symptoms appears when the symptoms are measured prospectively. Furthermore, investigating the pattern of symptom onset and qualities of cognitive processes that may be involved in subsequent SAD episode onset may have important implications for theories of etiology as well as for designing alternative psychological treatments for SAD.

### Summary

The majority of the existing literature on SAD focuses on epidemiological issues, description of the disorder, and on biological theories to account for the temporal pattern of symptom onset. Research suggests that seasonality may exist on a continuum (e.g., Kasper et al., 1989). In addition, preliminary findings indicate that different patterns of responses to seasonal changes may exist (e.g., Boulard & Sigmon, 1999). For example, there appears to be a distinguishable group of the general population who experiences vegetative symptoms with the same temporal patterns as SAD, but who do not develop mood symptoms (i.e., winter anergia; WA). It may be important to investigate WA in comparison to SAD as research on this group may indicate important protective factors that prevent the initial onset of SAD and may be used in the future to prevent recurrent episodes in those with SAD. In addition, work with this group may lend support to the dual vulnerability hypothesis. The WA group represents a group with a single vulnerability for developing the vegetative symptoms without the psychological vulnerability for developing depression.

Preliminary research on genetic heritability (e.g., Jang et al., 1997) offers support for a genetic role in the initial development of SAD. In addition, genetic research may uncover factors related to the increased prevalence of SAD among women.

Neurotransmitter theories (e.g., Thase & Howland) attempt to link findings from studies on the function of serotonin, non-seasonal depression, and biological rhythms governed by neurotransmitters. However, these hypotheses need more research and support in addition to treatment studies with anti-depressant drugs before they can be fully evaluated.

Other biological models have concentrated on investigating the role of light in physiological processes. The photoperiod model posits that SAD is due to insufficient length of daylight needed to maintain mood (Lewy, 1988). Investigators have offered more specific mechanisms to account for why decreased levels of light might affect mood. Researchers have posited that individuals with SAD may be shifted out of normal circadian phase with respect to natural light-dark cycles (e.g., Lewy, 1988). Another model posits that in the fall there is an insufficient amount of light energy for maintenance of mood (Rosenthal & Wehr, 1992).

In general, support for biological models comes from light therapy outcome studies. Light therapy represents the gold standard for SAD treatment, however, a significant proportion of individuals diagnosed with SAD do not respond to light therapy. Those who do respond generally do not experience complete remission of all symptoms. Therefore, it does not appear that light models can fully explain the phenomenon of SAD. In addition, light therapy has some troubling side effects and is not available for everyone, begging the exploration of alternative methods of treating SAD.

Studies investigating psychological correlates have begun to shed light on the role of cognitions in the onset of recurrent individual SAD episodes as well as the maintenance and experience of the disorder. Researchers have found that individuals with SAD report negative cognitions at levels similar to those with non-seasonal depression (Levitan et al., 1997). Studies have found evidence for dysfunctional cognitions about light and dark indicative of light-related schemas (Bouhuys et al., 1994; Rohan et al., 1999). In addition, cognitive impairments (e.g., spatial memory and learning) may be related to SAD (e.g., O'Brien, Sakaharian & Checkley, 1993) and cognitive interference may result from exposure to seasonal-related stimuli (e.g., Spinks & Dalgleish, 2001). Rumination (Nolen-Hoeksema, 1987) may also be related to the initial onset of SAD episodes or maintenance of the disorder (Rohan et al., 2001; Young, 1999b).

This study was proposed to investigate the role of rumination from prospective reports that can be related temporally to changes in symptoms. In addition, this study assessed the frequency of specific cognitions (i.e., automatic thoughts) related to the changing seasons and their relationship to symptom onset. Furthermore, this study investigated the presence of attentional bias in SAD related to WA and controls. Using the emotional Stroop task, this study attempted to examine interference effects of negative, seasonal and light related cues in SAD. Attentional bias is important to investigate because it may play a role in the development of subsequent SAD episodes. If SAD individuals attend more to negative stimuli than controls or those with WA, they are more likely to experience depressed mood.

Theories integrating findings from the biological literature and the psychological literature have focused on diathesis-stress models. Sigmon and colleagues (2001) integrate biological, cognitive and behavioral theory findings in their model. The researchers draw on Young and colleagues' (1991) dual vulnerability hypothesis, another diathesis-stress model, for the stressor in their model. The dual-vulnerability hypothesis proposes that individuals with a vulnerability to developing depression do so following the initial development of a core set of vegetative symptoms—those symptoms seen in WA. The vegetative symptoms (e.g., fatigue, hypersomnia, etc.) serve as a stressor. Young's model, however, has not received any direct published support and is based on the findings of one retrospective study. It is important to further test the component of both diathesis-stress models in order to establish whether this type of model has sufficient support to explain recurrent SAD episodes. The 1991 study served as the basis for formulating the dual vulnerability hypothesis, but it clearly has confounds. The proposed study tested the stressor component of the diathesis-stress models of SAD by utilizing a longitudinal design assessing the development of SAD symptoms during a recurrent episode onset. Furthermore, this design will allow the comparison of symptom onset and severity of individuals with SAD, WA, and controls.

#### Statement of Purpose

The current study sought to further investigate Young and colleagues' dual vulnerability hypothesis (1991) using prospective reports. Use of this methodology was intended to reduce or eliminate the selective attention bias that may exist for certain symptoms relevant to SAD. Replicating and improving the methodology of the previous study is important to establish support for the dual vulnerability hypothesis and diathesis-



stress models of SAD. According to both models, there is a primary core set of vegetative symptoms, followed by a secondary “reaction” of depressive symptoms. In the current study, similar rates of survival time for each set of symptoms were expected to emerge when the symptoms were measured prospectively as compared to previous findings with retrospective measurement. More specifically, the survival times for the core vegetative symptoms were expected to continue to cluster together and be shortened, whereas the remaining mood symptoms were expected to have longer survival time to onset when assessed prospectively. This design served to overcome the possible confounding effects of memory in the previous study (Young et al., 1991).

In addition, the present study further investigated the possible role of cognitive factors in vulnerability to developing SAD episodes. In the current study, participants monitored automatic thoughts about seasonal cues, degree of rumination, and SAD symptoms on a weekly basis. This component will contribute to the literature on cognitive factors regarding the onset and maintenance of subsequent SAD episodes by examining the onset of a subsequent SAD episode in relation to the frequency of negative automatic thoughts and degree of rumination. The prospective measurement of negative automatic thoughts and rumination in SAD allowed for the investigation of a possible mediating role for cognitive processes in SAD. Although hypothesized, little empirical evidence exists even in the non-seasonal depression literature of the mediating role of automatic thoughts (Kwon & Oei, 1994).

This study also addressed the role of cognition in SAD by investigating the degree of attentional bias and interference for words hypothesized to be more salient to individuals with SAD. In particular, this study was designed to address the effects on

response time in a Stroop task when stimulus words have depressotypic, seasonal or light-related valences. These words were compared to neutral valence words for comparisons between SAD, WA and Control groups. This component of the study expanded upon preliminary work that found individuals with SAD have specific cognitive structures (i.e., schema) sensitive to the negative and/or seasonal stimuli. In addition to replicating previous findings, this study included comparison groups, improved upon the previous methodology, and added light related stimulus words to investigate different types of possible schema.

The proposed study utilized individuals with winter anergia (WA) and individuals with no history of depression (i.e., Controls) as comparison groups with the SAD group. According to the dual vulnerability hypothesis, individuals in the WA group were expected to differ diagnostically from individuals in the SAD group only on mood symptoms. There is no evidence to suggest that the temporal onset of core symptoms should differ. Hypothetically, the WA group should only differ with regard to cognitive vulnerability to depression. In other words, the WA group was expected to be more similar to the Control group than the SAD group on measures of automatic thoughts, degree of rumination, attentional bias and cognitive interference (i.e., Stroop task), as well as overall depression symptoms. The Control group served as a comparison for the experience of the SAD and WA groups. The Control group was also used to further investigate the hypothesis that seasonality occurs on a continuum. Therefore, Control group participants were expected to exhibit lower frequency and severity of vegetative symptoms and depressive symptoms, including negative thoughts.

## Hypotheses

Hypothesis One—Prospective Symptom Measurement. Prospective measurement of the onset of SAD symptoms in the SAD group will demonstrate earlier temporal onset of vegetative symptoms (i.e., fatigue, increased appetite, hypersomnia) than mood symptoms (i.e. sadness, guilt, anhedonia). The WA group should exhibit a pattern of temporal onset for physiological symptoms similar to the SAD group, but the WA group should not exhibit onset of consistent *mood* symptoms, such that there is no calculable risk of onset for mood symptoms. Control participants should not exhibit onset of consistent symptoms, indicating that there is no risk of onset for a given symptom.

Hypothesis Two—Automatic Thoughts and SAD. Frequency of automatic thoughts and seasonal automatic thoughts will be correlated with depressed mood scores and will increase between summer and winter assessments. SAD participants will score higher on Automatic Thoughts Questionnaire and Seasonal Automatic Thoughts Survey overall than the WA and Control groups.

Hypothesis Three—Rumination and SAD. The SAD group should exhibit higher rumination scores on the Response Styles Questionnaire than WA and Control groups in both summer and winter assessments. Winter assessment scores should be higher for SAD participants.

Hypothesis Four—Stroop Task and SAD. In general, individuals experiencing affect related to the stimuli should have longer latencies to give responses to the color naming task. SAD participants will exhibit longer latencies to respond to seasonal words than WA and Controls in winter only. On depressotypic words, SAD participants will demonstrate a longer latency to respond than WA and Controls in winter only. On light

related words, SAD will have a longer latency to respond than WA and Controls in the winter only.

For SAD participants, negative, seasonal, and light words will result in a longer latency times at winter and summer than neutral stimuli or reaction time control stimuli (i.e., XXXX). No differences for the other two groups are expected between summer and winter measurements specific to stimuli type, but overall increased latency to respond is expected in winter across all diagnostic groups.

Hypothesis Five—Predicting SAD Episode Severity. Several cognitive variables will be examined for their utility in predicting the severity of subsequent episodes of SAD (i.e., SIGH-SAD scores). Seasonal automatic thoughts and trait rumination scores measured during the summer, as well as summer Stroop performance (i.e., latency to respond) on seasonal, depressed, and light words should predict severity of SIGH-SAD scores in winter.

## Chapter II

### METHODS

#### Participants

Participants ( $N = 67$ , 15 males and 52 females) were recruited during July through September 22 in two cohorts during 2001 and 2002. Participants were individuals who had participated in previous SAD research projects and indicated interest in future research projects ( $n = 11$ ), individuals who had responded to media advertisements/direct appeals for participants ( $n = 27$ ), and undergraduate psychology students who received course credit for participation ( $n = 29$ ). Undergraduate participants were screened for likelihood of falling into one of the three experimental groups (i.e., SAD history, Winter Anergia, or Controls).

The majority of the sample was Caucasian (66 Caucasian, 1 African American). Approximately half of the sample was married (55.2%), a third was unmarried and/or cohabitating (34.3%), with the remaining divorced (7.5%) or widowed (3%). Although most participants reported no medication use (59.7%), some individuals reported taking a stable dose of antidepressant medication (15%).

#### Assessments

The Structured Clinical Interview for DSM-IV (SCID; First, 1996) was used to diagnose participants with a history of SAD and to screen out individuals in the WA and Control groups with a history of depression episodes. In addition, participants were assessed using the Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder version (SIGH-SAD) to measure depressive symptom severity (Williams et al., 1992). The Global Symptom Severity (GSS) scale of the

Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Bradt, & Wehr, 1984) was used to screen for seasonal symptom profiles to identify potential participants within the university's pool of available research participants. The principal investigator and 3 other advanced graduate students conducted the assessment interviews. SCID interviews were audio taped and all interviewers listened to the available recordings of the diagnostic interviews in order to assess diagnostic reliability in this study. Fifty-four participants' summer interviews (80%) were reviewed, with 100% interrater reliability for SCID diagnosis. Thirty-five participants' winter interviews (56%) were reviewed, showing 94.7% interrater reliability of diagnosis by SCID. Unreviewed participant interviews were due to technical problems with audio equipment, unrecorded interviews and poor quality of recordings.

#### Exclusionary Criteria

Participants in the SAD-HX group ( $n = 23$ ) met DSM-IV (APA, 1994) criteria for past episode of major depression, recurrent, seasonal pattern and also met Rosenthal's (1984) research diagnostic criteria for past SAD episodes. Individuals in the SAD group were recruited through previous participation in SAD studies ( $n = 9$ ), media/direct appeals ( $n = 9$ ) or through undergraduate screening ( $n = 5$ ). Participants in the WA group ( $n = 19$ ) reported a history of significant seasonal changes in vegetative symptoms, as determined by a global symptom severity (GSS) score of  $> 12$  on somatic symptoms (i.e., sleep, appetite, weight, energy) and  $< 6$  on affective items (i.e., mood, social activity). All 19 participants were recruited through undergraduate screening. Control participants ( $n = 25$ ) reported no history of depression or SAD and evidenced few seasonal changes in vegetative symptoms, as evidenced by a GSS score of  $< 12$ . Control participants were

recruited through previous participation in SAD studies ( $n = 2$ ), media/direct appeals ( $n = 18$ ) or through undergraduate screening ( $n = 5$ ). Five participants dropped out of the study (3 SAD-HX, 1 WA, 1 Control) due to unexpectedly moving shortly after enrollment ( $n = 1$ ) or not returning for the winter session ( $n = 4$ ).

Participants did not meet criteria for any current Axis I disorder other than SAD (i.e., at winter assessment) or an anxiety disorder. Seven participants met criteria for anxiety disorder. In the SAD-HX group three participants met criteria for specific phobia of heights, generalized anxiety disorder (GAD), and social phobia. In the WA group one participant reported symptoms consistent with a history of panic disorder, social phobia and current GAD. In the Control group, 3 participants met criteria for specific phobias (i.e., heights, dentists, cats). Individuals using psychotropic medications were included in the study as long as they were on a stable dose of medication (i.e., no prescribed changes in dose for three months prior to study participation). For ethical reasons (i.e., no alternative treatment was offered following or during the course of the study) participants were not asked to discontinue antidepressant medication use. Stable antidepressant use was accepted in this study for naturalistic observation, and although it was acknowledged that its use may reduce reported symptoms, it should not lead to inflation of reported symptoms. Nine participants in the SAD-HX group and one in the WA were taking antidepressants during the study. Individuals were not asked to give the diagnosis that specific medications were prescribed to treat and some may have been taking the medications for diagnoses other than depression (i.e., anxiety disorder, chronic pain). Although a WA participant was prescribed an antidepressant, the individual did not meet criteria for past or current depressive episode.

### Experimenters

The principal investigator, who was completing her graduate training in clinical psychology, conducted the current study. She was assisted by three advanced graduate students who had training in assessment using the Structured Clinical Interview for the DSM-IV and the Structured Interview Guide for the Hamilton Depression Rating Scale—Seasonal Affective Disorder version. A licensed psychologist supervised the conduct of the study and the study was approved by the University of Maine Committee for Research Using Human Subjects (IRB). Undergraduate students with research experience assisted with clerical tasks and tracking participants' weekly responses after receiving training in IRB procedures.

### Measures

Structured Clinical Interview for DSM-IV for Axis I Disorders (SCID). The SCID (First et al., 1996) is used for diagnosing Axis I disorders according to DSM-IV criteria. The interview consists of an overview and nine modules. Reliability and validity data are not yet available for this newest version, but test-retest reliability ranged from kappas of .37 for current diagnoses to .68 for lifetime diagnoses for the DSM-III-R version (Williams et al., 1992). In addition, excellent inter-rater reliability was obtained for Major Depressive Disorder (.93), indicating that the SCID represents an acceptable diagnostic tool. The SCID is typically used in the literature to diagnose SAD.

Structured Interview Guide For the Hamilton Rating Scale For Depression-Seasonal Affective Disorder Version (SIGH-SAD). This guide (Williams et al., 1992) represents a semi-structured interview designed to assess the severity of depression in individuals with SAD. This instrument includes the 21-item version of the Hamilton



Depression Rating Scale plus eight additional items relating specifically to SAD symptoms. The 29 items cover somatic, cognitive, behavioral and affective symptoms. The original Hamilton Depression Rating Scale has demonstrated interrater reliabilities ranging from .86 and .96 (Gibbons, Clark & Kupfer, 1993) and good internal consistency, with a coefficient alpha of .76 in a depressed community sample, and good discriminant validity between depressed and normal individuals (Rehm & O'Hara, 1985). The modified SIGH-SAD measure is frequently used as an outcome measure in SAD light therapy studies (e.g., Tam et al., 1995; Oren et al., 1992) as well as studies of psychological factors related to SAD. Previous research has found adequate Cronbach's alphas ranging from .78 at summer assessment to .97 at fall measurement (Rohan, Sigmon & Dorhofer, 2003). Cronbach's alphas in this study were .82 at summer assessment and .90 at winter assessment.

The Seasonal Pattern Assessment Questionnaire. The Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal et al., 1987) is a self-report measure used to measure the extent to which individuals are affected by the changing seasons (i.e., seasonality). It comprises several scales (i.e., the months in which one feels best or worst, the degree to which one experiences seasonality as a problem), but the six-item seasonality scale (i.e., GSS) has received the most psychometric attention in the literature (e.g., Magnusson, 1996; Wirz-Justice, Graw, & Recker, 1993). The GSS has been shown to have high sensitivity (94%) and specificity (73%) for winter problems (Magnusson, 1996). It also differentiates SAD individuals with and sub-syndromal SAD (S-SAD) from those who have neither, but is less successful at differentiating SAD from S-SAD as compared to a structured interview (Magnusson, 1996).

