An Investigation into the Presence of Seasonal Symptoms in a Sample Treated for Depression

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Abstract

The “winter blues” would appear to be a global concept. This thesis examined features of depression, seasonal change and meteorological data in relation to theories on the development of seasonal depression and Seasonal Affective Disorder (SAD). It reviewed the neurotransmitter based theories of causation where increased exposure to light is thought to stimulate normal neurotransmitter production. It also examined the dual vulnerability hypothesis (DVH) which postulated that vegetative symptoms arose from a different vulnerability than depressive symptoms. Levels of vegetative symptoms and depression were analysed in relation to the different climate experienced in Auckland, New Zealand. Three studies were undertaken. In the first study, the sample consisted of 195 individuals in New Zealand who self-referred to participate in a separate research project examining the effects of “homework” and cognitive behavioural therapy for first time depression. Meteorological data were investigated in order to explore any potential vulnerability to seasonal depression in this sample. Additionally, age and gender were explored in relation to season of presentation. The second study involved the subsample (n = 81) who were assessed for therapy and examined season and symptom profile in relation to when the person presented. The third study traced the progress through therapy of 28 adults who were selected from the second study for CBT. The Beck Depression Inventory (BDI-II) provided additional data to test the related hypotheses Rate of change in depression scores and symptom expression in relation to seasonality were analysed using multilevel modelling (MLM). Daily hours of bright sunlight was found to have an unusual relationship to temperature in New Zealand when compared with previous research. In this setting increased sunshine was associated with lower temperatures. Therefore, the variables were separated in order to ascertain whether one affected results more than others. Bright sunshine hours affected the expression of vegetative symptoms with a decrease observed over time in relation to increased photoperiod. Subtle relationships between temperature and vegetative symptoms were observed. However, there was an overall lack of correlation between vegetative and depressive symptoms observed in the CBT sample, and sunlight was not observed to have any effect on typical depressive symptoms. The investigation provided partial support for the neurotransmitter basis of vegetative symptoms and for the dual vulnerability hypothesis. Gender and age were correlated with vegetative symptom endorsement, although over time only gender was found to have any ongoing significance in the presentation of seasonal symptoms with women more likely to exhibit vegetative responses over time.

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Contents

List of Tables

List of Figures

List of Appendices

CHAPTER ONE:
INTRODUCTION

Depression is thought to be one of the most common psychological disorders. A study by Andrews, Poulton, and Skoog (2005), indicated that nearly half of the population may experience depression in their lifetime. Consequently, there have been large volumes of research devoted to the diagnosis and treatment of this disorder both in New Zealand and overseas.

New Zealand (NZ) is known to have a comparatively high rate of depression among western nations, with up to 11% of the population diagnosed with depression over their lifetime (Ministry of Health: MOH, 2008). Currently, depression is commonly treated with medication and or psychotherapy, although electroconvulsive therapy (ECT) and antipsychotic medications are used as required for severe depression (Ellis, 2004). Despite these measures, up to 12% of sufferers do not experience a remission of their symptoms (Ellis, 2004). Thus, the long-term cost to the country through decreased productivity and use of the health care system by people who suffer from depression is considerable. Additionally, depression can also be associated with other conditions, for example, anxiety, substance abuse, HIV-AIDS, Parkinson's disease, thus augmenting the individuals distress (Ellis, 2004).

A large amount of research on depression in NZ has been performed as part of the longitudinal studies undertaken at Dunedin and Canterbury universities. The main focus of the Canterbury studies was on maternal depression and its effect on children (see Fergusson & Horwood, 1984; Fergusson & Lynskey, 1993; Fergusson, Horwood, & Lynskey, 1995). The focus of the Dunedin studies was primarily on depression in children through to adolescence and adulthood (see Bardone, Moffitt, Caspi, Dickson, & Silva, 1996; Kashani, et al., 1983; McGee, Anderson, Williams, & Silva, 1986; Moffitt, et al., 2007, as examples). Other research groups have produced studies on depression including the uses of depression prevention programs in adolescents (see Hetrick, Proctor, Merry, Sindahl, & Ward, 2007; Merry, 2007; Merry, Hetrick, McDowell, & Bir, 2004; Merry, McDowell, Wild, Bir, & Cunliffe, 2004a; Merry & Spence, 2007).

Additionally, there were a number of studies on other aspects of depression from these sources, but they are not reviewed in this project.

Within the umbrella diagnosis of Major Depressive Disorder (MDD) there are a number of specifiers. One of the documented specifiers of MDD is "Seasonal Pattern Specifier" (American Psychiatric Association: APA, 2000, p 425). This means that the depressive episode is associated with a particular season (usually winter or summer). Seasonal depression is currently diagnosed retrospectively in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) as requiring at least two previous major depressive episodes (MDE) associated with a specific season. These must occur without seasonal psychosocial stressors, for example, work fluctuations over the previous two years; and have no intervening non-seasonal episodes. Furthermore, the seasonal episodes of depression must outnumber non-seasonal episodes over the individual's lifetime (APA, 2000). Within this framework there are two main types of seasonal pattern: the 'winter type' where the depressive episode occurs mainly in the autumn or winter months and abates in spring; and the 'summer type', which exhibits the opposite temporal pattern (APA, 2000).

The name that is commonly used in research with regard to seasonal depression is Seasonal Affective Disorder (SAD). In 1984, a contemporary definition of SAD was first proposed by Rosenthal, et al. They considered SAD to be a "syndrome characterised by recurrent depression that occurred annually at the same time of year each year*"* (p.72). Additionally, seasonal depression or SAD was characterised by a group of atypical or vegetative symptoms commonly observed with the depressive episode. These vegetative symptoms are an increased sleep (hypersomnia), increased appetite (hyperphagia) and the associated carbohydrate craving and weight gain, and increased fatigue despite sleeping for longer periods (APA, 2000; Rosenthal et al., 1984). Because the terms seasonal depression and SAD are used to refer to the same concept in the literature, they will be used interchangeably throughout this thesis.

There has been a large amount of research done on seasonal depression and SAD despite its relatively recent addition as a pattern specifier to the DSM-IV-TR (APA, 2000). The main focus of this research has been on a winter depression. However, there has been no research to date on how season affects depression and whether seasonal depression is a significant problem in New Zealand (NZ). Although there has been some research on seasonal depression performed in Australia (see Murray & Hay, 1997; Murray, Allen, Rawlings, & Trinder, 2002; Murray, 2003; Murray, 2004; Parslow, Jorm, Butterworth, Jacomb, & Rodgers, 2004; Rock, Greenberg, & Hallmayer, 2003) these studies may not be applicable in a New Zealand context. Additionally, different treatments advocated in other studies are not readily available in NZ, nor is the knowledge of how to use them effectively.

NZ is situated in a different geographical location, and has a different population makeup and needs compared to other samples examined. Therefore, it is expected that there will be some differences as regards the effects of sunshine and temperature on the sample. In this thesis New Zealand's geographical uniqueness and how the various theories and the symptoms may be expressed here are discussed.

The prevalence of seasonality and the effect that it may have on depression statistics are unknown as these variables are also unexamined in a New Zealand context. However, there is anecdotal evidence which points to the possibility of the existence of seasonal depression in NZ (Patterson, 2007). The lack of NZ research may be due to the constraints of a small population and the numbers required for research purposes in order to produce satisfactory statistical models. Nevertheless, due to the large proportion of the population affected by depression in NZ, all vulnerabilities as related to depression should be explored as seasonal depression can potentially be treated efficaciously (Freed, 2006), thus reducing the long-term health care burden of depression on the country.

The main aim of this thesis is to examine how previous research, theories of causation, and treatments regarding seasonal factors in depression apply in a NZ context. This project was based within the confines of a larger study on treatment for depression

(see Appendix A). It was not possible to definitively allocate the diagnosis of Major Depressive Disorder with Seasonal Pattern (MDD-SP) or SAD to members of this study as there is a retrospective component to the diagnoses, that is, two previous episodes of seasonal depression, which this study was unable to cater for. However, it was possible to highlight seasonal factors in relation to the current presentation, symptomology and treatment course of those who may be considered to have seasonal depression. Additionally, it was anticipated that whether seasonal depression is present in NZ would be revealed.

The treatment of those with depression and seasonal symptoms in this study is examined in relation to Cognitive Behaviour Therapy (CBT). The effectiveness of CBT was being examined in the parent study of this investigation (see Appendices A and B). Additionally, the current literature on research into depression performed in NZ is explored.

The studies in this thesis will explore the relationships between meteorological variables (bright sunlight, temperature and rainfall) and presentation, assessment and therapy of individuals for depression. The associated demographic variables of gender and age will also be examined in relation to meteorological variables at the three aforementioned stages.

Study Limitations

Although there is a large body of literature on seasonal depression and SAD not all of it was reviewed in this thesis. This study focused on the literature associated with the phase shift hypothesis, the light deprivation hypothesis, and the dual vulnerability hypothesis as theories of causation. The symptoms, and the treatments currently used for seasonal depression are also explored.

The parent study that this thesis was nested in involved the assessment and treatment of first time Major Depressive Episode (MDE) sufferers (see Appendices A and B). Those with previous episodes of depression or who were on medications that would

affect the central nervous system were not admitted into the study. Because this study was primarily studying depression only the symptoms associated with seasonal depression which are considered to be 'atypical' of depression (hypersomnia, hyperphagia and fatigue) and the time of presentation (winter) were able to be used to differentiate those who may have had a seasonal depression from those with non-seasonal depression. Therefore, all results of this thesis should be interpreted in this light. Thus, the diagnosis of seasonal depression cannot be given to anyone in this study. It was thought however, that those who were diagnosed with a major depressive episode (MDE) with symptoms associated with a seasonal presentation would be more likely to have seasonal depression or SAD than those who had the "winter blues" or slight mood change in response to season. This is because depressive symptoms form the basis of the diagnosis of seasonal depression (APA, 2000). Therefore, it is believed that this study would have some validity as to the exploration of seasonal depression in NZ.

CHAPTER TWO: DIAGNOSTIC CONSIDERATIONS, RISK FACTORS AND SYMPTOMS OF SEASONAL DEPRESSION.

In this chapter seasonal depression and SAD and how it is diagnosed is discussed. The main symptoms that are associated with the diagnosis are reviewed as regards their research findings. Additionally, the risk factors believed to be precipitants are explored.

Seasonal Depression

The effect of season on mood has been postulated as far back as Hippocrates (460-370 BC) and Socrates (384-322 BC), both of whom hypothesised that the seasons not only produced physical ailments but also mood swings in relation to the humoral responses to temperature (Wehr, 1989). Mood changes as a result of the season were also described by Kraepelin (1921, cited in Abas & Murphy, 1987). Additionally, the variation of, reproduction and migration experienced by animals which has been associated with the seasons and available photoperiod, has been well known and researched (Jacobsen, Wehr, Sack, James, & Rosenthal, 1987).

There are currently two main diagnostic systems for the diagnosis of seasonal depression; the DSM-IV-TR, and the International Classification of Diseases, Tenth Edition (ICD-10: World Health Organisation: WHO, 1994). For the diagnosis of seasonal depression in the ICD-10, there needs to be the occurrence of three consecutive annual episodes of depression, with onset and remission within a specified time (WHO, 1994). Within ICD-10 definition, seasonal depression is not separated from non-seasonal depression in any aspect other than time, as differing symptomology is not considered (Michalak, Wilkinson, Hood, & Dowrick, 2002). Comparatively, the DSM-IV-TR notes the atypical/vegetative symptoms associated with seasonal depression (winter type) are if not sufficient, are then at least commonly observed components of the disorder (APA, 2000).

The vegetative symptoms that often characterise seasonal depression apart from seasonal occurrence are prominent anergy, hypersomnia, over-eating, weight gain and carbohydrate craving (APA, 2000; Rosenthal et al., 1984; Young, 1999). These symptoms are considered to be "atypical" of depression and make winter SAD relatively easy to observe and differentiate (Rosenthal et al., 1984). Conversely, 'summer type' seasonal depression sufferers tend to exhibit weight loss, decreased sleep and decreased appetite (Jacobsen et al., 1987). These symptoms are more consistent with non-seasonal depressive symptoms; therefore with summer seasonal depression, the seasonal component of the depression appears to be the main differentiating factor (APA, 2000). The difference between the 'types' of seasonal depression symptomology may also have influenced the focus of research, in that 'summer' seasonal depression may be perceived as more difficult to differentiate from non-seasonal depression.

Although historically seasonal depression within research focuses mainly on the winter type (see Chotai, Smedh, Johansson, Nilsson, & Adolfsson, 2004a; Lewy, Sack, Singer, White, & Hoban, 1989; Michalak et al., 2002; Rosenthal et al., 1984), it is unclear whether this is because winter depression is more prevalent or because it occurs in areas of increased population and research. A contributing factor may be that winter climatic variation is seen more readily in Europe and North America, which are major contributors to the body of research. Therefore, for the purposes of this thesis, seasonal depression and SAD will refer to the winter type unless otherwise specified. Another important differential in the diagnosis of seasonal depression was considered to be the duration of the seasonal depressive episode (5-6 months) compared with MDE, as in the northern hemisphere there are also significant social stressors present in the winter period which can trigger depressive symptoms for short periods (Partonen & Rosenthal, 2001).

The development of SAD as a definition of seasonal depression has served to make it the focus of research rather than seasonal depression per se. However, it would appear that the term SAD is applied loosely to research samples with subjects grouped under the umbrella diagnosis of SAD without full clinical assessment. In a study by Murray, Allen, & Trinder, 2003, for example, seasonal behavioural engagement is used as the way of

conferring the diagnosis of SAD which is a vague indication of the disorder and a limitation of this study. This indicates that the research tends to focus on the seasonal pattern of mood observed rather than meeting the strict retrospective criteria of the disorder. Therefore, this study although it has no ability to allocate the diagnosis of SAD is similar to other studies in its approach to the exploration of a current episode of depression with seasonal symptoms.

There is a smaller body of literature that focuses on seasonal variations of bi-polar episodes and also examines mania and summer depression. The majority of this literature is situated within the tropical climates, for example, Taiwan and Brazil. In these countries the greater summer temperatures are more noticeable as a climatic variable than the winter temperatures (Lee, Tsai, & Lin, 2007; Volpe, Tavares, & Del Porto, 2008). Additionally, these countries have produced research only comparatively recently which may reflect on the amount of research focused on the summer variation of SAD. Findings within these studies were replicated in a study by Avasthi et al. (2001), who documented a predominant pattern of summer SAD in North India, which included mania. Smaller numbers exhibited winter depressive symptoms in this study; however none showed the vegetative symptoms typically associated with SAD (Avasthi et al., 2001).

Defining seasons. Seasons are often classified using the solstice and equinox dates (see Murray et al., 2003; Reardon, 2007). This is because these dates are global and are related to the earth's position in relation to the sun. Therefore, they are objective measures of photoperiod with the equinox dates being when the sun is vertical at the equator and so day and night hours are evenly distributed (Sturman & Tapper, 2006). The equinox dates are the 21st March (autumnal equinox) and the 22nd September (spring equinox). Alternatively, the solstice dates change slightly over time, and are currently around the 21st June for the winter solstice and 21st December for the summer solstice in the southern hemisphere (Sturman & Tapper, 2006). In this study solstice and equinox dates are used to enable comparisons with other studies.

Additionally, winter can also be classified by the relative decrease in temperature and quantity and quality of sunshine at a particular time of year (Sturman & Tapper, 2006). Therefore, a decrease in temperature or sunshine hours may also be used to indicate the winter season in this study.

Subsyndromal SAD and Seasonality

Another facet of seasonal depression is the presence of subsyndromal SAD (S-SAD). The diagnosis of S-SAD is used to refer to a milder form of SAD and is explored using the Seasonal Pattern Assessment Questionnaire (SPAQ) designed by Rosenthal, Bradt, and Wehr (1987; cited in Rosenthal, 1998). For a classification of S-SAD there needs to be a score of 9-11 on the Global Seasonality Score (GSS), which looks at mood, appetite, weight, sleep energy, and socializing. The seasonality score is then combined with a mild to moderate experience regarding the seasonal change symptoms and feeling worse in the winter (Kasper, Rogers, Yancey, Schultz, Skwerer, & Rosenthal, 1989). Therefore, S-SAD requires moderate physical symptoms (GSS) that fluctuate in importance with the degree of cognitive symptoms expressed by the client. The presence of MDE is not required for people to be considered to have S-SAD. However, those with diagnosed seasonal depression are considered to have moderate to severe symptoms with reportedly up to 10% requiring hospital admission (Partonen & Rosenthal, 2001).

Seasonality was described in Chotai, Smedh, Nilsson, and Adolfsson (2004b) as the degree of variability in behaviour and/or mood experienced between the seasons. However this definition would fit S-SAD as well which indicates a degree of overlap which makes separating these two conditions difficult in the literature. Overall the literature would point to seasonality being more independent of negative cognitions regarding winter or whether the seasonal changes are considered a problem (Kasper et al., 1989). The concept of seasonality was considered to be an important aspect in providing support for the diagnosis of SAD (Bauer, 1992). Furthermore, in Terman (1989), 25% of subjects in a New York study reported mood, sleep and appetite disturbances in winter with another 35% noting these symptoms but being less distressed by them. The presence of physical symptoms in a large proportion of the sample lends

weight to the assertion that a large section of population 'normals' also experience seasonality (Lacoste & Wirz-Justice, 1989). Additionally, it should be noted that seasonal changes in mood and behaviour have been said to be adaptive in children (Kovalenko et al., 2000). Therefore, it is possible that these seasonal changes in mood have persisted in some individuals despite no longer serving an adaptive function. Consequently, over time these behaviours may have become of a source of distress. This is consistent with findings by Jacobsen et al. (1987), who noted that seasonal depression commonly began in the second or third decade but was observed less frequently in children.

Seasonality has been linked with violence and suicide in a number of studies (Kim et al., 2004; Lahti, et al., 2006; Lee et al., 2007; Marušič, Landau, & Tomori, 2003; Rock, Judd & Hallmayer, 2008). However the results were ambiguous, with some studies showing a positive association between seasonality and suicide (Lahti et al., 2006; Marušič et al., 2003), but not others (Kim et al., 2004; Rock et al., 2008). On the other hand, a number of studies showed similar findings regarding aggression. In these studies, non-lethal attempts at suicide and lower levels of violence tended to show greater seasonality than more extreme actions (Rock et al., 2008; Volpe et al., 2008). These studies also served to reinforce the correlation of anger and seasonal depression shown by Winkler et al. (2006).

Seasonal changes in behaviour have been linked to factors other than sunshine hours. Rainfall and temperature are also said to have significant effects on mood. A study by Lee et al. (2007), found that the aforementioned climatic variables can have a rapid effect on mood. However, as this study focused on bi-polar admissions some of the relationships found may be different to those experienced with unipolar depression. Additionally, Lee et al.'s study was based in Taiwan which experiences a minimal temperature range of 20- 26.8 ^{o}C. However, it remains possible that countries that do not experience large variation in actual photoperiod or temperature may still have people at risk for seasonal depression if they experience significant periods of rainfall. This is supported by Rosenthal et al. (1984) who found a number of people were adversely affected by ongoing cloudy weather. Thus countries similar to New Zealand whose

winters are typically wet rather than extremely cold and which do not experience a marked decrease in photoperiod, may have a population of seasonal depression sufferers that is undiagnosed or recognized. This may have ramifications as to what measures are best suited for the study of seasonal depression in a NZ context, that is, would rain or sunshine be considered to be a more appropriate variable in relation to mood?

On the other hand, a study by Huibers, de Graaf, Peeters, and Arntz (2010) explored weather and mood associations in a population screening for depression. They reported finding no association in their sample of 14,478 people and concluded that there was no relationship between weather and sad mood. However, their data were not longitudinal and so may miss subtle changes over time may be overlooked. Additionally, the data did show seasonal peaks for depression in summer and fall which indicates some measure of seasonality in the sample although this was not elaborated on (Huibers et al., 2010).

Overview of Weather Characteristics of Auckland

The main influence on weather in New Zealand (NZ) comes from its global position and mountainous terrain (Sturman & Tapper, 2006). New Zealand is situated in the mid-latitude band (34- 47 ^{0}S) which results in a weather pattern dominated by frontal systems from the west. These frontal systems produce frequent changes in air pressure which result in the regular formation of clouds and precipitation (Sturman & Tapper, 2006). Additionally, the clouds in the atmosphere scatter solar rays and therefore affect light intensity (Sturman & Tapper, 2006). Large amounts of water vapour are also present in the atmosphere which is a result of the regular precipitation, vegetation, and being surrounded by ocean, which serves to maintain stable temperatures. Water vapour is measured as humidity which ranges from 0-100% and indicates the closeness of the atmosphere to saturation with moisture (Sturman & Tapper, 2006).

Auckland is situated at latitude of 37 ^{o}S and experiences warm humid summers and mild winters with prevailing westerly winds (New Zealand Meteorological Service 1983, cited in Sturman & Tapper, 2006). The average humidity experienced in Auckland is 78.8% (National Institute of Water and Atmospheric Research: NIWA, 2009) and serves

as a stabilizing factor for temperature in this region (Sturman & Tapper, 2006). However, the humidity results in considerable amounts of cloud formation which serves to limit the amount of sunlight experienced. An example of this can be seen in the difference observed between sunrise and sunset times for a month and the average amount of bright sunlight hours recorded. In January of each year, sunrise is at approximately 0615 hours and sunset at approximately 2035 hours which gives a total daily photoperiod of just over 14 hours (Royal Astronomical Society of New Zealand: RASNZ, 2011). However the average daily hours of sunlight recorded in January was documented as being 7.5 hours per day (NIWA, 2009). Additionally, in winter, the average amount of daily bright sunlight was 4- 4.5 hours per day, compared with the total photoperiod of 10 hours at the winter solstice (RASNZ, 2011; NIWA, 2009). Thus total photoperiod does not accurately represent light exposure in this setting. The variability of bright sunlight experienced in the winter and summer is shown in Appendix D which is a graphical representation of sunlight hours over the duration of the study.

Auckland experiences approximately 2,050 hours of sunshine a year (NIWA, 2009). Bright sunshine is measured as being greater than 120 Wm^2 (NIWA, 2009). Watts per metre squared directly measures the intensity of solar radiation (Sturman & Tapper, 2006). This measurement is used in other research studies and is based on international measurements standards documented by the Commission for Instruments of Measurement Organisation (CIMO: World Meteorological Organisation: WMO, 2008). The other measurement commonly used in research studies exploring the effect of light intensity and therapy on seasonal depression is lux. It is not possible to directly convert watts per metre squared into lux as they measure different constructs; however, Partonen (2001) documented that outdoor light ranged from under 2000 lux on cloudy days to 50,000 lux in direct bright sunlight (p.66). Therefore, it is assumed that the recorded bright sunlight hours would represent adequate amounts of lux exposure to facilitate comparisons with other research.

Hypothesis

In light of these variable findings as regards seasonality and mood, and how weather is manifest in Auckland, this study plans to explore whether season influences presentation in New Zealand. Presentation in this study will involve initial enquiry about participating in a research project on therapy for depression. This question will be tested using the hypothesis:

Hypothesis 1: More people will present in the winter months (May-August) than over the rest of the year.

Physical and Cognitive Symptoms associated with Seasonal Depression

Physical Symptoms

In a landmark study by Rosenthal et al., (1984), 29 people were accepted into a study that described symptoms associated with SAD. Additionally, this study documented preliminary findings using light therapy (LT) as a treatment (Rosenthal et al., 1984). For three of the sample, seasonal cycles started in childhood but for the majority seasonal fluctuations in mood began after the age of 20 years. Many of the participants believed that the cycles became more severe with age and that it took some time for the pattern to be seen. All participants reported decreased physical activity. In 66% appetite increased and 79% reported carbohydrate craving and weight gain (2-5 kg). Additionally, some originally cited increased appetite as a symptom but as the depression deepened their appetite decreased. Ninety-seven percent of the subjects reported sleeping longer and waking later but not sleeping soundly. This was combined with drowsiness during the day and late afternoon worsening of energy and mood which was consistent with a phase delay. These physical symptoms are considered to be the hallmark vegetative or atypical features that are associated with seasonal depression, however not all subjects experienced all symptoms. Subjects in this study also found that their ability to concentrate decreased as well as their motivation; they generally withdrew socially and had experienced increased levels of irritability and suspicion. Light therapy was noted to be effective for those treated with bright light (n = 11). However for those treated with

dim light there was little change in mood and symptoms, and some appeared to worsen (*n* = 3). This study was the first to highlight SAD as a separate disorder as opposed to a variant of recurrent depression. Although the study mainly consisted of a group with bipolar disorder they were documented as having regular depressive episodes in winter. It is likely that this group was used because of their definite seasonal mood changes which made exploration of the disorder easier; however this was not expanded on which is a limitation of the study.

The symptomology of those with SAD was observed to be different to that commonly displayed with a typical MDE (Rosenthal et al., 1984). Additionally, the cyclical pattern and vegetative symptoms made the syndrome relatively easy to differentiate from non-seasonal depression. Furthermore the presence of vegetative symptoms was considered to be the best predictor of those with a seasonally bound disorder (Partonen & Rosenthal, 2001).

A temporal relationship, with the vegetative symptoms having an earlier onset than mood symptoms, was postulated by Young (1999), which facilitated differentiation from other mood disorders. This was supported by Whitcomb- Smith (2004), who found in a study exploring the relationship between vegetative and mood symptoms and cognitive factors, those with a history of SAD showed earlier vegetative symptom onset. Individuals with SAD also indicated more ruminative responses and negative automatic thoughts around the seasons (Whitcomb-Smith, 2004).

However, in a study by Reardon (2007), the above findings were not replicated. No temporal relationship was found between changes in energy and appetite and mood, although there were three clusters found which showed changes in energy/ motivation, cognition/affect, and appetite which seem consistent with Whitcomb-Smith's (2004) findings. It was possible that the samples in both studies were different which would account for the divergent findings. However, the sample size in Reardon's study was not documented in the published dissertation abstract, neither were any demographics available in either study that would facilitate comparison on these variables.

Therefore, although some studies indicate a temporal relationship between vegetative symptoms and the cognitions, this is not fully supported by research. An alternative view postulated by Rohan, Sigmon, and Dorhofer, (2003) is that that the relationship may begin as temporal, but over time and subsequent episodes, the linearity of the relationship may become less obvious. As a result, over time cognitions about the symptoms may present before the vegetative symptoms themselves and become an anticipatory factor which would result in a more simultaneous presentation. Therefore, the direction of this relationship remains unclear and requires further investigation.

Increased Appetite

Increased appetite is a common and distressing symptom commonly observed with seasonal depression. The distress is often related to the associated weight gain as much as to the increased appetite (Rosenthal et al., 1984). Historically, the majority of SAD sufferers appear to be female and overeating combined with weight gain has been cited as a trigger for depressive symptoms in women (Rawana, 2006). This is supported by Chotai et al. (2004a), who assessed gender differences in seasonally related mood and behaviour change in Sweden over October to February. The sample ($N = 2620$) consisted of 55.6% women and found that seasonal weight change was considered to be more of a problem by women compared to men. Additionally women who felt worse in winter were represented in the 'winter eaters' group significantly more often than men. Therefore, it would seem that women are more likely to have negative cognitions regarding increased appetite than men and the negative cognitions may have precipitated the development of seasonal depression in this sample.

In a study by Rawana (2006), 179 undergraduate and community participants were grouped into non-seasonal depressed, control, SAD and S-SAD groups. Those in the SAD group showed more atypical/vegetative symptoms. Additionally, those who were diagnosed with SAD tended to have stronger beliefs that engaging in restrained eating was positively associated with a sense of accomplishment. Rawana's subjects with SAD also tended to experience more stress associated with the vegetative symptoms and were

more preoccupied with thoughts about food than sub-syndromal or non-seasonal depression sufferers. It was found in Rawana's study that those with SAD could be separated from other depressed groups by their dysfunctional eating cognitions and behaviours.

In Rosenthal et al.'s (1984) study, eating was not associated with pleasure and participants used terms like "craving" and "compulsion" in describing their symptoms. These findings appeared to be in line with Rosenthal et al's concept that there was a psychological component as regards attitudes towards food with these terms indicating negative thoughts toward eating as much as a physical drive. However, in a contrary view Moller (1991) postulated that increased carbohydrate craving was physiologically based and was the body's response to low mood and decreased serotonin levels, and that the body was seeking to self-medicate. It was believed that increased carbohydrate consumption would increase levels of serotonin and result in the relief of vegetative and mood symptoms (Moller, 1991).

Overall, it appeared that the relationship between hyperphagia and the cognitions surrounding it had a greater effect as regards the development of seasonal depression than the physical symptom alone. Therefore, it appears that this symptom is important to consider when assigning a diagnosis of seasonal depression.

Increased Sleep

Increased sleep (hypersomnia) is also regarded as a classic symptom associated with seasonal depression. In Rosenthal et al.'s (1984) study 97% of the sample experienced increased sleep and lack of refreshment from sleep. Nine subjects in Rosenthal et al.'s (1984) study were examined with EEG sleep studies in summer and winter. In these subjects there was noted to be an increase of 17% in total sleep time and 23% in sleep latency. Additionally, there was a corresponding 46% decrease in slow-wave (delta) sleep, and a trend toward more frequent waking during winter (Rosenthal et al., 1984). The findings regarding changes in sleep were replicated in a paper by Jacobsen et al. (1987). In this paper support was provided for increased sleep length and decreased delta

wave sleep. Furthermore, a study by Rastad, Sjödén, and Ulfburg (2005) found that in a sample of 2500 individuals, those with SAD had comparatively greater increased sleep than other groups.

Delta wave sleep and sleep efficiency were found to improve with light therapy in a group of SAD patients (Skwerer et al., 1989). However, in this study, the control group, that is those with typical depression, exhibited the opposite effect when exposed to LT. This indicated that different mechanisms around sleep were activated in SAD patients compared to non depressed subjects (Skwerer et al., 1989).

Chotai et al. (2004b) found that men considered increased sleeping to be more of a problem than women. However, these results regarding males were not replicated in other studies (Jacobsen et al., 1987; Rastad et al., 2005; Rosenthal et al., 1984). Therefore, gender is unlikely to play a significant role in the manifestation of this symptom.

Additionally, as regards children with SAD, the majority reported having poor quality and decreased sleep (Sonis, 1989). Conversely, in the study by Rosenthal et al. (1984), a number of the sample had no change to sleep patterns or, although they originally had increased sleep, their sleep decreased as other symptoms worsened. Therefore, the differences observed in research findings indicate that increased sleep is not a symptom common to all sufferers and sleep disturbances can be experienced in a variety of ways and degrees.

Fatigue

Fatigue was noted as a symptom commonly associated with both depression and SAD (Beck, Rush, Shaw, & Emery, 1979; Rosenthal, Sack, Skwerer, Jacobsen & Wehr, 1989). As this symptom was believed to be primarily a result of sleep disturbances commonly observed in both seasonal and non-seasonal depression, it is unlikely that this symptom could serve as the basis for diagnosis or be in any way a significant point of

difference between them. Conversely, it would seem that this symptom is one of the overlapping symptoms that tie MDD and SAD together.

Overall, it appears that although physical symptoms are a major indicator of seasonal depression they are not always present to the same degree in all sufferers. Therefore, caution needs to be applied when deciding whether a person has seasonal depression purely on the presence of a single physical symptom. The presence of more than one vegetative or atypical symptom in line with a seasonal presentation would seem to be the more cautious and accurate approach. This would aid the accuracy of the diagnosis and facilitate better treatment approaches that would result in maximum benefit for the person involved. Furthermore, it would seem that the presence of depression would need to be the major consideration in the study of seasonal depression rather than the presence of physical symptoms alone. The concurrent depression would be especially relevant where the retrospective portion of the diagnosis was not examined or where predictive ability as regards future episodes is required.

Cognitive and Mood Symptoms

There are a number of studies that explore the cognitive and mood symptoms associated with seasonal depression. Additional to the symptomology commonly associated with MDE, people who suffered from SAD were noted to experience a greater dread of winter (Rawana, 2006). This factor may further serve to differentiate them from non-seasonal groups. Additionally, a study by Whitcomb-Smith (2004), found that in her study of 67 participants, those with SAD history had more ruminative responses and negative automatic thoughts about the seasons. Furthermore, Engasser and Young (2007) found that after controlling for vegetative symptoms, SAD patients in their study had a more ruminative response style and a greater internal attribution style for negative events. Moreover, subjects experienced more severe mood and cognitive symptoms associated with depression over winter (Engasser & Young, 2007). This result was in line with findings by Golden, Dalgleish, and Spinks (2006), who believed that underlying dysfunctional attitudes towards winter characterized those with SAD. In addition, Boulard (2004) found that those with a history of SAD exhibited negative thinking and

responding styles similar to those with major depression even during asymptomatic months.

Additional mood symptoms

Anger attacks have been associated more often with SAD clients than those with non-seasonal depression (Rock et al., 2008; Winkler et al., 2006). Moreover, increased levels of irritability and paranoia have been previously noted in those with SAD (Rosenthal, 1984). In Winkler et al.'s (2006) study, 36 SAD patients were compared with 24 non-seasonally depressed controls. In this study, anger attacks were associated with more vegetative symptoms and an increased number of behavioural outbursts (Winkler et al., 2006). Additionally, the ages of those with increased anger attacks appeared to be younger than SAD clients who did not have anger attacks. These results indicated that anger and irritability may be a cognitive symptom of interest in future studies exploring seasonal depression.

Another symptom associated with seasonal depression has been mania. The occurrence of episodes of hypomania or mania at the end of the seasonal episode serves to link the disorder to the bipolar disorders (APA, 2000). However, it was noted by Sato, Bottlender, Sievers and Moller (2006) that seasonality associated with depressive episodes was thought to be different to bipolar disorders, where a large seasonal peak in spring was observed for those with unipolar depression without depressive mixed states, whilst both bipolar and unipolar episodes with depressive mixed states experienced an autumn peak.

Hypotheses

The vegetative symptoms of seasonal depression including increased appetite, sleep and fatigue associated with a decreased mood are the cornerstone features of seasonal depression. Therefore how they are manifested in this investigation is important to explore. It is not possible to explore anger directly as it is outside of the confines of this study. Therefore, the use of irritability will be examined instead in order to gather some

experience of the symptom. These symptoms will be examined in more detail in the studies of this investigation and are listed here under the general hypotheses of:
Hypothesis 2: People who present in the winter period (May- August) will exhibit higher depressive and vegetative symptom scores than those who present over the rest of the year.
Hypothesis 3: Vegetative symptoms will be positively associated with depression scores.
Hypothesis 4: Irritability scores will be positively associated with vegetative symptom scores.

Overall, the studies exploring cognitive aspects of seasonal depression had similar limitations. The studies that conducted a more rigorous assessment as to whether the participants met the criteria for SAD or seasonal depression, tended to have smaller sample sizes (see Engasser & Young, 2007: $N = 59$) which does tend to limit the reliability of their findings. Other studies that used a larger sample size (see Rawana, 2006: $N = 174$) tended to rely on retrospective self report measures in order to assign diagnosis and so their findings may also have limitations as regards the ability to validate results and potential bias in the respondents.

Risk Factors

Age

Of those who experience seasonal depression, the majority are believed to be in the younger age bracket, that is, under the age of 25 years (APA, 2000; Chotai et al., 2004a). However, the reason for this is not well known as there is limited research in this area. Seasonal depression is known to exist in children although believed to be less common than in adults (Sweedo et al., 1995). Furthermore, from an evolutionary perspective, there has been some speculation that the seasonal fluctuations in mood serve a protective function in children, (Kovalenko et al., 2000). Since the decrease in sunlight is associated with stormy and cold weather, the changes in mood and energy levels are thought to keep children close to their parents, and the associated decrease in activity is believed to help children preserve energy during the colder months (Kovalenko et al., 2000).