The GSS demonstrates acceptable reliability for SAD participants across winter and summer ( $Rho = .57$ ; Wirz-Justice, Graw, & Recker, 1993). Although the reliability for S-SAD was poor across seasons ( $Rho = .09$ ; Wirz-Justice, Graw, & Recker, 1993), it should be noted that these correlations were calculated on a sample of 55 women, of whom only 6 were classified as having SAD and 11 as S-SAD. In addition, the SPAQ demonstrates good internal consistency for the six items of the GSS scale: sleep, social activity, mood, weight, appetite, and energy. Intercorrelation coefficients have ranged from .24 to .65 (Magnusson et al., 1997). In this study Cronbach's alphas for GSS were .92 (summer) and .94 (winter).

The Beck Depression Inventory-II. The Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1993) was originally developed by Aaron Beck in 1961, revised in 1971 and again in 1993. It is primarily used to detect depression symptoms in normal populations and to assess severity of depression in clinically diagnosed samples. In a 25 year review of the BDI (Beck, Steer, & Brown, 1993), the instrument shows good concurrent and discriminant validity, as well as internal consistency with coefficient alphas ranging .73 to .95 in the studies (Beck, Steer & Garbin, 1988). It is frequently used as both an ongoing assessment and outcome measure in cognitive behavioral therapy research (e.g., Beck et al., 1979; Dobson, 1989).

In order to assess symptoms specific to SAD, the BDI-II used in this study was modified by adding three SAD-relevant items to the instrument. These items have been used in previous research with SAD populations (e.g., Rohan et al., 2003) and address increased sleep, weight gain and increased food consumption. This instrument was used

to assess depression symptoms on a weekly basis. Internal consistency checks in this study were  $\alpha = .91$  (summer) and  $\alpha = .95$  (winter).

In order to distinguish winter anergia symptoms and SAD symptoms, face-valid subscales for mood and vegetative symptoms on the BDI-II were constructed to evaluate these symptom clusters. Items thought to reflect mood symptoms included sadness, loss of pleasure, crying, loss of interest, worthlessness, and irritability (i.e., Mood scale). Items included in order to reflect vegetative symptoms included loss of energy, change in appetite, tiredness/fatigue, increased sleep, increased eating, and weight gain (i.e., Veg scale). Previous research with an earlier version of the BDI with undergraduates supports a two factor-structure of the BDI for cognitive/affective and physiological components (Helm & Boward, 2003). An earlier study using a Major Depressive Disorder sample found a three-factor structure consisting of one cognitive and two somatic factors (Vredenburg, Krames, & Flett, 1985). Both scales demonstrated adequate internal consistency. Across the first 12 weeks of the study, Cronbach's  $\alpha$  for the Mood scales ranged from .76 to .93. Internal consistency checks for the Veg scale ranged from .73 to .87 across that same time period.

The Automatic Thoughts Questionnaire. The Automatic Thoughts Questionnaire (ATQ; Hollon & Kendall, 1993) was developed to assess the frequency of negative thoughts in depressed individuals consistent with Beck's theory of depression (Beck et al., 1979). The ATQ comprises 30 items, yielding scores ranging from 30 to 150 and has demonstrated high internal consistency (e.g., coefficient alpha of .94) in depressed participants (Hollon & Kendall, 1980). It has also demonstrated split-half reliability of  $r$

= .91 and high correlations with other depression measures (Hollon & Kendall, 1980).

Coefficient alphas for this study were .95 at both summer and winter measurements.

Seasonal Automatic Thoughts Survey. The Seasonal Automatic Thoughts Survey (SATS; Whitcomb, Sigmon & Kendrew, 2001) was developed to assess negative cognitions associated with the weather, light availability, and seasonal cues. It measures type and frequency of experience of negative thoughts about the changing seasons. The SATS consists of 22 items and has demonstrated excellent internal consistency ( $\alpha = .92$ ) and convergent validity with other measures of depression and seasonality (Whitcomb-Smith et al., 2001). Factor analysis has revealed 4 subscales (Depressed Mood, Vegetative Symptoms, Seasonal Expectation, and Noticing Change) accounting for 62% of the variance (Whitcomb-Smith et al., 2001). It has been shown to distinguish between SAD, S-SAD and Control groups (Whitcomb-Smith et al., 2002). This measure was used to monitor the weekly frequency of negative thoughts about the seasons. Cronbach's  $\alpha$  for this study were .96 and .95, respectively.

Response Styles Questionnaire. The Response Styles Questionnaire (RSQ; Nolen-Hoeksema et al., 1991) was designed to measure how an individual typically responds to depressed mood. The measure is composed of 32 items assessed with a four-point Likert-scale of frequency (where 0 = almost never and 3 = almost always). The RSQ contains two subscales, the Ruminative Response Scale (RRS) and the Distractive Response Scale (DRS). The RSQ has demonstrated good internal consistency, RRS = .89, DRS = .80 (Nolen-Hoeksema & Morrow, 1991) and the two subscales have been shown to be unrelated ( $r = .14$ ; Just & Alloy, 1997). The RRS demonstrated coefficient alphas

of .93 (summer) and .94 (winter). The DRS had coefficient alphas of .82 at both measurement occasions.

Response Styles Questionnaire-Diary Form. This shortened version of the RSQ was used weekly to assess for intensity of rumination (RSD; Nolen-Hoeksema, Morrow, & Frederickson, 1993). It contains 10 items empirically chosen from the longer RSQ scale and has demonstrated a coefficient alpha of .87. The RSD correlated with BDI-II scores ( $r = .54$ ) and with the HRSD ( $r = .44$ ) in a sample of 1122 adults (Nolen-Hoeksema, personal communication, April 23, 2001). Coefficient alphas in this study ranged from .91 to .96 over 18 weekly measurement occasions.

Profile of Mood States. The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971) represents a 63-item self-report measure of current mood state. The POMS is composed of six subscales: Depression-Dejection (i.e., depression), Tension-Anxiety (i.e., anxiety), Anger-Hostility, Fatigue-Inertia, Vigor-Activity, and Confusion-Bewilderment. Individuals rate each item on a 0 (not at all) to 4 (extremely) Likert scale according to their current experience. In the current study, only the Depression (POMS-D) subscale was used. This scale is designed to measure transient depressed mood. The POMS-D subscale has demonstrated excellent internal consistency ( $r = .95$ ) in an outpatient sample (McNair et al., 1971). The POMS-D was used to measure mood changes following the Stroop task so that current mood could be analyzed as a variable related to Stroop performance. It was given following the task, so as not to prime negative words that were included in the Stroop task. Cronbach's alphas for this study were .87 and .95, respectively.

Stroop Task Words. The stimulus words were stimuli previously used in the literature. Neutral words (i.e., rusty, statue, folded, structure, pile), seasonal-relevant words (i.e., seasonal, sleep, dark, November, winter) and depressotypic words (hopeless, lonely, immature, tense, pain) are from previous Stroop research in individuals with SAD (Spinks & Dalgleish, 2001; Williams & Nulty, 1986). The light relevant words (e.g., dim, dreary, dark, bleak, blackness) are also from previous research comparing groups of individuals with SAD, individuals with MDD and Controls (Sigmon et al., 2003).

### Procedure

Participant Recruitment and Assessment. Participants attended the initial session during the summer months (August through September 22; i.e., prior to the onset of the autumnal equinox). The study was explained to participants and written informed consent was obtained prior to participation in the study. Participants were assessed for inclusion in the study using the SCID-IV and SIGH-SAD interviews. Those who met criteria filled out the POMS-D questionnaire, completed the computerized Stroop task, and then completed the questionnaire measures. During the winter assessment (January through March 19; i.e., prior to vernal equinox), only the mood sections of the SCID and the full SIGH-SAD were administered. Participants then completed the POMS-D, Stroop task, and questionnaire measures again.

Stroop Task. At the initial session, participants completed the emotional Stroop task. Using a Dell Pentium III computer and E\*prime Beta software (Psychology Software Tools, Inc., Pittsburgh, PA) a modified Stroop task was presented to the participants. This task was designed to measure cognitive interference in individuals exposed to seasonal stimuli. Participants were given a practice trial consisting of 100

stimuli. The practice consisted of five trials of neutral words (i.e., ONE, TWO, THREE, FOUR, FIVE; Gotlib & McCann, 1984) that were randomly presented in each of the four colors used in the experiment (i.e., red, yellow, blue, and green).

Experimental stimuli were presented in five separate blocks and the order of presentation of the blocks was fixed. The fixed order was: control stimuli (e.g., XXXXX), neutral (i.e., folded, pile, rusty, statue, structure), negative (i.e., hopeless, immature, lonely, pain, tense), light-related (i.e., blackness, bleak, dim, dreary, night), and then seasonal words (i.e., dark, November, seasonal, sleep, winter). The neutral, negative, season-relevant, and light words were previously used in the literature (Sigmon et al., 2003; Spinks & Dalgleish, 2001; Williams & Nulty, 1986).

The order of the block presentation was designed to decrease the possibility of a Type II error due to practice effects (i.e., decreased naming speed) because the disorder-relevant blocks are presented last. Each block of a specific word type (e.g., neutral words) consisted of three trials of the 5 stimulus words. Within each block, stimulus words were presented in random order in each of the four randomly chosen colors. This design yielded a total of 60 stimuli per block. A total of 300 words were presented in the task. Participants were asked to indicate the color of the word by pressing the corresponding key from the four keys labeled on the computer keyboard with each of the experimental colors. The latency to respond to each word and block of words was automatically recorded by the computer program software.

Questionnaire Assessment. Participants also completed a short questionnaire packet in the lab containing all of the assessment measures. Participants recruited from the community received \$15 compensation for participating in each laboratory session

(i.e., summer and winter). Undergraduate participants received research credit for the summer assessment and \$15 for the winter assessment.

Weekly Diary Protocol. Participants were given monitoring packets containing sets of SATS, RSD, and modified BDI-II questionnaires (including the vegetative and mood symptom subscales) to be completed weekly. They were given instructions to begin monitoring symptoms using the weekly forms in the packet beginning the week of the autumnal equinox. Participants were contacted during that week by telephone to remind them to complete the weekly monitoring forms. Participants were given self-addressed stamped envelopes to return the forms to the investigator. After that time, participants were contacted only when weekly forms were not returned in a timely fashion.

Debriefing. Participants were thanked for their participation and all participants received a debriefing information sheet, information on SAD, and a list of psychological treatment referrals at the completion of the study.



### Chapter III

## RESULTS

### Participants

Participant demographic data were analyzed using several analyses of variance (ANOVA). Despite efforts to recruit equal groups (i.e., searching for older WA and Control group participants from the community, contacting older undergraduates that qualified for WA or Control group inclusion according to screening scores), there were significant differences across groups,  $F(2, 63) = 24.0, p < .001$ , education level  $F(2, 64) = 15.2, p < .001$ , and usual seasonal symptom experience (i.e., GSS),  $F(2, 61) = 11.0, p < .001$ . There were also significant differences between groups in years lived in this climate,  $F(2, 58) = 4.66, p = .013$ , however, it should be noted that this is likely due to the age differences between groups. Means for age, education, global seasonal severity (GSS) scores, and number of years in northern climate are presented in Table 3.1 below.

Significant differences between groups were further examined by Bonferroni post-hoc tests. SAD-HX participants were older than Controls ( $p < .001$ ) and WA participants, ( $p < .001$ ), and Controls were older than WA participants ( $p < .01$ ). Controls reported more years of education than SAD ( $p < .001$ ) and WA ( $p < .01$ ) participants. SAD-HX participants had lived in a northern climate longer than the other groups ( $p < .05$ ). SAD-HX participants ( $p < .001$ ) and WA participants ( $p < .01$ ) both reported more intense typical experience of seasonal symptoms than Control participants, but did not differ from each other. Although chi square analyses demonstrated that there was an uneven gender distribution among the groups,  $\chi^2(5) = 11.1, p < .01$ , further analyses showed that there was not a difference between groups in proportion of females,

Table 3.1

## Means and Standard Deviations for Demographic Variables

Variable	SAD-HX		WA		Control		Total	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age	40.4 <sup>a</sup>	13.4	19.4 <sup>b</sup>	3.5	28.6 <sup>c</sup>	9.0	29.9	12.7
Education	14.1 <sup>a</sup>	1.8	12.6 <sup>a</sup>	1.1	16.0 <sup>b</sup>	2.6	14.4	2.4
Years in northern climate	30.6 <sup>a</sup>	16.0	17.4 <sup>b</sup>	5.6	20.0 <sup>b</sup>	11.1	22.8	13.1
Global symptoms scale score	15.7 <sup>a</sup>	3.1	12.2 <sup>a</sup>	4.8	3.5 <sup>b</sup>	2.8	10.0	6.4
Gender ( <i>n</i> )	2m	21f	4m	15f	9m	16f	15m	52f

Note. Demographic variables of age, education, number of years one has lived in the northern climate, and baseline global symptoms scale score (Seasonal Pattern Assessment Questionnaire; Magnussen, 1986). Means with same superscripts are not significantly different at  $p < .05$  in the Bonferroni post-hoc tests. For gender data, m = males, f = females.

$\chi^2(2) = 1.2, p = .ns$  or males,  $\chi^2(2) = 2.92, p = ns$ . Furthermore, when the SAD group was compared to the population ratio of 4:1 for females to males, analyses revealed that proportion of males to females was as expected for the SAD group,  $\chi^2(1) = 1.8, p = ns$ . In examining the WA group, the proportion of males to females was more similar to the SAD population,  $\chi^2(5) = .01, p = ns$ , than to the 1:1 ratio of the general population  $\chi^2(1) = 6.4, p < .01$ .

### Hypothesis One

Longitudinal diary data were examined in several ways. First, the frequency of vegetative and mood symptom onset for all groups was examined across the 18 weeks of the study. Only 30 participants out of the sample of 67 completed all of the 18 weekly measurements ( $n_{\text{SAD-HX}} = 10, n_{\text{WA}} = 5, n_{\text{Control}} = 15$ ). Of the 67 participants who enrolled in the study, 63 completed at least 12 of the weekly assessments. All participants' mood and vegetative symptom profiles were examined, regardless of number of weeks completed. Vegetative and mood symptoms were evaluated through Mood and Veg subscales of the modified BDI-II, with each subscale consisting of 6 items. The onset of a symptom cluster (i.e., vegetative or mood symptom cluster) was determined by a score of 6 or greater on the Mood or Veg scale (indicating individuals had mild to moderate symptoms on a majority of items). The six point cutoff was used to indicate significant experience of symptoms. In order to reach six points, an individual must have experienced all symptoms comprising the subscale to a mild degree or have experienced a moderate to severe degree of several symptoms comprising the subscale.

As expected, none of the Control participants reached the 6-point cutoff score for Mood across the first 12 weeks. One individual in the Control group ( $n = 25$ ) reported

significant vegetative symptoms ( $> 6$ ) on the Veg scale at week 10. Only 6 of 18 WA participants reached the cutoff score ( $> 6$ ) for vegetative symptoms during the 12 weeks examined in the study. Contrary to expectations, all WA individuals who reached the cutoff did so between weeks 1 - 6. Unexpectedly, almost as many WA participants reported significant levels of mood symptoms; two at week 2, and one at week 6. Thus, the onset of mood symptoms in the WA group was contrary to a priori hypotheses stating that there would be no significant mood symptoms for this group.

Results of frequencies of significant symptom onset for SAD-HX participants are presented in Figure 3.1 below. It was predicted that there would be a relative symptom pattern of earlier temporal onset for vegetative symptoms as compared to mood symptoms. Overall, a pattern of early onset for both mood and vegetative symptoms was seen. The majority of SAD-HX participants ( $n = 19$ ) reported mild to moderate symptom onset of both mood and vegetative symptoms in September and October (i.e., Weeks 1 - 6). Consistent with predictions, more of the SAD-HX group met the Veg scale cutoff score in the first 6 weeks of reporting symptoms (80%), than met the Mood scale cutoff score in the first 6 weeks of reporting symptoms (70%). The median survival time (i.e., number of weeks from start of measurement until the cutoff criteria were met) for vegetative symptoms was 4 weeks ( $SE = 1.12$ ). This was slightly earlier than the 4.5 week ( $SE = 1.05$ ) median survival time to reaching the mood symptom cutoff point.

Order of symptom onset was also examined for each group and frequency of individuals meeting the cutoff score for vegetative symptoms first (or only met cutoff for vegetative symptoms), mood symptoms first (or only met cutoff for mood symptoms), or

neither symptom first (i.e., not meeting cutoff for either symptom or simultaneous onset) was analyzed. For participants in the SAD-HX group, 7 individuals had significant vegetative symptoms first, 5 had mood symptoms first and 7 had neither. Results were analyzed by using Chi Square analyses,  $\chi^2(2) = .42, p = ns$ , and suggested no onset pattern was more prevalent for the SAD-HX group, contrary to predictions that the onset of vegetative symptom first would be the more prevalent pattern. In the WA group, 6 participants reported vegetative symptoms first, with only one participant reporting mood symptoms first, and 10 with neither symptom experience meeting the cutoff scores. Analyses revealed that frequencies within the WA group for pattern of onset was different than chance,  $\chi^2(2) = 7.26, p < .05$ . Only 1 of 25 control participants met the cutoff score for significant symptoms (i.e., vegetative), none met the mood symptom cutoff and 24 participants did not meet cutoff of either symptom type. These results were also significant,  $\chi^2(2) = 44.69, p < .05$ ; as expected control participants did not report symptom onset.

Longitudinal mood and vegetative symptom data were examined by a repeated measures MANOVA using Wilk's  $\lambda$  for occasions (weeks =12) by group (SAD-HX, WA, Controls) by measure (mood, vegetative symptoms). Partial etas squared ( $\eta_p^2$ ) are reported for effect sizes. According to Cohen (1977),  $\eta^2 = .01$ ,  $\eta^2 = .06$ ,  $\eta^2 = .14$  reflect small, medium and large effect sizes (as cited in Stevens, 1996). Although  $\eta_p^2$  represents an overestimate of the actual effect size, it is a consistent measure and applicable to all  $F$  and  $t$  tests. Furthermore, in total sample sizes of 50 or more,  $\eta_p^2$  and  $\eta^2$  differ very little (Stevens, 1996). Due to a significant decrease in diary completion compliance during winter months, only the first 12 weeks (i.e, late September through mid-December) were

Figure 3.1 Frequency of Mood and Vegetative Symptom Onset for SAD-HX Group

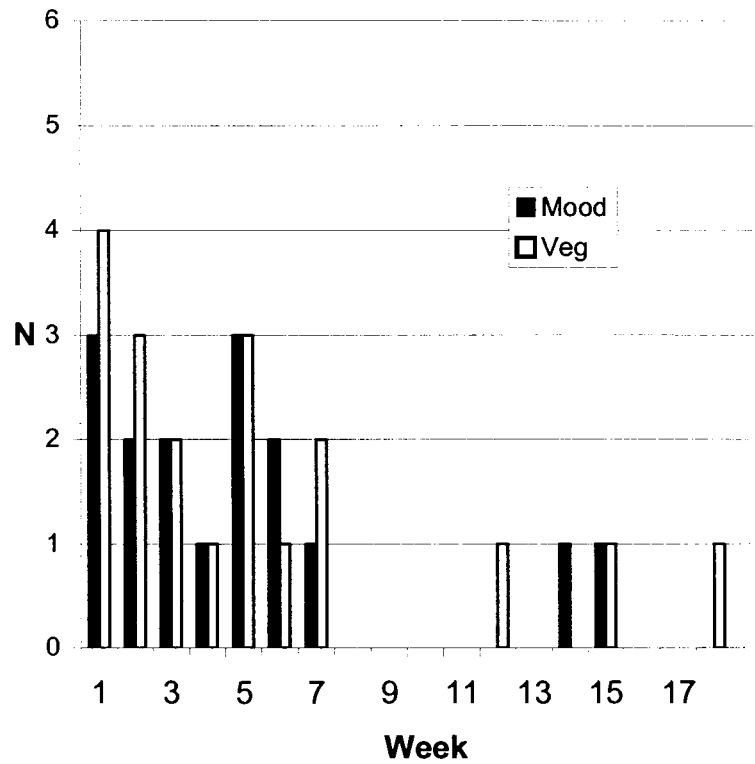


Figure 3.1 Frequencies of onset of mood and vegetative symptom clusters for SAD-HX group.

analyzed. This strategy also decreased the likelihood of introducing confounds due to the winter holidays. Only participants who completed all 12 weekly measurements were included in the longitudinal analyses. A MANOVA revealed significant effects for group,  $F(4,78) = 13.54, p < .001$ , effect size  $\eta_p^2 = .41$ ; Occasion,  $F(22,19) = 2.78, p < .05$ ;  $\eta_p^2 = .76$ ; and interaction of occasion by Group,  $F(44,38) = 1.93, p < .05$ ;  $\eta_p^2 = .69$ .

Results were further broken down by measure with planned repeated measures ANOVAs.

For Mood symptoms, there was not an interaction between Occasion and Group  $F(22,60) = 1.34, p = ns$ , nor a main effect for Occasion,  $F(11,44) = .87, p = ns$ , despite the large effect sizes  $\eta_p^2 = .33$  and  $\eta_p^2 = .30$ , respectively. There was an overall difference in mood symptom reporting between diagnostic groups,  $F(2,40) = 19.15, p < .001$ ;  $\eta_p^2 = .49$ .

Planned comparisons using the Bonferroni test, revealed that SAD-HX participants reported greater depressed mood than both WA and Control participants ( $p < .001$ ), but WA individuals in the group did not differ from Controls.

For Vegetative symptoms, a repeated measures ANOVA detected a significant interaction of Occasion x Group,  $F(22,60) = 1.77, p < .05, \eta_p^2 = .39$ . (see Figure 3.2 below). In a breakdown of the interaction, planned oneway ANOVAs demonstrated significant differences between groups at every week; week 1 was  $F(2,59) = 18.35, p < .001$ , week 2  $F(2,59) = 22.72, p < .001$ , week 3  $F(2,56) = 26.7, p < .001$ , week 4  $F(2,57) = 31.44, p < .001$ , week 5  $F(2,58) = 26.08, p < .001$ , week 6  $F(2,56) = 32.33, p < .001$ , week 7  $F(2,56) = 25.26, p < .001$ , week 8  $F(2,55) = 12.78, p < .001$ , week 9  $F(2,54) = 14.14, p < .001$ , week 10  $F(2,54) = 13.27, p < .001$ , week 11  $F(2,47) = 17.03, p < .001$ , and week 12  $F(2,45) = 13.82, p < .001$ . Further analysis with Bonferroni post-hoc tests ( $.05/12=.004$ ) revealed that at weeks 1, 2, 5, 6, and 7 SAD-HX individuals reported

Figure 3.2 Vegetative Symptoms by Group Across Fall Weeks

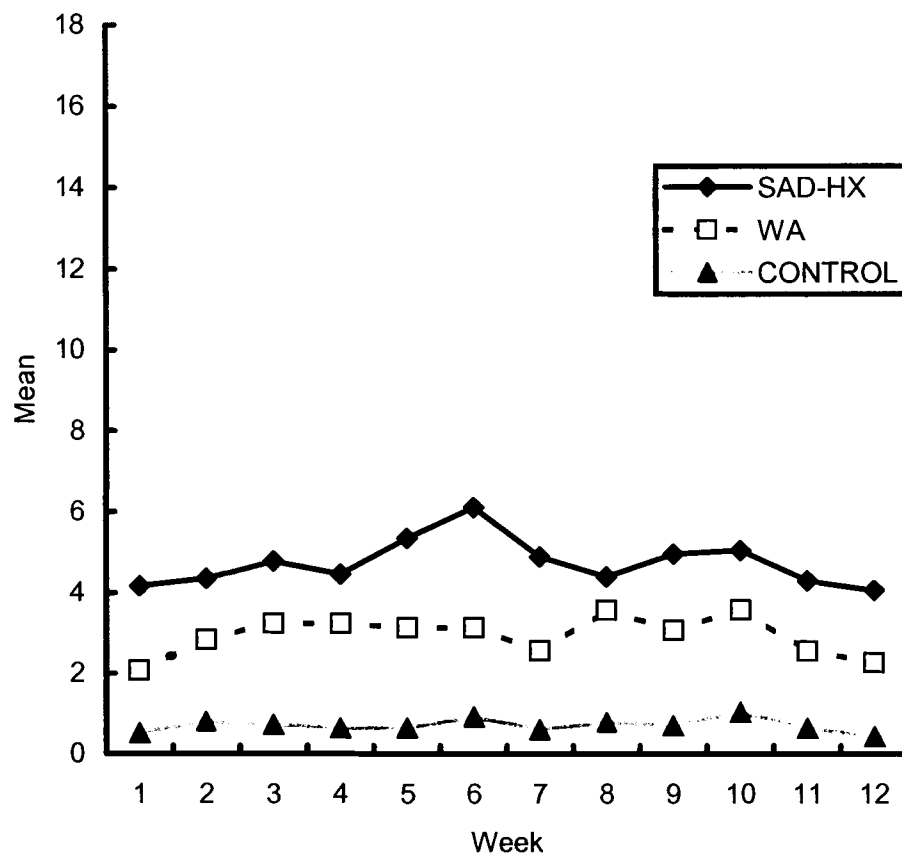


Figure 3.2. Interaction of occasion by group across fall weeks for vegetative symptom severity scores. At weeks 1,2,5,6, & 7 SAD-HX>WA>Controls. At weeks 3,4,8,9,10, & 11 SAD-HX,WA>Controls. At week 12, SAD-HX >Controls.



significantly more vegetative symptoms than the other two groups ( $p < .05$ ). During those same weeks WA participants reported more symptoms than Controls ( $p < .05$ ). At weeks 3, 4, 8, 9, 10, and 11 SAD-HX and WA participants reported more vegetative symptoms than Controls ( $p < .05$ ). At week 12, SAD-HX participants reported more vegetative symptoms than Controls ( $p < .001$ ).

Contrary to expectations, an examination of cognitive measures across the 12 weeks revealed no significant multivariate interaction  $F(22,58) = .974$ ,  $ns$ ,  $\eta_p^2 = .27$ , nor main effect for frequency of seasonal automatic thoughts for occasion using Wilk's  $\lambda$ ,  $F(11,29) = 1.68$ ,  $p = ns$ ,  $\eta_p^2 = .39$ . However, as expected, between-subjects analyses showed a significant group effect,  $F(2,39) = 30.98$ ,  $p < .001$ ,  $\eta_p^2 = .61$ . Planned comparisons using a Bonferroni test revealed SAD-HX participants reported significantly more automatic seasonal thoughts ( $M = 58.23$ ,  $SD = 2.94$ ) than WA ( $M = 35.33$ ,  $SD = 3.35$ ) or Control participants ( $M = 28.68$ ,  $SD = 2.43$ ;  $p < .001$ ). WA and Control participants did not differ significantly from each other.

Similar results were obtained for the RSQ diary version data. For rumination scores, a MANOVA detected a significant interaction for occasion x group,  $F(22,52) = 1.89$ ,  $p < .05$ ,  $\eta_p^2 = .44$  as expected. (See Figure 3.3 below). In a breakdown of the interaction, planned oneway ANOVAs demonstrated significant differences in rumination between groups at every week; week 1 was  $F(2,60) = 8.53$ ,  $p < .001$ , week 2  $F(2,58) = 24.69$ ,  $p < .001$ , week 3  $F(2,55) = 32.53$ ,  $p < .001$ , week 4  $F(2,57) = 31.82$ ,  $p < .001$ , week 5  $F(2,59) = 17.28$ ,  $p < .001$ , week 6  $F(2,54) = 27.70$ ,  $p < .001$ , week 7  $F(2,55) = 26.05$ ,  $p < .001$ , week 8  $F(2,55) = 20.73$ ,  $p < .001$ , week 9  $F(2,54) = 27.29$ ,  $p < .001$ , week 10  $F(2,54) = 31.44$ ,  $p < .001$ , week 11  $F(2,46) = 19.15$ ,  $p < .001$ , and week 12

Figure 3.3 Rumination by Group Across Fall Weeks

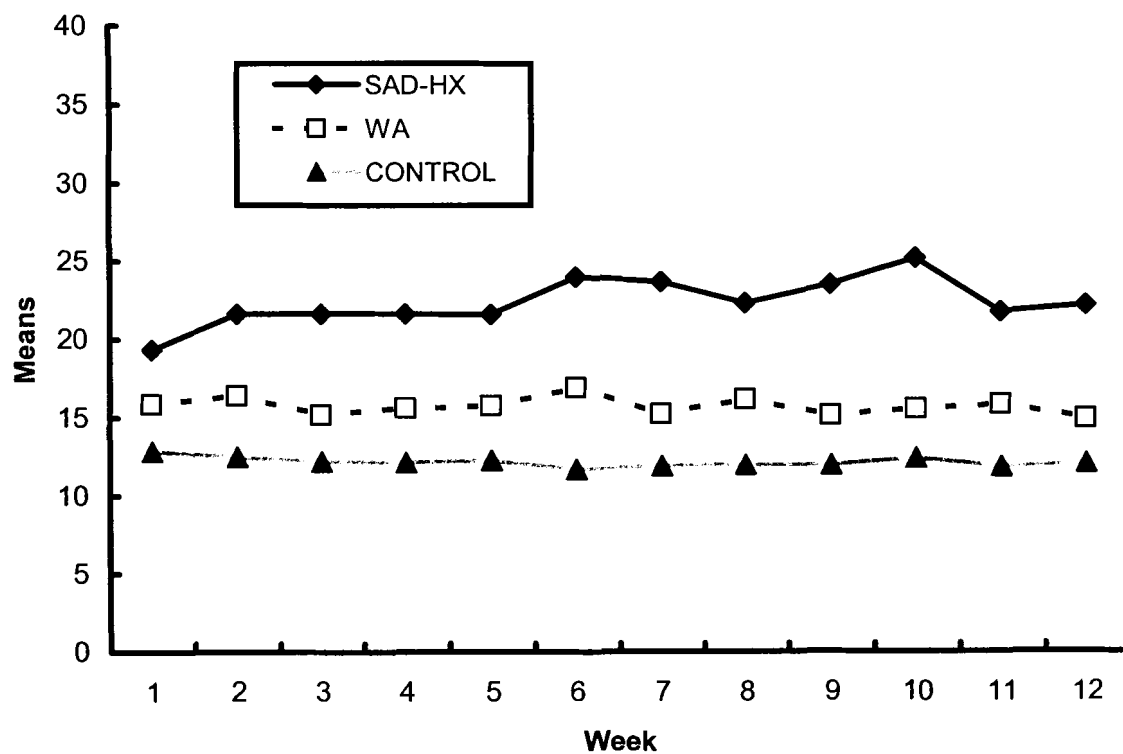


Figure 3.3. Interaction of occasion by group across fall weeks for rumination scores. At week 1 SAD-HX > Controls. At week 5 SAD-HX > WA, Controls. At weeks 2, 3, 4, 6, & 8 SAD-HX > WA > Controls. At weeks 7, 9, 10, 11 & 12 SAD-HX, WA > Controls.

$F(2,45) = 21.69, p < .001$ . Further analysis using Bonferroni test for multiple comparisons revealed that at week 1, SAD-HX participants reported more ruminative symptoms than Controls ( $p < .001$ ). At week 5, SAD-HX participants reported more rumination than WA or Controls ( $p < .01$ ). At weeks 2, 3, 4, 6, and 8, SAD-HX individuals reported significantly more rumination than the other groups and the WA group reported more rumination than Controls ( $p < .05$ ). At weeks 7, 9, 10, 11 and 12 SAD-HX and WA groups reported more ruminative symptoms than Controls ( $p < .01$ ).

Recognizing that not all of the SAD-HX group participants experienced a depression episode during the course of the study, the diary data for the SAD-HX group were further broken down in an exploratory analysis of the data. Means for individuals who developed a depressive episode during the study ( $n = 6$ ; SAD-E) and means for those individuals who did not develop a depressive episode during the study ( $n = 7$ ; SAD-H) were analyzed separately. Due to missing (i.e., unreported) data across diary collection, the sample size was very small which contributed to minimal statistical power.

Therefore, no significant differences were observed. However, examination of trends suggested that vegetative symptom reporting across the 12 weeks of fall was virtually identical for the two groups ( $p = .90, \eta_p^2 = .001$ ; see Figure 3.4 below). Conversely, SAD-E participants reported more mood symptoms than SAD-H individuals ( $p = .38, \eta_p^2 = .07$ ; see Figure 3.5 below). Examination of each subgroup of the SAD diagnostic group demonstrated that across weeks, SAD-E participants tended to report more depressed mood symptoms relative to vegetative symptoms ( $p = .48, \eta_p^2 = .08$ ), whereas this trend was reversed for the SAD-H participants although not significant ( $p = .21, \eta_p^2 = .28$ ). See Figures 3.6 and 3.7 below.

Figure 3.4 Fall Vegetative Symptom Means for SAD Groups

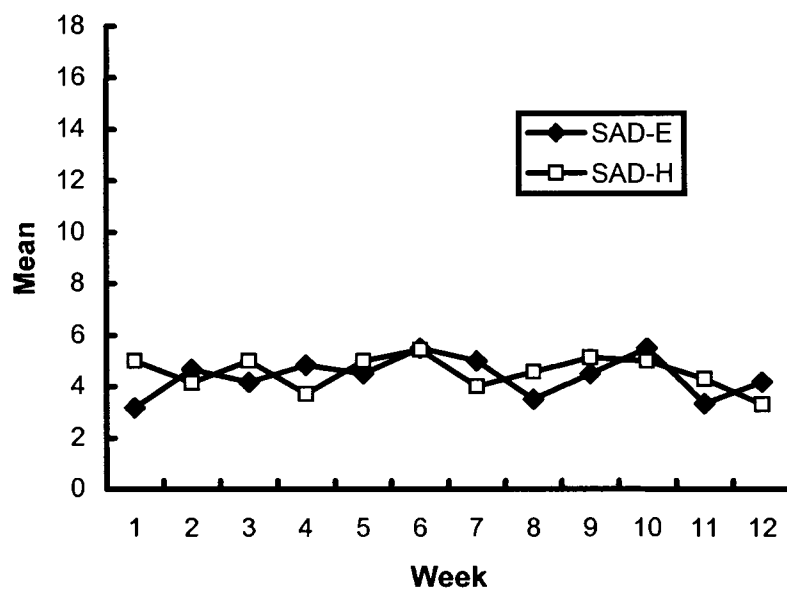


Figure 3.4 Vegetative means for SAD (SAD-E) and SAD history (SAD-H) groups across 12 weeks of fall symptom measurement.