According to Hoagwood and Johnson (2003), it was estimated that 16-22% of children had a diagnosable psychological disorder, with a severely limited number of them receiving any help. A factor cited as being of importance in the development of these disorders and their control was the school organizational culture (Hoagwood & Johnson, 2003). In relation to the development of seasonal depression it may be that the increasingly competitive school environment had led to a decrease in the natural sunlight exposure that children may have experienced in less competitive times. As a result of current school culture, outdoor work/play has been replaced by increased study or indoor activities that may trigger and maintain seasonal vulnerabilities in some. This supposition is partially supported by Sund, Morken and Linaker (2002) who explored children's calls to help lines. Their results showed an increase in the number of calls associated with decrease in length of the day, and increased latitude. Additionally, Morken, Sund and Linaker (2004) showed an increase in children's depression, and anxiety, which peaked in the winter months. These findings indicate that children may become more distressed when they are unable to experience a certain amount of natural bright light associated with being outside and the summer months.

The existence of SAD in children has been postulated to increase with the onset of puberty, with approximately 1-5% of children exhibiting symptoms consistent with the diagnosis of seasonal depression (Sweedo et al., 1995). It is also possible to speculate that current lifestyle practices served to maintain the childhood patterns mentioned in Kovalenko et al. (2000) and inhibit what may be part of physical development into adulthood. Furthermore, it is possible that long-term patterns may become established in adolescence by the increasing amount of time spent on computers and other indoor pursuits as well as the easy availability of high carbohydrate foods. Whereas previously young people would have been outside for longer periods as they became older, this is no longer the case. Additionally, one may speculate that the large amount of physical adjustment occurring within the bodies of young persons at this time may expose a vulnerability to depression that they are unable to counter without increased light exposure. Exposure to light is considered to be important in order to help regulate

neurotransmitter production during the winter months (Lewy, Wehr, Goodwin, Newsome, & Markey 1980; Lewy, Sack, Singer, White, & Hoban, 1989).

Furthermore, in Amons, Kooij, Haffmans, Hoffman, and Hoencamp (2006), it appeared that there was a frequent comorbidity (27%) found between SAD and Attention-Deficit /Hyperactivity Disorder (ADHD). The relationship between adult residual ADHD and SAD was also explored by Levitan, Jain, and Katzman (1999). Therefore, seasonal depression may be present in a number of children, but comorbid disorders serve to disguise the symptoms. Additionally, pediatric depression is recognized as difficult to diagnose due to the inherent difficulties involved in ascertaining the internal affect state of the child by either the parent or the child themselves (Ryan, 2001). Therefore, although seasonal depression is not commonly found in children, this may be a result of expectancy or demand effects and the inability of the child or parents to recognize patterns of symptom expression. This finding is supported by studies performed by Giedd, Sweedo, Clark, and Rosenthal (1997), and Sweedo et al. (1997), whereby a number of children were found to have SAD and were effectively treated with light therapy.

The majority of research on adolescence and seasonality appears to focus on suicide (Lahti et al., 2006; Marušič et al., 2003). Although there appeared to be a correlation between seasonality and suicide, the season in question appears to produce diverse findings. Mood changes are noted to be common among young people in Finland and a decrease in sunshine hours coincided with an increase in suicide (Lahti et al., 2006). This study showed an autumnal peak in suicidal behaviour. However, a study by Marušič et al. (2003) indicated a spring peak, which was more in line with the adults in the Lahti et al. (2006) study. Both aforementioned studies mentioned school as a stressor that may have influenced results. This served to reinforce Hoagwood and Johnson's (2003) belief regarding school culture having a significant effect on the development of psychological disorders in children. However, as the association between SAD, suicide and adolescence is understudied, these results are indicators of seasonal change in mood at most.

Nevertheless, the correlation between suicide and season indicates that young people may experience a greater depression in relation to season.

The supposition that younger people more frequently exhibit symptoms associated with seasonal depression is contrary to findings by Rosenthal et al. (1984). In their sample, individuals were noted to have increasing difficulties with age. Furthermore, Jacobsen et al. (1987) postulated that SAD was more prevalent in the second and third decades. Therefore the association between age and seasonal depression requires further investigation.

Hypotheses

There have been few studies which have examined age as a predictor of symptoms associated with seasonal depression. Age has been previously associated with depression especially in regards to irritability and suicide (Lahti et al., 2006; Kovalenko et al., 2000; Rock, et al., 2008). However, there is limited research into the expression of vegetative symptoms across the life span. Therefore, this investigation intends to use age as an independent variable in relation to presentation with symptoms consistent with seasonal depression.

Hypothesis 5: The age of persons presenting in the winter months (May –August) will be lower than those applying at other times of the year.

Hypothesis 6: Age at presentation will be negatively correlated with: a) vegetative symptoms; b) depression scores and c) irritability.

Gender

The majority of those who suffer from seasonal depression are reported to be female (APA, 2000). It was unclear as to whether there is an additional risk factor for women as regards seasonal depression compared to Major Depressive Disorder (MDD); however women appeared to comprise of 60%-90% of those with a seasonal pattern (Amons et al., 2006; APA, 2000; Levitan et al., 1999; Sweedo et al., 1995). Whether this was because women present more in the winter depression group as opposed to males who tended to feature more commonly with summer depression (as seen in seasonality and suicide and

violence studies, see Lahti et al., 2006; Marušič et al., 2003; Yip, Tang, & Qin, 2006) is unclear. Other gender differences were explored by Chotai et al. (2004a), who found that women tended to react negatively to temperature change more than men, whereas men reacted more to sunshine related changes (i.e. negatively to cloudy days). These findings are supported by Harmatz et al. (2000) who found that women had greater fluctuations in Beck Depression Inventory (BDI) scores in relation to seasonal variation in comparison to men. These differences indicated a possible difference in vulnerability factors between the genders that requires further research.

Hypotheses

Gender appears to have a significant association with seasonal depression. Therefore in this study gender will be explored in relation to the following hypotheses:

Hypothesis 7: Higher numbers of women will present in the winter months (May-August) compared to men.

Hypothesis 8: Women will have higher vegetative and depressive scores compared to men at presentation and on days with lower temperatures.

Hypothesis 9: Higher numbers of men will present during days of lower sunshine hours compared to women.

Hypothesis 10: Higher numbers of women will indicate increased appetite as a symptom compared to men.

Hypothesis 11: Men will have higher levels of irritability compared with women.

Aspects of Personality

The five factor model has also been examined in relation to SAD. A study by Enns et al. (2006) showed that participants with SAD had different profiles as compared with 'non-seasonal depressed patients' and non depressed participants ('norms'). The results revealed higher scores on openness for the SAD group compared with non-seasonal depression patients. This indicated that as a group those with SAD were more open to new experiences than non-seasonally depressed persons (McCrae & John, 1992). Additionally, SAD participants also recorded lower neuroticism scores than non-seasonal depressed patients, but their scores were higher than the norms group. Neuroticism scores

indicated a higher tendency to experience unpleasant emotions more easily than the non depressed group (McCrae & John, 1992). Moreover, the SAD group's neuroticism and extraversion scores were observed to change with the changing mood state; that is, with increased mood there was increased extraversion (Enns et al., 2006). The elevated openness scores appeared to be unique to SAD thus providing some support for the usefulness of personality assessment in patients with SAD in order to differentiate them from those who are non seasonally depressed (Enns et al., 2006).

Other personality components have been implicated in the development of seasonal depression. One such concept is the "larks" versus "owls" or the "morning" versus "evening" types of behavioural differences (Lacoste & Wirz-Justice, 1989). This concept was related to the phase shift hypothesis (PSH), in that some studies found that "evening types" shift their sleep onset and wake-up times throughout the year whereas "morning types" do not, resulting in an altered duration of sleep with the seasons (Lacoste & Wirz-Justice, 1989). Overall, findings indicate that evening types tended to be represented more in SAD samples (Lacoste & Wirz-Justice, 1989). However, prudence needs to be considered before ascribing eveningness as a risk factor, as other factors including cultural and social conditions may be underestimated in their involvement (Natale, Adan, & Scapellato, 2005).

Other conceptualisations of personality characteristics have also been explored. One such study by Maeno et al. (2005) used the Tri-dimensional Personality Questionnaire (TPQ). A sample of $N = 6{,}135$ was used in the study. Results found that those with winter SAD had higher levels of "Harm Avoidance" in relation to their level of depression. This result was consistent with previous results by Sachs, Jain, Truman, Blais, Otto, and Hirschfeld (1996) where high harm avoidance was thought to resemble negative automatic thoughts (NATs) in depressed patients. The levels of NATs were higher for the SAD group than controls and were similar to those experienced by those with non-seasonal depression (Maeno et al., 2005). These findings suggest that the presence of NATs in seasonal depression would make treatment with cognitive therapy a reasonable choice or a viable alternative to traditional therapies. This is related to the

theory of Anxiety Sensitivity (Reiss, 1987 cited in Young 1999) where by these individuals are more likely to monitor vegetative symptoms and report higher levels of distress around these symptoms. Therefore these individuals are likely to be more motivated to seek help around these symptoms.

The higher harm avoidance observed in Maeno et al.'s (2005) study may show some relation to findings by Eagles et al. (2002), whereby SAD sufferers were found to be heavy users of the health care system. In this study, those with SAD presented with a wider range of symptoms, had more investigations, received more prescriptions and had more referrals to secondary care. These factors indicated a possible co-morbidity with personality factors (Eagles et al., 2002). Additionally, their sample appeared to display greater help-seeking behaviours than other groups.

Overall, exploration of personality factors suggests that those with seasonal depression use the health care system more often than other groups. Therefore, identifying seasonal depression and treating it effectively may be a significant cost-saving measure with positive flow-on effects for the public health system.

Genetics

The majority of mood disorders including MDD and seasonal depression appear to have a genetic component, in that those with first degree relatives who have depression are more likely to develop the disorder than those who do not (Carlson, Miller, Heath, Donahoe, & Martin, 2010). Additionally, heritability estimates for depression were thought to be 30 - 40%, which was almost double the risk for the individual without a family history (Burton, Westen, & Kowalski, 2009).Therefore, it would seem that there are inheritable factors that predispose people toward the development of mood disorders.

However, in a study by Michalak et al. (2002), 25 people diagnosed with SAD were interviewed. In that study, family history was not indicated as a significant factor of presentation in seasonal depression. However, for those in the same study with non seasonal depression, family history was indicative of diagnosis of depression (Michalak

et al., 2002). Contrary to other findings, a study by Madden, Heath, Rosenthal, and Martin (1996) showed that seasonal variations in mood and behaviour did show some familial tendencies, supporting the hypothesis of a biological predisposition. Therefore, it would seem that the role of genetics in the development of SAD as typical/ non-seasonal depression requires further clarification.

Environmental and Social Causes

In a study by Sweedo et al. (1995), mothers who reported their children's "winter blues" symptoms, were thought to possibly be projecting their own symptomology. The maternal responses raise the question of inheritability/genetics versus environmental causes. It was hypothesised that the mothers may have 'seen' what they would interpret as the winter blues, but the child or an independent observer may not agree (Sweedo et al., 1995). Consequently, the manifestation of vegetative behaviours that may be considered appropriate and evolutionarily adaptive in children may have been interpreted by the parent as indicative of depression as a result of parental beliefs around what was 'normal' versus pathological behaviour. Therefore, it was believed by the researchers that the mother would have treated the child as depressed, or may have facilitated the development of predisposing factors, by facilitating vegetative behaviours over time (Sweedo et al., 1995). The researchers believed that this in turn would have nurtured an environment whereby the child may have gone on to develop SAD. In light of the complexities surrounding family dynamics, separating family history and environment remains an ongoing challenge, especially in relation to diagnoses for children (Burton et al., 2009; Kendler, Karkowski, & Prescott, 1999).

Other risk factors suggested as predictive of SAD are numerous negative life events, decreased levels of social support, and being an immigrant (Kendler et al., 1999; Michalak, Wilkinson, Hood, Dowrick, & Wilkinson, 2003). Environment was also believed to play an important role in the underlying predisposition, with early childhood experiences indicated in the etiology of depression (Burns, Andrews, & Szabo, 2002).

Another factor that was thought to lead to depression especially in women was the lack of intimate relationships (Brown & Harris, 1989). Additionally, low self esteem and lack of social support were indicated as a moderators of SAD in some studies (McCarthy, Tarrier, & Gregg, 2002; Michalak et al., 2003). Furthermore, both of these factors when combined with anxiety symptoms were observed to result in the fastest onset of depression (McCarthy et al., 2002). Therefore, it would seem that there are a number of factors which indicate the importance of environment in the development of depression and possibly SAD. Additionally, culture, ethnicity and geography may also play a significant part in how seasonal depression is experienced.

Ethnicity, Geography, Culture and Seasonal Depression

Ethnic and cultural differences can be related to how depression is observed and manifested (Young, 1999). This may influence the interpretation of results from studies in countries that experience large variation in seasonal photoperiod. Extended periods of low light and temperature may influence winter behaviours and affect what is considered to be adaptive versus abnormal symptoms. An example of this may be seen in Axelesson, Stefánsson, Magnússon, Sigvaldason, and Karlsson's (2002) study, where a specific population group of native Icelanders (Inuit) were examined in relation to their symptoms of SAD. In this study, there was some evidence of SAD, but levels were not greater than other population groups (Axelesson et al., 2002). A limitation of this study was that there may be some differences as to what was considered to be 'normal' versus 'abnormal' behaviour in winter. In this situation vegetative symptoms and decreased activity may be considered to be normal and adaptive therefore there may not be the accompanying distress exhibited by these symptoms that would constitute a seasonal depression because of this. Therefore, what may be viewed by one population as normal adaptive winter behaviour versus what would constitute an abnormal increase in vegetative/depressive symptoms would depend on the context and social interpretation. Another limitation of this study is the specific population used may have stayed with their historical ways of living within the environment which would imply a greater flexibility to life in regards to season which may not be seen on other environments.

Differences between people from different countries have been explored in various studies and articles (see Young, 1999; Rosenthal, 1998) and have been found to affect how depression and SAD are observed. An example of this is discussed by Dr Rosenthal in his book "The Winter Blues" (1998). In a section of the book a person had lived for much of her life in Finland and was described as a 'gregarious person'. However, when she moved to the United States she was asked regularly whether she was depressed (Rosenthal, 1998). The person did not feel that she had changed in her mood. Nevertheless, she believed that others perception of her 'normal' behaviour was in relation to different cultural norms which may have led to an unfounded presumption of distress. Therefore, it is possible that normal winter behaviour in one country could be viewed as abnormal in another, thus making diagnosis flawed in relation to certain cultural population comparisons without appropriate assessment. This has implications for the application of the DSM-IV-TR as regards seasonal depression in that cultural norms need to be considered before making the diagnosis.

Another aspect of geographical differences would appear to be the type of seasonal depression manifested by specific countries. A study by Kasof (2009) found that winter SAD was more prevalent in countries with higher individualism and lower power distance, whereas the opposite pattern was exhibited for summer SAD. A major limitation of this study would seem to be that this division could as easily be explained by meteorological factors as countries more often associated with summer SAD are also known for their comparatively high summer temperatures and sunshine e.g. India (see Avasthi et al., 2001). Added to this is the research that indicates those with darker skin and eye color require greater exposure to sunlight for health and mental well being (Kimlin, Harrison, Nowak, Moore, Brodie & Lang, 2007). This would point to some ethnic differences that may be important to New Zealand especially as regards those who have emigrated from countries with a higher amount of sunlight than is experienced in this country.

Overall, the concepts of culture often include ethnicity and geography and may be difficult to ascertain in many studies. This can make findings from these studies

complicated to apply to New Zealand. Findings around cultural differences in the diagnosis of seasonal depression are important for New Zealand in relation to its stance as a bicultural country. Therefore, the need to be aware of what constitutes depressive symptoms and abnormal physiological changes in response to seasonal change would need to be investigated appropriately for both Maori and Pakeha in order to be able to make the diagnosis a valid one for each group (Clark, 2008). There are reported differences between Maori and non-Maori in NZ as regards their presentation and diagnostic patterns with depression (Tapsell & Mellsop, 2007). However, other studies have indicated that depression is not viewed differently by Maori in comparison to non-Maori (Marie, Forsyth, & Miles, 2004; Marie & Miles, 2007). These findings indicate that seasonal depression may also be viewed in a similar manner across cultural groups in NZ however this requires further investigation.

Apart from being a bi-cultural country, NZ is also the home to a large percentage of Pacific peoples, and has a considerable percentage of the population documented as being of Asian extraction. The varied makeup of the population in comparison to other research samples may make direct comparison of findings difficult. Although there are some similarities between sample characteristics, there are a number of cultural, ethnic and geographical characteristics which may influence results. Overall, culture is an aspect of seasonal depression that needs to be considered carefully. Additionally, this may influence the amount of exposure that the person receives as some cultures can require a greater amount of indoor living and covering of the face and body than others (Kimlin et al, 2007).

The Debate over the Diagnosis of Seasonal Depression

There has been some debate over the diagnosis of seasonal depression. The presence of diagnostic criteria for seasonal depression in the DSM-IV-TR and the ICD-10 does not seem to be enough for some to consider the diagnosis valid. Although the research reported here would appear to validate the condition, detractors tend to view seasonal depression as an ‘atypical’ variation of depression rather than a separate and valid diagnosis (Grof, 2002). An example of this debate appeared in an editorial for the

Canadian Journal of Psychiatry (2002). In his letter to the editor, Grof stated that he "was of the opinion that there was no justification for the diagnosis of SAD" (p.786). This led to an exchange with Michalak and Lam (2002) who believed that Grof's position was based on "idiosyncratic opinion, rather than any scientific basis*"* (p.786). As the diagnosis of seasonal depression has been subject to repeated revision since its first proposal in 1984 (Michalak et al., 2002), it would seem that the classification of the disorder may still be in its infancy. For some, the definition still needs further refining in order to distinguish it from more parsimonious explanations of atypical depression (Stewart, Quitkin, Terman, & Terman, 1990).

Atypical depression has been described as presenting with similar symptomology as SAD however atypical sufferers reported no seasonal pattern to their symptoms (Stewart et al., 1990). Additionally, they were also found to have a different treatment response. The effects of light therapy on this group have been observed to be minimal and improvements in symptoms have been related primarily to antidepressant medication (Stewart et al., 1990). However, the similarities in symptom expression between the groups may cloud diagnosis and lead to incorrect application of either term.

Further, support for the diagnosis of SAD was offered in a study regarding Quality of Life (QoL) in patients affected by seasonal depression. Here, Michalak et al. (2005) investigated perceived QoL of 26 patients diagnosed with SAD. The study showed a significant improvement in QoL scores during the summer months as opposed to their winter scores. The authors believed that these results provided support for the distinctness of SAD from atypical depression. The diagnosis of seasonal depression has also received support from Gorman (2005). In his article Gorman discussed the "clock" genes that regulate circadian activity and make SAD a real and predictable disorder for many people. It was believed that those who suffer from seasonal depression had an inability to shift their circadian rhythms in response to seasonal light levels (Bunney & Bunney, 2000).

The debate around the diagnosis of seasonal depression was echoed by Murray et al., (2003) who noted that SAD was typically the extreme end of a continuum with variations in the intensity of symptoms present within the normal population. Overall, it is perhaps the variation in symptom intensity and its overlapping presence in a large proportion of the population that makes seasonal depression difficult to differentiate from the "winter blues" and serve to detract from its impact. This is supported by an article (Velamoor, 1991) which examined the case of a woman who retrospectively was found to have SAD, but was undiagnosed and had been consequently hospitalized many times for depression. This resulted in a lifetime of disability for her that would have been more effectively managed had she been assessed and diagnosed effectively (Velamoor, 1991).

The variety of assessment tools used for ascribing the diagnosis of SAD for research samples further adds to the dilemma. The Seasonal Pattern Assessment Questionnaire (SPAQ) is used in a number of the larger studies as part of the entry criteria and a basis for ascribing SAD to participants (see Chotai et al., 2004a; McCarthy, et al., 2002; Michalak, et al., 2002; Murray et al., 2002; Rastad, et al., 2005, for example). However, it has been noted that it is unable to provide a diagnostically sound retrospective assessment of mood in relation to season and has limited reliability and validity as a diagnostic tool (Mersch et al., 2004; Murray, 2003). Therefore, the diagnosis of seasonal depression as specified in the DSM-IV-TR cannot be given using the SPAQ as the measure can only screen for a pattern of seasonality and mood. Additionally, the SPAQ is readily available on the internet which may affect the reliability and validity of the tool as it can promote a seasonal bias in respondents especially if they have seen or used it before.

Other studies which have smaller samples have used more stringent methods for screening their participants. Other measures include the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID), and or the Structured Interview Guide for the Hamilton Depression Rating Scale- Seasonal Affective Disorder Version (SIGH-SAD, see Sigmon. et al., 2007, for example). Therefore, it is likely that these samples reflect the diagnosis of seasonal depression more accurately. However, the intensive screening

required for the diagnosis is often not practical for larger studies. This has some implications for the validity of results in that many studies exploring SAD are in reality exploring a current episode of seasonal depression, rather than a chronic condition as SAD is currently specified. Nevertheless, this does not mean that the participants in larger studies do not have seasonal depression as the condition can wax and wane over time (Rohan, 1998), but caution needs to be applied when reviewing results.

Overall, the consensus would appear to be that seasonal depression is a distinct and valid diagnosis. However, the ability to separate the disorder from the less disruptive seasonality or S-SAD continues to provide scope for discussion around the diagnosis. The ability to make the distinction is required in order to provide a rationale for the treatment of the disorder and to differentiate the diagnosis from the "winter blues". Current screening tools focus on physical changes rather than exploring mood. However, it would seem that the presence of a MDE is of primary importance in the diagnosis of seasonal depression in order to suggest a level of distress that requires intervention and aids diagnostic clarification.

Summary

The development of seasonal depression is influenced by a number of factors. The associated risk factors for seasonal depression are similar to those associated with a MDE although some variation has been documented. The concept of seasonality or physical symptoms without depression serves to separate seasonal depression from being a purely biological or psychological disorder. The combination of cognitive and physical symptoms is necessary for the diagnosis of seasonal depression. However, the manifestation of symptoms may vary in intensity and duration and be influenced by cultural factors and their interpretation. The diagnosis of SAD is relatively new and remains the subject of debate. This is problematic when considering that many of the studies around SAD use seasonality as assessed through the SPAQ as a major determinant of the condition. This results in ambiguity as to whether the participants have diagnosable SAD or whether they are exhibiting a current seasonal depression. Additionally, the overlap between the terms of SAD, seasonal depression and seasonality can serve to confuse the reader especially as there is often a lack of clarity around the

definitions used in the studies. Therefore, more research is required to explore the concept and refine the diagnosis. Reports from the DSM-V committee indicate that a review is underway in order to improve the diagnostic clarification and research base of seasonal depression (Fawcett, 2008).

CHAPTER THREE: THE THEORETICAL BASIS AND TREATMENTS FOR SEASONAL DEPRESSION.

In this chapter the major theories around the development of seasonal depression are discussed. The major symptoms are considered to be atypical of depression (Stewart et al., 1990) and are thus related to the biological and cognitive theories of seasonal depression. Also the treatments that are based on theories of causation and presenting symptoms are examined and their relationship to this investigation is explained. Finally, the effectiveness of these treatments is investigated.

Theories around the Development of Seasonal Depression

There are a number of etiological theories that espouse different causal mechanisms of depression and its pattern specifiers. Some focus on a primary biological vulnerability and therefore form the basis of biologically focused treatments, while others focus more on the role of cognitive factors in the development of seasonal depression.

The main theories around the development of depression revolve around biological/genetic causation, psychodynamic or cognitive/behavioural causation. Additionally, many theorists believe that causes may combine the physical and psychological facets (Engasser & Young, 2007; Rohan, Sigmon, & Dorhofer, 2003; Young, 1999). Because seasonal depression is considered to be a specifier of depression, there is a great deal of overlap observed between the theories of causation for depression and SAD.

Biological and Environmental Theories of Depression

Neurotransmitters

A major component involved in the development of depression is thought to be lowered levels of serotonin and norepinepherine (Burton et al., 2009). The effectiveness of medications which increase the amount of these neurotransmitters in the synapses lends weight to this argument (Carlson et al., 2010). Alterations in levels of serotonin and

norepinepherine are also implicated in the disturbance of sleep and appetite (Burton et al., 2009). As these disturbances are commonly observed in seasonal depression it seems reasonable to assume the dysregulation of the same neurotransmitters are involved in the development of seasonal depression. This is supported by Lurie, Gawinski, Pierce, and Rousseau (2006) who noted the overlap of SAD with other disorders involving the same neurotransmitters. Additionally, a trial by Lam et al., (2006), which involved 96 subjects, found that anti-depressant medication (serotonin reuptake inhibitors) was as effective for treating SAD as light therapy. This finding lends weight to the belief that the same neurotransmitters are involved in the etiology of seasonal depression. However, as medication does not seem effective in other studies this indicates that other mechanisms rather than a purely biological perspective are important to consider from a treatment aspect.

The role of serotonin in seasonal depression has received recent emphasis. A number of studies have examined the effects of sunlight on serotonin production. A study by Praschak-Rieder, Willeit, Wilson, Houle, and Meyer (2008) found that there were seasonal differences in the rate of binding of serotonin at the synapses. In winter, increased binding resulted in lowered mood in 'healthy' individuals. However, in the summer in response to increased light, serotonin remained in the synapses for longer periods, resulting in improved mood. Additionally, a study by Barton et al. (2008) noted that those with MDE had a higher brain serotonin turnover than controls thus advocating the relationship between serotonin and mood. Their results further validated the association of sunlight and the production of serotonin as Barton et al. (2008) matched subjects on experienced sunlight hours (e.g. they were exposed to similar amounts of sunlight during the study) as part of the selection process in their study exploring serotonin turnover.

In a study by Lawrynowicz and Baker (2005) they postulated the role of retinal Melanopsin receptors which are linked to the circadian rhythm centre in the suprachiasmic nucleus, as a possible neurophysiological explanation and direction for treatment regarding light therapy. Melanopsin was previously thought to be a significant

contributor to the magnitude of photic responses (Ruby et al., 2002). However, as the previous research on Melanopsin was conducted on mice its relevance to human populations is yet to be determined.

Specific Biological Theories around the Development of Seasonal Depression

There are a number of biologically focused hypotheses around the development of SAD. Those most commonly found in research will be expanded on with the lesser hypotheses briefly mentioned.

Phase Shift Hypothesis (PSH).

Lewy's chronobiological model proposed that the depression observed in SAD was a result of a phase delay of the endogenous circadian oscillator, as regards the sleep-wake cycle (Lewy et al., 1989). Within this theory, the normal production of melatonin by the pineal gland was thought to commence after dusk and end at dawn in response to light. Previously, light had not been thought to affect human melatonin production (Lewy et al., 1989). However, researchers found this assumption was incorrect, as melatonin production could be suppressed by light provided the light was of sufficient strength (Lewy et al., 1980). This finding indicated that human biological rhythms were cued to bright sunlight rather than dim ordinary room light, and researcher postulated that bright light could therefore be used to alter these rhythms (Lewy et al., 1989).

The PSH findings were based on the Phase Response Curve (PRC) whereby the ability to alter circadian rhythms in the constant dark with short bursts of light was demonstrated. The PRC was grounded in animal studies and was dependent on when the light pulse was scheduled. For example, when the pulse was produced in the subjective night, it produced a greater magnitude of circadian shift than when it was given during the subjective day, with light given in the subjective day having little effect. Additionally, if the light burst occurred in the first part of the subjective night it produced a shift to a later time (phase delay) and if given later in the night it caused a phase advance (shift to an earlier time). For the majority of PRCs the closer to the middle of the night the pulse occurred, the greater the magnitude of the phase shift (Lewy et al., 1989,

p.296). As regards humans it was hypothesised that they would exhibit similar responses to light and have comparable PRCs.

Accordingly, this resulted in two types of mood disorder, whereby both the abnormal phase delay and the abnormal phase advance resulted in depression due to the subsequent effects on time and sleep. The changes in sleep patterns were found to be indicative of seasonal depression in the majority of studies undertaken and increased sleep and fatigue were considered to be main components of the disorder (Rosenthal et al., 1984). However, Lewy et al. (1989) noted that not every mood disorder would have a chronobiological component.

In regards to seasonal depression, it was hypothesized that the shorter and later arrival of the subjective day in winter, and the subsequent alteration to the light-dark cycle would result in a phase delay. This would result in depression because humans should cue relatively more to morning as opposed to evening light (Lewy et al., 1989). Therefore, exposure to bright light in the morning was thought to result in a phase advance and stop the production of melatonin which would help normalise the circadian rhythm and reduce levels of depression. Improved mood was thought to be achieved by alleviating the chronic fatigue and realigning the sleep cycle of affected individuals (Lewy et al., 1989). Conversely, exposure later in the day was thought to exacerbate the presenting phase delay and result in worsening mood (Lewy et al., 1989). However the relationship between mood and melatonin is not an overt one.

The majority of research would appear to support this hypothesis. A study by Murray et al. (2003), showed a significant association between winter pattern seasonality and within-subject phase delay in winter. In their study phase delay was measured through a shift toward ‘eveningness’ in the subjects; whereby improved mood in the evening was thought to indicate evidence of phase delay (Murray et al., 2003). Additionally, Lewy, Lefler, Emens, and Bauer (2006), undertook a study to find an optimum time to phase-type SAD patients (i.e. ascertain whether the person was experiencing the results of a phase-advance or phase delay) in order to ascertain optimal times to treat them with low

dose melatonin. Results from this study produced support for the PSH whereby melatonin would stimulate a phase advance and lead to the realignment of sleep/ wake cycles (Lewy et al., 2006). Furthermore, the most commonly used and efficacious treatment for SAD is phototherapy, which is directly related to this theory.

However, it has been documented that phototherapy can be delivered effectively during various times of the day (Sato, 1997). This would imply that there are factors other than phase delay that need to be considered. Nevertheless, morning light is generally considered to be more effective when compared to evening light (Lewy et al., 1989; Terman, 1989; Terman, Amira, Terman, & Ross, 1996). However, others have noted that the intensity of light may compensate for the time the light was administered (Sato, 1997). Additionally, it is believed that light may also help regulate the 'clock genes' which provide the mechanism for regulating circadian and seasonal rhythms (Bunney & Bunney, 2000; Gorman, 2005).

Light Deprivation Hypothesis (LDH)

Another factor involved in the development of seasonal depression is the chronic decrease in available photoperiod and intensity of light for the majority of the day in winter. In this respect the PSH is closely linked to the light deprivation hypothesis. The LDH is another biologically focused theory as regards the development of seasonal depression. Some individuals are thought to have a bio-rhythmic susceptibility to depression that is reliant on exposure to appropriate amounts of ambient light (Woodson, 2004). The theory postulated that the decreased photoperiod available in countries of high latitude, for example, Scandinavia, during winter, facilitated the development of seasonal depression (Michalak & Lam, 2002a).

The decrease in available light was thought to trigger neurotransmitter imbalances especially those related to serotonin and catecholamine (dopamine and norepinepherine) production (Lam & Levitan, 2000). However, it remained unclear as to whether treatment restored the disturbed interactions between these two systems or affected the

systems separately (Neumeister et al., 2001). The different neurotransmitter foci appeared to be one of the main points of difference between the PSH and the LDH.

The LDH has served as a way to focus the disorder on regions of high latitude. The geographical focus resulted in the majority of research being done within these countries (see Sato, 1997). Further support for this theory was provided by the positive results achieved by phototherapy (exposure to bright light) as the main treatment for SAD. However, it was noted by Michalak & Lam (2002a) that although there may be a correlation between latitude and SAD there was no such correlation found between SAD and specific racial subgroups. The result led to the impression that certain ethnicities may have adapted to the conditions (Magnusson & Partonen, 2005). Additionally, Woodson (2004) noted that latitude had a significant positive relationship with the prevalence of SAD only when no other factors were taken into account. However, with the inclusion of other variables, for example, age of sample and date of survey administration, the relationship was no longer significant (Woodson, 2004).

Thus the PSH and LDH would both appear to have neurotransmitter dysfunction associated with decreases in light availability in common, but whether it is a combination of all neurotransmitters as opposed to melatonin versus serotonin and norepinepherine does not appear to have been resolved. Furthermore, although neurotransmitter imbalance is the primary cause of the depression according to these hypotheses factors other than latitude may account for the quality of light including for example, pollution, water vapour and cloud (Sturman & Tapper, 2006). Therefore the LDH may not be latitude specific but relate more to light availability in general as the relationship between latitude and seasonal depression appears to be tenuous.

Latitude and Photoperiod Comparisons to the Southern Hemisphere

The main biological theories around the causation of seasonal depression are linked to latitude and sunshine or bright light exposure, with the majority of the research focused in the northern hemisphere; especially within the latitudes of 50-60 ^{o}N. However, the results of research within this focused area may not be applicable to New Zealand, as it lies at a different latitude. Therefore an exploration of southern hemisphere research and research from comparable latitudes in the northern hemisphere is warranted.

The only habitable land masses in the southern hemisphere at the latitudes of 55-60^{o}S are Tierra del Fuego and the Falkland Islands. The ability to make direct comparisons on latitude is limited as these regions in the southern hemisphere, although at similar latitude to the northern research populations have harsh climates and sparse populations. Thus the ability to contrast southern hemisphere seasonal depression with the northern hemisphere is restricted.

One study performed at similar latitudes to northern research was undertaken by Lawrynowicz and Baker (2005). They presented findings on the relationship between suicide and latitude in Argentina. Their samples included those from latitudes below 45^{o}S, including Tierra del Fuego. The researchers found that there was increased suicide rates in the more southern provinces compared to national averages. Explanatory factors for these results aside from socio-economic and racial differences were believed to be associated with temperature and light variation (Lawrynowicz & Baker, 2005).

New Zealand is situated in the temperate climactic zone of the southern hemisphere (latitude range 35- 46 ^{o}S). A northern hemisphere country that is of similar latitude as NZ would be Italy which extends from 38-45 ^{o}N. In a study by Muscettola, et al. (1994), there was found to be little difference in Italy as regards latitude and presentation of seasonal depression. Indeed their results were similar to studies performed in different countries at comparable latitudes, for example, the United States (Muscettola et al., 1994). In Muscettola et al.'s (1994) sample, (N = 543), over half of the sample reported

problems associated with seasonal change. These findings indicate that latitude and extreme variation in photoperiod may not be as fundamental to the commencement of seasonal depression as previously believed. A limitation of this study is the reliance on self report and the vague definition of seasonal depression used in this study.

Another study that did examine seasonal depression in a country with a limited difference in photoperiod was a case study in Israel at 32.6 ^{o}N (Moscovici, 2006). This study served to highlight the possibility of the condition in countries with limited seasonal variation in sunlight and focus on individual vulnerability (Martiny, 2006). The main limitation with this study was that it involved one person. In this study light therapy was found to be beneficial despite the relatively stable photoperiod indicating an individual vulnerability to slight decreases in available light in some people. This study would have relevance for treatment options in NZ due to climactic similarities. The effectiveness of bright light for vulnerable individuals would support findings by Rosenthal et al. (1989) where some individuals required a certain strength of light in order to obtain therapeutic benefit from it. Therefore, light therapy or additional light exposure of sufficient strength may be required in winter for some individuals as the strength of naturally occurring light would have decreased as a result of the earth's position relative to the sun (Sturman & Tapper, 2006). The decrease in light would result in symptom expression in vulnerable individuals.