Figure 3.5 Fall Mood Means for SAD Groups

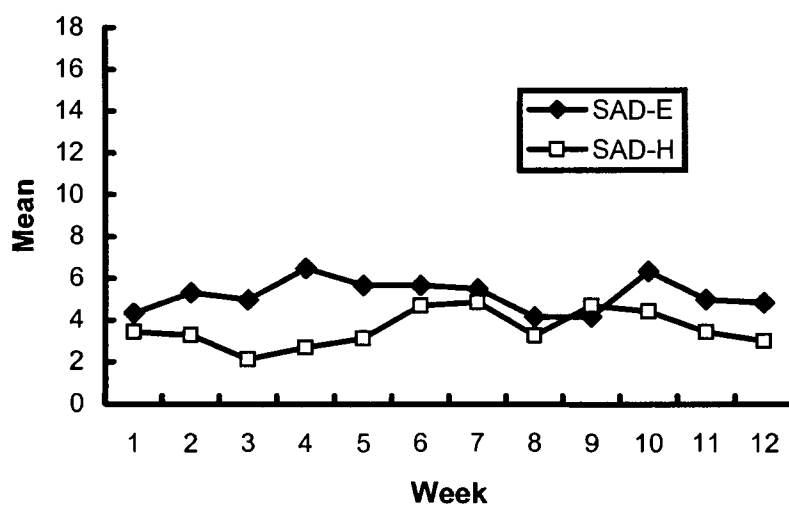


Figure 3.5. Vegetative means for SAD (SAD-E) and SAD history (SAD-H) groups across 12 weeks of fall symptom measurement.

Figure 3.6 Mood and Vegetative Symptoms  
for SAD Episode Group

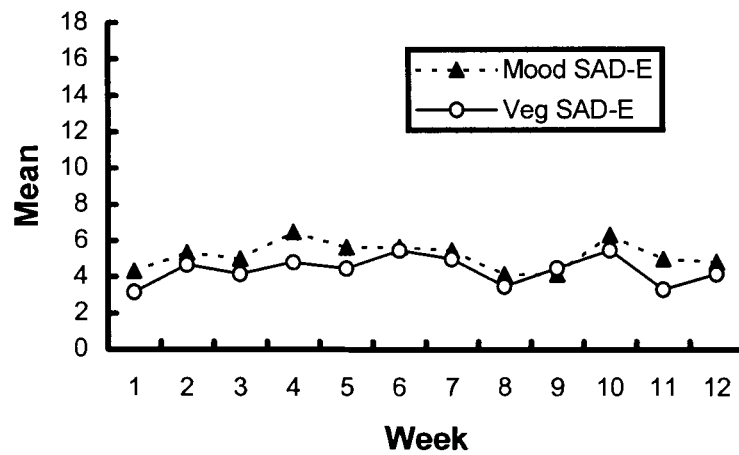


Figure 3.6. Mood symptoms relative to vegetative symptoms for those SAD group participants who developed an episode of winter depression during course of the study.

Figure 3.7 Mood and Vegetative Symptom Reporting  
for History of SAD Group

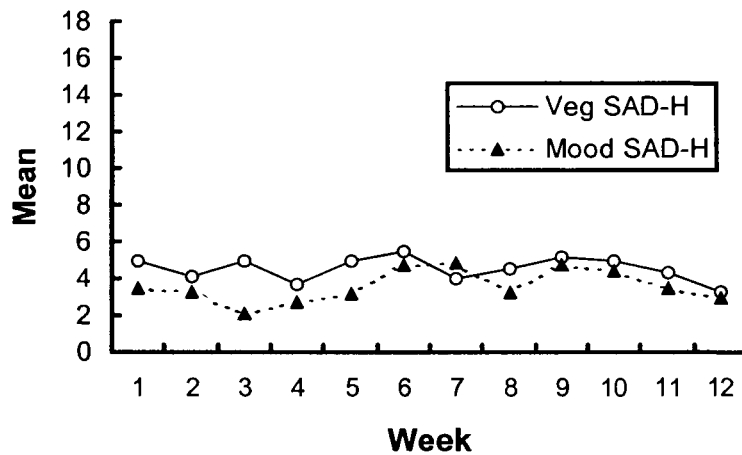


Figure 3.7. Mood symptoms relative to vegetative symptoms for SAD group participants who did not develop winter depression during the course of the study. Note: Figures 3.4-3.7 contain similar data but are presented separately to convey comparisons most clearly.

Although the sample is small, this finding suggests that relative symptom patterns may be predictive of developing a SAD episode later that year in those with a vulnerability to experiencing winter depression. It supports the hypothesis that mood and vegetative symptom onset are related to separate factors. This finding, however, does not support the hypothesis that mood symptoms appear as a reaction to vegetative symptoms.

### Hypotheses Two and Three

As expected, Pearson's  $r$  revealed a significant correlation between frequency of SAD-related cognitions (i.e., SATS and ATQ scores) and self-reported depression scores as measured by the modified BDI-II. At summer measurement, automatic seasonal and automatic negative thought scores were both significantly correlated with depression symptom scores,  $r = .76, p < .01$  and  $r = .80, p < .01$ , respectively. Similar results were obtained at winter measurement; depression scores were correlated with seasonal automatic thoughts,  $r = .84, p < .01$  and negative automatic thoughts  $r = .84, p < .01$ . Negative automatic thoughts and automatic seasonal thoughts scores were correlated with each other also, summer  $r = .72, p < .01$  and winter  $r = .76, p < .01$ .

Multivariate analysis of variance (MANOVA) using Wilk's  $\lambda$  was employed to assess differences between scores on negative automatic thoughts, seasonal automatic thoughts, and rumination (RRS) by group (SAD-HX, WA, Controls) and season (Summer and Winter). Analyses revealed the predicted significant main effects for season,  $F(3,57) = 3.66, p < .02, \eta_p^2 = .16$ ; and for group,  $F(6,114) = 13.6, p < .001, \eta_p^2 = .42$ ; and a marginal result for the interaction of group and season,  $F(6,114) = 2.16, p = .052, \eta_p^2 = .10$ . Means and standard deviations for group by season are listed in Table 3.2 below.

Planned univariate tests for within-subjects effects revealed a significant main effect for season of measurement on negative automatic thoughts as hypothesized,  $F(1,59) = 8.84, p < .005, \eta_p^2 = .13$ ; but not for seasonal automatic thoughts,  $F(1,59) = .97, ns, \eta_p^2 = .02$ ; or rumination,  $F(1,59) = .21, ns, \eta_p^2 = .003$ . There was also an interaction between negative automatic thoughts and season,  $F(3,59) = 6.43, p < .005, \eta_p^2 = .18$ , as predicted. Negative automatic thought scores increased overall from summer to winter across groups, but most noticeably in the SAD-HX group. See Figure 3.8 below. This pattern was also predicted for seasonal automatic thoughts and rumination, but was not supported by these findings.

Between subjects analyses revealed significant differences between groups on all three measures; negative automatic thoughts,  $F(2,59) = 26.9, p < .001, \eta_p^2 = .48$ ; seasonal automatic thoughts,  $F(2,59) = 31.5, p < .001, \eta_p^2 = .52$ ; and rumination,  $F(2,59) = 29.7, p < .001, \eta_p^2 = .50$ . Planned comparisons were calculated using the Bonferroni post-hoc test. Collapsing across season of measurement, SAD-HX participants' seasonal automatic thoughts and rumination scores ( $M_{SATS} = 53.7, SD = 17.2$ ;  $M_{RRS} = 27.1, SD = 8.5$ ) were significantly greater than WA ( $M_{SATS} = 36.2, SD = 11.1$ ;  $M_{RRS} = 15.2, SD = 9.6$ ), and Control participants' scores ( $M_{SATS} = 28.4, SD = 5.2$ ;  $M_{RRS} = 8.5, SD = 9.1$ ) and WA individuals' scores were significantly higher than Control individuals' scores. SAD-HX participants' scores on a measure of non-seasonal automatic negative thoughts ( $M_{ATQ} = 52.2, SD = 16.1$ ) were higher than WA ( $M_{ATQ} = 36.4, SD = 6.0$ ) and Control ( $M_{ATQ} = 33.2, SD = 4.6$ ) participants' scores, but WA and Controls did not differ from each other (see Table 3.2 below).

Table 3.2

Means for Cognitive Self-Report Measures by Group

Season	Measure	Group	N	Mean	Std. Deviation
Summer	ATQ	SAD-HX	20	47.6	15.2
		WA	18	36.4	7.2
		CONTROL	24	32.8	3.6
	SATS	SAD-HX	20	50	17.5
		WA	18	36.9	10.5
		CONTROL	24	28.5	5.4
	RRS	SAD-HX	20	26.5	7.5
		WA	18	15.4	9.7
		CONTROL	24	9.0	8.7
Winter	ATQ	SAD-HX	20	57.1	16.9
		WA	18	36.6	4.6
		CONTROL	24	33.7	5.4
	SATS	SAD-HX	20	55.4	15.9
		WA	18	36.2	12
		CONTROL	24	28.4	5
	RRS	SAD-HX	20	28.4	9.9
		WA	18	15.7	9.4
		CONTROL	24	8.3	9.6

Note: Means for each diagnostic group on self-report cognitive measures for summer and winter assessments. Cognitive measures assessed automatic negative thoughts (ATQ), seasonal automatic thoughts (SATS) and rumination (RRS).



Figure 3.8 Negative Automatic Thoughts by Group Across Season

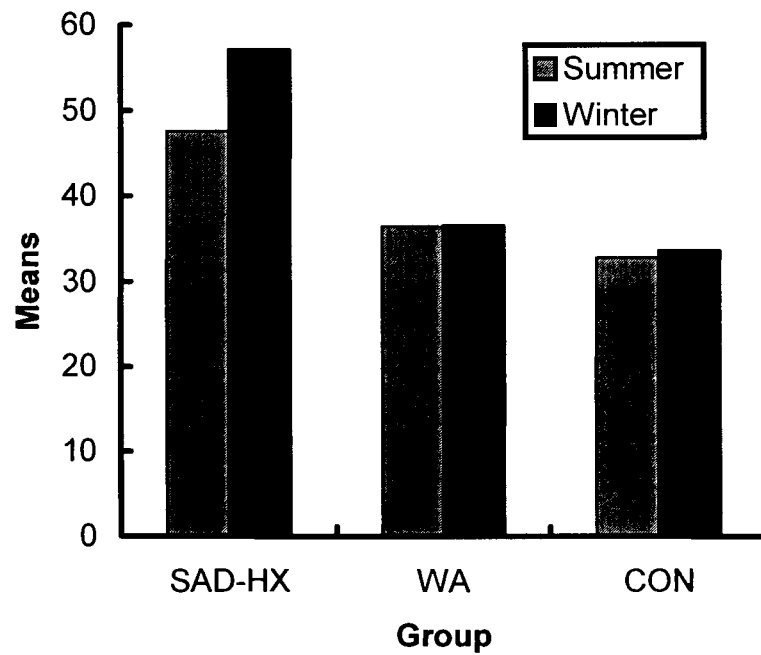


Figure 3.8. ATQ Means for SAD-HX, WA, and Control group participants across summer and winter measurements. SAD-HX group means are significantly different,  $t = -3.0$ ,  $p < .003$ .

### Hypothesis Four

Stroop results were analyzed by F tests with MANOVA using Wilk's  $\lambda$ . Overall, results indicated the expected main effect for word-type,  $F(4,56) = 6.25, p < .001, \eta_p^2 = .31$ . Planned t-tests with Bonferroni correction ( $.05/10 = .005$ ) revealed that across seasons and group, participants were slower to color-name when presented with negative words compared to light-related words,  $t(1,128) = 3.68, p < .001$ , or control stimuli (XXXXX),  $t(1,128) = 3.32, p < .001$ . In addition, neutral words elicited longer response latencies than control stimuli,  $t(1,128) = 3.18, p < .001$ , or light-related words  $t(1,128) = 3.18, p < .001$ . Marginal effects were also found for season,  $F(1,59) = 3.37, p = .071, \eta_p^2 = .05$ , and for interaction of wordtype x season x group,  $F(8,112) = 1.84, p = .077, \eta_p^2 = .116$ . Between subjects analyses revealed significant differences between groups overall,  $F(2,59) = 6.78, p < .01, \eta_p^2 = .19$ , as expected. Across season and wordtype, the SAD group had longer response latencies than WA ( $p < .01$ ) and Controls ( $p < .05$ ).

However, Stroop latency has been shown to decrease with age after 30-39 years of age (Uttl & Graf, 1997), an effect attributed to the general slowing seen with age (Verhaeghen & De Meersman, 1998). As noted above, there were significant differences in age across group, and when age was added as a covariate, no significant differences for the relevant variables were observed. There was a significant effect for age,  $F(1,57) = 18.33, p < .001, \eta_p^2 = .24$ . Effect sizes for the variables of interest, were small to medium (Cohen, 1977). Effect sizes for simple effects were  $\eta_p^2 = .065$  for wordtype and  $\eta_p^2 = .035$  for season. For interactions, effect sizes were as follows; wordtype x group,  $\eta_p^2 = .059$ , season x group  $\eta_p^2 = .003$ , wordtype x season,  $\eta_p^2 = .087$ , wordtype x season by group,  $\eta_p^2 = .107$ .

### Hypothesis Five

Several cognitive variables were examined for their utility in predicting severity of subsequent depressive episodes in individuals with a history of SAD (i.e., SAD-HX group). Summer measurement of seasonal automatic thoughts, rumination, and Stroop latencies for each of the 5 word-types (i.e., control XXXXX's, neutral, depressotypic, light-relevant, and season-relevant words) were entered into a stepwise linear regression model in order to determine the predictive utility of these variables for winter depression severity measured by winter SIGH-SAD scores. Results of the stepwise multiple regression analysis indicated that contrary to predictions, only rumination and seasonal automatic thoughts were significant predictors of depression symptom severity the following winter. Results are presented in Table 3.3 below. Rumination (i.e., RRS) accounted for 33.5% of the variance and both measures together accounted for 45.8% of the variance in winter SIGH-SAD scores across groups.

Table 3.3

Stepwise Regression Analyses for Predicting Winter Depression Symptom Severity

Variable	R	R <sup>2</sup> Δ	β	FΔ
RRS	.58	.34	.41**	30.2**
SATS	.68	.12	.40*	13.4*

*Note.* Results for stepwise regression analyses predicting winter depression symptom severity as measured by Structured Interview Guide for the Hamilton Rating Scale for Depression—Seasonal Affective Disorder version (SIGH-SAD). Note: RRS = Ruminative Response Subscale of the Response Styles Questionnaire. SATS = Seasonal Automatic Thoughts Survey. \*\* $p < .001$ , \* $p < .01$ .

## Chapter IV

### DISCUSSION

This prospective study assessing mood symptoms, vegetative symptoms, and cognitive factors in individuals with varying degrees of seasonal symptomatology was designed to test Young's (1991) Dual Vulnerability Hypothesis (DVH). According to the DVH, individuals must possess a biological vulnerability for developing vegetative symptoms and a psychological vulnerability for developing mood symptoms of depression in order to develop Seasonal Affective Disorder (SAD) episodes (Young et al., 1991). In general, the results of the present study provide support for the DVH.

#### Hypothesis One

As expected, participants in the SAD-HX group (i.e., all participants with a history of SAD episodes, regardless of whether they developed a winter depression episode during the course of the study) demonstrated significantly more depressed mood and vegetative symptoms across the fall compared to the other groups. The findings that the SAD-HX group reported more vegetative symptoms than the winter anergia (WA) group suggests that the SAD-HX and WA groups differ not only in terms of a depression factor, but also differ in the degree to which a seasonal (vegetative) factor is expressed.

The expression of the vegetative factor may be influenced by the effects of mood, or factors related to depressed mood (i.e., rumination) that may then affect the experience of vegetative symptoms in individuals with SAD as compared to WA. For example, frequently attending to or ruminating about vegetative symptoms and their causes may cause one to experience the symptoms more often or more severely than a strategy of thinking about other topics or distracting which may lead an individual to experience

symptoms less frequently or less severely. Individuals with a history of SAD, due to their prior experience with depression episodes, may be more likely to linger on the implications of vegetative symptoms. Furthermore, it has been recently suggested that rumination (about vegetative symptoms) may represent a depression vulnerability related to recurrent SAD episodes. For example, Young & Azam (2003) conducted a longitudinal study investigating the relation of SAD symptoms and rumination in 18 individuals with a history of SAD. The researchers found a significant interaction between fall vegetative symptoms and ruminative response style that predicted severity of winter non-vegetative depressive symptoms. Unfortunately, the authors did not examine whether a ruminative response style was predictive of vegetative symptom experience during the winter.

Another explanation for the differential intensity of vegetative symptoms is that individuals in the SAD-HX group have a history of developing SAD symptoms and therefore, may be more sensitized to and aware of changes in vegetative symptoms than individuals in the other groups. This increased sensitivity could produce more accurate ratings for the SAD-HX than the WA group or could result in over-reporting of vegetative symptoms in the SAD-HX group.

Alternatively, individuals in the SAD-HX group may be more susceptible to social desirability responding. For example, individuals with a history of SAD are probably aware of common SAD symptoms, identify with the diagnosis, and respond to study requests for volunteers with SAD. In contrast, the WA group participants were recruited through symptom pattern screening and may not identify with the descriptor of WA or be aware of the specific symptoms that researchers were examining. Therefore, it is possible that participants in the SAD-HX group may have felt more compelled to

report the symptoms and intensity of symptoms that someone with SAD “should” experience, compared to the WA group participants who may know little about seasonal vegetative symptoms.

Moreover, it is unclear how representative the individuals in the WA group are of the WA population because this group has not been systematically studied. It may be that individuals in the WA group in this study had less severe vegetative symptoms than those in the overall WA population. Additional considerations regarding the categorization of the WA group are discussed in more detail later.

In addition to finding differences in symptoms between diagnostic groups, there was a different relational pattern of depressed mood and vegetative symptoms observed in the SAD-HX group. This pattern, however, was dependent on whether the individuals developed a depressive episode later that winter or not. The SAD-HX group participants who developed a winter depression (SAD-E) reported slightly more depressed mood symptoms relative to vegetative symptoms across the fall, whereas those who did not develop a depressive episode (SAD-H) during the course of the study reported relatively more vegetative symptoms than mood symptoms. This differential pattern of reporting between the SAD-E and SAD-H supports the DVH proposal regarding separate independent vulnerabilities. Due to the small sample size in this study, however, there was not sufficient power to detect significant differences between the SAD-HX subgroups for mood and vegetative symptom reporting. Although any conclusions about this finding must be made with caution due to the small sample size, these preliminary results suggest that the relational pattern of symptoms even early in the fall may be

indicative of the likelihood of a subsequent SAD episode to follow later that fall or winter. Further study of this relational pattern of symptoms is warranted.

The DVH further posits that vegetative symptoms cluster together at an earlier temporal onset than mood symptoms (Young et al., 1991). A recent longitudinal study following changes in depression and anxiety symptoms also found a pattern of vegetative symptoms emerging earlier than mood symptoms (McCarthy et al., 2002). The researchers collected BDI-II data from September 1 to March 31 from participants with a history of SAD episodes for the subsequent onset of vegetative symptoms of sleep, fatigue, and appetite. Results indicated median survival times to be 2 weeks, 2 weeks, and 4 weeks, respectively. In comparison, median survival times for self-dislike (6 weeks), self-criticism (4 weeks), and worthlessness (4 weeks) were later.

In the present study, prospective measurement of SAD symptoms did demonstrate the expected temporal relation of vegetative symptom onset prior to mood symptom onset, albeit smaller (i.e., less than one week) than the differences in onset time previously observed (McCarthy et al., 2002; Young et al., 1991). Although these differences may be due to the smaller sample size in the present study, methodological differences may also play a role. For example, McCarthy and colleagues (2002) collected data bimonthly, rather than weekly, allowing for less specificity in measuring onset (i.e., they could not determine temporal differences of less than 2 weeks apart). In addition, individual symptoms were examined, which may be less reliable measures than composite scales (Anastasi & Urbina, 1997) comprising of several mood and vegetative symptoms. Furthermore, their criteria for symptom onset were less stringent (onset was endorsement of any single symptom at any degree of severity).

Methodological differences are also apparent between the present study and Young and colleagues' (1991) earlier work (upon which the DVH was based), which utilized a retrospective methodology. In the Young and colleagues (1991) study, participants experiencing SAD episodes were asked to recall the order of symptom onset, which in some cases was several months prior to the data collection. Thus, differences in symptom onset patterns across the current and previous symptom onset studies would likely be attributable to different methodology.

It is also possible that the 4-point symptom rating scale of the modified BDI-II was too truncated to pick up more subtle changes in individual vegetative and mood symptoms that would better differentiate symptoms measured at shorter time intervals. The BDI, however, was chosen for its proven reliability and validity, as well as the inclusion of items of interest that could be used to construct face valid scales for mood and vegetative symptoms. Previous longitudinal research with the full modified BDI-II found that individuals with a history of SAD scored significantly higher in the winter than in the fall or summer and also higher in the fall than the summer (Rohan, Sigmon & Dorhofer, 2003). Future research might include the development and validation of an instrument designed for longitudinal research of SAD that offers participants greater choice in responses to describe the experience of symptom severity.

Another reason that this study may not have found as strong a temporal pattern as Young et al. (1991) is reflected in the difference in how symptom onset was operationalized. Young and colleagues (1991) asked SAD participants to determine the start date of their current depression episode and measured symptoms from that time point, whereas the current study set a cutoff score for significant degree of symptoms to



determine symptom onset. Thus, symptom onset was not necessarily related to a depression episode. This method, however, allowed for comparison of experience of symptoms across season by group (i.e., WA and Controls do not have depression episodes by which to measure start of symptoms) regardless of diagnosis. It is also acknowledged that this strategy may capture changes in symptoms that SAD individuals would not attribute to SAD episode onset. The disparate findings may also be attributed to the difference in collecting data prospectively versus relying on memory for retrospective data collection.

### Hypotheses Two & Three

As noted above, the DVH hypothesizes that the subsequent onset of mood symptoms in SAD is related to a psychological vulnerability. This study sought to examine several cognitive factors as possible mediators of depressed mood and recurrent depression episode onset in SAD.

Significant correlations were observed between depression symptom scores and the frequency of seasonal and non-seasonal depression related cognitions. The results support continued validity testing for the Seasonal Automatic Thoughts Survey (SATS) which had not been used with a clinical sample prior to the present study. The current study demonstrated that the SATS was able to differentiate between SAD-HX, WA and Controls. Furthermore, these findings support a role for investigating seasonal thought frequency in the recurrence of SAD episodes.

Due to its predictable fall-winter onset and spring-summer remission of symptoms, cognitions in the form of depressive expectations and negative automatic cognitions about the changing seasons and one's self-efficacy to manage symptoms are

particularly suited to contribute to the development of subsequent SAD episodes.

Although self-efficacy was not explicitly explored in this study, items on the SATS address belief in the ability to cope with changes (e.g., “There is nothing I can do to stop depression from coming”, “I can’t control my food cravings”). A tendency to frequently experience negative thoughts about the seasons and self appear to contribute to a vulnerability for developing recurrent seasonal episodes of depression. Indeed, previous longitudinal research has found that low self-esteem was associated with faster onset of depressive symptoms in individuals with a history of SAD (McCarthy et al., 2002).

A significant correlation was also obtained for rumination scores and depression symptoms. These findings also lend continued support to cognitive theories of depression (i.e., Beck, 1967; Nolen-Hoeksema, 1987) and the role that cognitive factors may play in SAD, similar to their role in non-seasonal depression. Researchers that have compared individuals with seasonal and non-seasonal depression have also found similar levels of depressive cognitions (Hodges & Marks, 1998; Sigmon, Whitcomb-Smith, Kendrew, & Boulard, 2003) and negative attributional style (Levitan et al., 1998). Of course, correlational findings should not be used to imply that cognitive factors cause depression episodes. However, these findings add to the literature supporting a role for negative cognitions in SAD as has been suggested for non-seasonal depression episodes.

#### Hypothesis 4

The Stroop task was included to assess for differential attentional bias across groups and across SAD relevant versus control stimuli. Recent studies have found that both SAD and nonseasonal depressed groups had longer latencies for depressotypic and darkness-related words (e.g., Sigmon et al., 2003) than controls. In a recent study,

individuals with SAD evidenced an increased latency to respond to depressive and season-related words as compared to control stimuli (Spinks & Dalgleish, 2001). In the present study, however, significant differences in age were observed between the groups on the modified Stroop task. As a result, age confounds may have accounted for the differences measured across the groups. Unfortunately, due to low rates of participant recruiting, age and education matching of participants was not possible.

Unlike previous studies, the current study presented groups of words in blocks by word-type, rather than in random order. The blocks were presented in a fixed order such that the more SAD-relevant blocks (i.e., depressotypic and season-relevant) were presented last. This method was used to avoid Type II error related to practice effects. However, it may be that participants responded more quickly to the later stimuli as a result of practice effects, such that differential responses to the experimental stimuli could not be assessed.

### Hypothesis 5

Although the cognitive factor of attentional bias (i.e., Stroop results from summer measurement) was also not predictive of winter SAD symptom severity, summer scores for rumination and seasonal automatic thoughts were shown to be significant predictors of winter SAD symptom severity. Previously, researchers found that fall rumination scores predicted severity of winter depression symptoms in women with a history of SAD (e.g., Young & Azam, 2003) and was a better predictor than fall depressive symptoms as measured by the BDI-II (Rohan, Sigmon, & Dorhofer, 2003). It is recognized, however, that these cognitive variables cannot be ruled out as consequences of previous winter

depression experience. Therefore, these factors may be important to investigate with respect to the development and maintenance of recurrent episodes.

Unfortunately, the SAD-HX group size was too small to examine cognitive variables between those who went on to meet criteria for a depressive episode and those who did not. Examining the predictability of various cognitive factors across individuals with a history of SAD for development of subsequent winter depressive episodes represents an excellent area for future research. According to the DVH, individuals with SAD possess a psychological vulnerability to develop depression. This study lends support to the growing literature suggesting that cognitive factors may represent at least one component of the psychological vulnerability for developing recurrent SAD episodes (e.g., Rohan, Sigmon, & Dorhofer, 2003; Young & Azam, 2003).

### General Discussion

The DVH also details the need for a predisposition to developing season-related vegetative symptoms in addition to having the predisposition for depression for an individual to experience SAD episodes. It is assumed that individuals may possess only one (i.e., either the biological vulnerability or the psychological vulnerability) of the hypothesized vulnerabilities. Those with only a psychological vulnerability would be predisposed to non-seasonal episodes of depression. Although not previously an area of published study, extrapolating from the DVH suggests that there are also individuals who may be predisposed only to develop vegetative symptoms. Prior to this study, retrospective self-report questionnaire data from a convenience sample supported the existence of such a group (Boulard & Sigmon, 1999), but no reported studies prior to the

present study directly recruited and studied characteristics of this subgroup of sub-syndromal SAD (S-SAD).

By inclusion of the winter anergia (WA) group, this study served to support validation of the descriptor of winter anergia. Participants were recruited that met the criteria for significant seasonal vegetative symptoms with minimal seasonal mood symptoms. Results of this preliminary exploration of WA suggest that this group is worthy of further attention. Across the fall measurement, WA group mood scores were more similar to the Control group than to individuals in the SAD-HX group. In addition, vegetative symptom scores across the fall for the individuals in the WA group were significantly higher than Control group participants', and were frequently comparable to vegetative symptom levels in SAD-HX participants. On cognitive measures, WA group participants were more similar to Control group participants than SAD-HX group participants on a measure of non-seasonal depressive automatic thoughts. WA group participants scored lower on the seasonal automatic thoughts than SAD-HX and higher than Controls. Similar, unexpected results were also found for rumination, which might suggest that WA may represent a risk factor for developing SAD.

It must be noted that these findings on winter anergia are preliminary in nature and conclusions drawn from this sample must be made with caution. Of primary consideration is the reliability of the categorization of individuals with WA. Inclusion criteria for this group were primarily based on only 2 GSS items (i.e., mood and social activity). In addition to the problem of likely low reliability of essentially a two-item scale, one could argue social withdrawal is a vegetative symptom. However, social

activity is generally included as a mood symptom in the SAD literature (e.g., Sigmon et al, 2001; Young, 1991).

Furthermore, one might be tempted to conclude that due to the younger age of this group, combined with the rumination scores intermediate to SAD-HX and Controls, WA could serve as a precursor to the initial development of SAD. Therefore, individuals with WA participants would be vulnerable to developing SAD later in life. However, such generalizations about prevalence and demographics taken from this sample may be premature. This study was not designed to examine prevalence and demographic characteristics and the sample size was small, even compared to the other groups in the study.

Recruitment for both SAD-HX and WA group participants was difficult given that the experimenter was trying to enroll participants during the summer who had problems with winter symptoms. However, WA recruitment proved to be even more difficult. One possibility is that WA does not represent a symptom profile that most individuals identify with; it is not a commonly known term or condition. Despite efforts to recruit from the community, sample inclusion was limited to the undergraduates who were screened for winter anergia symptoms using the SPAQ and invited to participate for academic credit. This limitation on recruitment, however, contributed to age and education confounds. In addition, the WA group in the present study is likely to be more homogeneous than the other groups on factors not measured (e.g., activities, lifestyles, goals, etc.). These individuals may also differ from the other groups in terms of motivation, reasons for participating, and interest in the process of participating in the study. All of these factors could differentially affect results involving WA data.

Despite these limitations, the current study's findings, including results from the WA group, further support the DVH. As hypothesized, WA group individuals were similar to Controls on measures of mood symptoms, rumination and automatic thoughts about the seasons. Furthermore, participants in the WA group reported significantly more vegetative symptoms than Controls as hypothesized. WA group individuals, however, also reported significantly less vegetative symptoms than SAD-HX participants. It is not clear if severity of vegetative symptoms may play a role in the initial development or recurrence of SAD episodes or if the reports of individuals in the SAD-HX group might be elevated by sensitivity to vegetative symptoms through previous experience or through current depressive mood experience.

It should be noted that the criterion for the WA group individuals also falls within the traditional definitions and cutoff scores for global seasonality scores of individuals categorized as S-SAD. Because individuals in the WA group were only compared to individuals in the SAD-HX group and to never-depressed individuals in the Control group, this study cannot address how participants with WA differ from those classified as S-SAD or whether it should be considered a category distinct from S-SAD. Considering WA as a distinct category would yield a categorical view of seasonality from no changes to winter anergia to S-SAD to SAD. This categorical view, however, is typically applied as though an individual possesses the characteristics to fit into a given category as though it were a trait (e.g., questionnaires ask for "usual experience with the seasons"). Thus one "has SAD" or "has S-SAD." However, fluctuations are noted in the literature such that individuals with a history of SAD may be categorized as S-SAD during some winters (e.g., Rohan, Sigmon, & Dorhofer, 2003). Although categorical variables have heuristic

importance, the inclusion of a continuum view is also important in order to obtain a clearer picture of SAD and seasonality levels in the general and clinical population.

Recently, the DVH has been “revisited” to include a continuum in conjunction with the categorical view (Lam et al., 2001). According to Lam and colleagues (2001), individuals may have different degrees of loadings on seasonality (i.e., vegetative symptom vulnerability) and depression factors wherein each interacts with environmental and/or other external variables to produce variable expressions of symptomatology. The authors suggest that when an individual experiences the symptoms that mostly load on the seasonality factor, and has few or no symptoms that load on the depression factor, S-SAD represents the result. This explanation may account for why those individuals with a history of SAD do not develop winter depressions every year (e.g., interaction with external variables). In addition, the modified DVH was posited to explain how individuals with SAD have incomplete remission of SAD symptoms during the summer and possibly why they are less responsive to light therapy (i.e., higher loading on depression factor than “pure SAD”). Lam and colleagues (2001) further argue for the consideration of seasonality as a dimension separate from the category of SAD.

The findings of the current study are also consistent with Lam and colleagues (2001) modification of the DVH. The differences between groups on mood and vegetative symptoms support the idea of independent vulnerabilities and the variation within groups (i.e., not all SAD-HX participants developed a SAD episode during the course of the study) supports the idea of differential loadings and/or interaction with external factors. The inclusion of the WA group demonstrates that individuals may experience significant seasonal changes without any history of SAD or depression



episodes. Using the modified DVH formulation, WA individuals would fall under the label of S-SAD, but have essentially no symptoms that load on the depression factor, whereas the more traditional definition of S-SAD would include low levels of symptoms loading on the depression factor. Furthermore, research is needed to address how external factors influence the expression of seasonality and depression variables in SAD.

Taken together, the results of this study generally support the DVH, and the role of cognitive factors as at least part of the hypothesized psychological vulnerability that individuals with SAD possess. Although not a large difference, vegetative symptoms were observed to have an earlier temporal onset than mood symptoms in individuals with a history of SAD. Individuals in the Control and WA groups demonstrated less rumination and negative automatic thoughts than individuals in the SAD-HX group, and were significant predictors of winter SAD symptom severity.

### Limitations

The reliance on self-report measures reflects a major limitation of the study. An individual's responses can be affected by social desirability and expectations as well as by an individual's memory bias. Therefore, self-report measures can be less reliable than other methods of measurement (e.g., physiological measurements). However, there is no other way to directly measure specific cognitions or the experience of many symptoms other than self-report of some kind (i.e., even clinical interviews include a large degree of self-report, albeit filtered by clinical judgment). Furthermore, other experimental methods (i.e., observation, collateral data collection) are also limiting (i.e., in terms of intrusiveness to participants, increase in personnel/time of researchers to collect data, exclusive of those without available/willing collaterals). To combat the possible biasing

effects of using self-report measures alone, this study used a prospective, as well as retrospective, methodology in addition to other methods (i.e., retrospective structured interviews, Stroop task).