The majority of studies exploring the presence of seasonal depression in the southern hemisphere have focused on photoperiod. Boyce and Parker (1988) examined a convenience sample of women from the states of Victoria and New South Wales (Australia). The sample was recruited from an advertisement in a women's magazine. Of the 138 respondents, eighty reported depressive symptoms associated with a particular season. Thirty- five percent reported depressive symptoms in the winter, 35% in the summer, 20% in the spring and 10% in the autumn. The incidence of SAD in the study by Boyce and Parker (1988) appeared to be similar to those reported in studies conducted in the northern hemisphere. The authors concluded that photoperiod rather than temperature was likely to be the main contributor to the syndrome in their sample due to

the similarity of day length in northern hemisphere cities used as comparisons from other studies and Sydney, whereas there were large temperature differences between the cities (Boyce & Parker, 1988). A limitation of Boyce and Parker's study was the small sample size; which meant there was the possibility of type II error. Furthermore, the overall even distribution of winter and summer depression expression contributed to the non significance of the findings. These results may also be a result of the lower latitudes represented in analysis (28-38 ^{o}S) and the possible confounds of examining summer and winter seasonality within the same study. However, due to the similarity of the sample as regards those presenting with SAD in comparison to those in the larger northern hemisphere studies, it was possible that the sample is reasonably indicative of distribution of SAD in that region of Australia.

Conversely, Murray and Hay (1997) proposed that "perceived photoperiod" rather than actual light hours was more important as regards symptom presentation as they noted a large discrepancy between actual light exposures when compared with available light in some studies. In their study, photoperiod was examined in relation to the different environment that exists in Australia. The main hypotheses were that seasonality did not increase with distance from the equator and that seasonality was not separate from non-seasonal neurotic complaints (Murray & Hay, 1997). For this study the sample was comprised of female pairs of twins that resided in different latitudes, (from 12 to 43 ^{o}S) with a final sample size of $N = 526$. On examination of the results, there was found to be support for both of the hypotheses; however, the authors admit that latitude was an indirect measure of photoperiod (Murray & Hay, 1997). These results lend weight to the supposition that latitude (and indirectly photoperiod) are not the major contributors that they are presumed to be in some northern hemisphere studies. Winter, in this study was perceived from the results to be a "background stressor" (Murray & Hay, 1997, p.283) and a variety of psychological factors were thought to play a prominent role in the development of SAD in this population. Furthermore, the dominance of psychological factors in this study lends weight to the belief that cognitive interventions may be effective in the treatment of SAD in the southern hemisphere.

The use of twins in the study by Murray and Hay (1997) may have resulted in some lack of generalisability of the sample, especially given that genetics may play a role in the development of mood disorders. Additionally, the use of women only would further limit the applicability of findings. As noted by Murray and Hay (1997) results may have been a product of sample characteristics present in those twins who replied, therefore a non-twin study would be useful to replicate the results. Murray and Hay (1997) also noted that it was possible for a distinct seasonal variation to occur unconsciously in mood and behaviour, that is they do not experience any difficulties as a result of the variation. Therefore, some seasonal variation may not be reflected in the findings as a result of unawareness of the process in the sample. This may be reflected in the difficulty with diagnosis which was discussed earlier (Martiny, 2006; Wilson & Read, 2001). Furthermore, it was possible that the examination of both seasonality and non-seasonal neurotic complaints in the same sample may also have led to certain demand characteristics that have influenced the results. Exploring anxiety in this study may also have complicated findings due to the overlapping nature of depression and anxiety. However, it was possible that the findings related more to the presence and prevalence of seasonality in the sample rather than the presence of SAD itself.

The findings from other parts of the southern hemisphere indicate the presence of seasonality in this region. However, as the southern hemisphere is a large area and has a markedly different geographical composition to the northern hemisphere, that is, a greater proportion is ocean it is likely that these results may only apply to the areas in which they were studied. Additionally, Australia and South America are continents which further limit the applicability of their results to a group of islands such as New Zealand. Therefore, further research in this area is required.

The Vitamin D Hypothesis

In line with the previous hypotheses, the vitamin D hypothesis has a biological focus. This hypothesis postulates that the lack of sunshine (in winter) leads to a decrease in the vitamin D produced by the body. This is believed to result in depression and may be related to seasonal depression (Dumville et al., 2006). In other studies, hypovitaminosis

D has been documented as occurring more frequently in patients with fibromyalgia and anxiety and depression, although the exact nature of the relationship was unclear (Armstrong et al., 2007). Additionally, Wilkins, Sheline, Roe, Birge, and Morris, (2006) found that low levels of vitamin D were also associated with low mood and cognitive impairment in a cross section of older adults.

There were some studies whose findings did not support the specific association of hypovitaminosis D and the pathophysiology of depression. These studies have postulated that other factors may be influential, for example, different social situations (Schneider, Weber, Frensch, Stein, & Fritz, 2000). There was some support for the use of Vitamin D supplements in the treatment of SAD and depression (Berk et al., 2007; Gloth, Alam, & Hollis, 1999; Wilkins et al., 2006). However, the results were not consistent across trials. Furthermore, the supplementation of vitamin D in some samples has shown no resulting improvement of mood (Dumville et al., 2006). It may be concluded that at this time there is limited support for this hypothesis, but the area requires more research before definitive conclusions may be drawn (Dumville et al., 2006; Schneider et al., 2000).

Hypothesis

The majority of the biological theories around seasonal depression focus on the availability and intensity of light. The research is performed in areas which have noted seasonal fluctuations in light but there is less data from countries with a more stable photoperiod. Therefore in order to add to the knowledge around the subject, sunlight hours and their effect on this sample will be explored through the hypotheses:
Hypothesis 12: Hours of bright sunshine will be negatively correlated with a) depression scores and b) vegetative scores.

Cognitive Theories of Depression and Seasonal Depression

Beck's Cognitive Theory of Depression

Beck's cognitive model proposes that how an individual structures the world determines their behaviour and affect (Beck et al., 1979). Within the model are: schemas,

the cognitive triad (the view of how the person sees themselves, others and the future) and cognitive or thinking errors which when combined lead to a depressive episode (Rohan, 1998). Schemas are believed to develop in childhood and are based on childhood experiences (Wills & Sanders, 1997). When childhood experiences lead to the development of maladaptive schemas they are considered to be predisposing factors for depression. Although these schemas are often latent they can become activated under stressful circumstances which tend to be similar to the original experience that formed the schema (Beck et al., 1979). Once activated, maladaptive schemas are maintained by selective attention to information that reinforces the negative schema, leading to a downward spiral of affect (Beck et al., 1979).

Cognitive Theory in Relation to Seasonal Depression

In relation to seasonal depression, Sigmon et al., (2007) found that in their study of 45 individuals, there was an attentional bias observed that was specific to seasonal stimuli and distinct from over-generality of autobiographical memories exhibited in those with non-seasonal depression. This indicated that the extent of autobiographical (childhood) memories was not a major factor in the presentation of seasonal depression, and that general negative schemas appeared to have less impact on SAD sufferers in regard to perpetuating factors of their condition. However, a study by Rohan et al. (2003) hypothesised that there may be specific light-related schemas as seen by a negative psychological reaction to darker/winter stimuli that develops over time and contributes to the maintenance of the disorder. Nevertheless, the literature appears to be limited at this point and more research is required before there are definitive conclusions drawn.

Additionally, Sullivan and Payne (2007) found that in their study ($N = 93$), that the cognitive symptoms experienced by those with SAD were similar to those with non seasonal depression. This was supported by Rawana (2006), who found that SAD and depressed patients exhibited more typical (cognitive/ affective) symptoms than S-SAD or controls. These studies indicated that SAD may have a similar cognitive component to

typical depression thus reinforcing the need for a combination of physical and cognitive symptoms required for a diagnosis of seasonal depression..

Other theories postulate that the physical/vegetative symptoms appear first and that the depressive/cognitive symptoms form as a response (Young, 1999). However, Rohan et al. (2003) put forward the theory that cognitive schemas maintain the depressive responses without the necessity of physical symptoms being present in order to produce cognitive symptoms. It would seem from these positions that the relationship of cognitive and physical symptoms in the development of seasonal depression is complex and requires further study. However, these studies indicate that the cognitive aspects of SAD and depression are similar; therefore the notion that related treatments would also be effective seems reasonable.

The Dual Vulnerability Hypothesis (DVH)

A more recent hypothesis in relation to the development of seasonal depression was the DVH, which was first proposed by Young, Watel, Lahmeyer, and Eastman (1991). The major claim within this paradigm is that there are two main components in the development of seasonal depression. These components are: firstly, a genetic predisposition toward a vulnerability to depression and the development of physical symptoms; and secondly, the development of affective and cognitive symptoms (Young, 1999). The cognitive symptoms were predicted to be a response to the vegetative symptoms, that is, there was thought to be a temporal relationship (Young, 1999). The biological focus of previous hypotheses is included within this hypothesis. However, the inclusion of affective and cognitive factors serves to validate the diagnosis for sufferers who are in countries with little difference in light and temperature over the seasons, but who still suffer from a variety of seasonally induced physical and mood symptoms. The symptoms that are postulated to cause distress are the increases in vegetative functioning for example increased appetite and weight gain and increased sleep and fatigue (Young, 1999). Young (1999) stated that the vegetative symptoms were consistent cross-culturally but that cognitive and affective symptoms were found to be more culturally

specific. In this way the DVH serves to compensate for perceived cultural norms of mood and behaviour.

For hypotheses with a biological focus, it could be construed that the vegetative symptoms are the core of seasonal depression and that the affective and cognitive factors merely adjuncts. Nevertheless the DSM-IV-TR requires the diagnosis of seasonal depression to consist of a combination of biological and affective symptoms and thus cannot be obtained with vegetative symptoms alone (Young, 1999). The cognitive and affective variables are of equal importance in the diagnosis.

The cognitive aspects of the DVH are well supported in literature. Studies by Rohan et al. (2003) and Rohan et al. (2007) discuss the importance of cognitive processes in SAD. Additionally, quality of life which would relate to the individuals perception of their situation was documented as being markedly impaired in SAD patients (Michalak et al., 2005; Michalak, et al., 2007). This would be related to the negative response style discussed by Engasser and Young (2007) and Whitecomb-Smith (2004), who revealed that subjects with more dysfunctional attitudes, a more ruminative response style and a more internal attributional style, reported more severe mood and cognitive symptoms during winter. Dysfunctional attitudes were also noted in conjunction with SAD in research by Golden, Dalgleish, and Spinks (2006).

Nonetheless, the vegetative symptoms did not precede changes in cognitive/affective symptoms in a study by Reardon (2007) and no relationship was found between winter seasonality and symptom course. However, it was proposed that the onset and offset of SAD may have entailed different mechanisms and the broad range of seasonality among the sample may have affected results in this study (Reardon, 2007). These findings were also partially validated by Young (1999) who also found that the temporal precedence of vegetative symptoms was not always manifested in the disorder. Thus, different mechanisms may be involved in their respective onsets. Additionally, in a study by Rawana (2006), subjects were noted to have experienced a greater dread of winter. An anticipatory dread of winter may construct an argument for the temporal precedence of

cognitive symptoms and the development of related schemas postulated by Rohan et al. (2003).

Although there is some debate about the finer points of the DVH, overall it appears to be well supported in literature and research. Additionally, the focus on the cognitive aspects of the diagnosis serves to provide an alternate direction for treatment for seasonal depression. Due to the expense related to phototherapy and the need for cost effective treatment, cognitive therapy for SAD has become an area of increasing focus (Freed, 2006).

Treatment for Seasonal Depression

Physical Treatments

Phototherapy

The most common treatment designed specifically for SAD is phototherapy or light therapy (LT) (Freed, 2006; Martiny, 2006; Michalak et al., 2007; Sato, 1997). LT was developed as a direct response to the phase shift and latitude hypotheses. Phototherapy has also been noted to have an effect on those with seasonal variations in mood but who do not show the extreme symptomology of those with SAD (Kasper et al., 1989a). The effects of LT on 'normals' was believed to have public health relevance, as it may be that such individuals would benefit from outdoor or bright light exposure during winter months even if not clinically depressed (Jacobsen et al., 1987). It was thought that increased light exposure would translate to positive effects on productivity and wellness during the winter period (Jacobsen et al., 1987). Thus it was believed that LT may be effective for those with less severe symptoms as well as those with severe symptoms provided that there was a seasonal component to the depression (Rosenthal et al., 1989). This was supported by findings from Kasper et al. (1989) who noted that optimizing office lighting in winter would be effective for those with mild seasonal changes.

Historically, LT has been used for a variety of ills, with ultra-violet light being an important component along with total body exposure (Rosenthal et al., 1989). Currently,

LT is not considered to be a panacea but only useful for certain patients (Rosenthal et al., 1989). Additionally, modern LT contains little ultra-violet light and is focused mainly on the eyes as a portal of entry (Rosenthal et al., 1989; Sato, 1997). The majority of studies focused on LT as a therapy for seasonal depression due to the findings of Lewy et al. (1980). They found that bright light suppresses the secretion of melatonin and related its effects to those found in animals, suggesting its possible therapeutic value in humans (Rosenthal et al., 1989).

Lewy et al.'s (1980) findings showed that although bright light resulted in suppression of melatonin, ordinary room light did not. This result seemed self-evident as patients developed seasonal depression in the winter despite the use of ordinary room light (Rosenthal et al., 1989). The intensity of the light appeared to be an important factor: 2500 lux was found to be effective in Lewy et al.'s (1980) study and was considered to be appropriate for early studies in SAD. However, it was found that the intensity of light required varied from patient to patient with some deriving benefit from 300 lux whereas others required up to10 000 lux (Rosenthal et al., 1989). Sato's review (1997) also showed that a variety of light intensities used in studies was effective.

Additionally, there is also the question of how much light was enough. Initial studies found that five hours was better than two hours (Rosenthal et al., 1989). However, when this was combined with intensity, effectiveness could be maximized with decreased exposure, with two hours of 2500 lux being as effective as 30 minutes with 10 000 lux (Sato, 1997). When to administer the treatment was also explored. Although morning treatments were found to be slightly more effective, on the whole timing did not appear to be a significant factor (Avery et al., 2002; Ruhrmann, et al., 1998; Sato, 1997).

A study by Postolache et al. (1998), found LT was not as effective as summer for relieving symptoms. This finding raised the question around factors other than light as being effective or whether 'natural' sunlight is a more effective mood reliever than artificial substitutes. That bright sunlight has been estimated to produce up to 50 000 lux lends weight to its potential effectiveness (Partonen, 2001).

Findings by Putilov and Danilenko (2005) indicated that the most powerful mediator in regards to a person's response to bright light therapy were the associated psychological components. In this study the placebo effect which involved the patient's expectations and the researcher's enthusiasm was thought to account for a large portion of the clinical response. This result brought into question whether the patient's chronobiology was the major factor, or whether a variety of neuro-responses was involved in the improved mood associated with light therapy. If this is the case it may be that increased outdoor activity regardless of sunlight during the day would have similar positive effects on mood if the client believes that it will.

Although a 40-50% reduction of symptom severity was found in one week using LT, it has been noted that relapse is equally rapid (Sato, 1997). The rapid return of symptoms has led to the need for LT to be administered every day throughout the season of risk (Rohan et al., 2007; Sato, 1997). Additionally, relapse appears to be chiefly associated with the intensity of exposure, with those receiving higher doses (10 000 lux) exhibiting a relapse rate of 75% within a week of terminating treatment (Terman et al., 1990). These results indicate that the higher the dose given the more severe the relapse. Furthermore, a constancy of intensity of exposure has been observed as more effective and less likely to result in mood instability than frequent changes in lux administered (Rosenthal et al., 1984).

The most effective anatomical route for LT has been found to be through the eye (Wehr, Skwerer, Jacobsen, Sack, & Rosenthal, 1987). However, this route was observed to have some side-effects, for example, eyestrain, headaches, and insomnia (Sato, 1997). Hypomania was also associated with LT especially as regards unmonitored treatment (Meesters & van Houwelingen, 1998; Rosenthal et al., 1989). It would seem that although regarded as safe and readily available without prescription, LT still requires monitoring in order to be both effective and safe. LT was also noted to require a considerable time commitment which not all SAD patients adhered to (Rohan et al., 2007).

In New Zealand, LT is not commonly used or available. Therefore its use as an effective treatment for seasonal depression is limited and possibly contra-indicated given the adverse side-effects documented with unsupervised use. However, due to the relatively stable photoperiod and mild winter climate the use of outdoor bright sunlight is a viable alternative. Even on cloudy days sunlight has been documented to be comparable with 2000 lux and on sunny days up to 50 000 lux (Partonen, 2001). This means that outdoor activity can potentially be used as an alternative LT treatment for those with seasonal depression. The major drawback to this option is the significant periods of rain that may hinder exposure for prolonged periods during winter. This can result in a less effective treatment due the need for consistency in exposure (Rosenthal et al., 1984). Additionally, because consistency is required for effective treatment, relapse is possible due to fluctuations in weather patterns. Therefore, the use of an adjunct treatment, for example, therapy would be beneficial in order provide the individual with a way to counter physical symptoms and resulting schema.

Anti-Depressant Medication

For many sufferers of seasonal depression, the symptoms can be confused with those of atypical depression (Martiny, 2006). The confusion may be especially prevalent in those for whom it is their first presentation. As the DSM-IV-TR (APA, 2000) does not specify seasonal depression until there have been at least three episodes, there can be a resulting missed diagnosis or focus. The difficulty may be that once a diagnosis of depression (MDD) is given, further episodes are explained parsimoniously as recurrent episodes and cyclical causes may go unrecognized. Therefore, the most common treatment likely to be given for seasonal depression is medication.

The original major anti-depressants used in the treatment of seasonal depression were from the tri-cyclic and mono amine oxidase inhibitor (MAOI) families and the benzodiazepines which would address comorbid anxiety symptoms often observed with depression (Miller & Keane, 1983). However, these medications had numerous side-effects including dependence (Sarafino, 1994). Additionally, the time that these original

medications took to achieve a therapeutic level also made them less desirable for short term use (Sarafino, 1994).

Among the most commonly used medications in the treatment of seasonal depression currently are the selective serotonin reuptake inhibitors (SSRIs). As serotonin dysregulation has been thought to result in SAD, this group of anti-depressants was thought to be effective in the relief of seasonal symptoms (Jacobsen, Murphy, & Rosenthal, 1989). The main effect of SSRIs was believed to result from altering the amount of serotonin available in the synapses thereby enhancing mood (Barlow & Durand, 2002).

Fluoxetine is one of the more researched and readily available SSRIs. It has been shown to be effective in the reduction of depressive symptoms and associated with a marked improvement in quality of life for SAD patients (Michalak et al., 2002; Tam, Lam, & Levitt, 1995; Partonen & Lonnqvist, 1996). Fluoxetine has also been shown to lower melatonin levels in subjects in comparison to tricyclic medication and fluvoxamine (Childs et al., 1995). These results indicate that fluoxetine would be effective in treating SAD, however the study had few participants ($N = 10$) and so its results need replication to validate the findings (Childs et al., 1995). Additionally, fluoxetine has been found to assist in decreasing carbohydrate cravings which are common in SAD (Wurtman et al., 1993). The most recent study comparing fluoxetine with phototherapy, by Lam et al. (2006), showed similar effectiveness for either treatment but noted that phototherapy had faster response rates and fewer side effects. Rosenthal (1998) also noted that medication required at least two weeks for effectiveness to be found and that dosage may require titration. Therefore, with medication, symptoms of depression may continue for some time before relief is noted.

With SSRIs, side effects are common, including an increased risk of suicide (New Ethicals, 2008). Additionally, a study by Shen and Shapiro (2002) found that sleep quality was affected in SAD patients on fluoxetine. Another study by Swiecicki and Szafranski (2002), found that if combined with phototherapy, the SSRI-associated side

effects, for example nausea, confusion and hyperthermia, were increased in their sample. These treatment interactions were also found with patients on Sertraline (Swiecicki & Szafranski, 2002) where results were attributed to a serotonin effect potentialisation which was felt to be specific to these factors.

In a review of studies examining the use of SSRIs in children and adolescents in Australasia (based in the University of Melbourne), Fluoxetine was found to be useful in the reduction of symptoms for both children and adolescents (Hetrick, Merry, McKenzie, Sindahl & Proctor, 2007). However, in this study, medication use was also associated with increased suicidal ideation.

Anti-depressants are commonly prescribed and available from a General Practitioner (GP). Medication appears to be effective; however the difficulty with this treatment is that it does not focus on the underlying mechanisms of SAD and the need to alter aspects of the patient's lifestyle. This means that there may be effective symptom relief for the majority of sufferers, but re-emergence of symptoms occurs with seasonal change. Therefore, the affected person continues to have no insight into their condition, or access to education on how to reduce their problematic symptoms and cognitions. Additionally, it was noted that some antidepressant medications had the opposite effects on the same people at different times of the year (Rosenthal, 1998). Thus in winter medication may be activating with associated improvement in mood but in spring the same medication may make the patient lethargic and sluggish. Therefore, if the doctor does not recognize this pattern, they may believe that the patient requires more medication as opposed to less, thus compounding the problem and potentially leading to other adverse side effects (Rosenthal, 1998).

Other Medications

Melatonin and circadian rhythm treatment in conjunction with anti-depressants have been used to aid the assessment and adjustment of morning wakening difficulties and SAD (Lawrynowicz & Baker, 2005; Lewy, et al., 2006; Teicher et al., 1997). These treatments were believed to be in line with the PSH (Teicher et al., 1997). However,

Murray et al. (2005) found that the relationship between phase advance and symptom change was uncorrelated in their study. These results may in part be due to the measures used (the Beck Depression Inventory –II: BDI-II, and the Hamilton Depression Rating Scale: HRS) as there is some doubt as to the sensitivity of standardized measures when screening for SAD (Lurie, et al., 2006). However, a recent study by De Berardis et al. (2010) found that melatonin antagonists were effective in the treatment of depressive symptoms. This would add support for the PSH and the role of melatonin in maintaining depression.

The herb Saint John's Wort was also postulated as a possible treatment for depression and was considered to be safer than other anti-depressants (Barnes, Anderson, & Phillipson, 2001). However, its possible effectiveness as a treatment for seasonal depression has had limited consideration or research.

Use of Medication for Depression in New Zealand

A study by Wilson and Read (2001), examined the treatment practices of General Practitioners (GPs) in Auckland, NZ for depression. The study found that although many of those sampled believed in the value of psychotherapy, they prescribed anti-depressant medication in the majority of cases. This finding was despite the knowledge by GPs of the contribution of psychological and social factors to the cause of depressive episodes, and that medication does nothing to alter significant precipitants and perpetuating factors (Wilson & Read, 2001). More importantly, major traumatic events for example, rape were discussed by only a small percentage of respondents despite the GPs acknowledgement that addressing these factors is important to treatment and recovery (Wilson & Read, 2001). Time limitations, difficulty in accessing services, and the expense of counseling were presented as the main reasons for prescribing medication. Additionally, the diagnosis of depression as a disorder with associated 'symptoms' was thought to influence treatment because it predisposed GPs to a symptom-based "illness model" consistent with medication prescription (Wilson & Read, 2001). This study has a number of limitations, one of which was the low response rate to this questionnaire (39%) and it may be open to type II error. Also of note is that responders were only

surveyed from one area of Auckland, New Zealand. This study indicates that GPs are more likely to prescribe medication for 'depression' without an in depth assessment. If so it is unlikely that a seasonal component to the depression would be revealed which could suggest the use of another treatment as more appropriate.

A study on medication beliefs and antidepressant adherence in New Zealand by Russell and Kazantzis (2008) found that concerns about taking medication outweighed the necessity for medication adherence. Thus, adherence may be an ongoing issue for a number of those on antidepressants. This may result in chronic long-term difficulties with depression. If there are limited alternatives to medication available, the implications for mental health are concerning due to the ongoing costs of depression and medication without the focus on recovery and building resilience.

A study by Hetrick, Simmons, and Merry (2008) regarding medication side effects in adolescents found that engagement between clinicians and patients was imperative due to the severity of potential outcomes for example suicide. If the initial assessment was limited as indicated by Wilson and Read (2001), then it would be unlikely that rapport will be consolidated enough to facilitate medication adherence or to gauge the potential negative side effects of medication use especially in young persons. In general, these studies indicate that medication may not be the most beneficial way of treating depression but only the most accessible. An exploration of the effectiveness of alternative treatments in NZ would be useful.

Exercise

Exercise is another treatment that has received recent research. Findings by Leppamaki, Partonen, and Lonnqvist, (2002) have shown that exercise is effective in alleviating symptoms of depression especially in conjunction with bright light for seasonal depression sufferers. Additionally, Peiser (2009) postulated that exercise was an effective treatment for SAD. It would seem that exercise effects the circadian mechanisms which in turn lead to an improvement in mood (Peiser, 2009) however the explanation for the effect remains unclear and requires further investigation. Other

findings have also linked exercise to neurotransmitter production which may also serve to improve mood via regulating turnover in the body (Leppamaki et al., 2002).

Cognitive Behaviour Therapy

Cognitive-behaviour therapy (CBT) was originally developed as a treatment for non-seasonal depression (Beck et al., 1979). CBT usually involves a time-limited treatment of 15-20 therapy sessions and homework assignments (Beck et al., 1979). As seasonal depression is currently a specifier of depression, CBT would appear to have a place in its treatment. However the main treatments for seasonal depression originally had a biological focus, with little attention paid to the treatment of cognitive factors included in the diagnosis (APA, 2000). These previously unacknowledged cognitive factors may be involved in the relapse of patients who have discontinued other forms of treatment, especially LT. The belief that CBT would be beneficial in the treatment of SAD was due to the consideration that nearly half of SAD patients do not fully remit with LT alone (Rohan et al., 2007). Addressing cognitive factors in therapy was considered to be in line with the DVH, where it is believed that the interaction between cognitive and biological factors produce SAD (Young, 1999).

A study by Engasser and Young (2007), examined cognitive factors involved in SAD, and postulated that they were a result of substantial winter changes in vegetative symptoms. Engasser and Young (2007) also noted that there appeared to be the same pathogenesis for SAD and non-seasonal depression, and that their levels of depressive symptomology did not differ significantly on a variety of measures. However, they hypothesised that dysfunctional attitudes were a state marker for seasonal change due to the differences obtained between SAD and non-seasonal depression sufferers. Support was found for this belief in a study by Rohan et al. (2003) which showed that the rumination response style of SAD participants predicted the severity of their winter symptoms. In line with this, Engasser and Young (2007) hypothesized that the severity of cognitive and affective winter symptoms would be associated with an increased cognitive vulnerability to depression. In their study cognitive vulnerability was measured through

recruitment in the summer when participants were presumed to be symptom free and those with current depressive symptoms were excluded (Engasser & Young, 2007). However, there were some limitations to this study. Firstly, cognitive vulnerabilities may have been triggered by the advertisement (stressing winter vegetative symptoms) and led to attentional bias and exaggeration of symptoms, or may have led to the development of depressive symptoms in otherwise euthymic people. Secondly, participants may have been recruited into the study because of the emphasis on physical symptoms/seasonality rather than because they had seasonal depression.

Engasser and Young's (2007) results also showed that a ruminative response style predicted higher levels of associated seasonality in mood and cognitive symptoms. Vegetative symptoms were found to trigger brooding in those with a ruminative response style (Engasser & Young, 2007). Additionally, the brooding was thought to be more 'toxic' for depression prone individuals than 'normals' (Scott, 2008). It was believed that with CBT it was possible to reduce negative cognitions, helping reduce the development of cognitive responses to vegetative symptoms, and therefore reducing the possibility of a full episode of SAD; although due to the biological component of SAD, LT was advocated to achieve full symptom remission (Scott, 2008). This prescription raised the question of whether CBT alone would be an effective therapy for those in temperate climates where photoperiod was not a major issue and access to adequate sunlight was possible.

In the study by Rohan et al. (2007), CBT was delivered using a group therapy model. Sessions were 1.5 hours long, twice weekly for 6 weeks (Rohan et al., 2007). The CBT approach was modified for SAD and targeted dysfunctional attitudes, negative attributional style, negative automatic thoughts, rumination and behavioural disengagement (Rohan et al., 2007). The targeted factors were presumed to characterise psychological vulnerability to SAD (Rohan et al., 2007). The study was tailored in a number of ways: the behavioural activation and cognitive restructuring were placed within a seasonality framework in order to facilitate improved coping over the winter and presented as ways to develop winter interests and counteract behavioural disengagement

(Rohan et al., 2007). The effectiveness of structuring behavioural activation in relation to seasonality would be consistent with findings by Pinchasov Shurgaja, Grischin, & Putilov, (2000) who found that exercise and bright light had a significant effect on mood. The negative thoughts were restructured to SAD specific thoughts related to the winter season. Relapse prevention focused on early identification of negative thoughts about winter and SAD related behaviour change. A personalized relapse prevention plan in order to cope with mood change especially associated with seasonal change was developed (Rohan et al., 2007). Results showed that the antidepressant effects of CBT alone were noteworthy. In spite of this, the majority of participants expected CBT to be the least effective treatment and it was documented as the least preferred treatment when compared to LT or medication (Rohan et al., 2007).

The limitations of Rohan et al.'s (2007) study were related to the small sample size (*n* = 15 per group). Although this study built on previous studies and added to the knowledge base, more research is required as to the long term effectiveness of this therapy and to its adaptability in other populations. Rohan et al.'s study (2007) also had a reduced number of sessions in relation to traditional therapy which may have affected results to some degree. However, results in the study were promising and consistent with previous research findings.

The anti-depressant effect of CBT would support findings by Scott (2008), who documented that CBT had been associated with physiological changes in the brain similar to those produced by antidepressant medication. The changes were found to be related to the functioning of specific sites in limbic and cortical regions. Differences in the directional changes in frontal cortex, cingulate, and hippocampus with CBT were postulated to reflect the modality-specific effects of CBT (Scott, 2008).

A number of studies have shown CBT to be as efficacious as LT in the treatment of SAD, and CBT was found to be a more cost effective alternative (Freed, 2006; Rohan et al., 2007). However, CBT is a relatively new treatment for SAD and there is limited data on its effectiveness with the majority of CBT studies having few participants (Lurie et

al., 2006). Nonetheless, Engasser (2005) believes that cognitively orientated psychological interventions may be useful as an alternative treatment for those with SAD. Furthermore, Rohan et al. (2007) found that at one year follow-up, a combined treatment of group CBT and LT maintained treatment gains in comparison to LT alone. These results indicated that CBT would be beneficial in the treatment of SAD. However, there are few studies that explore the use of CBT for seasonal depression alone which is a limitation of the research thus far.

Cognitive Behavioural Therapy in New Zealand

An alternative to medication that is available in NZ is therapy. However, little has been documented about the effectiveness of therapy for the treatment of depression in NZ. Perhaps the most research has been on the use of Cognitive Behavioural Therapy (CBT), which has been proposed as an effective method in treating depression in NZ (Neimeyer, Kazantzis, Kassler, Baker, & Fletcher, 2008).

A NZ study by Merry et al. (2004a) showed that CBT based programs worked well when compared with similar programs without the cognitive component. This study was based on a sample of 13-15 year olds ($N = 392$). Furthermore, it would appear that the cognitive components were effective aspects of the treatment with an overall significant clinical benefit found on decreased risk and depression scores for the CBT based therapy compared with the control program (Merry et al., 2004a).

In a number of studies, the use of homework has been implicated in the effectiveness of CBT and homework compliance was expected to be a predictor of treatment effectiveness (Kazantzis & Lampropoulos, 2002). However, the client's perception of their homework completion appears to be more important as regards their improved mood rather than independent assessment of their compliance (Niemeyer et al., 2008). What a client views as effective may have direct implications as regards the effectiveness of the therapy and subsequent symptom reduction. This has implications for those with seasonal depression or seasonality, where tasks involving exposure to sunlight or bright light e.g. outdoor activities would be beneficial if the client was involved in the rationale

through psychoeducation. This is in line with findings by Putilov and Danilenko (2005) around the usefulness of LT involving a psychological component that is not well addressed in traditional treatment regimes.

Overall CBT would appear to show some benefit in the treatment of depression for people in NZ. However, continuing research is necessary in order to validate the therapy in a variety of conditions. Currently there appears to be little research on effectiveness of CBT with different cultural groups which is important given the bi-cultural ethos of the country. However, a study by Blampied (1991) indicated that CBT may be adapted effectively for use with Maori clients.

Summary

The differing theories around SAD have resulted in a variety of associated treatments. The theories with a physical basis have a greater focus on physically based treatments and, those with a cognitively based treatment have resulted from a greater focus on cognitive factors. However which type of treatment is more effective is still open to debate due to the relatively recent nature of cognitively based interventions for seasonal depression. Further, because the treatment studies reviewed above generally did not use large numbers of participants, randomised control trials (RCTs) or report effect sizes, it was difficult the ascertain the effectiveness of the interventions.

CHAPTER FOUR:
SUMMARY OF LITERATURE AND OVERVIEW OF STUDIES

Depression is an ongoing major issue in New Zealand (Ellis 2004). However, there has been limited research performed on depression in this country and none found on seasonal depression. One of the major difficulties with conducting research was the small population from which any sample can be based. Small sample size is a major limitation to any research's ability to generalize its findings (Whitley, 2002). There remains a need to conduct more studies in order to produce an accurate picture of depression and the factors that are implicated with its presence in NZ. Additionally, the comparative isolation of the country and its unique geographic and population composition serve to make studies from other countries less valid in this context.

The vast majority of research on seasonal depression has been conducted in the northern hemisphere. There are inherent restrictions in generalizing the findings of this research to NZ. The closest neighbour to NZ is Australia which has produced divergent findings on seasonal depression which has added to the uncertainty as whether it is possible to generalise research done in different geographical locations. NZ is a small group of islands in the South Pacific whereas the other countries (including Australia) are continents. Therefore, it is presumed that their weather patterns were different.

There has been vigorous debate over the diagnosis of seasonal depression. The overlap between seasonal depression, SAD and seasonality and sometimes unclear definitions associated with the research can add to the confusion around the diagnosis. Although the preponderance of the evidence points to the validity of such a diagnosis, it is possible that it may not exist or have any major implications for study on depression in NZ. An exploration into the presentation of seasonal/vegetative symptoms associated with seasonal depression within a NZ context may help to clarify the extent of SAD and whether additional and more focused studies would be valuable.

Although age is commonly cited in research on seasonal depression there appears to be none regarding whether specific weather variables have differing effect on different age groups. Additionally, how age is associated with presentation, assessment and therapy have not been examined in NZ in conjunction with SAD and depression. This project aims to explore the effects of age on symptom expression and relief.

Should seasonal depression be present in NZ, there is also the question of how to treat the condition. Although Light Therapy (LT) had been shown to be effective, there is evidence of notable side-effects. In addition, LT, needed to be administered consistently to be effective which may not be a reasonable option for a number of people in NZ. Medication, although readily available may not help with the negative schemas that may a considerable part of the condition for some people. Therefore, neither of the aforementioned treatments may be the best treatments for seasonal depression in a NZ context.

Cognitive Behaviour Therapy (CBT) is proposed as an effective treatment for SAD in other countries (Engasser, 2005; Rohan et al., 2003). CBT is currently a widely used and available treatment within NZ. The parent study for this project involves the administration of CBT for depression (Appendix A). Although the therapy in this study was not specifically designed for seasonal depression an investigation into how those with seasonal symptoms progress through therapy and how gains are maintained through follow-up would be of value. It is possible that these results would facilitate the adaptation of CBT for use with specific populations and the study may serve to validate the use of the treatment for seasonal depression.