Another limitation involving the measures represents the period of time that symptoms were assessed. In this study, only the first 12 weeks (i.e., September through December) were used to assess mood and vegetative symptom onset. It is possible that using this time frame did not capture the onset of winter depressive symptoms for all of the participants. Some participants may not develop depressive symptoms until January or February or even later. However, a significant number of participants reported significant symptoms starting as early as September. Because the study design did not call for repeated diagnostic evaluations to pinpoint episode onset, it is not clear if the symptoms reported represent the start of winter depression episodes much earlier than individuals with a history of SAD notice.

A further methodological limitation of the study represents the use of the Mood and Veg symptom subscales derived from the Beck Depression Inventory-II. These scales, although representative, were not exhaustive collections of possible SAD mood and vegetative symptoms. Indeed, many of the symptoms assessed by Young and his colleagues (1991) in the original proposition of the DVH were not included in these scales (e.g., psychomotor agitation, suicidal ideation, social withdrawal). Examination of the relative onset for each and every possible SAD symptom may yield slightly different results, but it would need to be synthesized in order to make meaningful use of the enormous number of results (i.e., comparing 20+ symptoms across three groups). In the current study, creating scales of common SAD symptoms was one way to meaningfully

synthesize the data. Furthermore, although the choice of items was made rationally and was informed by theory and clinical experience, the inclusion or exclusion of alternative items in either mood or vegetative subscales may have resulted in different findings.

The small sample size reflects another significant limitation of the study. This limitation was particularly a factor in the modified Stroop analyses and longitudinal data. Small sample sizes created low power to detect differences with the smaller effect sizes. Even with very large effect sizes, 16 participants are needed to detect differences for three groups in multivariate analyses (Stevens, 1996). Due to missing data these group  $n$ 's were not always large enough for adequate power.

Related to sample size, the proportion of individuals in this study with a history of SAD who developed a SAD episode during the fall or winter (50%) was lower than reported in previous studies (e.g., Rohan, Sigmon & Dorhoffer, 2003; Schwartz, Brown, Wehr & Rosenthal, 1996). One possible explanation for the lower rate in the current study may be due to the weekly self-monitoring of depression symptoms and cognition. Self-monitoring has been shown to affect individual's behavior and mood in positive directions (Harmon, Nelson, & Hayes, 1980; Kazdin, 1974; Nelson, Boykin & Hayes, 1982; see also Nelson & Hayes, 1981). By completing the diary questionnaire packets over several months, participants may have been more aware of their mood, thoughts, and behavior. Thus, some participants may have made adjustments to improve mood or unhelpful cognitive patterns such that they were able to reduce symptomatology to subsyndromal levels. Alternatively, some participants had participated in previous SAD research projects ( $n = 8$ ) and may have been more educated about their disorder and ways

to cope with SAD than other participants. Despite the lower rate of SAD episode development in the current study, the longitudinal data provided important findings.

In addition, there was significant homogeneity of the sample in terms of gender (i.e., mostly women), race (i.e., almost entirely Caucasian) and diagnosis (i.e., exclusion of Axis I disorders). Although women comprise the majority of SAD sufferers, the proportion of women in this study was higher than in the SAD population in general (Eagles, Mercer, Boshier & Jamieson, 1996; Kane & Lowis, 1999; Kasper et al., 1989; Lee & Chan, 1998). Although the sample was mostly Caucasian, it is not unrepresentative of the community in Northern Maine. Homogeneity with respect to the WA sample was discussed above (e.g., age, education, lifestyle) as a limitation of the findings relating to the population representativeness of the WA sample. Such homogeneity of the WA sample, as well as the overall study sample limits the generalizability of the results to the population as a whole. Although limits on comorbidity are necessary when attempting to detect effects attributed to SAD, it also limits generalizability to real world settings where psychiatric comorbidities, especially with depressive disorders, are common (e.g., Maser, Weise & Gwirtsman, 1995).

Furthermore, it should be reiterated that the definition issues for WA elaborated on above represent limitations for this study. In the current study, a diagnosis of WA relied on GSS scores alone (and the SCID to rule out history of depression and exclusionary criteria). Diagnosis according to 6 typical symptoms item responses is likely not a reliable diagnostic indicator. However, this is the first study to attempt to define and recruit WA participants. As such, it supports the validity of such a diagnostic

group and may serve as a catalyst for future work on reliably defining and examining characteristics of this group.

Several confounds also limit the findings of this study, most notably the age and education differences observed between diagnostic groups. For example, it could be that the WA group represents a pre-SAD group (i.e., they have vulnerability to developing SAD episodes, but have not received sufficient external stressors yet in their lives). Another confound that must not be overlooked is the fixed order of the Stroop task. It was designed so that any results for the salient variables could not be attributed to practice effects. Therefore, any practice effects may have decreased the experimental effect size such that it was no longer statistically significant. A better design would utilize a random presentation of the blocks of word types, in addition to the random presentation of words within each block. Unfortunately, this design was not possible with the available software.

### Future Research

Further study of the WA group may also elicit identification of protective factors that reduce vulnerability to developing seasonal depression episodes. Future study of winter anergia should include further refinement of a definition for this diagnostic category. Although suggested by theory and previously identified through examination of convenience sample GSS scores (Boulard & Sigmon, 2001), there is no published, structured criteria for classifying WA. Future research should address the reliability and validity of such a classification, as well as developing a standardized measure/interview for identifying those who may be categorized as suffering from WA. In addition, future study should include collection of prevalence data and population characteristics (e.g.,

age, gender, family history) as well as inclusion in investigations of light therapy efficacy for individuals with WA. This treatment has been hypothesized to most effectively treat the biological vulnerability of SAD (Lam et al., 2001), which is the vulnerability hypothesized to be shared by individuals with WA. Future research should also include comparison with S-SAD groups in order to determine if WA can be distinguished from general S-SAD such that it constitutes a discrete category rather than a sub-type of S-SAD.

Further longitudinal research of the subsequent development of SAD symptoms comparing those who develop a SAD episode with those who do not is likely to offer more clues about factors related to the recurrence maintenance of SAD episodes. Of course, most beneficial would be a large-scale longitudinal study of the general population that might capture individuals experiencing their first episode of winter depression and measure the factors that predict initial winter depression onset prior to meeting SAD diagnostic criteria. It is not clear whether factors related to recurrent SAD episode onset (e.g., negative thoughts, rumination) are present prior to an initial lifetime occurrence of a SAD episode.

In order to be diagnosed, individuals with a history of SAD must have already experienced at least 2 episodes of clinical depression. Therefore, findings from studies examining individuals with a history of SAD are limited to exploration of factors related to subsequent, rather than initial episodes of winter depression. It is not known how the previous experience of SAD episode affects factors related to subsequent episodes. Critics of cognitive theories of depression have argued that cognitive factors (e.g.,

tendency to ruminate in response to depressed mood) are symptoms and/or consequents rather than antecedents of depressive episodes (Coyne & Gotlib, 1983).

The field of SAD research can continue to benefit from studies designed from both the categorical and dimensional continuum perspective. This research supports the need for diathesis-stress models to more fully understand the complex disorder of SAD. In addition, the findings presented here support the consideration of a dynamic, rather than static continuum for seasonality. Any given individual cannot accurately be placed along a continuum of seasonality based on a measure of general experience of the seasons because that experience is likely composed of biological, genetic, and psychological internal factors as well as exposure to current and ongoing external factors (i.e., stress, ambient light, activity level).

### Summary

This study contributes to the growing literature supporting a diathesis-stress model approach to SAD. Beyond supporting existing findings, it makes several unique contributions to the literature. For one, it includes the prospective measurement of seasonal symptoms and cognitive factors across the fall months and suggests a differential symptom pattern for those who develop a recurrent SAD episode and those who do not. Furthermore, the inclusion of control groups allows for comparison of the experience of symptoms between those who often have SAD episodes during the course of the winter and those whose experience of seasonal changes does not include or lead to a depression episode. In addition, this is the first study to attempt to identify individuals with a seasonal vulnerability for vegetative symptoms who lack the psychological vulnerability for depression (i.e., WA). Although important theoretically and in comparison to the

SAD group's experiences, this category had not been considered and examined separately from S-SAD, which includes those with sub-clinical mood symptoms as well. The findings from this study suggest that WA may be viewed and measured as a category separate from S-SAD.

Despite the limitations due to small sample sizes and age differences across groups, these findings support the DVH (Young et al., 1991). In addition to the expected temporal relationship of mood symptom onset preceded by vegetative symptom onset, differences between the groups for vegetative symptom onset and intensity support the idea of a vulnerability to seasonal vegetative changes separate from a depression vulnerability. Furthermore, cognitive factors that are hypothesized to be related to seasonal depression (i.e., rumination and seasonal automatic thoughts) were greater in the SAD group than in the WA or Control groups. These factors were also shown to be predictive of severity of winter depression symptoms. The findings related to cognitive factors are in line with previous research that demonstrates a role for cognitive factors in seasonality and SAD (e.g., Hodges & Marks, 1998; Rohan, Sigmon, & Dorhofer, 2003).

Research is underway to determine the effectiveness of cognitive behavioral treatments for SAD (Rohan et al., 2003; Rohan, et al., in press; Sigmon, Whitcomb-Smith, & Boulard, 2001) and to examine cognitive schemas and attentional biases that may be related to recurrent SAD episodes (e.g., K.J. Rohan, personal communication, November 16, 2001). The search for alternative treatments for SAD is important because significant proportions of individuals do not respond to light therapy (Terman et al., 1989), and light therapy must be used every winter to treat recurrences. New research suggests that cognitive behavioral therapy may have a prophylactic effect, reducing



subsequent SAD symptoms such that recurrent SAD episodes are prevented (Rohan et al., 2003; Rohan, et al., in press). In addition, research supports diathesis-stress models that view SAD as a complex disorder which may require several modalities of treatment to address multiple factors in order to achieve maximal relief of the disorder.

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## APPENDICES

### Informed Consent for Seasonality Study

Because I am at least 18 years old I am being asked to participate in this study. I am being asked to participate because I either feel depressed in the winter, I feel more tired, hungry and sleepy during the winter or I do not notice any significant changes in my feelings across the fall and winter. The purpose of this study is to find out more about the effects of the changing seasons on mood and thinking in people, from those who do not experience seasonal symptoms to those who meet criteria for a diagnosis of Seasonal Affective Disorder.

First, I will undergo an assessment to see if I qualify for the study. This assessment will take approximately 1-2 hours and will involve answering questions during structured interviews (e.g., In the past month, has there been a period of time when you were feeling depressed or down for most of the day nearly every day? How has your appetite been this past week?)

If I qualify for this study, based on my responses in the interviews, I will be asked to complete several tasks. First, I will complete a task that is designed to measure my reaction time to naming colors. I will be asked to identify the colors that words are written in as fast as I can. Following the short reaction time task, I will be asked to complete a brief questionnaire about how I am feeling at that moment (i.e., how unhappy are you feeling right now?) Last, I will be asked to fill out some questionnaires involving:

- questionnaires about how I feel (i.e., I feel sad much of the time).
- questionnaires about changes across seasons (e.g., To what degree does your appetite change across seasons?)
- questionnaires about how often I have certain thoughts about myself and the seasons (e.g., I feel worthless, I wish the days were longer)
- questionnaires about how I react to certain situations (e.g., I think about how hard it is to concentrate)

I will receive a \$15 stipend for participating in the interview and tasks.

From the third week of September through January (approximately 4 months), I will be asked to complete three brief questionnaires once a week and return them to the researcher. These questionnaires will assess my feelings (e.g., I feel the same about myself as ever), my thoughts about the seasons (e.g., I am dreading the next few months) and my responses to situations (e.g., I think “Why can’t I get going?”). The questionnaires will be returned to the researcher in self-addressed stamped envelopes. The researcher will contact me to remind me when to start completing the questionnaires. The researcher may also contact me if questionnaires are not returned to her.

In January, I will be contacted about returning for the completion of the study. At that time, I will undergo a brief interview similar to the first one designed to assess my mood. At the return meeting in January, I will again complete the reaction time task and I will fill out the same brief questionnaire to assess my mood at that moment. Lastly, I

will complete the same questionnaires that I completed in the summer assessment. I will again receive \$15 compensation for participation in the study.

In order to ensure confidentiality, an ID number will be assigned to my questionnaires and interview information. The researcher will maintain a list of the assigned numbers and names throughout the duration of my participation. This information will only be used in the event that questionnaires are not returned and it becomes necessary to contact me. The information connecting my name to the ID number will be destroyed when the study ends. All of my written responses and taped interviews will be stored in a locked room and will be destroyed once all of the data analyses have been completed. No identifying information about me will be released. If the data from this study is presented or published, only data that would not identify me as an individual will be presented.

This research will not involve any risks greater than normally encountered in daily life. All of the questionnaire measures have been used in previous research and there have been no negative after-effects associated with using them. Possible benefits from this study include gaining additional insight into the relations between my feelings, thoughts, behaviors and the changing seasons. In addition, my participation may increase the knowledge base about Seasonal Affective Disorder and seasonality (i.e., the effects of the seasons on our moods and behaviors). I may withdraw my participation at any point in the study without penalty. If I desire, I will be offered counseling referrals upon ending my participation in the study.

This study is being conducted by Stacy Whitcomb, doctoral candidate in psychology, as her dissertation project. This study is being supervised by Sandra T. Sigmon, a licensed psychologist. If I have any questions about this study at any point, I may contact either Stacy Whitcomb at 581-2030, or Sandra Sigmon at 581-2038. They are both located at 5742 Little Hall, University of Maine, Orono, ME 04469.

I acknowledge that I have received a copy of this consent form.

Signature \_\_\_\_\_ Date \_\_\_\_\_

Printed name \_\_\_\_\_

Address \_\_\_\_\_ SS # \_\_\_\_\_

\_\_\_\_\_

## Informed Consent for Seasonality Study Student Version

Because I am at least 18 years old I am being asked to participate in this study. I am being asked to participate because I either feel depressed in the winter, I feel more tired, hungry and sleepy during the winter or I do not notice any significant changes in my feelings across the fall and winter. The purpose of this study is to find out more about the effects of the changing seasons on mood and thinking in people, from those who do not experience seasonal symptoms to those who meet criteria for a diagnosis of Seasonal Affective Disorder.

First, I will undergo an assessment to see if I qualify for the study. This assessment will take approximately 1-2 hours and will involve answering questions during structured interviews (e.g., In the past month, has there been a period of time when you were feeling depressed or down for most of the day nearly every day? How has your appetite been this past week?)

If I qualify for this study, based on my responses in the interviews, I will be asked to complete several tasks. First, I will complete a task that is designed to measure my reaction time to naming colors. I will be asked to identify the colors that words are written in as fast as I can. Following the short reaction time task, I will be asked to complete a brief questionnaire about how I am feeling at that moment (i.e., how unhappy are you feeling right now?) Last, I will be asked to fill out some questionnaires involving:

- questionnaires about how I feel (i.e., I feel sad much of the time).
- questionnaires about changes across seasons (e.g., To what degree does your appetite change across seasons?)
- questionnaires about how often I have certain thoughts about myself and the seasons (e.g., I feel worthless, I wish the days were longer)
- questionnaires about how I react to certain situations (e.g., I think about how hard it is to concentrate)

I will receive research credit for participating in the interview and tasks.

From the third week of September through January (approximately 4 months), I will be asked to complete three brief questionnaires once a week and return them to the researcher. These questionnaires will assess my feelings (e.g., I feel the same about myself as ever), my thoughts about the seasons (e.g., I am dreading the next few months) and my responses to situations (e.g., I think “Why can’t I get going?”). The questionnaires will be returned to the researcher in self-addressed stamped envelopes. The researcher will contact me to remind me when to start completing the questionnaires. The researcher may also contact me if questionnaires are not returned to her. I will receive research credit for completing the weekly questionnaires.

In January, I will be contacted about returning for the completion of the study. At that time, I will undergo a brief interview similar to the first one designed to assess my



mood. At the return meeting in January, I will again complete the reaction time task and I will fill out the same brief questionnaire to assess my mood at that moment. Lastly, I will complete the same questionnaires that I completed in the summer assessment. I will receive \$15 compensation for participating in the final assessment and tasks.

In order to ensure confidentiality, an ID number will be assigned to my questionnaires and interview information. The researcher will maintain a list of the assigned numbers and names throughout the duration of my participation. This information will only be used in the event that questionnaires are not returned and it becomes necessary to contact me. The information connecting my name to the ID number will be destroyed when the study ends. All of my written responses and taped interviews will be stored in a locked room and will be destroyed once all of the data analyses have been completed. No identifying information about me will be released. If the data from this study is presented or published, only data that would not identify me as an individual will be presented.

This research will not involve any risks greater than normally encountered in daily life. All of the questionnaire measures have been used in previous research and there have been no negative after-effects associated with using them. Possible benefits from this study include gaining additional insight into the relations between my feelings, thoughts, behaviors and the changing seasons. In addition, my participation may increase the knowledge base about Seasonal Affective Disorder and seasonality (i.e., the effects of the seasons on our moods and behaviors). I may withdraw my participation at any point in the study without penalty. If I desire, I will be offered counseling referrals upon ending my participation in the study.

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I acknowledge that I have received a copy of this consent form.