Overview of Studies

Based on the literature review this research project is divided into three studies which explore different facets of the theories, and previous research findings in a New Zealand context. A visual map of the studies is presented in order to provide an overview of the design, variables and hypotheses explored in each study. A complete list of the hypotheses is listed in Appendix C

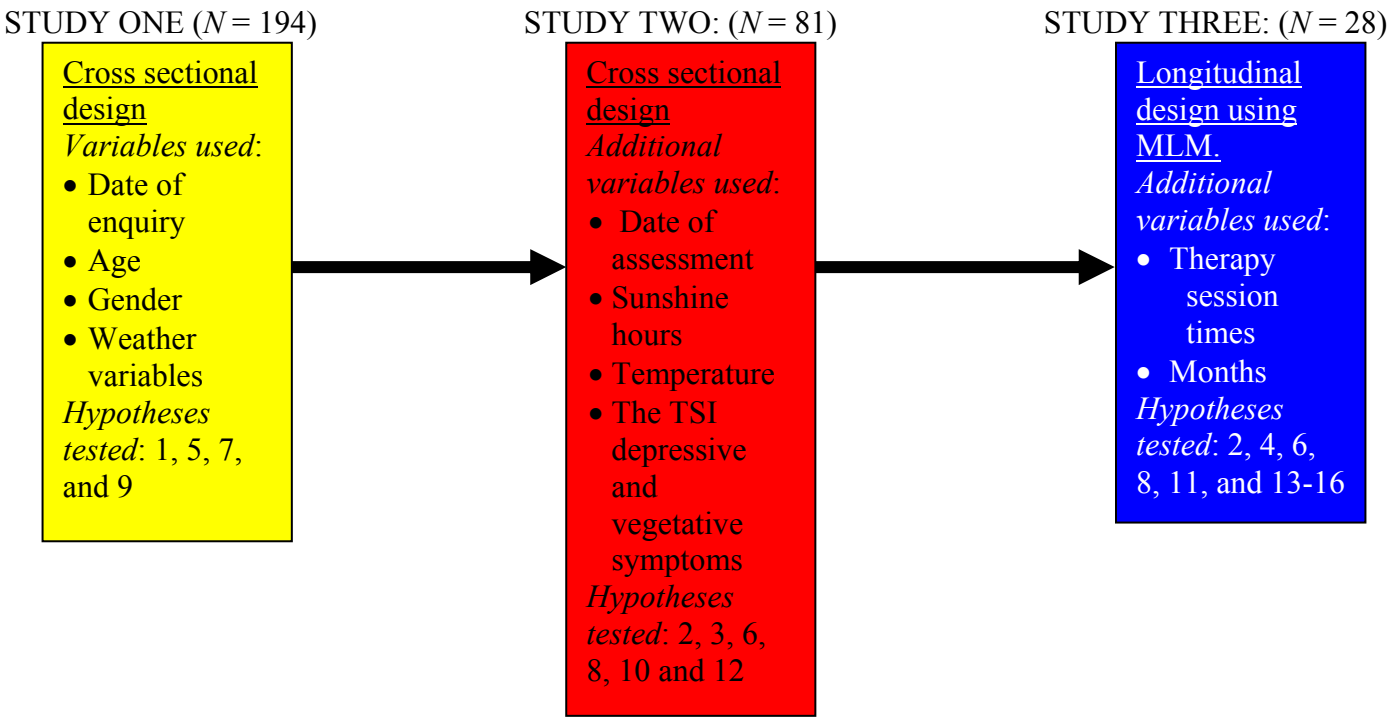

Figure 1. Visual Map of Studies

Note. A full list of the hypotheses is presented in Appendix C.

Study 1. The primary aim of this study is to investigate the relationship between season and initial enquiries around therapy for depression. It employs a cross-sectional design and a convenience sample of respondents and explores factors that affected initial enquiry for therapy for depression in the parent study in the sample ($N = 194$). Additionally, it seeks to provide a New Zealand context for weather variables during the period of the study. This study explores whether initial enquiry for the parent study (see Appendix A) is related to seasonal or meteorological variables (using daily bright sunshine hours and daily temperature recordings). This study also examines whether there are any relationships between date of enquiry, meteorological factors and the age and gender of the sample. These relationships are tested using hypotheses 1, 5, 7 and 9. The results are also used as indicators of which variables would be useful in pursuing in later studies.

Study 2. This study used the portion of the original applicants that fitted preliminary entry criteria for the study ($N = 81$). Entry criteria consisted of affirming at least five out of the possible eight symptoms for depression in a screening questionnaire. This sample was examined for the presence of depressive and vegetative symptoms in the presenting problem. Data were analysed in order to explore whether the presence of vegetative symptoms was related to meteorological variables, age, irritability and the severity of depression at assessment. These relationships are tested using hypotheses 2, 3, 6, 8, 10, and 12.

Study 3. The final study looked at the portion of the original sample that met entry criteria for the therapy arm of the parent study ($N = 28$). These individuals were believed to be experiencing a first episode MDE on the basis of interview and elevated depression scores. The main aim of this study was to explore depressive symptom relief over the course of therapy and follow-up of up to six months in relation to meteorological variables, age, gender and season. In a similar manner vegetative symptom expression was explored in relation to change of season. This was achieved using multilevel modelling (MLM) which is explained in the introduction of this study along with review of the literature related to seasonal change over time. The use of MLM in this study

enables the longitudinal nature of the data to be accounted for and the related hypotheses tested are documented in Study 3 (hypotheses 13-16). Additionally, Study 3 explores whether depressive and vegetative symptoms are linked or may be considered separate symptom clusters in line with the DVH. Additional symptom expression was explored more fully using hypotheses 2, 4, 6, 8 and 11 from the literature review in this study.

Ethics approval for the collection and analysis of data was obtained from the Northern Regional Ethics Committee and the Massey University Ethics Committee (NXT/06/07/085) for the parent study examining CBT for depression. Additional approval was obtained for the specific analyses in this study. As no additional data were collected from participants, ethics approval was obtained under the original ethics number.

CHAPTER FIVE:
STUDY ONE

Aims

The aim of Study 1 is to explore whether season, gender and age are related to presentation in a group of people who enquire about participation in a study on therapy for depression. Additionally, Study 1 aims to explore how sunshine and temperature data are manifest in relation to season over the course of the parent study. The hypotheses are taken from the literature review and specified for this data set.

Hypotheses

Hypothesis 1: More people will enquire about therapy for depression during the winter period (May- August) than during the rest of the year.

Hypothesis 5: Younger people will enquire more often in the winter months than at other times of the year.

Younger people will enquire more often on days with lower temperatures and lower sunshine hours and days of higher rainfall.

Hypothesis 7: More woman than men will enquire about therapy in the winter months

On days with lower temperatures more women than men will enquire about treatment for depression.

Hypothesis 9: More men than women will enquire on days with lower sunshine hours.

Method

Participants

The sample consisted of 194 people who responded to advertisements regarding participation in a study on CBT for depression. This sample consisted of 130 women (66%) and 64 men (34%), χ^2 (1, N = 194) = 19.81, $p < .001$. Age was not documented for 40 participants; however, for the remainder that applied, ages ranged from 18 to 75 years (M = 44.57 years, SD = 12.95years). No other demographic information was collected at this time.

Of those who enquired about the study, 56 (29%) were assessed as having had previous episodes of depression. Furthermore, 42 people (22%) were currently on medication that affected the central nervous system (CNS). The main medications documented were anti-depressants or sleeping medication/sedatives. On visual examination of the data there appeared to be some overlap between those currently on medication and those who had previously experienced a MDE. However on analysis, there was found to be no significant relationship χ^2 (1, N = 194) = 1.17, p >.05.

Measures

Date of Inquiry

The dates of initial contact of the participants were recorded and used to determine the weather data and the seasonal period.

Winter Months

Solstice (when the period of daylight and darkness are equal in length) and equinox (when the hours of darkness or daylight reach their peak) dates were used to categorise dates of respondent enquiry. The solstice and equinox dates were sourced from the website of the Weather Doctor's Diary (Heidorn, 2008). The categories used placed people in either an autumnal/winter group, (1 = May to August) or a spring/summer group, (0 = September to April). This facilitated the division of the sample for the purposes of testing winter versus other times of the year hypotheses.

Meteorological Measures

Temperature, hours of bright sunshine and levels of rainfall were collected from 1st of April 2007 to the 11th August 2009 (the full length of the study) in order to provide the daily weather characteristics over the duration of this project. A graphical representation of the distribution of temperature and sunlight hours and rain over the study period is presented in Appendix D. Data were sourced from the National Institute of Water and Atmospheric Research (NIWA) which provided the data from an Auckland weather station at -36.96177 degrees latitude and 174.77642 degrees longitude (NIWA, 2009).

Daily Temperatures. Temperature data for this study were gathered on two indexes: the daily maximum and the daily minimum temperatures. The daily maximum temperatures showed a range of 11.6-25.1 ^{o}C, (*M* = 17.3, *SD* = 2.77). The daily minimum temperatures showed a range of 1.3–19.8 ^{o}C, (*M* = 11.1, *SD* = 3.44).

Daily Hours of Sunshine. Hours of bright sunshine per day over the two year period ranged from 0-13.1 hours/day, (*M* = 4.39, *SD* = 3.12). Sunshine hours' data were normally distributed over the two year course of the study period. However there were noted daily fluctuations.

Daily Rainfall. Rainfall was collected in order to assess the relationship with other meteorological measures. Daily rainfall ranged from 0-56.6 mm/day (*M* = 3.17, *SD* = 6.94). However rainfall was intermittent; therefore for some of the hypotheses it was categorised into two groups: 0 = no rain, versus 1 = rain, but for other hypotheses it was treated as a continuous variable in analyses.

Procedure

Dates of initial enquiry were collected over a two year period from March 2007 to January 2009. Recruitment advertisements from the Massey University Corporate Communications Office (Appendix A) were placed in a variety of media six times over the course of the study (March 2007, July 2007, August 2007, September 2007, April 2008, and October 2008). This resulted in newspaper articles, webpage postings and radio interviews with the Primary Investigator for the parent study. In this study initial contact information was used and correlated with weather variables using a cross-sectional design with data collected only at the one time point.

All initial enquiries were by telephone. Some participants were referred by another person, either a family member or health professional. However, all assessment data were collected directly from the participant. Selection criteria were based on presentation

involving a first-time episode of MDE (see DSM-IV-TR; APA, 2000) and the lack of medication affecting the central nervous system. All participants were required to be between 18-65 years old. Additional exclusion were lack of prominent Axis II traits that may impact on therapy, currently not suicidal with plan or intent or history and suitable for outpatient therapy and can attend sessions twice weekly initially and be available for the duration of the therapy period (Appendix B).

Data Analysis

In order to test the hypotheses the Statistical Package for the Social Sciences (SPSS) version 17 for Windows was used (SPSS Inc, 2008). A chi–square was used to test for the significance of differences in proportions of males and females applying in winter versus other months (Brace, Kemp & Sneglar, 2003). Pearson's *r* was used to examine the strength of relationships between continuous variables (Dancey & Reidy, 2004). *T*-tests and Levene's test for equality of variances were computed and Multiple Analysis of Variance (MANOVA) was used to explore the variance between groups on weather variables. Due to the exploratory nature of this study the level of significance was set at $p < .05$.

Results

Preliminary Analysis of Weather Variables

Table 1 shows the correlations between minimum temperature, maximum temperature and hours of bright sunshine. Analysis of meteorological data showed that there were no significant relationships observed between sunshine hours and maximum temperature (Table 1). However, minimum temperatures and the number of sunshine hours were significantly correlated ($r = -.19$, $p < .05$).

There was no significant difference found between the genders in the proportions between the 'no rain' (%) and 'rain' (%) groups, (χ^2 (1, 194) = 0.33, $p > .05$). However, the age of people who enquired on days with rain was significantly younger when

compared with the ages of people who enquired on days with no rain, (r = -.16, p < .05, one tailed).

Hypotheses Testing

When exploring season in relation to enquiries more people enquired in the winter months (71%). Proportionally more people telephoned in the winter months than other times of the year, χ^2 (1, N = 194) = 34.66, p < .001. Additionally, significantly more women enquired during the winter months (76%) compared with men (60%), χ^2 (1, N = 194) = 5.40, p < .01.

When age was explored in relation to season of presentation there was no significant difference found when the sample was grouped into winter (M = 44.56, SD = 12.85) versus other time of year (M = 44.60, SD = 13.40), t (152) = -0.02, p > .05. Additionally, daily maximum temperatures (r = -.04), daily minimum temperatures (r = .07) or daily sunshine hours (r = -.10) showed no significant correlations with the ages of the respondents enquiring about CBT (Table 1).

Table 1

Correlation between Weather Variables and Age (N=194)

	1.	***2.***	***3.***	***4.***	***5.***
1.Minimum temperatures (^{0}C)	-	.68**	- .19*	.07	.03
2. Maximum temperatures (^{0}C)	-	-	.03	-.04	.01
3.Sunshine hours	-	-	-	-.10	-.07
4. Rain group[1]	-	-	-	-	-.16*
5.Age/years	-	-		-	-
Mean	11.18	17.29	4.39	0.53	44.57
SD	3.57	2.71	3.12	0.50	12.95
N	193	194	194	193	154

Note: [1] Point bi-serial correlation
*p < .05, **p < .01, one tailed

Table 2
Comparisons of Previous Episodes, Current Medication, Season, Age and Weather variables by Gender (N=194)

	Men *n = 64*		Women *n = 130*		Total *N = 194*		
	Yes *n (%)*	No *n* (%)	Yes *n* (%)	No *n* (%)	Yes *n* (%)	No *n* (%)	χ^2 (*df* =1)
Previous episodes	16(25)	49 (75)	40(31)	88(69)	56(29)	137(71)	0.92
Previous medications	17(26)	48(74)	25(20)	103(80)	42(22)	151(78)	1.11
Winter	40(61)	26(39)	98(77)	30(23)	138(71)	56(29)	5.40**
Rain Group	33(50)	33(50)	69(54)	58(46)	102(53)	91(47)	0.33
Temperature	***M***	***SD***	***M***	***SD***	***M***	***SD***	***t(df=1)***
Maximum	17.38	2.76	17.25	2.70	17.29	2.71	0.33
Minimum	11.23	3.67	11.15	3.69	11.18	3.57	0.14
Sunshine	4.18	2.96	4.50	3.21	4.39	3.12	-0.67
Age (*N=154)*	44.56	13.64	44.58	12.64	44.57	12.95	-0.01

***p = .01, one tailed.*

Table 2 shows the means and standard deviations between the genders in relation to seasonal variables. Additionally, a MANOVA was performed between gender (male vs. female) and weather variables (maximum and minimum daily temperature, and hours of daily sunshine). This revealed that there was no significant multivariate difference between the genders on the temperature variables or on number of sunshine hours, Wilks' Lambda (Λ) = 0.995, F (3, 189) = 0.29, p = .83.

Discussion

General Findings Regarding Weather Variables

The results of the correlation found between minimum temperature and sunshine hours are in line with the geographical location of NZ. This result indicated that clearer skies were associated with increased sunshine hours and less humidity. This meant that there was a corresponding decrease in temperature related to less water vapour in the atmosphere to contain heat (Sturman & Tapper, 2006). Additionally, temperature displayed a more consistent seasonal variation, but sunshine did not (see Appendix D). This indicates that whereas there was an association observed between season and

temperature observed in this study there was no corresponding association seen between sunshine and season observed. The lack of clear association between temperature and sunshine signifies that in this investigation they are best regarded as separate variables in order to observe their effects on other factors. Additionally, as the relationship between daily low temperatures and sunshine hours yielded an observable relationship compared to high temperatures and sunshine hours, the low temperature readings are the most appropriate to retain for use in subsequent analyses. However, as the association between temperature and sunshine is weak it may not indicate a relationship with any practical application.

Initial Findings Regarding the Respondents

Twenty-nine percent of the participants were considered to have had depressive episodes before. Since this study was advertised as being for those with a first episode, of depression, it was possible that some of those who enquired may not have recalled a previous episode, and may have believed that this was their first episode. The lack of recall would be consistent with findings by Wells and Horwood (2004), who found that forgetting prior episodes of depression was not unusual if there were no significant risk factors (for example suicidal ideation) involved.

Twenty-two percent of the sample was currently on medication at application. The most common medications were for depression, anxiety, or symptoms related to depression (mainly sleeping difficulties).These people appeared to have experienced little relief from medication which may have been the reason they made contact with the study. Alternatively, there may have been compliance issues or side effects involved (see Russell & Kazantzis, 2008). However, these issues were not explored in this study. Medication is often used by General Practitioners in New Zealand as a primary treatment due to the lack of available alternatives (Wilson & Read, 2001). However, medication for depression is not effective for all people (Ellis, 2004). Additionally, some people may have been incorrectly diagnosed with another condition. Those diagnosed with primary sleeping difficulties or another Axis I disorder, for example anxiety, may also have felt depressed (McCarthy et al., 2002). Sleeplessness can be an indication of depression and

anxiety can be co-morbid with depression (Westrin & Lam, 2007). Therefore, medication given for a specific symptom may not have treated the underlying depression effectively, thus prompting those individuals to seek additional help. Therefore, it would appear that a number of the sample had Major Depressive Disorder (MDD). However, as there was little data collected from these people the implications are uncertain.

Season and Initial Presentation

The majority of the participants applied to the study in winter. This result implied that more participants felt depressed in the winter months than at other times of the year. However some of this may have been related to the lack of advertisements at this time. This result is partially supported by other research with Michalak et al. (2004), who found that in some areas there appeared to be a relationship between season and depression. However, as Michalak et al.'s (2004) findings were around severity of symptoms within the sample, rather than increased referral numbers within the area, there were some limitations to the comparison. Nevertheless, a study by Sullivan and Payne (2007), found that seasonal depression was more prevalent than MDD (28% versus 8.6%) in their sample. The substantially larger number of seasonal depression sufferers in Sullivan and Payne's (2007) study may show some comparison to the results apparent in Study 1. In Study 1, specific symptoms were not examined. However, the proportionally larger numbers of people who enquired in the winter months imply some seasonal relationship with depression or low mood in this sample.

Age and Initial Presentation

Younger people were not found to enquire more frequently on days with lower temperature or sunshine hours. The lack of association between age of respondent and lower temperatures was contrary to other studies where the incidence of seasonal depression was more prevalent in younger individuals (Chotai et al., 2004a; Kovalenko et al., 2000; Rastard et al., 2005; Sweedo et al., 1995). However, increased enquiry on rainy days was associated with lower ages although the relationship was weak. Where this fits with previous findings is unclear as periods of rainfall have been associated with decreased mood in some countries where temperature does not play a significant part in

climate but these findings were not explored in relation to age (Lee et al., 2007). Additionally, prolonged periods of cloudy weather (such as experienced with rain in Auckland) have been associated with decreased mood (Rosenthal et al., 1984). However, in neither of these studies was age mentioned as a moderating factor, therefore, further research is required to clarify these results.

Gender and Initial Presentation

Overall, women referred more in the winter months. This is consistent with previous research whereby women are found to be more prevalent in SAD samples (Chotai et al., 2004; APA, 2000; Terman & Terman, 2007). However, when gender was examined in relation to temperature no specific relationships were found. On further analysis of the sample, women and men were found to be randomly distributed across all temperature categories. These findings contrast those found by Chotai et al., (2004a) where temperature and gender were found to be significantly related to depressive symptoms and SAD.

Additionally, there was no support for the hypothesis that men would enquire on days of lower sunshine compared to women. Once again the sample was uniformly distributed across the range of sunshine hours. Given that there was no discernable pattern observed for sunshine hours in relation to season in this study, these results were not unexpected. However, the variable distribution of sunshine hours (Appendix D) may reflect the relatively minimal overall decrease in photoperiod. Furthermore, the comparatively mild winter conditions experienced in Auckland (NZ) enable greater exposure to what sunlight is available. Mild weather may also serve to minimize the effects of photoperiod on this sample in comparison to other studies. Therefore, results seen by Chotai et al. (2004a) may not have been replicable in this study.

Other studies indicated that photoperiod may be related to the onset of SAD; yet they found no relationship between daily hours of sunshine and seasonal depression (Young et al., 1997). Findings by Martiny (2006) indicated that SAD can occur in places with a small photoperiod difference and that it can be treated effectively by LT. These results

signified that there can be a relationship between sunlight and seasonality in vulnerable individuals without the involvement of large photoperiodic differences. However, whether the person developed depressive symptoms, as a result of decreased light exposure, appears to be more a product of individual vulnerability in temperate settings. It is also worth considering that the photoperiodic difference in NZ may be comparatively too small to be reflected in the results of this study.

Limitations

This study had a number of limitations. Firstly, the data were based on a convenience sample and as such may not be representative of the population. Secondly, the differing information given through the various media may have influenced the presentation of the sample. Additionally, the spread of the recruitment may have limited potential summer participants however this was due to the lack of people available for assessments over the summer period and previous investigations indicating a consistent lack of response to advertisements over the summer period (N Kazantzis, personal communication, September 2008). Furthermore, there was little in the way of early demographic data collected. Individual differences such as ethnicity, occupation and socio-economic status were not examined as regards initial responders. Not all of the ages of the sample were collected; therefore, any analyses around age as a variable are open to interpretation due to the limited nature of the data. Some of the differences between men and women although significant are weak. Additionally, as findings around temperature and sunshine hours in relation to gender pertained only to presentation it is possible that a more subtle relationship was present but not captured in this study. Furthermore, a lack of power in the group analyses may have produced results that were a result of type II error. Finally, the lack of a control group may further serve to limit findings. Overall, the relationships between temperature and sunshine were found to be inconsistent in this study, therefore some caution must be used when applying the results.

This study indicated some relationship between season and initial enquiry about therapy for depression. However it was not able to draw any conclusions based on the

data analysed. This study may best be viewed as a preliminary exploration of certain factors in order to ascertain whether there was any point to further exploration of this topic with this sample. Its ability to use the majority of the people who enquired was a strength of this study as a larger sample did help with the generalisability of results. Additionally there was no expectation that there would be any findings consistent with theory which enabled a broad approach to be used in order to explore the data rather than focus on certain aspects which may yield insignificant results. This reduced the potential for researcher bias which is another strength of this study.

Study 1 Summary

Overall, the results for Study 1 were mixed, with season and gender found to influence presentation, but meteorological variables having little impact. As weather has been cited in previous research as being directly implicated in seasonal depression it is possible that meteorological variables may be related to symptoms rather than presentation alone. However, due to the inconclusive results regarding sunshine, temperature and rain; the potential importance of additional/cognitive factors in relation to the preponderance of winter presentations is indicated.

Study 2 will examine the weather variables in relation to the types of symptoms that the sample presented with. Rainfall was not used in further analyses due to the infrequent nature of the variable. Study 2 explored both vegetative and depressive symptoms; therefore, the psychological aspects of the presentation will be examined as well as the physical symptoms. In this way the subtle aspects of presentation may be examined more fully.

CHAPTER SIX:
STUDY TWO

Aims

The aim of Study 2 is to examine the sample for the presence of vegetative symptoms and symptoms of clinical depression combined with presentation during the winter period (May-August). Potential correlates between the features of depression, seasonal changes and meteorological data are explored in relation to theories on the development of seasonal depression. Additionally, Study 2 will explore the data in relation to the climate experienced in Auckland, NZ and examine the sample for the presence of seasonal factors in depression at initial assessment. The resulting data will be analyzed in order to expose any potential vulnerability to seasonal depression in this group.

Hypotheses

Hypothesis 2: People who are assessed in the Auckland winter period will have higher seasonal vegetative and depression scores, than those who are assessed at other times of the year.

Hypothesis 3: Vegetative scores will be positively associated with depression scores.

Hypothesis 6: Older members of the sample will have fewer vegetative symptoms and lower depression scores compared to younger members.

Hypothesis 8: Women will have higher depressive and vegetative symptom scores than men on days with lower temperatures.

Hypothesis 10: More women will indicate increased appetite as a symptom of their depression compared to men

Hypothesis 12: Hours of sunshine on date of assessment will be negatively correlated with depression and vegetative scores.

Method

Participants

Of the respondents who met criteria for Study 2, the age range was 18 to 62 years (M = 43.43, SD = 12.44). There were 53 (65%) women and 28 (35%) men, χ^2 (1, N = 81) = 7.72, $p < .01$. Of this sample, 17% were considered to have had a previous episode of depression and 13% reported being on medications that affected their CNS. No other demographic data were collected.

Measures

Meteorological Measures

Date of assessment. For this study the date of the assessment was used. For many participants this was different to the date of initial enquiry. The main reason for the differences between these dates was the availability of researchers to perform the assessment. However, the use of the assessment date provides consistency within the sample and thus improves the reliability of the data collected. In this study the date was used to ascertain how people felt at the time of assessment as regards their mood and vegetative symptoms.

Winter months. As in Study 1, date of assessment was categorized into winter or non-winter for analyses using the solstice and equinox dates (Heidorn, 2008).

Hours of daily sunshine. The hours of bright sunshine were taken from the same NIWA weather station as Study 1 and encompassed the dates from the beginning to the end of data collection (NIWA, 2009). The readings were based on the total hours of bright sunshine for the day and range from 0 - 13.10 hours of bright sunshine/day (M = 4.16, SD = 3.30) and a skew of 0.63.

Daily temperatures. Temperature data were collected in the same manner as sunshine data. Minimum temperatures had a range of 1.3 - 20.6 ^{o}C (M = 11.21, SD = 3.97) and a skew of -0.39.

Assessment Measures

Telephone Screening Interview (TSI)

The measure used for this study was the Telephone Screening Interview (TSI) questionnaire (see Appendix E). The TSI was designed for use in the parent study as a means of exploring first episode depression from which data for this study was obtained. The aim of the first eight items of the TSI was to ascertain whether the criteria for depression were met in the prospective sample. The second part of the TSI was larger and included questions around suicide, medical conditions and drug abuse or dependence (see Appendix E).

Reviews were conducted by the principle investigator of the parent study in order to ascertain the ability of the TSI to perform as required before implementation into the study (N. Kazantzis, personal communication, September, 2008). The TSI screened for the presence of a variety of symptoms and was based on the Zimmerman Interview Guide for Evaluating DSM-IV Psychiatric Disorders and the Mental Status Examination (1994: N. Foster, personal communication, August, 2008). Only the portion relating to the screening for depression was utilised in this study. This portion consisted of the first eight items of the TSI.

Reliability and validity of the TSI. As the TSI was developed for screening for the parent study there was no reliability and validity data available. Therefore, an internal consistency reliability analysis was conducted for the present study. This involved only the first eight items measuring depressive symptoms. Analysis yielded a Cronbach's alpha of $\alpha = .64$ for the sample in Study 2. However, with the removal of question 5 in the TSI the reliability increased to $\alpha = .67$. Because the reliability of the measure increased without question 5, it was removed from subsequent analyses. This was likely because the question around psychomotor agitation and/or retardation it was difficult to conceptualise for many clients. Overall, the TSI was estimated to be a moderately reliable screen for depression (Tabachnick & Fidell, 2001).

Concurrent validity for the TSI was ascertained using the Beck Depression Inventory – II (BDI-II: Beck, Steer, & Brown, 1996). Analyses were conducted using data obtained from 48 of the participants who were re-interviewed at a different time point for inclusion in the treatment program. Time differences between administrations of the TSI and the BDI-II varied from 9 days to 1 month. A correlation of $r = .30$, $p < .05$ between the TSI depression scores and the BDI-II scores was found which indicated that the TSI captured similar constructs to the BDI-II. This was not a strong correlation and may indicate the differing number of questions between the 2 measures. Furthermore, the weakness of the relationship limits the usefulness of the TSI in ascertaining the validity of the results.

The TSI in this study. In the present study only the first eight items in the depression section of the interview were used. Each question was scored using a 0 if the answer was negative and 1 if the answer was positive. Thus an individual could receive a score of 0-8 points for their number of depressive symptoms. For admission into the parent study, a score of five or greater was required. Three of the items in the depression screening section of the TSI measured vegetative symptoms. The vegetative symptoms were increased appetite, increased sleep and increased fatigue. In the present study, if the symptoms were present there was an additional question asked regarding how the symptom presented: for example, "*Has your appetite… increased or decreased?*" or "*Have you gained or lost any weight?*" and a separate score was recorded for each of the vegetative symptoms. An example of this would be for question 3 which examined appetite:

1. Have there been changes in your appetite? No = 0, Yes = 1 (general depressive symptom question)
2. Has your appetite increased/decreased? Decreased = 0, Increased = 1 (specific vegetative question)

Thus there were two independent resulting scores: one for the presence of a depressive symptom and an additional score for the presence of a vegetative symptom. Therefore a person may receive a score of 1 if they experienced changes in appetite. But if the appetite were reduced they would receive a score of 0 for vegetative symptoms. On the

other hand, if the appetite was increased a score of 1 was given for the endorsement of a vegetative symptom.

When the symptoms were combined with the date of assessment it was possible to ascertain if the vegetative symptoms were in line with seasonal depression. If the person presented in the winter period and had at least one vegetative symptom they were categorized as "seasonal" for the purposes of this study as symptom expression has been noted to vary in the literature. Conversely if the individual manifested with vegetative symptoms but not in the winter period they were classified as "non seasonal".

The responses to the TSI were gathered in order to gain both a depressive symptoms score and a vegetative symptoms score. The total depression and vegetative scores were then analysed against weather and seasonal data in order to explore relationships between the variables. Vegetative symptoms were examined in order to determine which symptoms were the most commonly endorsed and whether their distribution was similar to other studies. Total scores for depression and vegetative symptoms were explored in relation to gender and age.

Procedure

Over the two year period from March 2007 to January 2009, 95 respondents from Study 1 were contacted for a 30 minute telephone screening interview. The decrease was often due to comorbid conditions that may affect the study, medication use or previous depressive episodes. Of the 95 contacted only the participants who met the initial criteria for Major Depressive Episode (APA, 2000) were included in Study 2. This was ascertained by an endorsement of 5 out of the 8 items on the depression screen on a Telephone Screening Interview (TSI: Appendix E). Eighty four percent of the original sample of ninety five endorsed five or more items ($N = 81$). The data for Study 2 included all interviewees who met the inclusion criteria for clinical depression, even if they did not meet the research criteria for the offer of outpatient treatment.

Vegetative symptoms in this study are defined as increases in appetite, sleep and fatigue as reported by the participant. This provides consistency with other research investigating seasonal depression and seasonality.

Assessment interviewer characteristics. Assessment interviews were conducted by three post graduate students enrolled in Doctoral studies in Psychology. They received training in the administration of the questionnaires used in this study in order to improve the reliability of results.

Data Analysis

As for Study 1, tests for hypotheses in Study 2 were performed using SPSS version 17 for Windows (SPSS Inc, 2008). Hypotheses testing involved the use of Pearson's *r* in order to ascertain the strength of the relationship between the continuous variables. For dichotomous variables, *t*-tests with Levene's test for equality of variances were computed. Due to the involvement of more than one DV, MANOVA was used to examine the significance of differences in vegetative and depressive symptoms for season and gender (Dancey & Reidy, 2004). As the data used in Study 2 were taken from a larger study examining different aspects of depression, the possibility of finding a significant result is reduced. Therefore, Study 2 was considered to be exploratory in nature and the level of significance is set at $p < .05$.

Results

Depression

For the 95 people who completed the telephone screening interview (TSI), depression scores ranged from 1-8 ($M = 6.44$, $SD = 1.63$) and vegetative scores from 0-3 ($M = 1.54$, $SD = 0.80$). For this sample, ($N = 81$) the depression scores were normally distributed, ranging from 5-7 ($M = 6.36$, $SD = 0.78$), with a skew of -0.73.

Vegetative Symptom Endorsement

The data for vegetative symptoms were also normally distributed, ranging from 0-3 (*N= 81, M* = 1.67, *SD* = 0.76). Increased appetite was endorsed by 42% (*n* = 34), increased sleep was endorsed by 30% (*n* = 24), and increased fatigue was endorsed by 93% (*n* = 75) of the sample. There was no significant correlation observed between the depression and vegetative scores, ($r = .08$, $p > .05$); therefore these measures were presumed to be independent of each other. Therefore hypothesis 3 was unsupported in this analysis.

The initial sample for Study 2 (*N* = 95) showed a number of people who presented with vegetative symptoms but did not meet the threshold of 5 out of 8 depressive symptoms that was needed for inclusion into the study.

Symptoms in Relation to Season

A season by depression and vegetative scores MANOVA was computed. There were no significant multivariate differences between the groups on depression or vegetative symptom endorsement, Wilks' Lambda (Λ) = 0 .99, F (3, 77) = 0.37, $p = .69$. On depression there were no significant differences between total depression scores of those who were assessed in the non- winter period (*M* = 6.29, *SD* = 0.90) and those who were assessed in the winter period (*M* = 6.36, *SD* = 0.74), F (1, 80) = 0.24, $p > .05$. This implied that those who were assessed in the non-winter months were as depressed as those assessed in the winter months. Therefore, the hypothesis that those applying in the winter periods would have higher depression scores was unsupported.

For vegetative symptom endorsement it was expected that there would be an increase in the number of vegetative symptoms present in those who were assessed in the winter months. However, there was found to be no significant difference in the vegetative scores between those who were assessed in the non-winter (*M* = 1.76, *SD* = 0.70) and the winter months (*M* = 1.63, *SD* = 0.78), F (1, 80) = 0.44, $p > .05$. Therefore, the hypothesis that those assessed in the winter period would show higher vegetative symptom endorsement was not supported in this analysis.

Age and Symptom Expression

Age and numbers of vegetative symptom scores on interview were expected to be negatively correlated. However a significant relationship was not found in this sample, although a trend was noted on analyses $r = -.17$, $p < .10$, one tailed (see Table 3). Additionally, age was not found to be negatively related to numbers of depressive symptoms, $r = -.08$, $p > .05$. Therefore, the hypothesis that age would be negatively correlated to depressive and vegetative symptom was not supported.

Gender Differences

A MANOVA was performed between gender and total depressive symptoms and total vegetative symptoms, Wilks' Lambda (Λ) = 0.94, F (2, 78) = 2.58, $p = .08$ ($p < .10$). On total depression scores women ($M = 6.47$, $SD = 0.70$) scored higher than men ($M = 6.14$, $SD = 0.89$), F (1, 80) = 3.36, $p < .10$. However on total vegetative scores women *(*$M = 1.75$, $SD = 0.73$) did not score significantly higher than men ($M = 1.50$, $SD = 0.79$), F (1, 80) = 2.10, $p > .10$. However as these analyses did not reach significance of $p < .05$ hypothesis 8 was not supported.

Symptoms and Sunshine Hours

To test hypotheses around the relationship between sunshine hours and depressive and vegetative symptoms a correlation was performed between number of sunshine hours and the number of symptoms elicited from the phone screening questionnaire (see Table 3). There was no significant correlation between the number of hours of sunshine and an increased number of depressive symptoms, $r = .11$, $p > .05$ recorded. However, a significant correlation was found between the number of sunshine and number of vegetative symptoms, $r = .22$, $p < .05$. Nevertheless, as this correlation was not in the expected direction, this hypothesis was not supported.

Table 3

Age and Meteorological Associations with Depression and Vegetative Symptom Endorsement (N=81)

	Depressive symptoms	Minimum temperature	Sunshine hours	Age	Vegetative symptoms
Depressive symptoms		.12	.11	-.08	.08
Minimum temp. (^{o}C)			-.22*	.14	-.31**
Sunshine hours				-.01	.22*
Age (yrs)					-.17†
M	6.36	11.21	4.16	43.43	1.67
SD	0.78	3.97	3.27	12.44	0.76

†$p < .10$, one tailed; *$p < .05$, **$p < .01$, two tailed.

Temperature was expected to be associated with higher depression and vegetative symptoms for women but not for men. For men no significant correlations were observed between their depressive and vegetative symptoms and temperature (see Table 4). Significant negative correlations were observed in the women's group between vegetative symptom endorsement and minimum temperature, $r = -.38$, $p < .01$; and between minimum temperature and depressive symptoms, $r = .27$, $p < .05$ (see Table 4). Therefore this hypothesis has been partially supported as women reported higher vegetative symptoms on days with lower temperatures. However, the correlation between the number of depressive symptoms endorsed and lower temperatures was not in the expected direction, therefore; this aspect of the hypothesis was unsupported.

Higher numbers of vegetative symptoms were reported by men on days of higher sunshine hours, $r = .45$, $p < .05$. However women showed no significant relationship between sunshine hours and vegetative symptoms.

Table 4

Comparisons of Men's and Women's Correlations on Temperature, Sunshine Hours, Depressive and Vegetative Symptom Endorsement (N = 81)

	Women (n =53)		Men (n = 28)	
	Depressive symptoms	Vegetative symptoms	Depressive symptoms	Vegetative symptoms
Minimum Temperature	.27*	-.38**	-.05	-.12
Sunshine hours	.02	.11	.13	.45*
Vegetative symptoms	-.15		.31	
M	6.47	1.75	6.14	1.50
SD	0.70	0.73	0.89	0.79

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

A chi-square analysis showed that there was no significant difference in proportions of women who endorsed increased appetite as opposed to men, χ^2 (1, N = 81) = 0.69, p > .05. Therefore, this hypothesis was unsupported with women no more likely to present with changes in appetite compared to men.

Discussion

Sample Characteristics

Overall, the Study 2 sample was met preliminary criteria for depression as they endorsed the majority of the TSI depression screening items, although the measure may be considered to be an indication of depression rather than a diagnostic tool. Changes in sleep, appetite and increased fatigue were experienced by varying proportions of the sample. At least one vegetative symptom was endorsed by nearly half of the sample. However, only 13% of the sample endorsed all the vegetative symptoms. This result was in line with findings by Magnusson and Partonen (2005) and Murray and Hay (1997) where 10-20 % of their samples were considered to have seasonal depression. However, in some studies the incidence of seasonal depression was found to be lower (1-3%) in

temperate climates (Corral, Wardrop, & Zhang, 2006; Morrissey, Raggatt, James, & Rogers, 1996). However, the endorsement of individual symptoms in this study, for example, increased appetite or sleep, was similar to results found in previous Australian studies (see Boyce & Parker, 1988; Murray & Hay, 1997). As in Study 1, the gender breakdown was consistent with pervious research (Amons et al., 2006; APA, 2000; Levitan et al., 1999; Sweedo et al., 1995).

Seasonality in the Sample

Seasonality has been defined as the presence of vegetative symptoms associated with the change in season but not the presence of an MDE (Rosenthal, 1989). The incidence of seasonality was noted as being up to 19% in some studies (see Chotai et al., 2004b; Morrissey et al., 1996; Rastad et al., 2005). Seasonality in this sample was not formally assessed however a number of potential participants (n = 14) failed to reach the clinical threshold for depression in this study but nonetheless experienced some vegetative and low mood in relation to a seasonal presentation. This finding supports literature that suggests that seasonal variation in vegetative symptoms and mood may be a global concept as New Zealand has not previously been included in the data sources.

Vegetative and Depression Scores

The lack of correlation observed between depression and vegetative symptoms indicates that these variables may be regarded as independent of each other. This would be in line with the DVH whereby the depressive symptoms were presumed to present as a reaction to the appearance of the vegetative symptoms (Young, 1999). In the DVH it was thought that vegetative symptoms would develop as a response to biological and meteorological factors rather than an atypical aspect of the depression per se. This is consistent with research by Stewart et al. (1990) where SAD and atypical depression were believed to be different disorders.

Although increased appetite and sleep were normally distributed, increased fatigue was not. For those who endorsed increased fatigue, 38% also cited fatigue as their only vegetative symptom. Additionally, the fact that the majority of this sample endorsed

fatigue irrespective of other vegetative symptoms indicates that fatigue is more aligned with depression and mood disorders in general rather than specifically with seasonality or SAD in this sample.

Season and Symptom Endorsement

The relationship between season and depression and vegetative scores was explored. The lack of relationship found between season and depressive symptoms was in line with other findings that depression was not related to season (Michalak et al., 2004). However, that there was no relationship between vegetative symptoms and season found was unexpected. An increase in vegetative symptoms would be expected if the PSH and other neurotransmitter based theories were correct. However, the lack of variance between the winter and non winter groups indicated that the photoperiodic base of these theories may not play a major role in the development of seasonal depression in this sample. This result is in line with findings by Murray and Hay (1997), who also noted that photoperiod appears to play a limited part in the development of SAD in their study (based in Australia). The non significant result may have been due to the small numbers of the sample that presented with more than one vegetative symptom. However, there were significant differences in the proportions of people who applied in the winter versus the non-winter period. This indicated that there may be a subtle relationship between presentation and season that were not revealed in this study.

Age and Symptom Expression

In hypothesis 6 the use of age as an independent variable was examined. There was limited support found for the decrease of vegetative symptoms with increasing age that was in line with other findings (Sweedo et al., 1995; Kovalenko et al., 2000). However, the relationship was weak and not significant in this sample. It is possible that there were a number of older people in the original sample that had no vegetative symptoms, but who did not meet entry criteria for the study. However, there was no significant difference found between age and depressive symptoms which indicated that depression presented similarly across the age spectrum in this study. Conversely, the findings regarding men having a negative relationship with vegetative symptoms and age as

compared to women indicated that women appear to present with similar symptom expression regardless of age, whereas, men's may alter with age.

Gender, Symptoms and Meteorological Variables

Overall, women tended to endorse more depressive symptoms than men. This result was in line with other findings whereby women are presumed to be more affected by depression then men (APA, 2000). Hypothesis 8 indicated that women experienced a greater relationship between both their depressive and vegetative symptom endorsement and temperature. This supported findings by Chotai et al. (2004a). However the findings were weak in this study and so may not be reliable.

However, the positive relationship found between vegetative symptoms and sunshine hours in the men's group was unexpected (see Table 4) as a negative relationship would be more in line with other findings (Chotai et al., 2004a). Therefore, the origin of these results requires further clarification. Additionally, the results found in relation to vegetative symptom expression for the men's group indicated that there were aspects of gender or the relationship between temperature and sunshine hours that were not exposed in this study. Alternatively, this may be further evidence for the presence of atypical depression in this sample due to the presence of vegetative symptoms that do not appear to respond to light exposure.

Although previous findings would point to women being more likely to be represented in the group with increased appetite, (Chotai et al., 2004a) this result was not found in this study as examined in hypothesis 10. However, these results were in line with the generally contrary nature of findings regarding gender and symptom endorsement in this study thus far.

Hours of Sunshine and Symptom Expression

The lack of correlation between depressive symptoms and sunshine hours indicated they were not related in this sample. This result was similar to Young et al. (1997) where

they also found a lack of significance in the relationship between daily hours of sunshine and symptoms of depression.

A significant negative correlation between vegetative symptoms and sunshine hours would support the LDH, whereby decreased photoperiod is related to decreased neurotransmitter production and increased vegetative symptoms. Furthermore, the reduction in neurotransmitters is known to result in altered sleep and appetite (Burton et al., 2009). However, the results in this study were unexpected with a significant positive correlation found between vegetative symptoms and sunshine hours. As the correlations were not in the expected direction, where they fit with other studies is uncertain. It may be that these results are indirectly due to the negative correlation observed between temperature and sunshine hours. In Study 1 sunshine hours were found to be negatively correlated with temperature indicating that clearer/sunnier skies were associated with cooler temperatures. Thus, the result reinforced previous notions that in this study, temperature may have affected the sample subtly as well as directly.

Additionally, data indicates that vegetative symptoms are independent of season in this study. This lends weight to the notion that a decrease in bright light may influence the development of vegetative symptoms regardless of season. In this study there was no significant correlation between season and sunshine hours found which indicates that there were periods of low light in the summer months as well as in the winter months. This would result in vegetative symptom development in vulnerable individuals if the LDH were valid. Alternatively, there may be a portion of the sample who have presented with an atypical depression consistent with findings by Stewart et al. (1990).

Limitations

Study 2 examined a group of people who were assessed for the presence of symptoms of depression. This study has a number of limitations. Firstly, the TSI was not completed by all of the original participants, which would affect the data analysis and the ability to clarify some of the ideas presented in this study. Additionally, the moderate

screening ability of the TSI may have served to eliminate people from this study who may have been experiencing depression. Furthermore, the TSI is a self report measure and as such has inherent bias which may have influenced results. The low internal consistency of the TSI indicated that it would not be useful as a stand- alone screening tool for depression and might have resulted in type II errors. Also, the dichotomous nature of the TSI might have affected the size of the correlation coefficients between the TSI and the BDI-II as the strength of the variables was not able to be explored fully. The use of the TSI assessment date as opposed to original presentation date for this study may serve to limit the findings in relation to Study 1. However, as the dates were often within a week of each other it was not considered to significantly affect the results.

Secondly, there were no significant relationships found between the season and the endorsement of depressive or vegetative symptoms. This result may be due to the small numbers who presented with more than one vegetative symptom in this study. The majority of people in this study indicated fatigue as their only vegetative symptom. Because most of the sample indicated that they experienced sleep disturbance (decreased or increased) it would be expected that those who were getting limited sleep would be fatigued/tired as well as those with increased sleep/seasonal symptoms. Therefore, there was a large overlap between the measures of sleep disturbance and fatigue. This meant that fatigue was difficult to separate as a vegetative symptom, as opposed to a 'typical' symptom of depression. As fatigue was associated with non-seasonal depression, as well as seasonal depression, this may have influenced the results. Consequently, it is possible that a larger sample may have improved the power of this analysis.

Another limitation was the persistent lack of relationship found between sunshine hours and season. This may be a result of the single data point used to collect meteorological variables. However, this finding raises the notion that vegetative symptoms can manifest in summer in New Zealand as a result of periods of decreased sunshine (from cloud). Although this result would support the effect of sunshine on neurotransmitter production it would be unexpected from a seasonal depression perspective. Additionally, this would be in contrast to previous findings. However, the

consistency of these findings with those in Study 1 may reinforce the belief that photoperiod is not a major contributor to depressive or vegetative symptoms in this study. Furthermore, although temperature was found to have significant relationships for women in regards to their initial assessment, the relationships were not large and so may be a result of type II error.

Although Auckland experiences approximately 2,050 hours of sunshine/year, (M = 5.62/day: NIWA, 2009), the means found in Study 2 were lower which may influence the generalisability of these findings.

Overall the use of correlations in this study is a weakness as it limits the ability of the study to explore causality in a meaningful way. However, as this thesis is an exploratory one, looking at the strength of the relationships was appropriate as the data was taken from another study and so could not be used in a definitive way to ascertain levels of seasonal depression. Nonetheless, this study did allow for the exploration of seasonality within this sample and served to validate the use of a more focused approach in future research.

Summary

Overall, the majority of people who were assessed with the TSI were thought to be clinically depressed. Additionally, this sample had vegetative symptom endorsement consistent with other research. In Study 2 results showed that vegetative and depressive symptoms may be regarded as independent of each other. These findings would support the DVH in this sample. There were some difficulties around the usefulness of the endorsement of fatigue in this study due to its presence in the majority of the sample not just those with additional vegetative symptoms. Therefore, the ability of fatigue to differentiate between seasonal and non-seasonal depression may be limited. Once again the majority of the sample presented in the winter months, however, the winter group was not found to be more depressed or to have significantly more vegetative symptoms than the non-winter group. This finding has implications for the presence of seasonal

depression in this sample as the defining features of the condition were not clearly observed.

Sunshine hours were found to have no relationship with depressive symptom endorsement. However, a trend was noted with increased sunshine hours and increased vegetative symptoms. It is possible that this result pertained to previous findings where higher sunshine hours were associated with lower temperatures. Thus, indirectly temperature may have affected these results. Alternatively, findings may point to the presence of atypical depression. Therefore, it would appear that temperature requires further exploration in order to clarify these results. Overall, temperature was found to have an effect on overall depressive and vegetative symptom endorsement in women but not in men.

There was no significant relationship found between increased appetite and women which is unexpected from the results of previous studies. In addition, age was found to have a negative trend noted as regards total vegetative symptom endorsement, which was consistent with other research.

How meteorological factors, gender, age and season effect change in depression over time will be the focus of Study 3. Data will be collected longitudinally in order to explore how these differences effect change within and between the participants.

CHAPTER SEVEN:
STUDY THREE

Aims

In the previous studies of this investigation, the symptoms of depression and expression of seasonal symptoms were examined at presentation. The main aim of Study 3 is to explore the symptoms of depression and vegetative behaviours in response to meteorological factors and the course of seasonality over the course of therapy. Literature related to symptom change over time and use of multilevel analysis as a way to explore this are reviewed. The studies aims are achieved through an initial examination of the descriptive breakdown of the sample's presentation over time for both general depressive symptoms and vegetative symptoms. This is followed by the use of multilevel modelling (MLM) to ascertain whether seasonal factors have a significant effect on depression or vegetative symptoms in this sample. Subsequently, the change over time in vegetative symptoms is investigated in order to see whether it is predicted by gender, age, meteorological factors and season. Finally, irritability is explored in relation to the expression of seasonality in this sample.

Depressive and Vegetative Symptom Change

It has been previously stated that a change in depressive symptoms can result from a decrease in vegetative symptoms (Rosenthal et al., 1984; Young, 1997). The temporal precedence whereby a decrease in vegetative symptoms led to a decrease in depressive symptoms has been demonstrated in a number of studies (Lewy et al., 1989). However, the Dual Vulnerability Hypothesis (DVH) postulates that depressive and vegetative symptoms arise from separate vulnerabilities that exist in certain individuals (Young et al., 1991). Consequently, it is reasonable to hypothesize that the rate of change over time for each group of symptoms (general depressive and vegetative) would also be different and influenced by different variables. However, there appears to be little research on the subject with the decrease in the depressive and vegetative symptom clusters associated with seasonal depression not investigated as separate and change for both assumed to be temporal. This is partially due to the assumptions of the majority of theories around

seasonal depression which link the disorder to biological factors and the common treatments for SAD which reflect that ethos.

Various studies have explored how depression is related to weather variables. A study performed by Denissen, Butalid, Penke, and van Aken, (2008) examined the relationship between mood and weather over the course of two years using multilevel analysis. This study found no relationship between temperature, sunlight and wind on negative affect over time. However, the authors did not use direct measures to assess depression but a diary and self report in order to explore the five factor model of personality in relation to mood. Therefore, this study can only provide an indication around the effect of weather variables on mood. However, their results are in line with Michalak et al. (2004) who also found weather to have little impact on depression. Nevertheless, Denissen et al.'s study (2008) did observe a relationship between tiredness and sunlight and also found that sunlight mediated the effects of tiredness on rain and air pressure. These findings indicate that sunlight affects levels of fatigue, thus providing support for the effect of sunlight on vegetative symptoms although this would need to be explored more fully. Therefore, there is some support for the belief that depressive and vegetative symptoms may be affected by different variables.

Other studies have explored the relationship between weather, season and mood. Huibers, et al. (2010), found that numerous weather variables did not affect mood but that season was associated with the prevalence of major depression and sad mood. They were unable to account for this finding in relation to weather variables which are the main determinants of the seasonal depression hypotheses. However, Huibers et al. (2010) noted that weather variables fluctuated daily as well as between seasons which meant that their impact was difficult to ascribe in a particular way. The relationship found between season and mood implies that there are other aspects involved in their findings that were not fully explored in this study. As the study by Huibers et al. (2010) did not specifically explore seasonal depression, the role of vegetative symptoms in this study was not examined which limits the application of this research. However, it provides some support for the effect of season specific schemas as postulated by Rohan (1998).

Exploration of general seasonal symptom change over the course of therapy has been performed (Rohan et al., 2003; Rohan et al., 2007). However, these studies examined overall symptoms of seasonal depression rather than separating out the symptom clusters in order to ascertain if depressive and vegetative symptoms were affected differently by therapy.

Therefore, whether depressive and vegetative symptoms have similar rates of change is a question that has not been directly addressed in previous research and will be explored in this study. Additionally, whether a decrease in vegetative symptoms is necessary in order for a decrease in depressive symptoms to occur has not been fully explained. Furthermore, there is little data on whether vegetative symptoms can return seasonally in some individuals without a corresponding significant increase in depressive symptoms. Consequently, exploring symptom expression over season change would provide data on the separateness of the symptom clusters.

Hypotheses

MLM Hypotheses

Hypothesis 13: BDI-II scores will show higher rates of change than vegetative scores over time.

Hypothesis 14: Higher vegetative symptom expression will be associated with lower sunshine hours over time.

Hypothesis 15: Sunshine hours will show a significant effect on rate of change in depressive and vegetative symptom scores.

Hypothesis 16: Temperature will show a significant effect on rate of change in depressive and vegetative symptom scores.

Additional Hypotheses around Symptom Expression explored in Study 3

Hypothesis 2: Higher vegetative symptoms will be associated with the winter season of presentation.

Hypothesis 4: Levels of irritability will be positively correlated with levels of vegetative symptoms and levels of vegetative symptom severity

Those who are considered to be 'seasonal' will show higher levels of irritability than those considered non-seasonal.

Hypothesis 6: Age will be negatively correlated with levels of irritability.

Hypothesis 8: Women will have higher BDI-II and vegetative scores in comparison to men.

Hypothesis 11: Men will report higher levels of irritability than women.

Method

Participants

This sample consisted of those who were accepted into the treatment phase of the parent study ($N = 29$). However, in this study one person was excluded from analyses due to their early exit from the study and the limited data points (3) that they were able to contribute to the study. Therefore, the final sample for this study was $N = 28$. Their symptoms were consistent with a first episode MDE and they were considered to be suitable for outpatient treatment. Also the participants indicated they were able to receive therapy twice each week for eight sessions and then weekly until the conclusion of therapy (up to 20 sessions).

The sample consisted of 10 men (36%) and 18 women (64%), with an age range of 20- 63 years, ($M = 44.61$ years, $SD = 11.71$). Ninety-six percent of the sample ($n = 27$) was NZ/European; however this included a variety of people originally from other countries, who had lived in NZ for varying lengths of time. Thus ethnicity in this sample was difficult to define. Thirty nine percent of this sample ($n = 11$) were university graduates, with only 11% having no tertiary education ($n = 3$). Thirty-six percent of the sample ($n = 10$) had received some previous therapy or medication for another psychological disorder, with the remainder having had no prior contact with mental health services. Approximately half of the sample was employed (57%, $n = 16$), with 25% self-described as being housewives ($n = 7$). The majority of the sample had children, with only 29% of the sample childless. Thirteen of the group were married or in a de

facto relationship (47%), with 15 members (53%), single, divorced, separated or widowed.

Therapist Demographic and Training Information

The therapists were psychology students enrolled in doctoral programs (for psychology) at Massey University ($N = 7$). The therapist's ages ranged from 23- 50 years. All of the therapists were women. The therapists' previous experience in providing therapy ranged from 1-5 years. Additionally, the therapists all had completed post-graduate psychology degrees to at least Honors level.

The therapists provided cognitive behaviour therapy (CBT) for the study over various periods of time. For the period of 2007- 2008, three of the therapists were involved in providing therapy, after that from mid 2008-mid 2009 another four therapists provided therapy for the sample. Each therapist provided therapy for 2-9 clients. The number of clients seen by each therapist was dependent on the client's length of involvement in the study and the client allocations made at the time.

The therapists attended two, one week block courses run by Massey University on Cognitive Behaviour Therapy and Cognitive Behaviour Therapy for Depression, (80 hours). Part of their training involved submitting three videos of interactions between the therapist and a 'client'. Overall, in excess of 100 hours total training in cognitive behaviour therapy (CBT) was provided for each therapist. Weekly supervision was provided for each therapist in order to maximize therapeutic gains and monitor client and therapist progress and safety. This was achieved by regular viewing of recorded sessions with clients which enabled the provision of feedback on processes and skills used as well as the early identification and intervention of any observed difficulties in the session.

Procedure

After the assessment procedure was completed and admission into the study was finalized, participants all received therapy twice each week for the first eight sessions, and then weekly until completion of therapy. Follow-up sessions were provided at the

two month and six month time points after therapy was completed. Follow-up was in order to assess the continued effectiveness of therapy after completion and care for any ongoing issues that may be causing concern.

CBT administration overview. In this study CBT was administered in a similar manner to other therapy manuals (see Greenberger & Padesky, 1995, for example). Therapy generally began with behavioural strategies including activation and pleasant activity scheduling, which would include outdoor activities. Then therapy would move on to more cognitively based strategies beginning with automatic thoughts and moving on to deeper level thoughts and finally schemas and core beliefs.

Measures

Daily Weather Variables and Season

The measures for Study 3 were the same as for Study 2. These variables included daily sunshine hours and daily low temperatures (NIWA, 2009). Daily weather variables were collected for each session and follow-up sessions attended. All data were normally distributed. Season was defined as in the previous studies by using solstice and equinox dates (Heidorn, 2008).

Demographic Information

Age and gender data were collected for use in demographic and hypotheses analyses (see Appendix F). Additional data on marital status, employment, education, and previous therapy was used only in initial demographic breakdown of the sample.

Beck Depression Inventory (BDI-II)

The BDI-II is a 21 item self-report measure used to screen for depression severity with possible scores of 0 to 63 (Beck et al., 1996). For research purposes, clients are usually classified as non-depressed if their BDI-II scores fall below 13, with 14 to 19 classed as mild depression, 20 to 28 moderate depression, and 29 to 63 for severe depression (Beck et al., 1996). The questions were measured along a Likert scale, for example, question 1 around sadness: with a score of *0* being no symptom presence, "*I do*

not feel sad" to *3- "I am so sad or unhappy that I can't stand it."* The BDI-II was used in the assessment of each client. The measure has been found to have good reliability and validity and has previously been used in a number of studies examining seasonal depression although only as an indication of depression in the samples (Michalak et al., 2002; McCarthy et al., 2002; Rohan et al., 2003; Rohan et al., 2007). A reliability analysis of the BDI-II was performed for this study and the results are presented in Appendix G.

The BDI-II was administered prior to each session and both the BDI-II and BDI-II change scores were collected for each session. The BDI-II change score was calculated by subtracting the current BDI-II score from the previous BDI-II score and used to provide an alternative perspective of change in depression between each session. The data were also analysed in order to assess whether the person had vegetative symptoms indicating the possibility of seasonal depression, or would be considered to be 'non-seasonally' depressed. This was achieved by examining the number of vegetative symptoms. The vegetative symptoms were identified as increased appetite (question 18), sleep (question 16) and fatigue (question 20). Additionally, the severity of increased vegetative symptoms were also recorded and analysed. For example, one person may have slight appetite increase and would record it as a 1 on the BDI-II indicating that their symptoms although present were mild compared to someone who experienced the symptom as a 3 which indicated a constant craving for food. However, should the sleep or appetite variable be considered to be 'typical' i.e. decreased, the severity of the symptoms would not be counted as a vegetative symptom score.

Data Collection

Measuring Symptom Change over Time

For a diagnosis of seasonal depression or an exploration of seasonal patterns in depression to be considered, there needs to be a change in season involved in the course of data collection (Murray, 2001). Although a variety of analyses can be employed to accomplish this process it is believed that multilevel modelling (MLM) would provide the most appropriate way of analysing change over time.

Multilevel analysis. Longitudinal data analysis is known by a number of names including: *individual growth models, random coefficient models, multilevel models, mixed models* and *hierarchical linear models* (Singer & Willett, 2003). These multilevel models (MLM) enable change to be measured over time despite the direction or variation of the change trajectory (Singer & Willett, 2003). Additionally, in multilevel analyses, change can be observed both within and between individuals.

An advantage of using individual growth modelling techniques is that multilevel modelling is designed to deal with the varying number of entries per person, and variable spacing between the data points (Singer & Willett, 2003). Previously, if data were missing from a portion of the study for a participant then all data from that participant was removed from the analysis (Kwok et al., 2008). However, because time is treated as a continuous variable in MLM, the analysis is able to use all data in an estimation of model parameters provided data is missing completely at random or; missing at random (Kwok et al., 2008). Additionally, data does not need to be collected in evenly spaced waves, in order to still be considered to be a valid measure, as change in a sample is expected to fluctuate over time (Singer & Willett, 2003).

Previously, large samples were required for accurate analyses. However, the increased number of data entry points (or waves) per person provided with MLM has been found to provide accurate results even with limited sample numbers (Willett, Singer, & Martin, 1998). The number of data points for each individual can be defined by the investigator (Singer & Willett, 2003). Given that individual growth modelling (or MLM) uses a person- period data set, each predictor can take on a specific value for each measurement occasion (Pan, Rowe, Singer & Snow, 2005). Therefore, it is possible to collect multiple data points (or waves of data) for each individual at set times. However, at least three waves of data are required if using linear individual growth modelling (Willett et al., 1998). Additionally, the use of multiple data points allows for the simultaneous examination of Level 1 (within person or individual change over time) and Level 2 (between persons) questions (Pan, et al., 2005).

Level 1 measures change in the individual over time which is considered to be relatively stable (Holden, Kelly, & Agarwal, 2008). However, it is also possible to examine intra-individual variability which refers to fluctuations in the trajectory of change. It is important to examine these fluctuations as they can influence understanding of change in relation to other members of the sample (Willett et al., 1998). Additionally, unexplored variability can either mask or result in mistaken conclusions about the nature of change in the sample (Holden et al., 2008). In the exploration of sunshine and temperature variables it was noted that their affect may not translate from one level to another (Huibers et al., 2010) and so they are best inputted first where their effect can be viewed simply.

On the other hand, Level 2 analysis measures change between individuals over time, and offers information about the relationship between individuals and answers questions around the consistency (or otherwise) of change between them (Holden et al., 2008). Here variables that are time invariant or less likely to change over the duration of the study for example gender and age are explored. Additionally, level 2 variables can be used as control variables in order to explain potions of the variance found at level 1 (Denissen et al., 2008; Singer & Willett, 2003).

Fitting a statistical model invokes assumptions around the relationship between the outcome and the predictors. Therefore, analysis is not complete without checking the tenability of these assumptions (Singer & Willet, 2003). However, only the observed properties of the sample quantities can be examined (Singer & Willett, 2003, p.127). Therefore, assumptions checks are most appropriately achieved through checking:

1. the shape of the data, through graphical representation,
2. the normalcy of distribution and,
3. The Homoscedasticity (Singer & Willett, 2003, p.129).

Alternatively Raudenbush (2004) indicated that assumption checking should involve clarification of the following:

1. Are the distributional assumptions realistic?

2. Are the results likely to be effected by outliers or influential observations?
3. Have important variables been omitted or non-linear relationships ignored?

It was felt that these questions could be addressed by "means of analysis of residual files*"* for level 1 and 2 (Raudenbush, 2004, p.15).

Importantly, the use of time as an explanatory variable is essential for the model to be conceptualized as a longitudinal model (Holden et al., 2008). However, time can be measured in a number of ways with data collection usually at times thought by the investigator to reflect the times when change was most likely to occur (Singer & Willett, 2003). In addition, strategic use of data points allow for the effects of therapy on the individual and so therapy could be taken into consideration without analysing therapy directly per se. In this study, a greater decrease in depression scores was anticipated at the beginning of therapy as opposed to change being evenly distributed throughout the course of therapy. This was based on previous research whereby expectations around being helped were involved in pre-therapy and early therapy change (Meyer et al., 2002; Poston & Hanson, 2010). Therefore, by increased data collection where periods of rapid change were expected and more data were available, it was expected that time should reflect the pace that was related to the study (Singer & Willett, 2003). Furthermore as there was no contact with clients for extended periods of time after therapy was completed it was not possible to collect data at evenly spaced intervals.

Additionally, different markers of time are able to be used if required (Singer & Willett, 2003). It has been hypothesized that 'session' may not be the most appropriate marker of time for the seasonal aspect of this study. Therefore, an alternative method of recording time has been put forward. The alternative method of documenting time involves the use of months as the time variable in order to take season into account.

Therefore, using multilevel modelling (MLM) to ascertain the rate of change for the depressive symptoms and vegetative symptoms (increased appetite, fatigue and increased sleep) over the course of therapy is expected to reveal differences between the

participants that may otherwise remain unobserved. These assumptions will be tested using the following hypotheses.

All therapy sessions were videotaped (with the client's permission, see Appendix B) in order to provide data for the Parent Study and to facilitate supervision of the therapists.

At the beginning of each session the client completed a Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996), which was used in the data analysis to track changes in mood over time and in relation to meteorological data. Questions on increased sleep (question 16), appetite (question 18), and fatigue (question 20) were used for exploration of the vegetative symptom profiles of each client. Additional questions in the BDI-II (i.e. question 17 on irritability) were used to correlate against other variables for example age and gender.

Terms. The term seasonal depression implies a chronic pattern with previous episodes of depression associated with a season. However, this study was unable to provide the retrospective component for this diagnosis. Therefore as regards Study 3 an episode of *seasonal depression* is operationalised in this study as: a current depressive episode which includes at least one vegetative symptom and occurs during the winter period with a remittance at the end of the season.
Seasonal: A person who presents in the winter period with at least 1 vegetative symptom from increased sleep or appetite.

Data Analysis

In this study the sample was analysed with different variables in order to ascertain which factors influenced BDI-II score change within the individual over time. In order to explore how individuals changed over time, a number of random predictors were initially explored. These factors included: sunshine hours, temperature, and the season of therapy. Additionally another model was constructed to explore the change over time in the

number of vegetative symptoms present with the same random predictors used. In order to ascertain if there were any differences between the individuals on these variables, individual growth trajectories were constructed and are presented in the results section (see Figure 4).

Data Management

Data shape. The possibility of inconsistency in the data and the shape of the data were considered. This meant that data transformations were a possibility. The shape of the data was ascertained through the examination of descriptive graphs showing the individuals BDI-II scores over time (see Figure 1). This was cited by Singer and Willett (2003) as the primary way of ascertaining whether the data were linear as assumed or manifested another shape. Because there were 28 participants in the study it was not expected that all would show the same data shape which was confirmed through examination of individual graphs. The summary of each individual's growth trajectory used a nonparametric approach in order to "let the data speak for itself" (Singer & Willett, 2003, p.26). This was intended to reveal the most appropriate trajectory form. A curve fit analysis performed in SPSS (2008) showed negligible differences between linear, curvilinear, quadratic or cubic fit models which indicated that any form may be used (Singer & Willett, 2003). Therefore, in Study 3 a linear trajectory was selected as being the most reasonable overall form as it enabled the participants to be compared using the same set of numerical summaries (Singer & Willett, 2003). Furthermore, the residual scatterplots presented in Appendix G showed random even scatter indicative of linearity.

Missing values. A number of the sample did not complete all sessions. This was for a variety of reasons including loss of contact with the client and rapid relief of depression. The expectation-maximization (EM) method was used to calculate the proportions of missing data. In this study the missing data were considered to be "missing completely at random" (MCAR) as the reason for the missing data was considered to be unpredictable or to have no discernable pattern (Tabachnick & Fidell, 2001). Additionally, it was believed that the 'missingness' was unrelated to the observed outcome (Singer & Willett,

2003). The proportion of missing data was calculated and found to be not significant (*N* = 1, percentage missing 0.4%, *ns*).

Normality and assumption checks. Normality was assessed via the use of Normal Probability Plots whereby the residuals for BDI-II scores, vegetative symptom scores (Figures 2,3,5, and 6), and sunshine and temperature readings (see Appendix F) were checked in order to see if they formed a generally diagonal line which would indicate a normal distribution. Additionally, the residual plots were examined in order to ascertain whether they were randomly scattered around the centre of the plot as opposed to the top or bottom as a further indicator of normality (Tabachnick & Fidell, 2001). The linearity of the relationship was checked via determining whether the distribution was approximately rectangular. Furthermore, residuals were checked for even distribution across the graph in order to check for homoscedasticity (Tabachnick & Fidell, 2001).

In this study, an intervention (therapy) was also in place over time. It was considered to be important to consider this variable as therapy would be considered to have an effect on depression scores. However, because all clients received therapy, it was not possible to compare them on the effects of therapy as an adjunct to the seasonal variables. Therefore, for the purposes this study it was accepted *a priori,* that therapy should be treated as an intrinsic part of the "time" variable. This was considered to be a reasonable supposition in that all participants showed a reduction in depressive symptoms regardless of the therapist involved, the length of their therapy, or when therapy took place. This indicated that therapy was beneficial for the entire sample and that differing results between individuals were attributed to alternative factors.

Measuring time. In this study data were collected for each client for up to nine time points during the course of therapy. Therefore, the data collection times were:

1. At initial assessment – in order to provide a base line for severity
2. Session 1- to establish any change from first contact. This may be related to relief around the prospect of receiving help (Meyer et al., 2002)
3. Session 3- to monitor change in the first week of therapy

4. Session 7- to ascertain change during the initial twice weekly therapy sessions which may indicate "flight to health" in some clients and rapid gains in therapy (Stulz, et al., 2007)
5. Session 10- to ascertain progress halfway through therapy
6. Session 12- to determine change as some discontinued treatment at this time point
7. Session 15- to monitor therapy at the three quarter mark
8. Session 20- to discover total treatment gains over the course of therapy
9. Last contact session – to ascertain total therapy gains

Months was used as an alternative time marker. Here the session dates were used, and the data were recorded as months of the year. The month of each session was documented and then further broken down into weeks in the month and recorded. Furthermore, when the client continued therapy over to the following year (e.g. from June 2008- June 2009) time was recorded as a continuous variable. For example, client 57 commenced therapy in November, therefore, for this person: *time 1* = 11. 4 (November); *time 2* = 14 (beginning of February the next year); *time 3* = 14.2 (in February); *time 4* = 14.5 (February); *time 5* = 15.4 (March); *time 6* = 16 (April)…..*time 8* = 17.4 (May); and *time 9* = 19.5 (July). Client 69 began therapy in June, therefore their time points were: *1* = 6.8; *2* = 7; *3* = 7.2; *4* = 8; *5* = 9; *6* = 9.5; *7* =10.5; *8* =12; and *9* = 18.8. The use of the decimal point in this data collection was intended to aid clarification on when in the month the client attended, for example 18.8 would signify contact in the last week of the month. This was because as previously noted therapy was twice a week initially which meant that there were a number of sessions that would appear to occur at the same time. Separating the data more precisely would also aid visualization of the data. Using this alternative method for measuring time it was hypothesized that season/month ergo "seasonality" may be explored as a time variable. It was also believed that this may more accurately account for the time variance experienced between members of the sample. Additionally, graphical display of data collected in this manner enabled the visualization of seasonal fluctuation in vegetative symptoms to be observed.

Waves of data. In this study five to nine waves of data were collected for each individual.

Introductory Information for Model Building

Graphs. Descriptive graphs were produced for each of the dependent variables used in this study (Figures 1 and 4). The most important reason for performing exploratory analyses of the data were in order to ascertain the nature and idiosyncrasies of each individuals pattern of change (Singer & Willett, 2003). Additionally, this enabled the exploration of symptom severity and stability of symptoms to be assessed.

Having established two possible time variables, the use of graphs revealed how individuals changed over time in relation to these variables. This enabled visualization of comparable data in order to ascertain which variable would provide the most accessible model for the hypotheses. Empirical growth plots revealed how each person changed in relation to session and time of the year in relation to both absolute terms (overall) and relative terms (in relation to others) (Singer & Willett, 2003). In this study both BDI-II scores and vegetative symptom scores were analysed in this way and are presented in the results section (Figures 1 and 4). This was intended to facilitate preliminary comparisons and reveal the stability of vegetative symptoms in comparison to change in other 'typical' depressive symptoms.