Signature \_\_\_\_\_ Date \_\_\_\_\_

Printed name \_\_\_\_\_

Address \_\_\_\_\_ SS # \_\_\_\_\_

\_\_\_\_\_

## Demographics Information

ID # \_\_\_\_\_

Sex    M       F

Age \_\_\_\_\_

Ethnicity (check one)

Caucasian            ☐African American    ☐Latino                ☐Asian/Pacific Islander ☐Native American     ☐Other \_\_\_\_\_ ☐

Income Level (circle one)

\$ 0-15,000    \$16-30,000    \$31-50,000    \$50,000+

Highest grade completed (High school diploma or GED equals 12 years)

\_\_\_\_\_

Current medications:

\_\_\_\_\_  
\_\_\_\_\_

Have you ever been treated for seasonal symptoms?        Y        N

If yes, what treatments have you tried?

\_\_\_\_\_

# STRUCTURED INTERVIEW GUIDE FOR THE HAMILTON DEPRESSION RATING SCALE SEASONAL AFFECTION DISORDER VERSION

(SIGH-SAD)

Janet B. W. Williams, D.S.W., Martha J. Link, B.S.,  
Norman E. Rosenthal, M.D., Leora Amira, Ph.D., and Michael Terman, Ph.D.

PT'S NAME: \_\_\_\_\_

PT'S ID: \_\_\_\_\_

INTERVIEWER: \_\_\_\_\_

DATE: \_\_\_\_/\_\_\_\_/\_\_\_\_

DAY OF THE WEEK: \_\_\_\_\_

Interview setting: 1 - phone; 2 - live

Protocol: \_\_\_\_\_

No. days on protocol: \_\_\_\_\_

Evaluation is based on how many days? \_\_\_\_\_

Evaluation period: 1 - retrospective; 2 - current

FOR FEMALES (pre-menopausal): When did your last period begin? \_\_\_\_/\_\_\_\_/9 \_\_\_\_

This interview guide is based on the Hamilton Depression Rating Scale by Max Hamilton, M.D. and an addendum to the scale for seasonal affective disorders by Norman E. Rosenthal, M.D. This work was supported in part by Biomedical Research Support Grant 903-E7595 from the Research Foundation for Mental Hygiene, Inc., and by National Institute of Mental Health Grants MM-00461 and MM-42931.

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For correspondence: Dr. William or Dr. Terman, New York State Psychiatric Institute, 722 West 168th Street, New York, New York 10032.

For masters: SAD Assessment Tools Packet, Society for Light Treatment and Biological Rhythms, P.O. Box 473, Wilsonville, OR 97070.

**STRUCTURED INTERVIEW GUIDE FOR THE HAMILTON DEPRESSION  
RATING SCALE SEASONAL AFFECTION DISORDER VERSION**

(SIGH-SAD)

Janet B. W. Williams, D.S.W., Martha J. Link, B.S.,  
Norman E. Rosenthal, M.D., Leora Amira, Ph.D., and Michael Terman, Ph.D.

OVERVIEW: I'd like to ask you some questions about the past week, since last (DAY OF WEEK). How have you been feeling since then?

H1: What's your mood been like this past week (compared to when you feel OK)?	DEPRESSED MOOD (sadness, helpless, worthless):
Have you been feeling down or depressed?	0 - absent
	1 - indicated only on questioning
Sad? Hopeless? Helpless? Worthless?	2 - spontaneously reported verbally
	3 - communicated non-verbally, i.e., facial expression, posture, voice, tendency to weep
In the last week, how often have you felt (OWN EQUIVALENT)? Every day?	4 - VIRTUALLY ONLY; this in spontaneous verbal and nonverbal communication
All day? Have you been crying at all?	

IF SCORED 1-4 ABOVE, ASK: How long have you been feeling this way?

H2: IF OUTPATIENT: Have you been working this week (in or out of the home)?  
IF NOT: Why not?

IF WORKING: Have you been able to get as much (work) done as you usually do (when you're feeling OK)?

How have you been spending your time this past week (when not at work)?

Have you felt interested in doing (THOSE THINGS), or do you feel you have to push yourself to do them?

Have you stopped doing anything you used to do? IF YES: Why?

Is there anything you look forward to?

A1: In the last week, have you been as social as when you feel well? IF NO: Tell me which fits you best. (READ DOWN ANCHOR DESCRIPTIONS AND RATE ACCORDINGLY.)

#### WORK AND ACTIVITIES:

- 0 - no difficulty
- 1 - thoughts and feelings of incapacity, fatigue or weakness related to activities, work or hobbies
- 2 - loss of interest in activity, hobbies or work - by direct report of the patient or indirect in listlessness, indecision and vacillation (feels he has to push self to work or do activities)
- 3 - decrease in actual time spent in activities or decrease in productivity. In hosp, pt. spends less than 3 hrs/day in activities(hospital job or hobbies) exclusive of ward chores
- 4 - stopped working because of present illness. In hospital, no activities except ward chores, or fails to perform ward chores unassisted

#### \*SOCIAL WITHDRAWAL:

- 0 - interacts with other people as usual
- 1 - less interested in socializing with others but continues to do so
- 2 - interacting less with other people in social (optional) situations
- 3 - interacting less with other people in work or family situations (i.e., where this is necessary)
- 4 - marked withdrawal from others in family or work

H3: This week, how has your interest in sex been? (I'm not asking about actual sexual activity, but about your interest in sex - how much you think about it.)  
Has there been any change in your interest in sex (from when you were not depressed)?  
Is it something you've thought much about?

IF NC: Is that unusual for you, compared to when you feel well? (Is it a little less or a lot less?)

H4: How has your appetite been this past week? (What about compared to your usual appetite?)  
Have you had to force yourself to eat?  
Have other people had to urge you to eat? (Have you skipped meals?)  
Have you had any stomach or intestinal problems? (Have you needed to take anything for that?)

H5: Have you lost any weight since you started feeling depressed or down? IF YES: Did you lose any weight this last week? (Was it because of feeling depressed?) How much did you lose?

IF NOT SURE: Do you think your clothes are any looser on you?

GENTAL SYMPTOMS  
(such as loss of libido, menstrual disturbances):

0 - absent  
1 - mild  
2 - severe

SOMATIC SYMPTOMS  
GASTROINTESTINAL:

0 - none  
1 - loss of appetite but eating without encouragement  
2 - difficulty eating without requests or urging; requires laxatives or medication for G.I. symptoms

LOSS OF WEIGHT (Rate either A or B):

A. When rating by history:  
0 - no weight loss  
1 - probable weight loss due to current depression  
2 - definite (according to patient) weight loss due to depression  
3 - not assessed

B. When actual weight changes are measured:  
0 - less than 1 lb. loss in week  
1 - greater than 1 lb. loss in week  
2 - greater than 2 lb. loss in week  
3 - not assessed

NOTE: AVOID CODING "3" IF POSSIBLE

- A2: Have you gained any weight in the last week? IF YES: Was it because of feeling depressed or down? How much did you gain?
- \*WEIGHT GAIN:
- 0 - no weight gain
  - 1 - probable weight gain due to current depression
  - 2 - definite (according to patient) weight gain due to depression
- A3: In the past week, has your appetite been greater than when you feel well or OK? IF YES: Do you want to eat a little more, somewhat more, or much more than when you feel well or OK?
- \*APPETITE INCREASE:
- 0 - no increase in appetite
  - 1 - wants to eat a little more than usual
  - 2 - wants to eat somewhat more than normal
  - 3 - wants to eat much more than usual
- A4: In the past week, have you actually been eating more than when you feel well or OK? IF YES: A little more, somewhat more, or much more than when you feel usual well or OK?
- \*INCREASED EATING:
- 0 - is not eating more than usual
  - 1 - is eating a little more than usual
  - 2 - is eating somewhat more than usual
  - 3 - is eating much more than normal
- A5: In the last week, have you been craving or eating more starches or sugars? IF YES: Have you been eating or craving starches or sugars more than when you feel well or OK, much more, or has it been irresistible?
- \*CARBOHYDRATE CRAVING OR EATING (in relation to total amount of food desired or eaten):
- 0 - no change in food preference or consumption
  - 1 - craving or eating more carbohydrates (starches or sugars) than before
  - 2 - craving or eating much more carbohydrates than before
  - 3 - irresistible craving or eating of sweets or starches
- Has it been mainly starches or mainly sweets? Which specific foods have you been craving? LIST:
- CIRCLE ONE    Mainly                      Mainly  
OR BOTH:       starches                      sweets

Have you been actually eating more  
starches or sweets, or just craving them?

CIRCLE ONE

OR BOTH: Craving Eating

Has the (CRAVING OR EATING)

USUAL TIME OF CRAVING OR  
EATING:

usually occurred at any particular time  
of day? ( \_\_\_\_\_ o'clock)

0 - it comes and goes at  
various times

1 - usually morning

2 - usually afternoon or  
evening

3 - virtually all the time

RATER NOTE: IF BOTH CRAVING AND EATING, RATE TIME OF  
EATING. DO NOT COUNT ABOVE SCORE IN SCALE TOTALS.

H6: I'd like to ask you now about your sleeping  
during the past week.

INSOMNIA EARLY  
(INITIAL INSOMNIA):

Have you had any trouble falling asleep  
at the beginning of the night? (Right after  
you go to bed, how long has it been taking  
you to fall asleep?)  
How many nights this week have you had  
complaints of nightly

0 - no difficulty falling asleep

1 - complains of occasional  
difficulty falling asleep  
-i.e., more than 1/2 hr

2 - trouble falling asleep?  
difficulty falling asleep

H7: During the past week, have you been waking  
up in the middle of the night? IF YES:  
Do you get out of bed? What do you do?  
(Only go to the bathroom?)

INSOMNIA MIDDLE:

0 - no difficulty

1 - complains of being  
restless and disturbed  
during the night

2 - waking during the night –  
any getting out of bed  
(except to void)

When you get back in bed, are you able  
to fall right back asleep?  
Have you felt your sleeping has been  
restless or disturbed some nights?

H8: What time have you been waking up in  
the morning for the last time, this past week?  
IF EARLY: Is that with an alarm clock,  
or do you just wake up yourself?

INSOMNIA LATE  
(TERMINAL INSOMNIA):

0 - no difficulty

1 - waking in early hours of  
morning but goes back  
to sleep

2 - unable to fall asleep again  
if gets out of bed

What time do you usually wake up (that is,  
when you feel well?)



A6: Have you been sleeping more than usual past week? IF YES: How much more?

\*HYPERMOMNIA sleep this (Compare length to euthymic and NOT to hypomanic sleep length. If this cannot be established, use 8hrs)

IF NO: What about weekends?  
(What time have you been falling asleep?  
Have you been taking naps? That means  
you've been sleeping about \_\_\_\_ hours a  
day altogether? How much time do you  
usually sleep when you feel well?)

- 0 - no increase in sleep length
- 1 - at least 2 hour increase in sleep
- 2 - 2-hour increase
- 3 - 3-hour increase
- 4 - 4-hour increase

Sleep length used (circle one):  
euthymic (\_\_\_\_ hrs)                      8-hour

H9: How has your energy been this past week?

SOMATIC SYMPTOMS  
GENERAL:

IF LOW ENERGY: Have you felt tired?  
(How much of the time? How bad has it  
been?)  
This week, have you had any aches or  
pains? (What about backaches, headaches,  
or muscle aches?)  
Have you felt any heaviness in your limbs,  
back or head?.

- 0 - none
- 1 - heaviness in limbs, back or head. Backaches, headache, muscle aches. Loss of energy and fatiguability
- 2 - any clear-cut symptom

A7: IF ACKNOWLEDGED FEELING TIRED  
ON PREVIOUS ITEM:  
How much of the time have you felt tired?  
(Every day? How much of each day?)

Very tired, or just a little?

- \*FATIGABILITY (or low energy or feelings of being heavy, leaden, weighed down):
- 0 - does not feel more fatigued than usual
  - 1 - feels more fatigued than usual but this has not impaired function significantly;
  - 2 - more fatigued than usual; at least one hour a day, three days a week
  - 3 - fatigued much of the time most days
  - 4 - fatigued almost all the time

H10: Have you been putting yourself down, this past week, feeling you've done things wrong, or let others down?

IF YES: What have your thoughts been?

Have you been feeling guilty about any-rumination over thing that you've done or not done? What about things that happened a long time ago?

Have you thought that you've brought (THIS DEPRESSION) on yourself in some way? Do you feel your being sick is a punishment?

H11: This past week, have you had any thoughts that life is not worth living? IF YES: What about thinking you'd be better off dead? Have you had thoughts of hurting or killing yourself?

IF YES: What have you thought about? Have you actually done anything to hurt yourself?

H12: Have you been feeling especially tense or irritable this past week? IF YES: Is this more than when you are not depressed or down?

Have you been unusually argumentative or impatient?

Have you been worrying a lot about little things, things you don't ordinarily worry about?

IF YES: Like what, for example?

#### FEELINGS OF GUILT:

- 0 - absent
- 1 - self-reproach, feels he has let people down
- 2 - ideas of guilt or past errors or sinful deeds
- 3 - present illness is a punishment; delusions of guilt
- 4 - hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

#### SUICIDE:

- 0 - absent
- 1 - feels life is not worth living
- 2 - wishes he were dead or any thoughts of possible death to self
- 3 - suicidal ideas or gesture
- 4 - attempts at suicide

#### ANXIETY PSYCHIC:

- 0 - no difficulty
- 1 - subjective tension and irritability
- 2 - worrying about minor matters
- 3 - apprehensive attitude apparent in face or speech
- 4 - fears expressed without questioning

H13: In this past week, have you had any of the following physical symptoms? (READ LIST, PAUSING AFTER EACH SX FOR REPLY, CIRCLE POSITIVE SXS.)

Have you had these only while you've been feeling depressed or down? IF YES:

How much have these things been bothering you this past week?

How bad have they gotten?

How much of the time, or how often, have you had them?)

Do you have any physical illness or are you taking any medication that could be causing these symptoms?  
(IF YES, RECORD PHYSICAL ILLNESS OR MEDICATION, BUT RATE SYMPTOMS ANYWAY:

ANXIETY SOMATIC  
(physiologic concomitants of anxiety, such as

GI - dry mouth, gas, indigestion, diarrhea, stomach cramps, belching

C-V - heart palpitations, headaches

Resp- hyperventilating, sighing

Having to urinate frequently  
Sweating):

0 - absent

1 - mild

2 - moderate

3 - severe

4 - incapacitating

H14: In the last week, how much have your thoughts been focused on your physical health or how your body is working (compared to your normal thinking)? (Have you worried a lot about being or becoming physically ill? Have you really been preoccupied with this?)

Do you complain much about how you feel physically?

Have you found yourself asking for help with things you could really do yourself? IF YES: Like what, for example? How often has that happened?

HYPOCHONDRIASIS:

0 - not present

1 - self-absorption (bodily)

2 - preoccupation with health

3 - frequent complaints, requests for help, etc.

4 - hypochondriacal delusions

H15: RATING BASED ON OBSERVATION INTERVIEW.

INSIGHT:

0 - acknowledges being OR not currently depressed

1 - acknowledges illness but attributes cause to bad food, overwork, virus, need for rest, etc.

2 - denies being ill at all

H16: RATING BASED ON OBSERVATION  
INTERVIEW.

IF TELEPHONE INTERVIEW: Do you feel that your speech or physical movements are sluggish? Has anyone actually commented on this?

RETARDATION (slowness of thought and speech; impaired ability to concentrate; decreased motor activity):

- 0 - normal speech and thought
- 1 - slight retardation at interview
- 2 - obvious retardation at interview
- 3 - interview difficult
- 4 - complete stupor

H17: RATING BASED ON OBSERVATION  
INTERVIEW.

IF TELEPHONE INTERVIEW: As we talk, are you fidgeting at all, or having trouble sitting still? For instance, are you doing anything like playing with your hands or your hair, or tapping your foot? Do still others notice that you are restless?

AGITATION:

- 0 - none
- 1 - fidgetiness
- 2 - playing with hands, hair, etc.
- 3 - moving about, can't sit
- 4 - hand-wringing, nail biting, hair-pulling, biting of lips

TOTAL 17-ITEM HAMILTON DEPRESSION  
SCORE (without starred items):

\_\_\_\_\_

Over the past week, in the first few hours after waking up have you been feeling better or worse or no different from are before you go to sleep?  
If NO diurnal variation, mark none:

DIURNAL VARIATION TYPE A:

- A. Note whether symptoms worse after awakening or before sleeping.
- 0 - no variation OR not currently depressed
  - 1 - worse after awakening
  - 2 - worse before going to sleep

RATER NOTE: DO NOT COUNT ABOVE SCORE IN SCALE TOTALS.

H18: IF VARIATION: How much worse do you feel in the (MORNING OR EVENING)? B. When present, mark the severity of the variation:

IF UNSURE: A little bit worse or a lot worse?

- 0 - none
- 1 - mild
- 2 - severe

- A8: This week, have you regularly had a slump in your mood or energy in the afternoon or evening?
- IF YES: Is it mostly in your mood or your energy? Does it occur every day? At what time has the slump usually begun?
- (\_\_\_\_\_ O'CLOCK). When has it ended? Has that been at least an hour before you go to sleep? How big a slump do you have - would you say it's generally mild, moderate, or severe?
- CIRCLE ONE Mood Energy  
OR BOTH: slump slump
- NOTE: RATE ONLY SLUMPS THAT ARE FOLLOWED BY AT LEAST AN HOUR OF RECOVERED MOOD OR ENERGY BEFORE SLEEP.
- H19: In the past week, have you ever suddenly had the sensation that everything is unreal, or you're in a dream, or cut off from other people in some strange way? Any spacey feelings? IF YES: Tell me about it. How bad has that been? How often this week has that happened?
- DEPERSONALIZATION AND DEREALIZATION (such as feelings of unreality and nihilistic ideas):
- 0 - absent  
1 - mild  
2 - moderate  
3 - severe  
4 - incapacitating
- H20: This past week, have you thought that anyone was trying to give you a hard time or hurt you?
- What about talking about you behind your back?  
IF YES: Tell me about that.
- PARANOID SYMPTOMS:
- 0 - none  
1 - suspicious  
2 - ideas of reference  
3 - delusions of reference and persecution
- H21: In the past week, have there been things you've had to do over and over again, like checking the locks on the doors several times, or washing your hands? IF YES: Can you give me an example?
- Have you had any thoughts that don't make any sense to you, but that keep running over and over in your mind? IF YES: Can you give me an example?
- OBSESSIVE AND COMPULSIVE SYMPTOMS:
- 0 - absent  
1 - mild  
2 - severe

TOTAL 21-ITEM HAMILTON DEPRESSION  
SCORE (without starred items):

\_\_\_\_\_

TOTAL 8-ITEM ATYPICAL SCORE (starred  
items only):

\_\_\_\_\_

TOTAL 29-ITEM SIGH-SAD SCORE

\_\_\_\_\_

ATYPICAL BALANCE SCORE (total 8-item  
atypical score divided by total 29-item SIGH-  
SAD score, multiplied by 100):

\_\_\_\_\_

NOTE: If patient is not depressed and score is derived primarily from symptoms of hypomania (e.g., items H4, H5, H6, H7, H8, H12, H17), administer HIGH-SAD and report both scores.

## SPAQ

DATE: \_\_\_\_\_ SEX: \_\_\_\_\_ AGE: \_\_\_\_\_ RACE: \_\_\_\_\_ CURRENT WEIGHT: \_\_\_\_\_  
 CURRENT TIME OF DAY: \_\_\_\_\_ YRS. OF EDUCATION: \_\_\_\_\_  
 MARITAL STATUS: \_\_\_\_\_  
 PLACE OF BIRTH: CITY AND STATE: \_\_\_\_\_  
 COUNTRY: \_\_\_\_\_  
 HOW MANY YEARS HAVE YOU LIVED IN THIS CLIMATIC AREA?: \_\_\_\_\_

**The purpose of this form is to find out how your mood and behavior change over time. NOTE: We are interested in your experience; not others you may have observed.**

**To what degree do the following change with the seasons? Please indicate the appropriate number in front of the item.**

	no change 1	slight change 2	moderate change 3	marked change 4	extremely marked change 5
_____ a. sleep length					
_____ b. social activity					
_____ c. mood (overall feeling of well being)					
_____ d. weight					
_____ e. appetite					
_____ f. energy level					

**In the following questions, circle all applicable months. You may have one circle for a single month or you may have several circles for a cluster of months, or any grouping of months.**

**At what time of the year do you:**

a. feel best	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
b. tend to gain most weight	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
c. socialize most	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
d. sleep least	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
e. eat most	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
f. lose most weight	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
g. socialize least	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
h. feel worst	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
I. eat least	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month
j. sleep most	Jan Feb Mar Apr May Jun Jul Aug Sep Oct Nov Dec	No particular month

Using the following scale, indicate how the following weather changes make you feel.  
Place your response in front of the item.

- 3 = in very low spirits or markedly slowed down  
 -2 = moderately low/slowed down  
 -1 = mildly low/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  
 4 = don't know

- \_\_\_\_\_ a. cold weather  
 \_\_\_\_\_ b. hot weather  
 \_\_\_\_\_ c. humid weather  
 \_\_\_\_\_ d. sunny days  
 \_\_\_\_\_ e. dry days  
 \_\_\_\_\_ f. grey cloudy days  
 \_\_\_\_\_ g. long days  
 \_\_\_\_\_ h. high pollen count  
 \_\_\_\_\_ i. foggy, smoggy days  
 \_\_\_\_\_ j. short days

How much does your weight fluctuate during the course of the year? (circle one)

0-3 lbs.      4-7 lbs.      8-11 lbs.      12-15 lbs.      16-20 lbs.      over 20 lbs.

Approximately how many hours of each 24-hour day do you sleep during each season?  
(include naps)

- \_\_\_\_\_ a. winter (Dec. 21 - Mar. 20)  
 \_\_\_\_\_ b. spring (Mar. 21 - June 20)  
 \_\_\_\_\_ c. summer (June 21 - Sept. 20)  
 \_\_\_\_\_ d. fall (Sept. 21 - Dec. 20)

Do you notice a change in food preference during the different seasons?

Yes\_\_\_ No\_\_\_      If yes, please specify the changes.

---

If you experience changes with the seasons, do you feel that these are a problem  
for you? Yes\_\_\_ No\_\_\_

If this is a problem, circle the severity: Mild Moderate Marked Severe Disabling

How many years has this been a problem for you? \_\_\_\_\_ years and months



## BDI-II with Addendum

This questionnaire contains 21 groups of statements. Please read each group of statements carefully and then pick out **one statement** in each group that best describes the way you have been feeling during the **past week (7 days), including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
  - 0 I do not feel sad.
  - 1 I feel sad much of the time.
  - 2 I am sad all of the time.
  - 3 I am so sad or unhappy that I can't stand it.
2. Pessimism
  - 0 I am not discouraged about my future.
  - 1 I feel more discouraged about my future than I used to.
  - 2 I do not expect things to work out for me.
  - 3 I feel my future is hopeless and will only get worse.
3. Past Failure
  - 0 I do not feel like a failure.
  - 1 I have failed more than I should have.
  - 2 As I look back, I see a lot of failures.
  - 3 I feel I am a total failure as a person
4. Loss of Pleasure
  - 0 I get as much pleasure as I ever did from the things I enjoy.
  - 1 I don't enjoy things as much as I used to.
  - 2 I get very little pleasure from the things I used to enjoy.
  - 3 I can't get any pleasure form the things I used to enjoy.
5. Guilty feelings
  - 0 I don't feel particularly guilty.
  - 1 I feel guilty over many things I have done or should have done.
  - 2 I feel quite guilty most of the time.
  - 3 I feel guilty all of the time.
6. Punishment feelings
  - 0 I don't feel I am being punished.
  - 1 I feel I may be punished.
  - 2 I expect to be punished.
  - 3 I feel I am being punished.
7. Self-Dislike
  - 0 I feel the same about myself as ever.
  - 1 I have lost confidence in myself.
  - 2 I am disappointed in myself.
  - 3 I dislike myself.

## 8. Self-criticalness

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

## 9. Suicidal Thoughts or Wishes

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

## 10. Crying

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

## 11. Agitation

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to sit still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

## 12. Loss of Interest

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It is hard to get interested in anything.

## 13. Indecisiveness

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.
- 3 I have trouble making any decisions.

## 14. Worthlessness

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to be.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

## 15. Loss of Energy

- 0 I have as much energy as ever
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

## 16. Loss of Interest in Sex

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

## 17. Changes in Sleeping pattern

- 0 I have not experienced any change in my sleeping pattern.

---
- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.

---
- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.

---
- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

## 18. Irritability

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

## 19. Changes in appetite

- 0 I have not experienced any change in my appetite.

---
- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.

---
- 2a My appetite is much less than before.
- 2b My appetite is much greater than before.

---
- 3a I have no appetite at all.
- 3b I crave food all the time.

## 20. Concentration Difficulty

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

## 21. Tiredness or Fatigue

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

## 22. Increased sleep

- 0. The amount of time I spend sleeping (including naps) has not increased.
- 1. I wake up about an hour later than usual.
- 2. I wake up about 2-3 hours later than usual.
- 3. I wake up several hours later than I used to and find it hard to get out of bed.

## 23. Increased Eating

0. I haven't been eating any more than usual.
1. I have been eating a little more than usual.
2. I have been eating somewhat more than usual.
3. I have been eating much more than I usually do.

## 24. Weight gain

0. I haven't gained much weight, if any, lately.
1. I have gained more than 5 pounds.
2. I have gained more than 10 pounds.
3. I have gained more than 15 pounds.

I am purposely trying to gain weight.

\_\_\_\_\_ Yes                      \_\_\_\_\_ No

# ATQ

Instructions: Listed below are a variety of thoughts that pop into people's heads. Please read each thought and indicate how frequently, if at all, the thought occurred to you over the last week. Please read each item carefully and put the number in the blank that most closely corresponds to your answer (1 = "not at all," 2 = "sometimes," 3 = "moderately often," 4 = "often," and 5 = "all the time").

1	2	3	4	5
not at all	sometimes	moderately often	often	all the time

- \_\_\_ 1. I feel like I'm up against the world.
- \_\_\_ 2. I'm no good.
- \_\_\_ 3. Why can't I ever succeed?
- \_\_\_ 4. No one understands me.
- \_\_\_ 5. I've let people down.
- \_\_\_ 6. I don't think I can go on.
- \_\_\_ 7. I wish I were a better person.
- \_\_\_ 8. I'm so weak.
- \_\_\_ 9. My life's not going the way I want it to.
- \_\_\_ 10. I'm so disappointed in myself.
- \_\_\_ 11. Nothing feels good anymore.
- \_\_\_ 12. I can't stand this anymore.
- \_\_\_ 13. I can't get started.
- \_\_\_ 14. What's wrong with me?
- \_\_\_ 15. I wish I were somewhere else.
- \_\_\_ 16. I can't get things done.
- \_\_\_ 17. I hate myself.
- \_\_\_ 18. I'm worthless.
- \_\_\_ 19. Wish I could just disappear.
- \_\_\_ 20. What's the matter with me?
- \_\_\_ 21. I'm a loser.
- \_\_\_ 22. My life is a mess.
- \_\_\_ 23. I'm a failure.
- \_\_\_ 24. I'll never make it.
- \_\_\_ 25. I feel so helpless.
- \_\_\_ 26. Something has to change.
- \_\_\_ 27. There must be something wrong with me.
- \_\_\_ 28. My future is bleak.
- \_\_\_ 29. It's just not worth it.
- \_\_\_ 30. I can't finish anything.

## Seasonal Automatic Thoughts Survey

Directions: Below is a list of thoughts that people might have during the day. Please read each thought and indicate how often, if ever, you had each thought in the past week. Please read each item carefully and write the number in the blank that best describes how frequently you had the thought in the past week.

1	2	3	4	5
not at all	occasionally	moderately often	often	
all the time				

---

- \_\_\_\_\_ 1. I worry that I won't have enough energy.
- \_\_\_\_\_ 2. I'm going to feel worse when the days get much shorter.
- \_\_\_\_\_ 3. I am overwhelmed.
- \_\_\_\_\_ 4. I shouldn't be eating this much.
- \_\_\_\_\_ 5. No one else feels this way every year.
- \_\_\_\_\_ 6. The amount of daylight has been changing lately.
- \_\_\_\_\_ 7. I am dreading the next few months
- \_\_\_\_\_ 8. I'm too tired to do anything.
- \_\_\_\_\_ 9. I am worried about how bad I will feel this winter.
- \_\_\_\_\_ 10. I wish spring would get here sooner.
- \_\_\_\_\_ 11. It is hard to get out of bed in the morning.
- \_\_\_\_\_ 12. I am always going to feel down in the winter months.
- \_\_\_\_\_ 13. There are signs that the season is changing.
- \_\_\_\_\_ 14. I can't control my food cravings.
- \_\_\_\_\_ 15. I'm going to be depressed until spring.
- \_\_\_\_\_ 16. I can't do this much longer.
- \_\_\_\_\_ 17. There is nothing I can do to stop depression from coming.
- \_\_\_\_\_ 18. I dread the cold.
- \_\_\_\_\_ 19. Everything is so dark and dreary.
- \_\_\_\_\_ 20. I just want to crawl into bed for a long time.
- \_\_\_\_\_ 21. There are not enough hours in the day to accomplish everything I need to do.
- \_\_\_\_\_ 22. It is hard to be social.

### Response Styles Questionnaire

People think and do many different things when they feel 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 down, sad, or depressed. Please indicate what you generally do, not what you think you should do.

0 = Almost Never

1 = Sometimes

2 = Often

3 = Almost Always

- \_\_\_ 1. Think about how alone you feel
- \_\_\_ 2. Think "I won't be able to do my job/work because I feel so badly"
- \_\_\_ 3. Think about your feelings of fatigue and achiness
- \_\_\_ 4. Think about how hard it is to concentrate
- \_\_\_ 5. Try to find something positive in the situation or something you learned
- \_\_\_ 6. Think "I'm going to do something to make myself feel better"
- \_\_\_ 7. Help someone else with something in order to distract yourself
- \_\_\_ 8. Think about how passive and unmotivated you feel
- \_\_\_ 9. Remind yourself that these feelings won't last
- \_\_\_ 10. Analyze recent events to try to understand why you are depressed
- \_\_\_ 11. Think about how you don't seem to feel anything any more
- \_\_\_ 12. Think "Why can't I get going?"
- \_\_\_ 13. Think "Why do I always react this way?"
- \_\_\_ 14. Go to a favorite place to get your mind off your feelings
- \_\_\_ 15. Go away by yourself and think about why you feel this way
- \_\_\_ 16. Think "I'll concentrate on something other than how I feel."
- \_\_\_ 17. Write down what you are thinking about and analyze it
- \_\_\_ 18. Do something that has made you feel better in the past
- \_\_\_ 19. Think about a recent situation, wishing it had gone better
- \_\_\_ 20. Think "I'm going to go out and have some fun"
- \_\_\_ 21. Concentrate on your work
- \_\_\_ 22. Think about how sad you feel

- \_\_\_ 23. Think about all your shortcomings, failings, faults, mistakes
- \_\_\_ 24. Do something you enjoy
- \_\_\_ 25. Think about how you don't feel up to doing anything
- \_\_\_ 26. Do something fun with a friend
- \_\_\_ 27. Analyze your personality to try to understand why you are depressed
- \_\_\_ 28. Go someplace alone to think about your feelings
- \_\_\_ 29. Think about how angry you are with yourself
- \_\_\_ 30. Listen to sad music
- \_\_\_ 31. Isolate yourself and think about the reasons why you feel sad
- \_\_\_ 32. Try to understand yourself by focusing on your depressed feelings



### Rumination Diary Form

Instructions: 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 thought each one when you felt sad, down, or depressed this past week. Please indicate what you actually did, not what you think you should have done.

	Never	Sometimes	Often	Always
1. I think about how alone I feel	1	2	3	4
2. I think about my feelings of fatigue and achiness	1	2	3	4
3. I think about how hard it is to concentrate	1	2	3	4
4. I think about how passive and unmotivated I feel	1	2	3	4
5. I think "Why can't I get going?"	1	2	3	4
6. I think about a recent situation, wishing it had gone better	1	2	3	4
7. I think about how bad I feel	1	2	3	4
8. I think about all my shortcomings, failings, faults, and mistakes	1	2	3	4
9. I think about how I don't feel up to doing anything	1	2	3	4
10. I think "Why can't I handle things better?"	1	2	3	4

## POMS

Below is a list of words that describe feelings people have. Please read each one carefully. Then fill in the space beside the answer with the number that best describes HOW YOU ARE FEELING RIGHT NOW.

Not at all	A little bit	Moderately	Quite a bit	Extremely
0	1	2	3	4

- \_\_\_\_\_ Unhappy
- \_\_\_\_\_ Sorry
- \_\_\_\_\_ Sad
- \_\_\_\_\_ Blue
- \_\_\_\_\_ Hopeless
- \_\_\_\_\_ Unworthy
- \_\_\_\_\_ Discouraged
- \_\_\_\_\_ Lonely
- \_\_\_\_\_ Miserable
- \_\_\_\_\_ Gloomy
- \_\_\_\_\_ Desperate
- \_\_\_\_\_ Helpless
- \_\_\_\_\_ Worthless
- \_\_\_\_\_ Terrified
- \_\_\_\_\_ Guilty

## BIOGRAPHY OF THE AUTHOR

Stacy Whitcomb-Smith was born in Waterville, ME and lived on her parents' farm in Newburgh, ME throughout her childhood. She graduated in 1991 from Hampden Academy, Hampden, ME, as a National Honor Society member, captain of her soccer and basketball teams, and officer of several service organizations. She attended Williams College in Massachusetts, where she continued to balance academics, athletics, service to her community, and employment. There she completed the Bachelor of Arts with Honors in Psychology, and a concentration in neuroscience in 1995. Her undergraduate thesis was a multimodal study of the behavioral and neurochemical effects of postnatal cocaine exposure in rats. She was elected to Sigma Xi and 1960's Scholars honors. After college, she spent several years as a senior research coordinator for psychotropic pharmaceutical trials and co-authored an article on refractory schizophrenia. She then returned to Maine to attend the Clinical Psychology program at The University of Maine, where she co-authored 3 journal articles, a book chapter, and 13 research presentations at the annual conventions of the Association for the Advancement of Behavior Therapy. She was awarded for her research by the University's Association of Graduate Students in 2002 and served as the Associate Director of the Psychological Services Center. She completed her required internship at the Albany Psychology Consortium in Albany, NY, where she initiated interdisciplinary research on preventing falls in the geriatric primary care clinic of the Stratton VA Hospital and co-wrote a behavioral medicine treatment manual for chronic pain patients. She is a candidate for Doctor of Philosophy in Psychology from The University of Maine in December, 2003.