Regressions. Regression analyses were performed with BDI-II and vegetative symptoms scores over time. The regressions produced an intercept (constant) and slope for each client which was used for future correlations (see Table 6). This allowed correlations to take the longitudinal nature of variables into account. The use of ordinary least squares regression (OLS) was used to further explore the data and provide data for summary statistics, that is, each person's R^2 and residual variance statistics in order to ascertain their goodness of fit

Correlations of prospective predictors. Correlations were performed between the BDI-II scores and sunshine and temperature raw data. Additionally, vegetative symptom

number and severity, and irritability were calculated along with age and gender in order to see if significant relationships existed between the variables (see Table 5). Further correlations were performed around mid and end BDI-II scores, the number of sessions a client had, age, and gender (Appendix I). Additional correlations were performed using the intercept and slopes of the individual change trajectories of the BDI-II in order to see whether there was any correlation between age, initial status and rate of change (see Table 6). This information was considered important for the building of the multilevel model using age as a predictor.

Multilevel Model Building

In this study two separate models were built in order to explore the predictors of change in BDI-II and the predictors of change in vegetative symptom expression. Therefore, for each model level both aspects will be explored. The general syntax used is documented in Appendix J.

Model A. "Model A", or the "Unconditional Means Model" (UMM: Singer & Willett, 2003), was constructed first. The UMM was based on the premise that at Level 1 the true change trajectory for the individual is flat (Willet et al., 1998). Additionally, the lack of a time predictor meant that there was no slope. The Level 2 aspect of the sub-model indicated that the elevation of the trajectories may differ (Singer & Willett, 2003). Model A had no predictors and does not describe change but describes only the outcome variation and serves as a baseline for model comparison (Singer & Willett, 2003). In Study 3, the dependent variables used for each of the MLMs constructed in Model A were BDI-II and vegetative scores. In this model the raw scores were entered into the model alone. This base model indicated whether the BDI-II scores and the vegetative scores were not equal to zero in order for there to be a rationale for future model building. In this case the participants BDI-II and vegetative scores differed from each other and differed individually over time. Therefore, there was enough variance at Levels 1 and 2 that were not explained to justify the addition of predictor variables in an attempt to explain the variance.

Model B. "Model B", is also known as the "Unconditional Growth Model" or UGM (Singer & Willett, 2003). 'Time' was added to Model A in the form of *session* for examining the factors that effected BDI-II scores, or *month* for the model exploring vegetative symptom scores (Table 7) in order to ascertain whether the scores were still significantly different to zero after 'time' was accounted for. All the variables in this equation were random and had an assumed normal distribution of error in the population (Cohen, Cohen, West, & Aiken, 2003). It was expected that there would be variance within individuals over time. This model was intended to show how much variance was accounted for by time at Level 1 or 2, as seen by a decrease in the variance components from Model A to Model B. If the variance remained significant after the decrease this indicated that not all of the variance had been explained at that point. Pseudo R-Square statistics were determined by calculating the percentage change between the models in terms of the variance components which gave an exact percentage of the variance explained at Level 1 and 2 by time. For Level 1 the individuals BDI-II score at each time point was considered to be the result of their initial status (Π_{0i}), and rate of change (Π_{1i}) (see Table 7). Level 1 variables were considered to be time varying predictors as they could potentially change over time and therefore are measured at each time point in order to be used in the model (Holden et al., 2008).

Goodness of fit statistics is also obtained at this point. These included the Deviance statistic (2-Log Likelihood) which compares the current model with a *saturated model* and quantifies how much worse the current model is in comparison, that is the smaller the statistic the better the fit (Gelman & Hill, 2007). Additionally, two *ad hoc* criteria were used to explore relative goodness-of-fit of the models which are based on the deviances statistic but decrease the log likelihood in response to specific criteria (Singer & Willett, 2003). The Akaike Information Criterion (AIC; Akaike, 1973) was one of the statistics gathered. An observed reduction in the AIC revealed a corresponding reduction in the out-of-sample prediction error, thus the statistic compensated for the number of parameters in the model in order to reveal the actual state of the model fit (Gelman & Hill, 2007). The Bayesian Information Criterion (BIC; Scharwz, 1978) was also collected. This statistic made adjustments for the sample size and the number of

parameters used in the analyses (Singer & Willett, 2003). Both the AIC and the BIC can be compared if they are fit to the same data; therefore, whichever statistic has the smaller information criteria is considered to fit better (Singer & Willett, 2003).

These statistics are used because adding variables to the model even if they were not informative would increase the "fit" of the model (Gelman & Hill, 2007). A decrease of 1 could be expected merely through the addition of another 'noise' variable (Gelman & Hill, 2007). Therefore, a decrease of at least 2 would be considered to be necessary for any improvement of fit to be attributed to the variable added. However, small improvements may not be statistically significant but the variables may be retained if it is considered to 'fit' the ethos of the study (Singer & Willett, 2003).

Model C. In "Model C" time varying or main predictors are added to the model. The predictors chosen were associated with theories around the development and maintenance of seasonal depression. The variables used were added in order to determine whether they were predictors of initial status and or change (Singer & Willett, 2003). In this study, both daily sunshine hours and daily low temperatures were used as predictors for both BDI-II scores and vegetative symptom models. Because they both showed significant correlations with different aspects of the BDI-II scores there was no indication that one variable was more appropriate than the other (see Table 5). The same variables were used in both models in order to ascertain whether there were any similarities between depression and vegetative symptom change in their trajectories at either Level 1 or Level 2. The variance components for each factor were assessed in order to ascertain whether the need to explore the effects of other time varying predictors was required. Gender and age are also added to the depression model at this level in order to ascertain their affect on BDI-II scores.

Model D. Time invariant predictors were added at this point. They were used as ways to control for the effects of the time varying predictors that were considered to 'fit' the model better. This enabled any potential groups that may be significant to be explored and the effects of these variables controlled for in the analyses (Singer & Willett, 2003).

In this study, the variables used were age and gender. It was expected that gender would be the stronger predictor in light of previous research, however the variable that explained the most variance is the one kept for further analyses. Because these variables only show effect between individuals at Level 2 they were not inputted earlier into the vegetative symptom change model.

Post hoc analyses. After the model had been constructed additional analyses were run using alternate variables in the "Depression" model. Season of therapy was entered at model C in order to ascertain whether this variable explained more of the variance than temperature or sunshine alone. As an alternative to season of therapy, season of presentation was also used as a time invariant predictor in order to ascertain whether this variable influenced rate of change between individuals.

General Data Analysis

Data were collected longitudinally over the course of therapy and follow-up. The data analyses were carried out using SPSS for Windows version 17 (SPSS Inc., 2008). The data were primarily analysed using SPSS MIXED. The syntax used for MLM analyses was obtained from the Applied Longitudinal Data Analysis website (ALDA: Appendix I; Singer & Willett, 2003a). Although models of change for depressive and vegetative symptom change were constructed, they were considered to be preliminary models due to the exploratory nature of this study and the small sample size. Therefore, a $p < .10$ level of significance was considered appropriate for this study in order to increase the powere of the analyses. Additional hypothesis testing was conducted using chi squares, correlation and t-tests in order to ascertain the presence of significance in the relationships between the variables.

Results

General Results

Twenty eight participants took part in the study. Three participants commenced therapy in the autumn (11%), nineteen in the winter (68%) and six (21%) in the spring. No participants commenced therapy in the summer period. The sample attended therapy from 6 – 22 sessions, $M = 19.14$, $SD = 4.21$. The majority ($n = 19$) attended at least 1 follow-up session. However, only 5 were able to be contacted for the final follow-up session

Initial correlations presented in Table 5 showed that sunshine was associated with change in BDI-II scores and temperature with BDI-II scores. However, there were no significant associations between meteorological variables and vegetative symptom total or severity. Of note were the findings around irritability which showed significant correlations with vegetative symptoms, and low temperature. Other correlations performed between age and session and BDI-II scores showed no significant relationships (Appendix I).

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Table 5

Relationships between Vegetative Symptoms, Irritability, Meteorological Symptoms, BDI-II (BDI) Scores and Change in BDI-II (chBDI) over 235 measurement occasions (N=28)

	Sunshine hours	Low temp	Vegetative severity	Vegetative total	Irritability	BDI	chBDI	fatigue	Sleep & Appetite
Sunshine	-	-.12*	.01	.03	-.04	-.02	-.14**	-.02	.04
Low temp	-	-	.01	.07	-.16**	-.13**	.01	.01	.03
Vegetative severity	-	-	-	.68***	.25***	.39***	-.22***	.38***	.75***
Vegetative total	-	-	-	-	.35***	.36***	-.19***	-	-
Irritability	-	-	-	-	-	.64***	-.19***	.53***	.18***
BDI	-	-	-	-	-	-	-.21***	.74***	.18***
chBDI	-	-	-	-	-	-	-	-.13**	-.16*
Fatigue	-	-	-	-	-	-	-	-	.12***
M	5.615	10.94	0.63	1.25	0.91	19.24	2.51	0.94	0.56
SD	3.5585	4.35	0.81	0.89	0.81	12.48	5.63	0.84	0.66

*p <.10, **p <.05, ***p <.01

Season as a predictor of change. A Chi square analysis of proportions showed that there were significant differences in the distribution of vegetative symptoms compared on what time of year (season) the participants commenced therapy, χ^2 (6, N = 28) = 17.27, $p < .05$. For those who commenced therapy in the autumn (n = 3), one presented with vegetative symptoms. Of those who commenced therapy in the winter period (n = 19), 13 had vegetative symptoms and a further 5 presented with fatigue only. There was 1 person in the winter group who had no vegetative symptoms. The participants who started therapy in spring (n = 6) consisted of two people who had vegetative symptoms and one with increased fatigue, the other 3 members of this group had no vegetative/atypical symptoms. No participants began therapy in the summer period.

At the end of therapy no significant difference in proportions found in distribution of vegetative symptoms, χ^2 (4, N = 28) = 4.87, $p > .10$. For those who commenced therapy in the autumn, 2 of the 3 people had vegetative symptoms at the end of contact despite this no longer being in the winter period. Those who received therapy beginning in winter, 10 endorsed no seasonal symptoms at the end of contact, and 3 experienced a return of vegetative symptoms in the following winter follow up period. All 3 who had a return of vegetative symptoms the following winter period were women. However, 6 of the winter group continued to have residual vegetative symptoms despite it no longer being winter. For those who began therapy in the spring, 1 person was noted to have vegetative symptoms associated with the winter season, that is, they developed spontaneously as the season changed into winter during therapy. The remaining members of this group had no vegetative symptoms or fatigue at end of contact.

Results indicated that the winter season was significantly associated with vegetative symptom expression; therefore the hypothesis is met.

Preliminary Information for Model Building and Assumption Testing

Change in general depression scores. All of the sample experienced symptom relief relative to initial BDI-II scores (see Figure 1). However the rate of change was different for each client. The initial BDI-II scores ranged from 13-52, (M = 30.68, SD = 10.55). At the midpoint of therapy (session 10) the BDI-II scores ranged from 3-53, (M =16.89, SD

= 11.69). The final BDI-II scores ranged from 0-31, (*M* = 9.32, *SD* = 8.09). The number of waves of longitudinal data collected for each participant ranged from 5–9 waves, (*M* = 8.36, *SD* = 1.06). Half of the sample was considered to have mild to moderate depression and the other half severe depression at the beginning of therapy. At the midpoint of therapy contact nearly 50% of the sample no longer met the threshold for clinical depression and by the end of contact 68% no longer met the threshold for clinical depression. Of those who were still clinically depressed at the end of contact: 6 were classified as mild, 2 as moderate and 1 remained severely depressed. The person who remained severely depressed had a final BDI-II score of 33 and an initial BDI-II score of 53. Therefore, they still experienced significant therapeutic gains whilst in therapy. These results indicated that therapy was effective for all participants in the study; however the degree of change varied for each participant.

Table 6

Correlations on the effects of initial status, rate of change and age on BDI-II scores (N = 28)

	Initial status	age	Rate of change
Initial status	-	-.121	-.**440***
age	-.121	-	-.226
Rate of change	-.440*	-.226	-
M	27.70	45.07	-1.02
SD	11.11	11.58	.69

$^{*}p < .05$

Correlations performed using the regression slope analyses involving the constant and slopes of the individual trajectories indicated that there was a significant association between the initial status and rate of change (Table 6). This meant that those with higher initial BDI-II scores showed a greater rate of change in BDI-II scores. However, there were no significant relationships found between age with either initial status or rate of change.

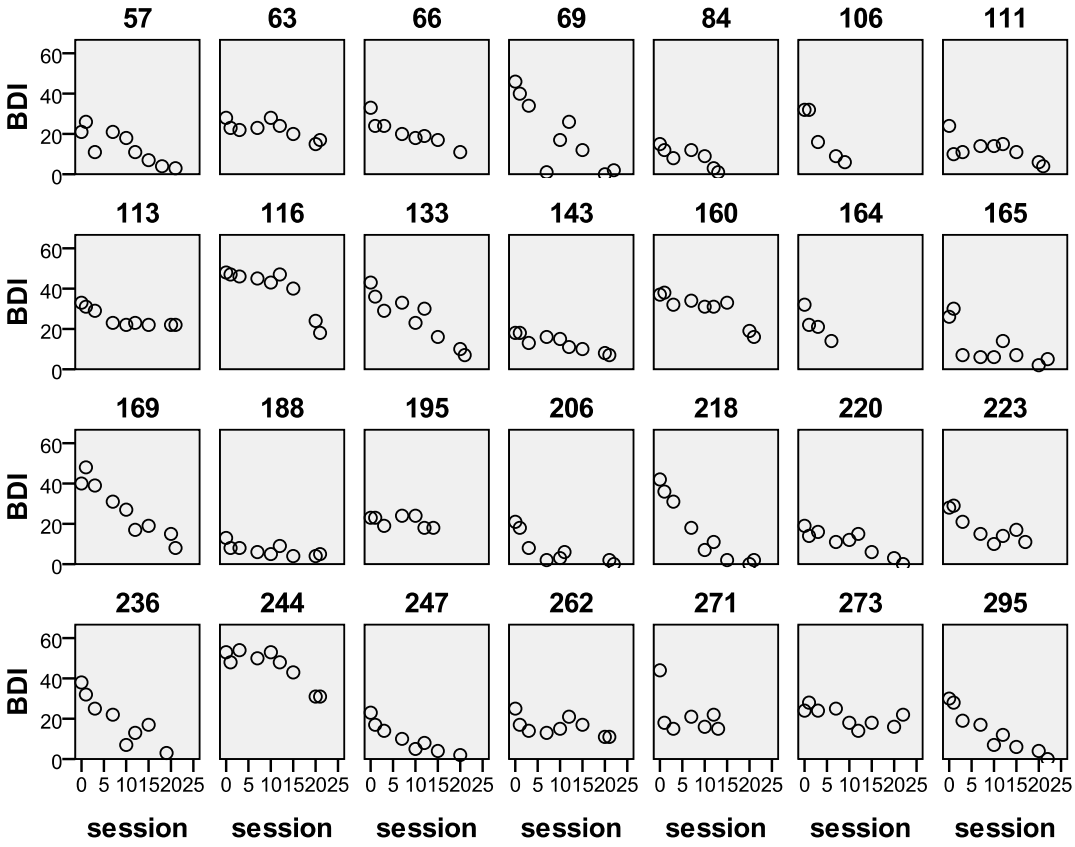

Figure 2. Individual Trajectories of Change in BDI-II scores over sessions by participant number ($N = 28$)

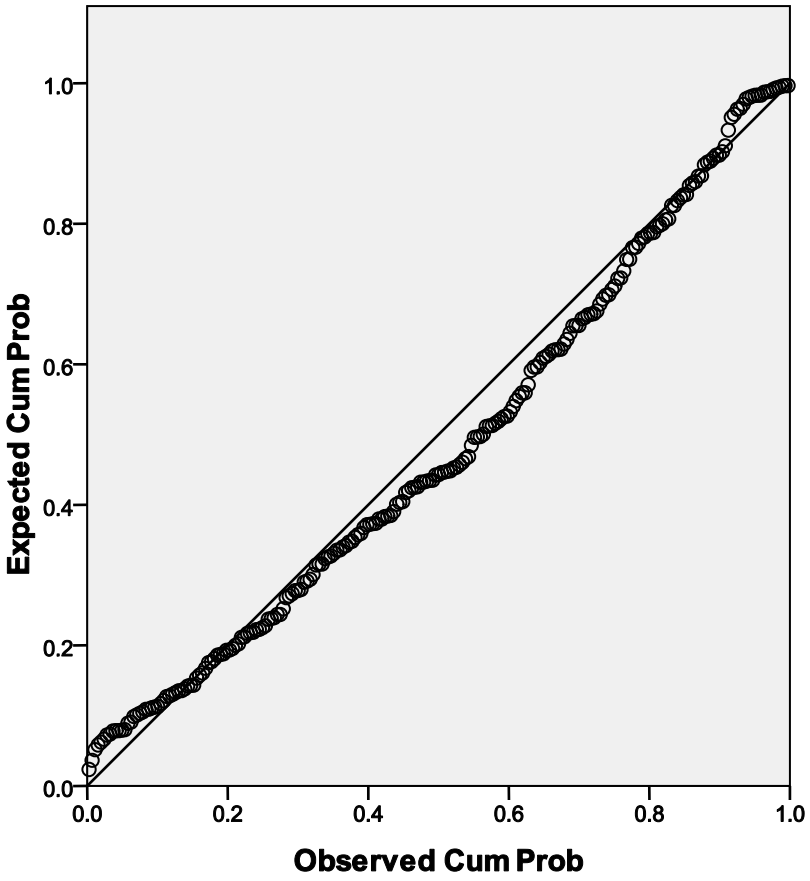

Figure 3. Normal P-Plot of regression standardised residuals over time for BDI-II scores.

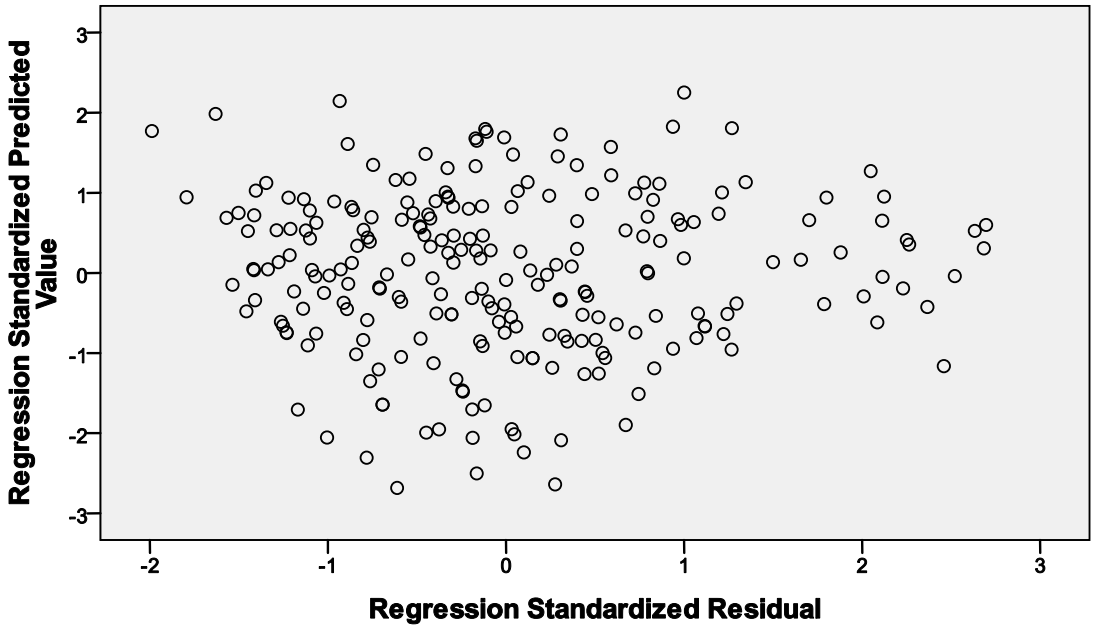

Figure 4. Regression standardized residual scatterplot of BDI-II scores over time.

In Figure 2 the BDI-II score residuals assume a generally diagonal line with only slight deviations from normality. Additionally, the scatter plot (Figure 3) had an approximately rectangular shape, was centred on the middle of the graph with an equal width. These features indicated that the assumptions of normality, linearity and homescedasticity were sufficiently met as regards the BDI-II data.

Change in vegetative symptoms. The majority of the sample had vegetative symptoms as part of their presentation (see Figure 4). Only 6 clients reported no changes in sleep and appetite and only 1 client reported no increase in fatigue. The rate of change for vegetative symptoms was observed to be slower than for general BDI-II score change, with symptoms showing greater stability/chronicity throughout therapy (see Figure 4). Session was used as the time variable when exploring the differences between

depressive and vegetative symptoms as it allowed for direct comparisons of change between the individuals to be made.

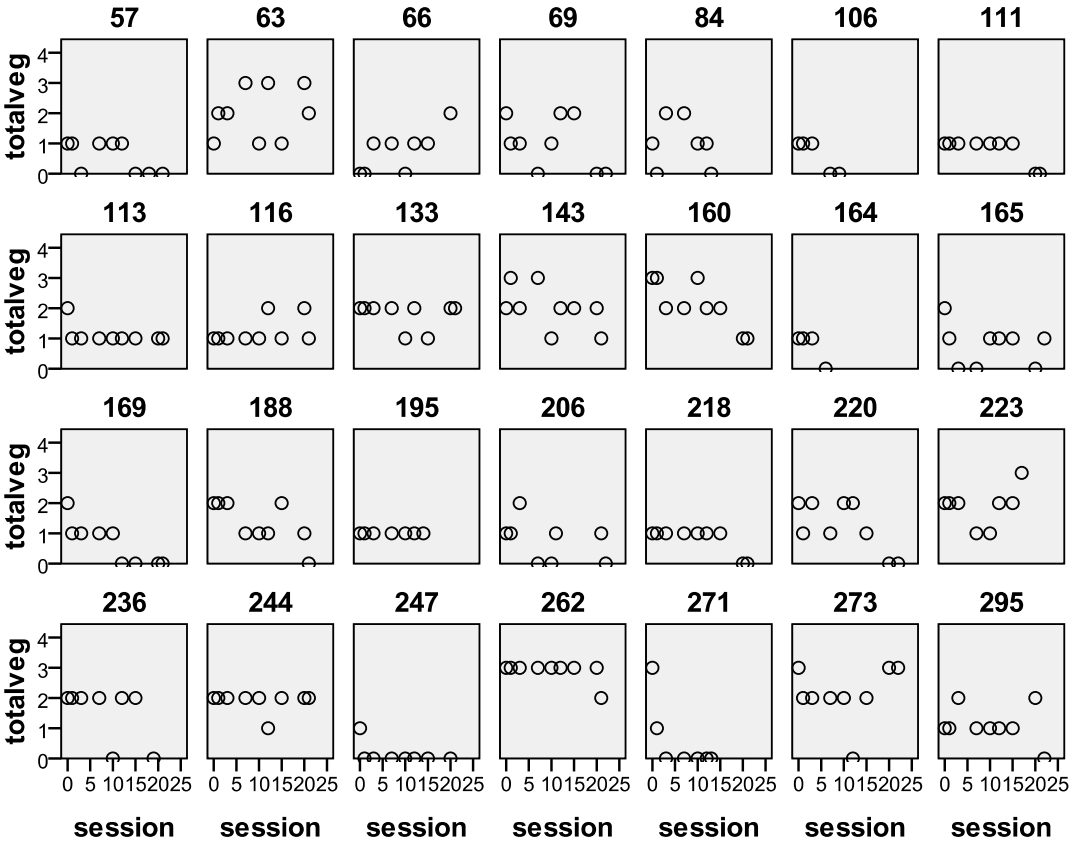

Figure 5. Individual trajectories of change in total vegetative symptoms over time by participant number ($N = 28$)

At the end of study contact, approximately half of the sample (n = 15) had no vegetative symptoms. Of the group who had no vegetative symptoms only two met the criteria for mild depression, the remainder of the sample was considered to no longer meet criteria. Eight of the sample had vegetative symptoms that had persisted throughout therapy and remained despite the change in seasons. Of these three no longer met the threshold for clinical depression, three met criteria for mild depression, one for moderate depression and one for severe depression. Three members of the sample experienced a re-emergence of vegetative symptoms in the winter period at the end of contact. Of those three, one did not meet criteria for depression; one met criteria for mild depression and one met criteria for moderate depression.

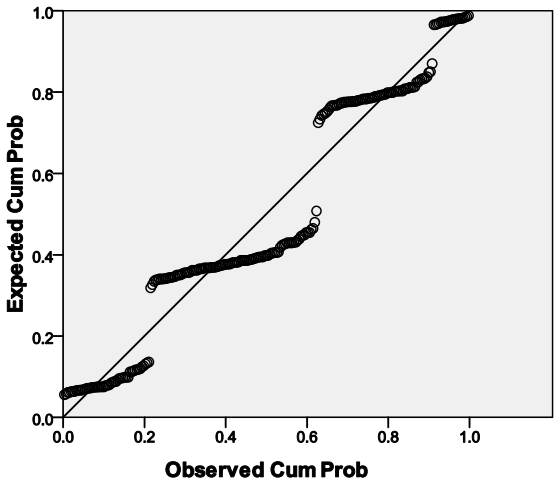

Figure 6. Normal P-Plot of regression standardised residuals over time for total vegetative scores over months.

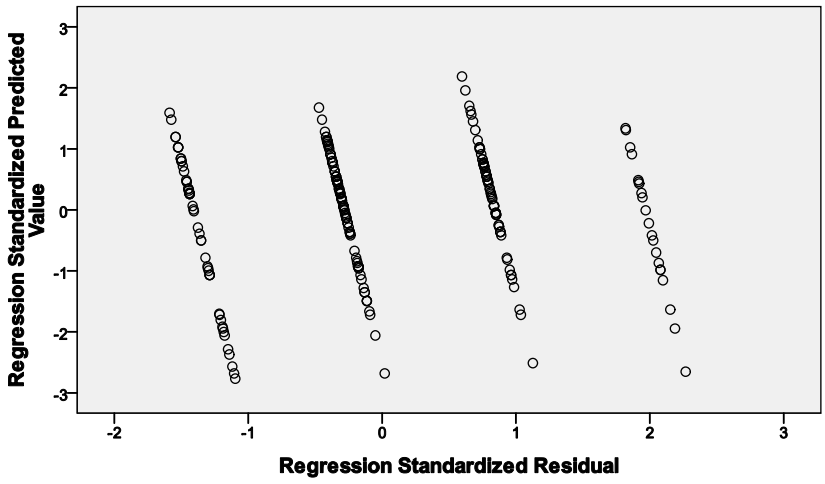

Figure 7. Regression standardized residual scatterplot of total vegetative scores over months.

In Figures 5 and 6, the residuals for vegetative symptoms were examined for normality, linearity and homoscedasticity. Although the residual P-Plot was not clearly linear, a curve fit analysis indicated little difference between linear, cubic or quadratic estimations. Therefore, the linear fit was accepted as reasonable for use with this data. Additionally, the scatter plot indicated a normal distribution for linear analysis.

Additionally, season of therapy (see Figure 7) and season of presentation (Figure 8) were explored as alternative predictors of change in BDI-II scores. The graphs indicated that there were observable differences between the trajectories of change between each of the seasons.

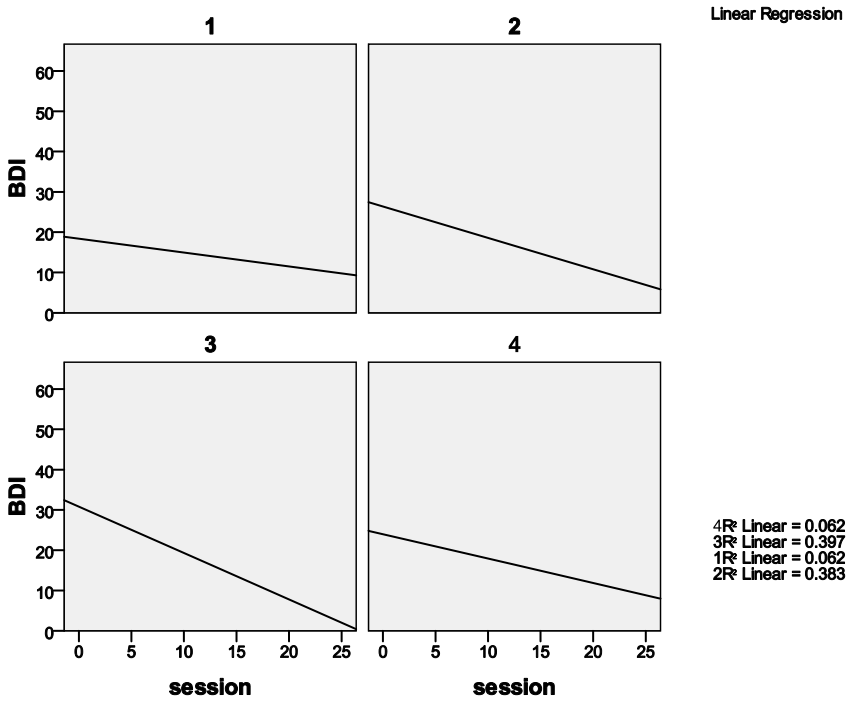

Figure 8. Changes in BDI-II scores in relation to season of therapy (*N* =28). The number above each graph refers to a season: 1 = summer, 2 = autumn, 3 = winter, 4 = spring.

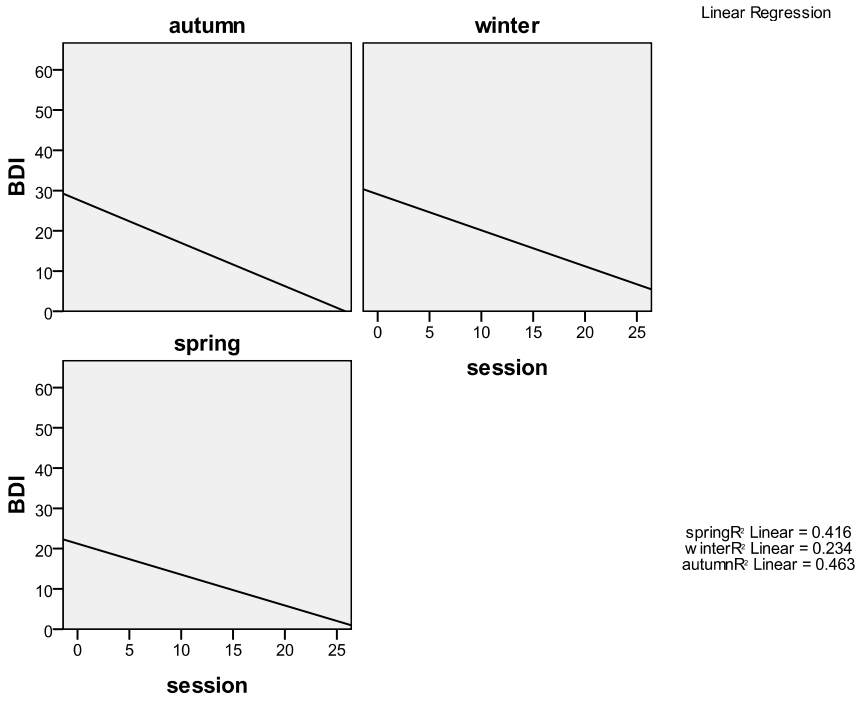

Figure 9. Season of presentation and change in BDI-II scores over time. Autumn (n = 5), winter (n =17), spring (n = 6).

Multilevel Model Building

Predicting Change in Depression Scores over Time

When viewed graphically, rate of change between individuals was noted to vary for BDI-II scores over sessions (see Figure 1). In order to ascertain whether meteorological variables affect BDI-II and change in BDI-II (chBDI) initial correlations were performed in order to explore the effects of these variables as predictors (see Table 5). BDI-II was observed to be significantly correlated with temperature (r = -.13, p < .05) but not sunshine hours. However, chBDI was significantly correlated with sunshine hours (r = -.14, p < .05) but not temperature.

Model A and B

Using MLM, Model A represented the fitting of the unconditional means model (UMM: Table 7). The intercept was 19.11 (p <.001). With the fitting of Model B the unconditional growth model (UGM), it was estimated that the average BDI-II score on

admission to the study (γ_{00}) was 27.5 ($p < .001$) and the average rate of change in BDI-II scores (γ_{01}) was -0.95 per session ($p < .001$). The negative significant score indicated that a decrease of BDI-II scores was observed. As expected session was a significant predictor of BDI-II scores. When months was used as an alternative UGM, the intercept rose to 43.29 ($p < .001$) with an average rate of change of -2.28 points per session ($p < .001$). This indicated that "Month" was also a significant predictor of BDI-II scores. The deviance statistics decreased with both variables indicating that either would be suitable for use in the model. However, the Deviance and the AIC and BIC statistics were lower using "Session" as the UGM variable as opposed to "Month" (see Table 7). Therefore, the session variable was adopted for Model B and further analyses. Furthermore using "Session" for the UGM showed that 70% of the within person variation was explained by this variable as opposed to 57% of the variance explained when months was used. This indicated that session was a superior variable in this situation. The final model for change in BDI-II scores over time is presented in Table 8.

Table 7

Model Building for Change in BDI-II Score: Model A with Alternate Time Variables for Model B using "Session" and "Months" (N = 28)

Level	Variable	Parameter	Model A	Session	Months
	Fixed Effects				
Initial status π_{0i}	Intercept	γ_{oo}	19.11***	27.50***	43.29***
			(1.68)	(2.07)	(3.43)
Rate of change π_{1i}	Intercept	γ_{01}		-0.95***	-2.28***
				(0.097)	(0.24)
	Variance Components				
Level 1	Within person	σ^2_e	83.02***	24.55***	35.81***
			(8.15)	(2.62)	(3.83)
Level 2	In initial status	σ^2_0	69.37***	111.55***	242.95***
			(21.1)	(31.89)	(85.73)
	In rate of change	σ^2_1		0.22***	0.87***
				(0.09)	(0.41)
	covariance	σ_e		-2.38***	-11.62***
				(1.16)	(5.36)
	Pseudo R^2 and Goodness-of-fit				
	R^2_ε		-	.70	.57
	R^2_0		-	-	-
	R^2_1		-	-	-
	Deviance		1763.67	1551.79	1618.42
	AIC		1769.46	1563.79	1630.42
	BIC		1779.85	1584.55	1651.18

Note. *** p <.001

Model C

The effects of climatic variables on rates of BDI-II change. Residual plots for bright sunshine hours and temperature data are presented in Appendix G. Using the meteorological variables of sunshine hours and low daily temperature as predictors in the model gave non-significant results (Appendix K). There was little difference between data points as regards the initial status of BDI-II in relation to either temperature or sunshine. When temperature was used as a predictor of initial status and change the results showed that the estimated initial BDI-II for the average subject was 29.63 (p <.001) and the estimated differential in initial BDI-II between the different temperatures

(γ_{01}) was -0.21 (p >.10*).* The estimated rate of change (γ_{10}) in the sample remained significant (-1.08, p< .01) indicating that further variables are involved in change at this level. However the estimated differential on temperature (γ_{11}) was not significant (-0.01, p >.10). This meant that the sample continued to change however the differences between them as regards temperature did not appear to effect how much they changed. The variance components at both level 1 ($\sigma^2\varepsilon$) and level 2 initial statuses (σ^2_0) and rate of change (σ^2_1) showed no significant difference associated with temperature. These results indicated that temperature had no significant influence as a predictor on the rate of change of BDI-II. Additionally, the deviance statistics showed no improvement in the fit of the model with the addition of this predictor with an increase observed in the AIC and BIC statistics. Therefore, temperature was not kept for further analyses in this model.

When sunshine was used as alternative predictor (Appendix K) the estimated BDI-II for the average subject was 27.14 (p < .01) and the estimated differential was 0.06 (p >.10). The estimated rate of change in the average person on this variable was -1.02 (p <.01) and the estimated differential was 0.01 (p >.10). These results indicated that, sunshine hours as a predictor had no significant influence on BDI-II scores over time. Additionally, the variance levels showed that sunshine explained a small amount of the variance at Level 1 but accounted for less of the variance at Level 2. Further to that, there were negative Pseudo R^2 findings which indicated that this variable accounted for change at Level 2 only and should be interpreted with caution (Singer & Willett, 2003). These results meant that sunshine accounted for little change above session number as a predictor for BDI-II scores. However, the deviance statistics indicated that sunshine did improve the fit of the model above what was considered “noise” as there was a reduction of 3 points noted. Therefore, this variable was kept for further analyses as it appeared to fit the model better than temperature. However, as the AIC and BIC showed no reduction with this variable it was not considered to be a significant improvement.

Neither temperature nor sunshine hours were found to influence BDI-II scores over time. Therefore the hypotheses that they would produce greater levels of change, has not been met.

The effects of gender and age on changes in BDI-II scores. Gender was also included in the analyses as a predictor in Model C (Appendix K). The initial status and the intercept remained significant (29.34, $p < .001$) and the rate of change (-0.97, $p < .05$) was also significant. Additionally, there was some between individual variance noted in initial status (σ^2_0). However, there was no improvement in the fit of the model observed. This indicated that gender was not a significant predictor of change in BDI-II scores over time.

Age was also examined in order to ascertain whether it had an impact on BDI-II scores. Once again initial status remained significant (33.38, $p < .001$); however, the intercept for the rate of change was no longer significant (-0.56, $p > .10$). Variance components also showed little change at level 1 and some change at level 2. These results indicated that age had little effect on initial status and rate of change in BDI-II scores. However as there was some improvement noted in the fit of the model this variable was retained for further analyses as a control variable.

Overall, none of the variables used in this study adequately explained significantly more of the variance within persons over session from Model B. Most of the variance observed in the sample was at Level 2 with age showing more effect on BDI-II change than gender.

Model D

Gender was used as a control variable for sunshine in Model D in order to ascertain whether this improved the model fit (Appendix K). There was insignificant change in the variance noted from the use of gender alone. However there was some improvement in the goodness of fit observed although this was not significant. When age was used as an alternative control variable, there was also little change in the variance at level 1 or level

2 (Appendix K). Nevertheless, there was a slightly better fit for age observed. These results indicated that neither of these variables was estimated to have a significant effect on BDI-II scores in these analyses.

Post Hoc Analyses

Season of therapy was entered into Model C in order to provide an alternative variable for the effects of season as a combined variable compared to sunshine or temperature alone. However, there were no significant effects found for either of these variables. These results are presented in Appendix K.

Summary of depression model. The final model is presented in Table 8. There was no support for the effect of meteorological factors or season as predictors of BDI-II score or rate of change. Additionally, age and gender appeared to have limited effect as predictors of BDI-II change. Model D which combined sunshine hours and age showed some applicability as there was an overall decrease in the deviance of 218.76 and in the AIC and BIC statistics indicating that these variables did improve the fit of the overall model. Although most of the variance was accounted for by session (70%), the addition of sunshine hours and age accounted for 20% of the between individual variance at level 2. This result indicated that these variables may be of use in further research although no significant results were found in this study.

Table 8

Final Model for Change in Depression Scores (N = 28)

Level	Variable	Parameter	Model A	Model B	Model C	Model D
	Fixed Effects					
Initial status π_{0i}	Intercept	γ_{oo}	19.11*** (1.68)	27.50*** (2.07)	27.14*** (2.26)	33.50*** (8.42)
	Sunshine	γ_{01}			0.06 (0.17)	0.07 (0.17)
	Age	γ_{02}				-0.14 (0.18)
Rate of change	Intercept	γ_{10}		-0.95*** 0.097	-1.02*** (0.12)	-0.67* (0.39)
	Sunshine	γ_{11}			0.01 (0.01)	0.01 (0.01)
	Age	γ_{12}				-.008 (.008)
	Variance Components					
Level 1	Within person	σ^2_e	83.02 (8.15)	24.55 (2.62)	24.19 (2.58)	24.08 (2.57)
Level 2	In initial status	σ^2_0	69.37 (21.1)	111.55 (31.89)	112.54 (32.14)	109.72 (31.36)
	In rate of change	σ^2_1		0.22 (0.09)	0.19 (0.07)	0.18 (0.07)
	covariance	σ_e		-2.38 (1.16)	-2.43 (1.16)	-2.55 (1.15)
	Pseudo R2 and Goodness of Fit statistics					
	R^2_ε			.70	.70	.70
	R^2_0				#	.02
	R^2_1				.22	.18
	Deviance		1763.67	1551.79	1548.36	1544.91
	AIC		1769.46	1563.79	1564.36	1564.91
	BIC		1779.85	1584.55	1592.03	1599.5

****p* < .001; ***p* < .05, **p* <.10

Predicting Change in Vegetative Symptoms

Model A and B

Vegetative symptoms had greater stability over time in comparison to BDI-II scores (see Figure 4) although they were noted to vary both within and between individuals. The UMM for vegetative symptoms was found to not be equal to zero (Appendix L), therefore the null hypothesis was rejected. As this study was examining vegetative

symptoms in relation to seasonality it was believed that "Months" would fit the research questions for change in vegetative symptoms more appropriately. Furthermore, it was felt that the use of months would also encompass the ethos of the study more appropriately. A comparison UGM of "Session" and months was performed and as results were similar it was considered reasonable to continue using months as the time variable in this case. Table 9 presents the results of the final model building for change in vegetative symptoms.

The fitting of the UGM (Model B) indicated that months had a significant effect on both the individual status intercept (1.95, p <.001) and the rate of change intercept (-0.07, p <.001). Additionally months also accounted for 18% of the variance at Level 1 indicating that null hypothesis could be rejected. Additionally, there were decreases in the deviance statistics which indicated the improved fit of the model (see Table 9).

These findings indicate that vegetative symptoms were subject to slower rates of change compared to depressive symptoms. Therefore the hypothesis that BDI-II scores will show increased rates of change compared to vegetative symptom scores was met.

Model C

Fitting of the meteorological variables are displayed in Appendix L. Using "sunshine" as a predictor indicated a significant effect on initial status (2.32, p < .001) and a significant trend on vegetative symptoms (-0.08, p <.10). There was also a significant effect on the rate of change of vegetative symptoms (-0.11, p <.05). Although there was slight within individual change in variance most of the change occurred at Level 2 on this variable. This indicated that sunshine hours affected some individuals more than others. Goodness-of-Fit statistics showed that this variable accounted for an additional 7% of the variance between individuals. Additionally, there were significant decreases observed in the deviance statistics which indicated that this variable improved the fit of the model.

Model "temperature" showed no significant effect on vegetative symptom change, however the intercept remained significant. Additionally, there was a decrease in the amount of variance explained at Level 2 and negative Pseudo R^2 results which indicated that this variable should be interpreted with caution. Furthermore, there was an increase in the goodness of fit statistics. In light of these findings this variable was abandoned as there appeared no reason to retain it in future models due to the lack of significant results at this level.

Therefore the hypothesis that sunshine hours would show a significant rate of change in vegetative symptoms was met. However, the hypothesis that temperature would significantly affect rate of change in vegetative symptoms was not met.

Gender was not inputted at level C in this model. This was because there had been a previously non significant effect for gender found in the depression model. Additionally, in Study 2 there were contradictory findings observed which may confound results in this study.

Model D

Gender and age were used as control variables in this model. The results of fitting these variables are presented in Appendix L. When gender was used as a control variable for sunshine hours in Model D it produced a significant intercept on initial status (2.66, p <.001). Main effects were shown for both gender (-0.93, p <.05) and sunshine (-0.09, p < .05) on initial status. Significant effects were also shown for the intercept on the rate of change (-0.13, p < .001) which indicated that higher vegetative symptoms were associated with lower sunshine hours when gender was controlled for. Additionally, there was a significant change in the variance components at Level 2.Goodness-of-fit statistics indicated that gender accounted for 40 % of the between person variance in initial status at this level. However, there was no significant difference observed in the rate of change between individuals on this variable at Level 2. The deviance statistics also indicated an improved fit for the model with sunshine and controlling for gender accounting for a decrease in deviance. Therefore, hypothesis 8 was supported.

The use of age as a control variable for sunshine also showed a significant intercept (2.52, $p < .05$). However, there was no significant difference noted between individuals as regards their rate of change. Additionally, goodness-of-fit and deviance statistics indicated that this variable did not contribute to the overall model (Appendix L).

The Final Model for Vegetative Symptom Change

This model is presented in Table 9. The results indicated that both gender and sunshine hours had an effect on the relationship between vegetative symptom expression at initial status. When gender was used as a control variable, sunshine hours had a significant effect on symptom expression over time. However, temperature and age were not significant effects on symptom expression over time in this sample. As there was not a large decrease in deviance statistics in the final model, much of the variance remains unexplained by the variables used.

Table 9

Final Model for Change in Vegetative Symptoms (N = 28)

Level	Variable	Parameter	Model A	Model B	Model C	Model D
	Fixed Effects					
Initial status π_{0i}	Intercept	γ_{oo}	1.22*** (0.12)	1.95*** (0.22)	2.32*** (0.3)	2.66*** (0.32)
	Sunshine	γ_{01}			-0.08* (0.04)	-0.09** (0.04)
	Factor	γ_{02}				-0.92** (0.43)
Rate of π_{1i}	Intercept	γ_{10}		-0.07** (0.02)	-0.11** (0.03)	-0.13*** (0.03)
	Sunshine	γ_{11}			0.008 (0.003)	0.01** (0.003)
	Factor	γ_{12}				0.06 (0.04)
	Variance Components					
Level 1	Within person	σ^2_e	0.44 (0.04)	0.36 (0.04)	0.35 (0.04)	0.35 (0.04)
Level 2	Initial status	σ^2_0		0.57 (0.38)	0.53 (0.37)	0.34 (0.31)
	Rate of change	σ^2_1		0.004 (0.003)	0.004 (0.003)	0.004 (0.003)
	covariance	σ_e		-0.03 (0.03)	-0.03 (0.03)	-0.02 (0.02)
	Pseudo R^2 and Goodness of Fit Statistics					
	R^2_ε			.18	.18	.18
	R^2_0				.07	.40
	R^2_1				0	0
	Deviance		530.59	507.81	501.05	496.41
	AIC		536.59	519.81	517.05	516.41
	BIC		546.93	540.57	544.72	551.01

****p< .001, ***p < .05, *p <.10*

Irritability

Irritability and vegetative symptoms. In order to answer hypotheses around the relationship between vegetative factors and irritability a number of correlations were performed (see Table 5). The number of vegetative symptoms was found to be

significantly correlated with the degree of irritability ($r = .35$, $p < .01$). The severity of irritability was significantly correlated with the severity of increased appetite and sleep ($r = .25$, $p < .01$) and the severity of fatigue ($r = .53$, $p < .01$). Therefore, irritability was significantly related to vegetative symptoms. However, there was no significant difference in proportions found between those who were seasonal and those who were not on their degree of irritability $\chi^2 (3, N = 28) = 4.85$, $p > .10$.

Irritability and meteorological variables. Temperature was noted to be significantly correlated with irritability $r = .-16$, $p < .05$ but sunshine hours were not.

Age, gender and irritability. Age showed no correlation with irritability in this sample, $r = -.06$, $p > .10$. Additionally, there was no support found for the hypothesis that men ($M = 1.40$, $SD = 0.52$) were more irritable than women ($M = 1.44$, $SD = 0.86$), $t = 0.15$, $p > .10$. Therefore neither of these hypotheses was supported.

Discussion of Results

Sample Characteristics

Overall the sample in Study 3 was consistent with other studies as regards gender breakdown (Amons et al., 2006; Levitan et al., 1999). The majority of the sample had some form of tertiary education and was employed during the course of the investigation. Additionally, this sample was mainly of European extraction which would limit the generalisability of results to be applied to other cultures.

Symptom Severity

Symptom severity of BDI-II scores decreased over the course of therapy with only one person considered severely depressed and 68% no longer met criteria for MDD at final contact. These results are in line with other studies whereby CBT had been found to be an effective therapy in the treatment of depression (Beck et al., 1979; Freed, 2006; Rohan et al., 2003; Rohan et al., 2007).

Vegetative symptoms were also found in the majority of the sample at the beginning of therapy. However, their prevalence had greatly diminished by the end of contact. Additionally, the significant distribution of vegetative symptoms at the beginning of therapy and the non significant distribution at the end of contact indicated that a portion of the sample produced vegetative symptoms in line with a current episode of seasonal depression. The return of symptoms in three of the sample with the beginning of a new winter indicated that 10.7% of the sample had symptom expression consistent with a seasonal pattern. This is supported by two out of the three indicating some level of increased depressive symptoms. This finding is in line with results by Murray and Hay (1997) where 10-20% of their sample was considered to have SAD. However, the results in this study were higher than other studies based in temperate climates (see Corral et al., 2006; Westrin & Lam, 2007) where sample numbers with seasonal depression were found to be lower (1-3%). Therefore, where this finding fits in relation to other research requires further clarification.

For a number of the sample, vegetative symptoms did not appear to be related to season. In this group vegetative symptoms persisted throughout therapy for some and a number of them remained depressed at the end of contact (n = 6). This would support findings by Goel, Terman, and Terman (2002) and Stewart et al. (1990) whereby atypical depression was found to be distinct to seasonal depression. Additionally, those with atypical features remained more depressed over time (see Figures 1 and 4); thus indicating that their vegetative symptoms were part of a chronic depressive picture rather than a specific biological response (Stewart et al., 1990). The lack of seasonal pattern observed and no decrease in vegetative symptoms in the summer periods would reinforce this assumption.

Analysing change in BDI-II scores over time

Overall, when exploring the influence of meteorological variables on depression, the only factor that significantly predicted change in BDI-II scores over time was session. Neither, meteorological factors, gender, nor age, had any significant effect on rate of BDI-II score change. That the meteorological factors used showed no effect, is

inconsistent with preliminary correlations which did show a significant relationship between BDI-II and chBDI scores and weather variables. However, these correlations were not longitudinal and did not take time into account, which may explain the variation in findings.

Alternatively, therapy would account for these findings. Furthermore, a portion of therapy included behavioural activation which would increase bright light exposure for the individual and may account for some of the variance found at this level. This would be consistent with previous research whereby exercise programs were found to be beneficial for those with seasonal depression (Leppamaki, Partonen, & Lonnqvist, 2002; Peiser, 2009). However, overall, these findings were consistent with Michalak et al. (2004) and Denissen et al. (2008) whereby daily weather variables were found to have no effect on the rates of depression.

That there were no effects found for gender was surprising as women are more frequently associated with clinical depression compared to men (APA 2000; Chotai et al., 2004a). This finding contradicts results in Study 2 where women were found to have higher levels of depressive symptom endorsement than men. Therefore, the effects of gender require further investigation.

Other studies have indicated that the onset of a MDE is associated more often with younger persons and that earlier onset was associated with greater chronicity and poorer response to treatment (Barlow & Durand, 2002). However, age was not found to have any relationship with severity of depression or rate of change over time in this study. The results found in this study were contrary to previous research, but replicated findings in Study 2. Therefore, the effects of age on depression require further investigation in New Zealand.

Analysing Change in Vegetative Scores over Time

Sunshine and change in vegetative symptoms. The main effects for sunshine on vegetative scores over time were surprising as Studies 1 and 2 had shown no relationship

between sunshine hours and symptom expression on presentation. However, this finding is consistent with the biological theories of seasonal depression whereby bright light is thought to relieve vegetative symptoms and the associated depression (Lam & Levitan, 2000; Lewy et al., 1980; Rosenthal et al., 1989). This effect is found to generalize to countries where minimal variation in photoperiod is experienced (Martiny, 2006) which may validate findings in this setting. It would appear however that the effect of sunshine is specific to vegetative symptoms in this study as there was no similar effect found for BDI-II scores. This was supported by Denissen et al. (2008) who found in their multilevel study that sunlight had a main effect on tiredness, but that there was significant random variation between individuals. Additionally, the effect of sunshine may also provide an alternative theory to atypical depression in regards to the chronicity of vegetative symptoms for some as there were documented periods of low sunlight present even in summer (see Appendix D). However, this needs further clarification as the amount of variance explained by this variable is small.

Temperature and vegetative symptoms. The lack of effect found for temperature was inconsistent with findings from Studies 1 and 2. However, as there had been consistent and significant relationships previously demonstrated between temperature and sunshine readings, it is possible that the variables may fluctuate in importance over time. Previous studies have noted that the fluctuations in daily weather characteristics make their association with symptoms difficult to ascertain (Huibers et al., 2010). Furthermore, temperature is not directly implicated in the etiology of seasonal depression in other studies which may indicate the understated nature of this variable. Additionally, temperature has been found to have no effect on mood in other studies (Denissen et al., 2008).

Gender and age and vegetative symptoms. For gender, a main effect was found on initial vegetative scores and rate of change. This is consistent with other findings whereby differences have been noted with women more likely to have symptoms associated with seasonal depression (APA, 2000). However, in this investigation gender has produced conflicting results which make conclusions difficult to determine. In this

study, a greater percentage of men had atypical vegetative symptoms at final contact when compared with women. Additionally, three women showed a re-emergence of seasonal vegetative symptoms at follow-up consistent with a seasonal pattern. The re-emergence of symptoms was not associated with a depressive episode in one woman however the other two did show mild to moderate depressive symptomology. This indicates that CBT for depression had some ongoing effectiveness, but that some specific tailoring with clients who exhibit a seasonal pattern is warranted.

Conversely, age was not found to have an effect on vegetative symptoms over time apart from sunshine. This finding was contrary to initial correlations which showed a significant relationship between age and a decrease in vegetative symptom severity and the total number of vegetative symptoms. These preliminary findings were consistent with other research whereby vegetative symptoms were more commonly associated with younger age groups (Kovalenko et al., 2000; Sonis, 1989). However, as there were no children or adolescents involved in this study, potential differences may not have been observed.

Irritability and its Relationship to Vegetative, Meteorological and other Depressive Symptoms

The findings around irritability suggested that it was highly related to both the number and severity of vegetative symptoms in this sample. However, the lack of relationship found with seasonality supported the view that the symptoms themselves rather than the season per se that influenced irritability. This was in contrast with the findings from other studies where season was found to be related to outbursts of anger and violence (Lahti et al., 2006). However, as that research was from countries where seasonal variation was more defined (Scandinavia); results may have been produced that were specific to those conditions. Nevertheless, the decreased temperature associated with increased irritability found in this study, indicated that warmer climatic conditions did not appear to affect levels of irritability in this sample. Furthermore, the lack of association found in this study as regards the relationship between age and irritability also is inconsistent with previous findings (Lahti et al., 2006; Marušič et al., 2003).

Limitations

There are a number of limitations associated with this study. The first is the small sample size ($N = 28$). Although the use of longitudinal data analysis and decreased level of significance ($p < .10$) improved the ability of the data to yield differences between and within the subjects, the small number of participants meant that some analyses would not have enough power to detect the small differences that may have been present. Additionally, data were not clustered in this study (*disaggregated analysis*) due to its exploratory nature (Singer & Willett, 2003). Therefore any potential group structures were ignored which may have led to alpha inflation (Cohen, et al., 2003).

Secondly, although all time points were intended to be the same for each client this was not always possible. Some clients did not complete the 20 sessions of therapy with a number of them (6 clients) discontinuing therapy early for a variety of reasons. Additionally, the time points were not consistent for each client even though the session times were superficially the same, as there were some times when the client or the therapist could not present for therapy (e.g. illness and holidays).

Thirdly, the climatic conditions may have affected results in unforeseen ways, for example extensive periods of low light in the summer months, which may further limit the ability of the data to reveal patterns of interest. Finally, this study used measures for assessing depression rather than any specific measures for SAD. Therefore, results must be interpreted in that light and the measures used may not have had the ability to assess the sample for seasonal depression with specificity.

CHAPTER EIGHT:
FINAL DISCUSSION

The overall aim of this investigation was to explore a sample of first episode depression sufferers from New Zealand in order to ascertain whether a seasonal pattern was discernable in any of them. This was chiefly undertaken because there was no research on seasonal depression based in New Zealand found in the literature. Because New Zealand is situated within the mid latitudes and exhibits a temperate climate whether any discernable seasonal patterns would be found was open to question. Therefore, this investigation was broken up into three studies in order to explore the possibility of seasonal depression.

Implications and issues arising from the literature

One of the major difficulties with the current literature exploring seasonality, seasonal depression and SAD is the inter-changeability of the terms used. Often one is left to guess what the term indicates specifically as they are often not well defined in the study and this can result in confusion around the findings and where each study fits with the others can be vague. This was the main reason that the studies used in this literature review were discussed using the terms used in the article. In this investigation the definition of the terms used to describe the sample was intended to address this perceived short coming in the literature. Because this study was not able to diagnose a seasonal depression with specificity the terms used in this investigation were defined in order to enable easier comparisons on the data gathered.

Often the terms used in research are based around the theoretical hypotheses of causation but there are a variety of measures used to assess the samples. The most common used in the literature is the SPAQ which has been noted to potentially overestimate the prevalence of SAD in the general population (Murray, 2004). This may have risen to an increased perception amongst the public that SAD is a common disorder with prevalence documented as being up to 30% in some studies. This may in turn have resulted in response bias that has been noted to be a possibility in some studies (Murray

et al, 2004) and served to perpetuate the perception of commonality. More rigorous studies do place the prevalence of seasonal depression as being lower however they are based on continents and may have limited application in this country. Therefore this study was designed to address previous limitations in the research by providing an observed manifestation of current depressive and vegetative symptoms in line with current season without retrospective bias of symptom profile over time.

Overview of studies and their results

Study 1.

Study 1 served as a preliminary exploration of whether there were any discernable differences found in season of presentation especially focusing on gender and age. It enabled the exploration of the largest available sample ($N = 194$) in order to maximise the chances of observing any potential relationships. Should none of the variables of interest have been found to have any relationship with season then the chance of finding relationships in the following studies would have been unlikely. That there was a significant winter presentation compared with other seasons and some gender differences observed were encouraging. Overall, it is likely that this result indicated the presence of seasonality or S-SAD in a significant portion of depression sufferers. Seasonality or S-SAD in this context would be seen as increased cognitive distress with the change of seasons which was in line with other research (Chotai et al, 2004b; Magnussen & Partonen, 2005; Rosenthal, 1989). However, as the presence of specific physical symptoms was not examined in this sample this interpretation is broad and other factors may account for the way the sample presented. An example of this would be the placement of advertisements for the parent study as they were not published in the summer months. Another important finding of this study was the inconsistent weather that was present over the time of the study. The exploration of weather variables in this study and the lack of relationship found between sunshine and temperature were also important as it enabled the results of this study to be placed in context. This meant that how findings related to other studies could be analysed more accurately.

Weather and geography. Because New Zealand is situated in a unique geographical location compared to other countries, it was important to explore the differences in weather conditions that are experienced here as opposed to other settings. In order to do this, daily bright sunshine and daily low temperature readings were collected for the duration of the study (NIWA, 2009). These variables were chosen due to the way they fitted the theoretical constructs for the development of seasonal depression. The data collected in this investigation showed a markedly variable pattern of daily bright sunshine with noticeable periods of decreased light even in the summer months (Appendix E). On the other hand, temperature readings were more consistent with seasonal variation. Interestingly, the results showed a negative correlation between hours of bright sunshine and temperature. This indicated that in New Zealand, lower temperatures were often associated with higher sunshine hours. This was in contrast to previous studies based in northern populations which showed a more consistent low sunshine with low temperature pattern (Chotai et al., 2004b; Michalak & Lam, 2002; Rastard et al., 2005; Rohan et al., 2003; Rosenthal, 1998).

Study 2.

A smaller portion of the original sample (N = 81) was assessed regarding their basic symptoms of depression in regards to season, gender and age. This enabled a preliminary analysis for the presence of vegetative symptoms within this sample. It also facilitated the comparison of how these symptoms were manifested between the genders and whether their expression was consistent over time, that is, did younger participants present in a similar manner as older participants. The result that depressive symptoms and vegetative symptoms had no significant correlation with each other was a valuable finding as it contributes to the literature on the dual vulnerabilities thought to exist in the DVH (Young, 1991). Additionally, it meant the variables could be explored separately in future studies which would enable increased accuracy of results around these symptom groups. This would have potentially important ramifications for the direction of future research in this area by enabling a focus on vegetative symptom change in comparison to depressive symptom change in other conditions. It also serves to validate the premise that vegetative symptoms may not cause concurrent depressive symptoms and that their

presence can lead to a variety of responses specific to the individual (Kasper et al, 1989). This would also show support for the prevalence of seasonality or S-SAD in New Zealand despite its inconsistent weather patterns.

Study 3.

This study used MLM in order to explore how a group of people receiving therapy for depression ($N = 28$) changed over time, both as regards their depressive symptoms and their vegetative symptoms. A major finding of this study was the disparity of rate of change between the two symptom groups (typical vs. seasonal). The observed chronicity of the vegetative symptoms despite depressive symptom change contradicts previous hypotheses where it was the decrease of the vegetative symptoms that was considered to result in the decrease of depressive symptoms. This questions the premise of the PSH and the expected order of change. However, the lack of effect found for meteorological variables on depression supports previous findings. The effect of sunlight when combined with gender on vegetative symptoms was unexpected however throughout this study findings around gender were contradictory. Nonetheless, it does support the premise of specific vulnerabilities to small variations of light in some people but as the variance described was small this finding remains inconclusive. Additionally, the overall results around gender remain inconclusive and it is unclear how they fit with other literature but as other research findings have also been divergent the area is likely to remain open to discussion.

The seasonal factors in depression explored in this study involved a seasonal presentation (in this case winter); and expression of at least one of a group of vegetative symptoms. As previously stated in order to meet DSM-IV-TR criteria for MDD-SD (seasonal pattern specifier) at least two previous episodes of seasonal depression were required, consequently, it was not possible to definitively state whether individuals had seasonal depression within this sample. However, there were a number of individuals who reported vegetative symptoms and presented in the winter season. Furthermore, a portion of the sample reported a return of vegetative symptoms in response to seasonal change. This longitudinal pattern is consistent with seasonal depression. As a result, it is

reasonable to suggest that this sample comprised of a number of persons who had a current episode of seasonal depression. Additionally, this portion would also appear to be of a similar percentage to other studies which explored the prevalence of seasonal depression in other studies (10-20%: Murray, 2004; Parslow et al. 2004). Nevertheless, it should be noted that this sample was smaller than other studies which limits the generalisability of the findings.

Because there were a large number of hypotheses which were tested in different studies a visual map of the studies and which hypotheses were supported is presented on the next page order to clarify the findings of the study

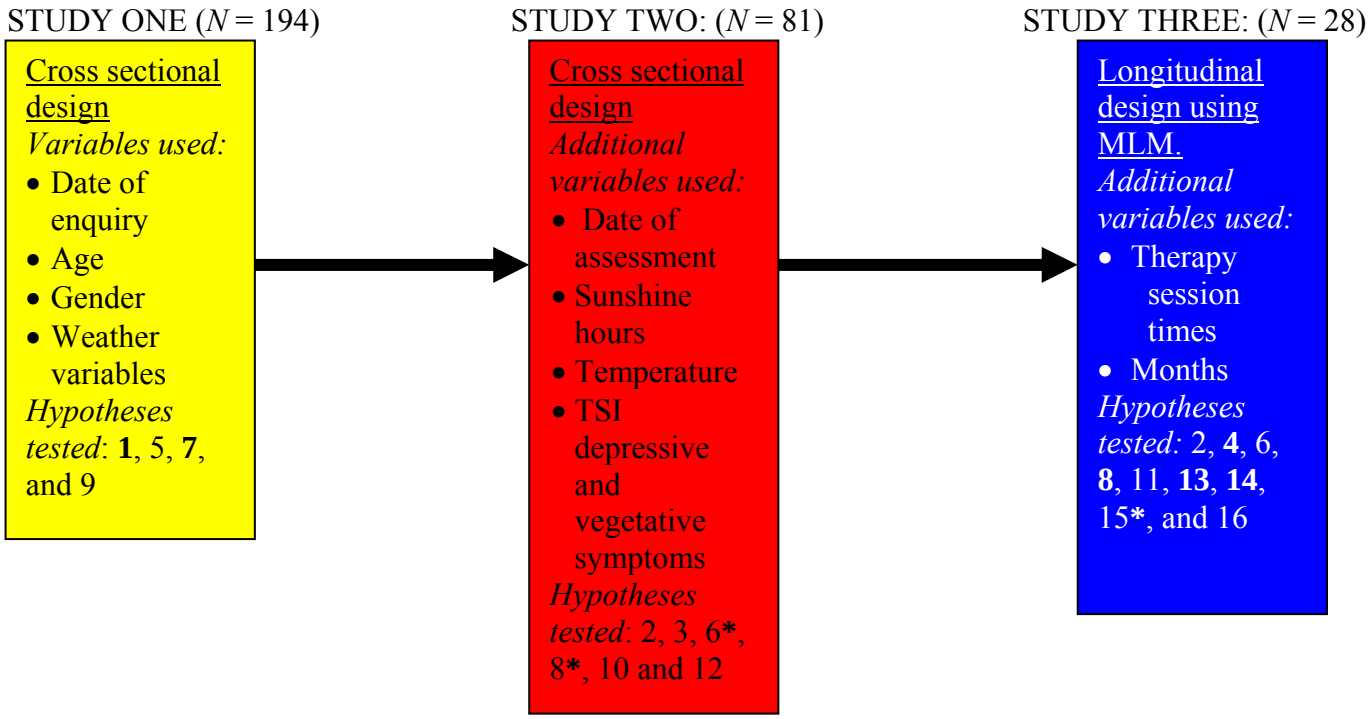

Figure10. Visual Map of Studies with Supported Hypotheses.

Note: for list of hypotheses see Appendix C. The hypotheses in **bold** were supported in the analyses. With those partially supported indicated by *

Findings in Relation to Meteorological Variables and Seasonal Change

In this investigation it was decided to split the variables and examine sunshine separately from temperature in order to ascertain if there were differences in their effect on vegetative and depression scores. In Study 2 of this investigation, hypothesis 12 was tested and there was no significant relationship between sunshine hours and levels of depression at assessment observed. This was consistent with results in Study 3 where hypothesis 16 proposed that sunshine hours would affect rate of change in depressive symptoms over time but this was not supported with sunshine hours not significantly affecting BDI-II scores over time. This was in line with a number of studies which showed that daily sunshine hours had little effect on levels of depression (Michalak et al., 2004; Young et al., 1997).

In this investigation hypothesis 16 in Study 3 also tested the effect of bright light as a factor in the reduction of vegetative symptoms. This hypothesis was supported with a decrease of vegetative symptoms in relation to increased sunshine hours over time observed. Vegetative symptoms were observed to be more stable over time than depressive scores, indicating that overall seasonal change rather than daily sunlight hours was of more benefit as regards symptom relief in this sample. This finding is in line with research by Denissen et al. (2008) who documented that responses to daily sunlight may not translate to seasonal responses and vice versa. As daily sunshine hours were found to vary widely in this study the cumulative effect of light and seasonal change is likely to have been implicated in this result.

The results in this study indicated that the vegetative symptoms associated with seasonal depression can be alleviated through general increased light exposure consistent with seasonal change even if this increase is not observed to be large. As bright sunlight has been postulated to be around 50, 000 lux, this should provide a rapid response in symptom relief with even short exposures (Partonen, 2001; Sato 1997). This is in line with other studies (see Martiny, 2006) where seasonal depression was observed and treated with LT in countries with similar latitudes to New Zealand. These findings are consistent with theories proposed by Murray and Hay (1997) in which vulnerable

individuals were more susceptible to vegetative symptom expression in response to even small changes in light. These results provided some support for the PSH and the LDH in regards to the effect of decreases in light on the individual although further investigation is warranted

The effects of light on the symptoms associated with a seasonal depression have been well accepted in the literature (Rosenthal et al, 1984; Lewy et al, 1989). However in this study there was no consistent relationship found between sunshine and depressive symptom change. Additionally, vegetative change was difficult to ascribe to sunlight directly and the change of season as a whole appeared to be more in line with the findings. This is consistent with other studies based in Australia whereby factors other than photoperiod were considered to be more important to change (Murray & Hay, 1997). It is possible that this indicates a schema focus regarding winter amongst sufferers in countries with little difference in seasonal photoperiod; however it was not possible to explore this in this investigation. Nonetheless this has highlighted a direction for potential future research. Furthermore, the re-emergence of symptoms in three of the sample in response to change of season indicates that this may be a variable worth further exploration in New Zealand. This study has served to demonstrate that New Zealand may also have a small percentage of people who experience a seasonal pattern of vegetative symptoms and depression. Therefore how various treatments can help these people is a valuable consideration as New Zealand has a significant number of people who are diagnosed with depression and how to best help them is an ongoing problem.

Additionally, the majority of previous research has been based in the Northern Hemisphere and at greater latitudes than New Zealand. Moreover its place as an island that is comparatively distant from other land masses serve to explore season and depression in a new and unique setting. The findings around the meteorological variables serve to accentuate the distinctive geographical location. Additionally, this research adds to the research on temperate locations and research based in the Southern Hemisphere, both of which are comparatively smaller. That some of the findings are consistent with other studies despite the geographical uniqueness serve to support the concept of

seasonal depression as a valid condition that can present in a variety of climates rather than limiting its presentation to the extreme latitudes that it has been historically associated with.

The effects of temperature on symptom expression were tested in Study 3 (hypothesis 17). As regards the effects of temperature on BDI-II scores, the findings were inconclusive. Although temperature demonstrated a clear correlation with season (see Appendix E), results showed limited translation of this construct onto mood. This was in line with previous findings by Boyce and Parker (1988) who also found that temperature contributed little to symptom expression. This indicated that the effects of temperature may be transient and readily mitigated by access to warmth. This also may be due to the relatively mild temperatures experienced in Auckland. Therefore, replicating this study in an area with greater diversity of temperature recordings may produce different results.

In Study 1 rainfall showed little association with mood, but young people tended to respond to advertisements for therapy on depression on rainy days. This may be due to younger persons engaging in more outdoor activities which would then be associated with low mood when unavailable. However, due to the inconsistent nature of this variable any conclusions are supposition at this point and require further study.

Relationships between Depressive and Vegetative Symptoms

Hypothesis 14 in Study 3 tested whether the rate of change in depression scores would be greater than vegetative scores. This was supported indicating that the symptoms were independent and that it was possible to treat depression separately from the symptoms associated with seasonal depression. The lack of direct effect of sunshine on depression scores as opposed to vegetative symptoms served to reinforce that belief. These findings also provided support for the concept of seasonality in research by Chotai et al. (2004b) and Kasper et al. (1989), who observed that changes in physical symptoms associated with the seasons was separate from negative cognitions and depression that characterised SAD.

Additionally, the return of vegetative symptoms with change of season in three of the participants without an associated significant increase in depressive symptoms provided support for the DVH. These results showed that for some, the vegetative symptoms appeared to have a biological origin which was not under the cognitive control of the person. With the re-emergence of vegetative symptoms, one showed no corresponding depression, another had mild symptoms of depression and the other had moderate symptoms of depression (see Figures 1 and 4). This finding indicated that the re-emergence of vegetative symptoms was not contingent on parallel depressive symptom re-emergence. That there was a return of depressive symptoms in two of this group does indicate that a more accurate assessment and tailoring of treatment are indicated for those with seasonal factors in their depression.

Cognitive behaviour therapy (CBT) was found to be effective for the majority of the sample in this study, including those with seasonal factors. Additional support for the effectiveness of CBT in seasonal depression was observed in that those with and those without seasonal symptoms showed similar rates of change in depressive scores. This replicated the findings of other studies (Engasser & Young, 2007; Freed, 2006; Rohan et al., 2007) which also advocated the efficacy of CBT for SAD. From these results it would appear that CBT is a valid and effective treatment for the cognitive symptoms associated with seasonal depression, but it requires some tailoring in order to take into account the specific needs of the seasonally depressed group. This would include the use of psychoeducation, increased exposure to bright light, relapse prevention and schema work around winter in vulnerable individuals in order to effectively manage or reduce the vegetative symptoms and provide the person with an increased sense of relief and efficacy as well as long term benefits. This combination of CBT and increased exposure to light would appear to be especially important for vulnerable women due to the observed stability of their symptom profile over time.

This study also allowed other symptom profiles i.e. atypical depression to be seen and explored. This group of people may otherwise have been grouped with individuals with a seasonal pattern to their depression (especially if they presented in the winter

period) thus accentuating the perceived prevalence of seasonal depression. In this study the atypical group was differentiated and other possible interpretations and implications for vegetative symptoms and their depressive profile postulated. As atypical depression has had limited focus in the literature and was not the focus of this investigation, this was an interesting although unexpected finding. It was the exploratory focus of this study and the analysis of vegetative symptoms over time that allowed for this presentation to become apparent. As a result this group has emerged as a potential focus for future research due to the chronic manifestation of symptoms over time.

Demographic Variables and Depression

Age and Depressive and Vegetative Symptoms

Youth had previously been associated with higher levels of depression and increased irritability in response to seasonal change (Amons et al., 2006; APA, 2000; Chotai et al., 2004b; Lahti et al., 2003; Kovalenko et al., 2000; Marušič et al., 2003; Sweedo et al., 1995). In this study, age was explored in relation to season of presentation in Study 1 (hypothesis 5) in which there was no support found for the belief that a greater proportion of younger people would present to the parent study in winter. This result indicated that age and season of presentation were not associated in this sample. It was postulated in hypothesis 6 that age would be negatively associated with levels of depression, vegetative symptoms and irritability. This hypothesis was tested in Study 2 where there was found to be no significant relationships with depressive symptom expression. However there was a trend noted in relation to increasing age and decreasing vegetative symptoms noted in Study 2. This finding supports other research which postulated that the frequency of vegetative symptoms expressed would decrease as the individual got older (Kovalenko et al., 2000). The relationship between age and vegetative symptoms over time was further tested in Study 3 (hypothesis 6) where the results indicated that although age did impact on vegetative symptoms at presentation it did not significantly affect the rate of change in the sample. This result shows that the sample responded over time in a similar manner regardless of age and that therapy was effective for all ages represented. In addition, age was not found to have any significant

effect on depressive and vegetative scores over time in Study 3. Although there was a wide range of ages represented in this study, there were none who were younger than 20 years old, and so this study was unable to explore any child and adolescent aspects of the disorder.

The Influence of Gender

Throughout the literature women are found to be more likely to suffer from depression and SAD (APA, 2000; Chotai et al., 2004a; Chotai et al., 2004b; Murray, 2004; Rohan et al., 2003; Rosenthal et al., 1984). In this study gender was explored in Study 1 (hypothesis 7) which postulated that more women would present in the winter months. This belief was supported and was in line with previous research (Chotai et al., 2004a). Additionally, more women overall applied for admission into the study on therapy for depression. Hypothesis 9 was also explored in Study 1 which looked at whether more men would present for therapy on days with decreased sunshine hours. Contrary to other research there was no support found for this belief in this study. In Study 2, hypothesis 8 tested whether women endorsed higher levels of depression, which was supported however women did not endorse more vegetative symptoms than men in Study 2. Hypothesis 10 tested in Study 2 postulated that more women would endorse increased appetite as a symptom however this was not supported. Neither was hypothesis 11 tested in Study 3 which showed no significant relationship between men and irritability. Conversely in Study 3, women were observed to have higher vegetative symptomology over time and were also observed to have an increased response to bright sunlight as a method of decreasing vegetative symptoms. Those who showed a re-emergence of seasonal symptoms at the end of Study 3 indicative of an ongoing seasonal pattern were all women however there were a number of men who indicated vegetative symptoms that were non seasonal. These divergent findings indicate that further research is required to clarify the relationships between gender and vegetative symptoms and seasonal depression.

Implications for assessment and treatment

This raises a number of issues around current assessment and treatment options available in New Zealand. As was previously noted, GPs tend to prescribe medication for those they presume to be depressed; however this is often done without a thorough assessment of the presenting symptoms (Wilson & Read, 2001). Therefore it is possible to misdiagnose people and so begin a process of inadequate treatment which can have chronic disability associated with it (see Martiny, 2006). This study highlighted the differing patterns of symptom expression and supports the need for a comprehensive assessment to be performed in order to differentiate those who may be seasonally depressed from the atypical or non seasonal depression sufferers. This is especially as a majority of the presentations did occur in winter which may result in a seasonal bias present in either the client or the clinician as to the diagnostic choice.

A traditional treatment for seasonal depression is LT which is usually delivered via a dedicated light box which are available without prescription. Therefore, they are potentially open to misuse. This can be by those either without a seasonal depression who misdiagnose themselves or by those who use the box wrongly; either not for long enough which can result in no relief of symptoms, or over use resulting in eye problems or mania (Sato, 1997). This study explored the use of natural sunlight as an alternative to LT. Results indicated that it was effective for the relief of vegetative symptoms in some people which meant it can be advocated as an alternative which has additional health benefits. Although there are some risks associated with sunlight the relatively short exposures required due to the comparative strength of light mean that potentially it is easier to use than light boxes and more pleasant as it can be combined with enjoyable activities.

Additionally CBT was indirectly examined in this study regarding its effect on those with a seasonal and non seasonal depression. Results here suggested that it was suitable for either group however potentially more effective for non-seasonal depression unless it was tailored to meet the specific requirements of the seasonal depression sufferer.

Although this study did not specifically address the effectiveness for therapy it was able to demonstrate improvement in mood for the entire sample. This supports other studies which have suggested that CBT can be effectively used for seasonal depression as a cost-effective intervention which has ongoing gains (Freed, 2006; Rohan et al, 2007). Specific tailoring would include light based outdoor activities and exercise which have been documented as effective for those with seasonal depression (Leppamaki et al, 2002). Additionally, some cognitive restructuring work around winter schemas would be beneficial. This would involve finding positive aspects to winter and engaging in pleasant activities or holidays in order to aid the process. An aspect that has been also indicated by Rohan (2007) as important is the use of relapse prevention and maintenance plans which are focused on reducing the impact of vegetative symptoms which can result in cognitive distress for seasonal depression sufferers.

Additionally, the benefits of exercise as a potential treatment option for seasonal depression would have a number of effects. These would not only include the improvement in mood but studies have also indicated that they may be beneficial for the regulation of neurotransmitters which would help with the increased sleep and weight symptoms expressed by many who suffer with seasonal depression. This has implications for the management of obesity in these people which have been noted to be a significant issue for the health of the country as a whole.

Limitations and Future Directions

This study has shown that the seasonal factors in depression can manifest in a New Zealand context. However, the true estimates of its prevalence in this country as a whole are difficult to estimate currently from this study. A limitation of this study was that it did not directly study seasonal depression which is a retrospective diagnosis (APA, 2000), but focused on those with first episode depression. Although this study showed a small portion of the population had sufficient severity of symptoms to be considered to have seasonal depression; admission criteria for the depression study may have excluded

a portion of the population that has a recurrent seasonal depression profile which was required for the DSM-IV-TR definition of the diagnosis.

Additionally, there are no indications of how this diagnosis would present in Maori and Pacific populations. Immigration has been observed as a possible precipitant for seasonal depression (Michalak et al, 2004). Additionally, those with darker eye and skin colour have also been shown to require greater sunlight exposure than those with lighter coloring (Kimlin, et al, 2007). This is hypothesised to be because countries where these characteristics are naturally present have stronger sunshine and these characteristics serve as a protective factor in the environment. However, it would also seem that such racial groups have a natural requirement for more sunlight than is often present in their current environment in order to feel the positive benefits of exposure (Kimlin et al, 2007). This may have implications for New Zealand as we have a large immigrant population which often originates in countries with stronger sunlight than is experienced here. Therefore whether this has an impact on mental health should be explored more rigorously, especially as Maori have been noted to have a different depression presentation in the literature (Tapsell & Melsop, 2007).

The sample involved in this investigation was small and so there was a corresponding lack of power to detect small effects and it was only possible to observe medium to large differences in the sample as related to the hypotheses. A number of factors contributed to the sample size, including the stringent criteria involved in the selection process for the parent study. It took over two years to accumulate the sample, which indicated that another factor may be the overall smaller population available, especially in comparison to other studies which were situated in areas with significantly more people (Chotai et al., 2004a; Lam et al., 2006; Michalak et al., 2004; Rosenthal et al., 1989). Therefore, additional studies involving a number of centres may provide a larger and more diverse sample. This would also enable exploration of how seasonal factors in depression or seasonal depression present within New Zealand as a country.

Another benefit of further research is that it would address the weakness of the findings regarding the meteorological variables. Some of the associations between sunshine and temperature in this study were weak and may not present an accurate picture of how weather presents in New Zealand as a whole or even Auckland over an extended period of time. Therefore although the associations found in this study were significant it is possible that the relationships discovered were a result of error and so findings should be viewed as preliminary rather than a true picture of the country as a whole. The geographical location would also impact on findings in the study. Auckland is located on an isthmus which means that weather conditions produced variable patterns which made the resultant analyses difficult. These conditions are different from those experienced in other parts of the country which may affect the generalisability of the findings. Furthermore, the negative relationship between sunshine and temperature was surprising and may have affected findings in unintended ways.

Furthermore, there was a corresponding gender inequality with significantly more women than men involved in this study. This may have served to accentuate any group differences observed. Thus this investigation is unable to add to the literature on this aspect. Future studies which have an even gender balance may help to provide more accurate information on this aspect would be beneficial. Additionally, exploring the genders separately may also provide a useful dimension to how this disorder presents as regards gender.

That this study did not explore the cognitive aspect of seasonal depression is also a limitation of this study. How the sample may have changed over the course of therapy in relation to their cognitive aspects of their depression especially as regards season could have contributed to the findings around seasonal and atypical depression in meaningful ways. Additionally exploration of schemas especially in relation to season would have added to the validity of findings around those who exhibited a seasonal pattern to their depression. The cognitive characteristics of seasonal depression observed in other studies make this a potential valuable focus for future study.

The need to stay out of the sun and protect the eyes from the sunlight has been advocated widely in NZ and Australia (Cancer Council Australia, 2006). These precautions were initiated in order to reduce the risk of melanomas and other skin cancers which result from prolonged sun exposure. Although the risk is real and deadly, it may be that the other side of the proverbial coin is a resulting increase in chronic vegetative symptoms within the vulnerable portion of the population. As the eyes were found to be the most effective route for LT (Sato, 1997; Wehr et al., 1987), their constant shielding may further limit exposure to light for those vulnerable to developing seasonal depression. This may result in not only ongoing mental health issues but also decreased productivity due to chronically low mood and fatigue (Rosenthal et al., 1984). One may speculate that these factors may also be a long term contributor toward the development of obesity in the population due to the increased appetite and associated weight gain of seasonal depression. Further study in this area would be valuable as regards public health especially as regards the effectiveness of increased light exposure for symptom relief.

Atypical depression. There were a number of individuals in this study who indicated chronic vegetative symptoms even in the summer months. This indicates that a portion of this sample had symptom expression more consistent with atypical depression whereby they present with similar symptoms to seasonal depression but show no seasonal pattern and little response to light exposure (Goel et al., 2002; Stewart et al., 1990).As the majority of these individuals also reported ongoing depressive symptoms after therapy, atypical depression appears to be an area that warrants further investigation as to the best means of helping this group of people. It may be that a tailored therapy is warranted involving longer therapy periods and that medication needs to be considered for long-term relief. As this group was larger than those who presented with a seasonal profile, they may have a greater impact on society as regards their long-term mental health than either of the other groups as they also showed a greater chronicity of depression.

Concluding Remarks

This study contributes to the findings on seasonal depression by exploring the factors associated with this diagnosis in relation to symptoms of depression. The seasonal differences in presentation are in line with other studies which indicate that New Zealand may have a portion of the population with seasonal depression that would be worth further exploration due to the long-term health costs associated with the disorder.

However, the group of atypical depression sufferers makes the number of those who potentially have seasonal depression difficult to ascertain from this study as they can present in a similar manner that may not be discernable without an accurate assessment and monitoring of treatment. This can be a potential problem due to the observed chronicity associated with this group.

The differing patterns of change observed by depressive and vegetative symptoms provides some support for the DVH around the individual vulnerabilities associated with the diagnosis but questions the accepted order of change. In this study depressive symptoms were more easily changed than vegetative symptoms which are contrary to previous studies which indicate that vegetative symptoms change should precede depressive symptom change. That this was not supported in this research indicates that the biological symptoms can fluctuate over time and season but that the person can respond to these symptoms differently over time. This is in line with other studies which have noted that a seasonal depression will not necessarily occur each year. This indicates that interventions focusing on cognitive aspects of change may have a large effect on levels of distress associated with symptoms. In this respect therapy can provide a valuable tool and skills in the long-term management of this disorder. This is encouraging as medication and LT may not be effective for some people and require expensive monitoring that may be unnecessary for those with a milder form of the disorder but who may benefit from a cognitive intervention.

References

Abas, M., & Murphy, D. (1987). Seasonal affective disorder: The miseries of long dark nights. *British Medical Journal 295*, 1987- 1988.

Akaike, H. (1973). Information theory as an extension of the maximum likelihood principle. In B. N. Petrov & F. Csaki (Eds.), *Second international symposium on information theory* (pp. 228-267). Budapest, Hungary: Akademiai Kiado.

American Psychiatric Association: APA, (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., rev.). Arlington, VA: APA.

Amons, P. J., Kooij, J.J., Haffmans, P.M., Hoffman, T.O., & Hoencamp, E. (2006). Seasonality of mood disorders in adults with lifetime attention-deficit/hyperactivity disorder. *Journal of Affective Disorders, 91*, 251-255.

Andrews, G., Poulton, R., & Skoog, I. (2005). Lifetime risk of depression: Restricted to a minority or waiting for most? Editorial. *British Journal of Psychiatry, 187*, 495-496.

Armstrong, D., Meenagh, G., Bickle, I., Lee, A., Curran, E., & Finch, M. (2007). Vitamin D deficiency is associated with anxiety and depression in fibromyalgia. *Clinical Rheumatology, 26*, 551-554.

Avasthi, A., Sharma, A., Gupta, N., Kulhara, P., Varma, K., Malhotra, S., & Mattoo, S. (2001). Seasonality and affective disorders: A report from North India. *Journal of Affective Disorders, 64*, 145-154.

Avery, D., Kouri, M., Monaghan, K., Bolte, M.A., Hellekson, C., & Eder, D. (2002). Is dawn stimulation effective in ameliorating the difficulty awakening in seasonal affective disorder associated with hypersomnia? *Journal of Affective Disorders, 69*, 231-236.

Axelsson, J., Stefánsson, J., Magnússon, A., Sigvaldason, H., & Karlsson, M. (2002). Seasonal affective disorders: Relevance of Icelandic and Icelandic-Canadian evidence to etiologic hypotheses. *Canadian Journal of Psychiatry, 47*, 153-158.

Bardone, A., Moffitt, T., Caspi, A., Dickson, N., & Silva, P. (1996). Adult mental health and social outcomes of adolescent girls with depression and conduct disorder. *Development and Psychopathology, 8*, 811-829.

Barlow, D., & Durand, V.M. (2002). *Abnormal psychology* (3rd ed.). Belmont:

Wadsworth. Thompson Learning.

Barnes, J., Anderson, L., & Phillipson, J. (2001). St John's wort (Hypericum perforatum L.): A review of its chemistry, pharmacology and clinical properties. *Journal of Pharmacy & Pharmacology, 53*, 583-600.

Barton, D., Esler, M., Dawood, T., Lambert, E., Haikerwal, D., Brenchley, C., Socratous, F., Hastings, J., Guo, L., Wiesner, G., Kaye, D., & Bayles, R., Schlaich, M., Lambert, G. (2008). Elevated brain serotonin turnover in patients with depression: Effect of genotype and therapy. *Archives of General Psychiatry, 65*(1), 38-46.

Bauer, M. S. (1992). Defining seasonal affective disorder(s). *Biological Psychiatry, 31*, 1185-1189.

Beck, A. T., Rush, A.J., Shaw, B.E., & Emery, G. (1979). *Cognitive therapy for depression*. New York: Guildford Press.

Beck, A. T., Steer, R.A., & Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonia, TX: The Psychology Corporation.

Beesdo, K., Pine, D., Lieb, R., & Wittchen, H. (2010). Incidence and risk patterns of anxiety and depressive disorders and categorization of generalised anxiety disorder. *Archives of General Psychiatry, 67*(1), 47-57.

Berk, M., Sanders, K., Pascoe, J., Jacka, F., Williams, L., Hayles, A., & Dodd, S. (2007). Vitamin D deficiency may play a role in depression. *Medical Hypotheses, 69*, 1316-1319.

Blampied, N. (1999). Cognitive-behaviour therapy in Aotearoa New Zealand. *The Behaviour Therapist, 22*(9), 173-178.

Boulard, N. E. (2004). Quantitative and qualitative measures of activity in seasonal affective disorder. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 65*(2-B), 1021.

Boyce, P., & Parker, G. (1988). Seasonal affective disorder in the southern hemisphere. *American Journal of Insanity, 145*, 96-99.

Brace, N., Kemp, R., & Sneglar, R. (2003). *SPSS for Psychologists: A guide to data analysis using SPSS for Windows* (2nd ed.). New York: Palgrave Macmillan.

Brown, G. W., & Harris, T.O. (1989). Depression. In G. W. Brown, & T.O. Harris

(Eds.), *Life events and illness*. New York: Guildford Press.

Bunney, W., & Bunney, B. (2000). Molecular clock genes in man and lower animals: Possible implications for circadian abnormalities in depression. . *Neuropsychopharmacology, 22*(4), 335-345.

Burns, J. M., Andrews, G., & Szarbo, M. (2002). Depression in young people: What causes it and can we prevent it? *Medical Journal Australia, 177*, S93-S96.

Burton, L., Westen, D., & Kowalski, R. (2009). *Psychology (Australia and New Zealand edition)* (2nd ed.). Australia: John Wiley & Sons.

Cancer Council Australia, (2006). *Eye protection from ultraviolet radiation*. Cancer Council Australia: Eye Research Australia. Retrieved from: http://www.cancer.org.au/file/policypublications/pseyeprotectionaug06.pdf.

Carlson, N., Miller, H., Heath, C.D., Donahoe, J., & Martin, G.N. (2010). The nature and causes of psychological disorders, *Psychology: The science of behaviour* (7th ed.): Allyn & Bacon.

Childs, P., Rodin, I., Martin, N., Allen, N., Plaskett, L., Smythe, P., & Thompson, C. (1995). Effect of fluoxetine on melatonin in patients with seasonal affective disorder and matched controls. *British Journal of Psychiatry, 166*, 196-198.

Chotai, J., Smedh, K., Johansson, C., Nilsson, L., & Adolfsson, R. (2004a). An epidemiological study on gender differences in self-reported seasonal changes in mood and behaviour in a general population of northern Sweden. *Nordic Journal of Psychiatry, 58*, 429-437.

Chotai, J., Smedh, K., Nilsson, L.G., & Adolfsson, R. (2004b). A dual vulnerability hypothesis for seasonal depression is supported by the seasonal pattern assessment questionnaire in relation to temperament and character inventory of personality in a general population. *Journal of Affective Disorders, 82*, 61-70.

Clark, T. (2008). Factors associated with reduced depression and suicide risk among Maori high school students in New Zealand. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 68*(12-B), 7926.

Climatetemp, (2010). Climate for Canberra. Retrieved from http://www.climatetemp.info/australia/canberra.html.

Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple*

regression/correlation analysis for the behavioral sciences (3rd ed.). Mahwah NJ: Lawrence Erlbaum Associates.

Corral, M., Wardrop, A., & Zhang, H. (2006). Seasonality of symptoms in women with postpartum depression. *Archives of Women's Health, 10*, 9-13.

Dancey, C., & Reidy, J. (2004). *Statistics without maths for psychology: Using SPSS for Windows* (3rd ed.). Essex, England: Pearson Education Limited.

Davies, G., Ahmad, F., Chant, D., Welham, J., & McGrath, J. (2000). Seasonality of first admissions for schizophrenia in the southern hemisphere. *Schizophrenia Research, 41*(3), 457-462.

De Berardis, D., Di Iorio, G., Acciavatti, T., Conti, C., Serroni, N., Olivieri, L., Cavuto, M., Martinotti, G., Conti, P., & Di Giannantonio, M. (2010). The emerging role of melatonin agonists in the treatment of major depression: focus on Agomelatine. *CNS & Neurological Disorders-Drug Targets, 10*(1), 119-132.

Denissen, J., Butalid, L., Penke, L., & van Aken, M. (2008). The effects of weather on daily mood: A multilevel approach. *Emotion, 8*(5), 662-667.

Denny, S., Clark, T., Flemming, T., & Wall, M. (2004). Emotional resilience: Risk and protective factors among alternative education students in New Zealand. *American Journal of Orthopsychiatry, 74*, 137-149.

Dumville, J., Miles, J., Porthouse, J., Cockayne, S., Saxon, L., & King, C. (2006). Can vitamin D supplementation prevent winter-time blues? A randomised trial among older women. *Journal of Nutrition, Health and Aging, 10*, 152-153.

Dykema, J., Bergbower, K., Doctora, J., & Peterson, C. (1996). An attributional style questionnaire for general use. *Journal of Psychoeducational Assessment. , 14*, 100-108.

Eagles, J., Howie, F., Cameron, I., Wileman, S., Andrew, J., Robertson, C., & Naji, S. (2002). Use of health care in seasonal affective disorder. *British Journal of Psychiatry, 180*(5), 449-454.

Ellis, P. (2004). Australian and New Zealand clinical practice guidelines for the treatment of depression. *Australian and New Zealand Journal of Psychiatry, 38*, 389-407.

Engasser, J. L. (2005). Cognitive vulnerability to depression in seasonal affective

disorder. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 66*(4-B), 2304.

Engasser, J., & Young, M. (2007). Cognitive vulnerability to depression in seasonal affective disorder: Predicting mood and cognitive symptoms in individuals with seasonal vegetative changes. *Cognitive Therapy and Research, 31*, 3-21.

Ennis, E., & McConville, C. (2004). Stable characteristics of mood and seasonality. *Personality and Individual Differences, 36*, 1305-1315.

Enns, M., Cox, B., Levitt, A., Levitan, R., Morehouse, R., Michalak, E., & Lam, R. (2006). Personality and seasonal affective disorder: Results from the Can-SAD study. *Journal of Affective Disorders, 93*, 35-42.

Fawcett, J. (2008). *Report of the DSM-5 Mood Disorders Work Group*. American Psychiatric Association. Available: http://www.dsm5.org/progressreports/pages/0811reportofthedsm-vmooddisordersworkgroup.aspx [2010.

Fergusson, D. M., & Horwood, L.J. (1984). Life events and depression in women: A structural equation model. *Psychological Medicine, 14*, 881-889.

Fergusson, D. M., & Lynskey, M.T. (1993). The effects of maternal depression on child conduct disorder and attention deficit behaviours. *Social Psychiatry and Psychiatric Epidemiology, 28*, 116-123.

Fergusson, D. M., Horwood, L.J., & Lynskey, M.T. (1995). Maternal depressive symptoms and depressive symptoms in adolescence. *Journal of Child Psychology and Psychiatry, 36*, 1161-1178.

Freed, M. C. (2006). Costs and cost-effectiveness of seasonal affective disorder treatment: Comparing light therapy, group cognitive-behavioural therapy and their combination. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 66*(9-B), 5086.

Gelman, A., & Hill, J. (2007). *Data analysis using regression and multilevel/hierarchical models*. New York: Cambridge University Press.

Giedd, J., Swedo, S., Clark, C., & Rosenthal, N. (1997). "Pediatric seasonal affective disorder: A follow-up report." *Journal of American Academy of Child and Adolescent Psychiatry 37*, 2.

Gloth, F., Alam, W., & Hollis, B. (1999). Vitamin D vs. broad spectrum phototherapy in the treatment of seasonal affective disorder. *Journal of Nutrition, Health and Aging, 3*, 5-7.

Goel, N., Terman, M., & Terman, J. (2002). Depressive symptomology differentiates subgroups of patients with seasonal affective disorder. *Depression and Anxiety, 15*(1), 34-41.

Golden, A., Dalgleish, T., & Spinks, H. (2006). Dysfunctional attitudes in seasonal affective disorder. *Behaviour Research and Therapy, 44*, 1159-1164.

Gorman, J. (2005). Sad seasons: Winter mood not always holiday blues. *CNS Spectrums, 10*(8), 605.

Greenberger, D., & Padesky, C. (1995). *Mind over mood: Change how you feel by changing the way you think*. London: The Guildford Press.

Grof, P. (2002). "Evidence supports validity of seasonal affective disorder": Reply. *The Canadian Journal of Psychiatry, 47*(8), 786-787.

Harmatz, M., Well, A., Overtree, C., Kawamura, K., Rosal, M., & Ockene, I. (2000). Seasonal variation of depression and other moods: A longitudinal approach. *Journal of Biological Rhythms, 15*(4), 344-350.

Hetrick, S., Merry, S., McKenzie, J., Sindahl, P., & Proctor, M. (2007). Selective serotonin reuptake inhibitors (SSRIs) for depressive disorders in children and adolescents: Cochrane Database of Systematic Reviews.

Hetrick, S., Proctor, M., Merry, S., Sindahl, P., & Ward, A. (2007). Selective serotonin reuptake inhibitors (SSRIs) for depression in children and adolescents, *Cochrane Depression, Anxiety and Neurosis Group*: Cochrane Database of Systematic Reviews.

Heidorn, K. (2008). *The Weather Doctor's Diary*, [website]. Weather Doctor. Retrieved from: http://islandnet.com/~see///weather/almanac/seasondate.htm [2009, 12.03.09].

Hetrick, S., Simmons, M., & Merry, S. (2008). SSRIs and depression in children and adolescents: The imperative for shared decision-making. *Australasian Psychiatry, 16*(5), 354-358.

Hoagwood, K., & Johnson, J. (2003). School psychology: A public health framework. I.

From evidence-based practices to evidence -based policies. *Journal of School Psychology, 41*, 3-21.

Holden, J., Kelley, K., & Agarwal, R. (2008). Analyzing change: A primer on multilevel models with applications to nephrology. *American Journal of Nephrology, 28*, 792-801.

Huibers, M., de Graaf, L., Peeters, F., & Arntz, A. (2010). Does the weather make us sad? Meteorological determinants of mood and depression in the general population. *Psychiatry Research, 180*(2), 143-146.

Jacobsen, F., Wehr, T., Sack, D., James, S., & Rosenthal, N. (1987). Seasonal affective disorder: A review of the syndrome and its public health implications. *American Journal of Public Health, 77*, 57-60.

Jacobsen, F., Murphy, D., & Rosenthal, N. (1989). The role of serotonin in seasonal affective disorder and the anti-depressant effect of phototherapy. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorder and phototherapy* (pp. 333-342). New York: Guildford Press.

Kashani, J., McGee, R., Clarkson, S., Anderson, J., Walton, L., Williams, S., Silva, P., & Robbins, A., Cytryn, M., & McKnew M. (1983). Depression in a sample of nine year old children: Prevalence and associated characteristics. *Archives of General Psychiatry, 40*, 1217-1223.

Kasof, J. (2009). Cultural variation in seasonal depression: Cross-national differences in winter versus summer patterns of seasonal affective disorder. *Journal of Affective Disorders, 115*(1), 79-86.

Kasper, S., Rogers, S., Yancey, A., Schultz, P. Skwerer, R., & Rosenthal, N. (1989). Phototherapy in individuals with and without subsyndromal seasonal affective disorder. *Archives of General Psychiatry, 46*, 837-844.

Kasper, S., Rogers, S., Yancey, A., Skwerer, R., Schultz, P., & Rosenthal, N. (1989a). Psychological effects of light therapy in normals. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders & phototherapy* (pp. 260-270). New York: The Guildford Press.

Kazantzis, N., & Lampropoulos, G. (2002). Reflecting on homework in psychotherapy:

What can we conclude from research and experience? *Journal of Clinical Psychology, 58*(5), 577-585.

Kendler, K. S., Karkowski, L.M., & Prescott, C.A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry, 156*, 837-848.

Kim, C., Lesage, A., Seguin, M., Chawky, N., Vanier, C., Lipp, O., & Turecki, G. (2004). Seasonal differences in male suicide completers. *Comprehensive Psychiatry, 45*, 333-339.

Kimlin, N., Harrison, S., Nowak, M., Moore, M., Brodie, A., & Lang, C. (2007). Does a high UV environment ensure adequate vitamin D status. *Journal of Photochemistry and Photobiology B-Biology, 89*(2-3), 139-147.

Kline, P. (1993). Introduction to reliability, validity and classical test theory, *The handbook of psychological testing* (pp. 5-41). London: Routledge.

Kovalenko, P., Hoven, C., Wicks, J., Moore, R., Mandell, D., & Liu, H. (2000). Seasonal variations in internalizing, externalizing and substance use disorders in youth. *Psychiatry Research, 94*, 103-119.

Kripke, D., Mullaney, D., Savides, T., & Gillin, J. (1989). Phototherapy for Non-seasonal Major Depressive Disorders. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders and phototherapy* (pp. 342-356). New York: The Guildford Press.

Kwok, O. M., Underhill, A., Berry, J., Luo, W., Elliott, T., & Yoon, M. (2008). Analyzing longitudinal data with multilevel models: An example with individuals living with lower extremity intra-articular fractures. *Rehabilitation Psychology, 53*(3), 370-386.

Lacoste, V., & Wirz-Justice, A. (1989). Seasonal variation in normal subjects: An update of variables current in depression research. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders and phototherapy* (pp. 167-230). New York: The Guildford Press.

Lahti, A., Räsänen, P., Karvonen, K., Särkioja, T., Meyer- Rochow, V.B., & Hakko, H. (2006). Autumn peak in shooting suicides of children and adolescents from northern Finland. *Neuropsychobiology, 54*, 140-146.

Lam, R., & Levitan, R. (2000). Pathophysiology of seasonal affective disorder: A review. *Journal of Psychiatry Neuroscience, 25*, 469-480.

Lam, R., Levitt, A., Levitan, R., Enns, M., Morehouse, R., Michalak, E., & Tam, E. (2006). The CAN-SAD study: A randomised controlled trial of the effectiveness of light therapy and fluoxetine in patients with winter seasonal affective disorder. *American Journal of Psychiatry* (163), 805-812.

Lambert, G., Reid, C., Kaye, D., Jennings, G., & Esler, M. (2002). Effect of serotonin and season on serotonin turnover in the brain. *Lancet, 360*(9348), 1840-1842.

Lawrynowicz, A., & Baker, T. (2005). Suicide and latitude in Argentina: Durkheim upside down. *American Journal of Psychiatry, 162*, 1022.

Lee, H., Tsai, S., & Lin, H. (2007). Seasonal variation in bi-polar disorder admissions and the association with climate: A population based study. *Journal of Affective Disorders, 97*, 61-69.

Leppamaki, S., Partonen, T., & Lonnqvist, J. (2002). Bright-light exposure combined with physical exercise elevates mood. *Journal of Affective Disorders, 72* (2), 139-144.

Levitan, R., Rector, N., & Bagby, M. (1998). Negative attributional style in seasonal and non-seasonal depression. *American Journal of Psychiatry, 155*, 428-430.

Levitan, R., Jain, U., & Katzman, M. (1999). Seasonal affective symptoms in adults with residual Attention-Deficit Hyperactivity Disorder. *Comprehensive Psychiatry, 40*, 261-267.

Lewy, A., Wehr, T., Goodwin, F., Newsome, D., & Markey, S. (1980). Light suppresses melatonin secretion in humans. *Science, 210*, 1267-1269.

Lewy, A., Sack, R., Singer, C., White, D., & Hoban, T. (1989). Winter depression and the phase shift hypothesis for bright light's therapeutic effects: History, theory and experimental evidence. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders and phototherapy*. New York: Guildford Press

Lewy, A., Lefler, B., Emens, J., & Bauer, V. (2006). The circadian basis of winter depression. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 103*, 7414-7419..

Lurie, S., Gawinski, B., Pierce, D., & Rousseau, S. (2006). Seasonal affective disorder,

American Family Physician.

Madden, P., Heath, A., Rosenthal, N., & Martin, N. (1996). Seasonal changes in mood and behavior: The role of genetic factors. *Archives of General Psychiatry 53*, 47-55.

Maeno, N., Kusunoki, K., Kitajima, T., Iwata, N., Ono, Y., Hashimoto, S., Imai, M., Li, L., Kayukawa, Y., Ohta, T., & Ozaki N. (2005). Personality of seasonal affective disorder analyzed by Tri-dimensional Personality Questionnaire. *Journal of Affective Disorders, 85*(3), 267-273.

Magnusson, A. (2000). An overview of epidemiological studies on seasonal affective disorder. *Acta Psychiatrica Scandinavica, 101*, 176-184.

Magnusson, A., & Partonen, T. (2005). The diagnosis, symptomology, and epidemiology of seasonal affective disorder. *CNS Spectrums, 10*, 625-634.

Marie, D., Forsyth, D., Miles, L. (2004). Categorical ethnicity and mental health literacy in New Zealand. *Ethnicity & Health, 9(3),* 25-252.

Marie, D., & Miles, B. (2007). Does social group status influence social distance in relation to depression? *New Zealand Journal of Psychology, 36*(1), 34-41.

Martiny, K. (2006). Bright light therapy for seasonal affective disorder in Israel (latitude 32.6N: A single case placebo-controlled study. *Acta Psychiatrica Scandinavica. , 114*, 218-219.

Marušič, A., Landau, S., & Tomori, M. (2003). Long-term trends, seasonality, weekly distribution and methods of suicide in Slovenia: A comparison between the younger and older population. *Archives of Suicide Research, 7*, 135-143.

McCarthy, E., Tarrier, N., & Gregg, L. (2002). The nature and timing of seasonal affective symptoms and the influence of self-esteem and social support: longitudinal prospective study. *Psychological Medicine, 32*, 1425-1434.

McCrae, R., & John, O. (1992). An introduction to the Five-Factor Model and its applications. *Journal of Personality, 60*, 175-215.

McGee, R., Anderson, J., Williams, S., & Silva, P. (1986). Cognitive correlates of depressive symptoms in eleven year old children. *Journal of Abnormal Child Psychology, 14*, 517-524.

Meesters, Y., & van Houwelingen, C. (1998). "Rapid mood swings after unmonitored

light exposure": Correction. *American Journal of Insanity, 155*, 710..

Merry, S., Hetrick, S., McDowell, H., & Bir, J. (2004). The effectiveness of psychological and or educational interventions for the prevention of depression in children and adolescents. *The Cochrane Library* (1).

Merry, S., McDowell, H., Wild, C., Bir, J., & Cunliffe, R. (2004a). A randomized placebo controlled trial of a school-based depression prevention program. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(5), 538-547.

Merry, S. N. (2007). Prevention and early intervention for depression in young people - a practical possibility? *Current Opinion in Psychiatry, In press*

Merry, S. N., & Spence, S. (2007). Attempting to prevent depression in young people - a systematic review of the evidence. *Early Intervention in Psychiatry, In press.*

Mersch, P., Vastenburg, N., Meesters, Y, Bouhuys A., Beersma, D., van den Hoofdakker, R., & den Boer, J. (2004). The reliability and validity of the Seasonal Pattern Assessment Questionnaire: A comparison between patient groups. *Journal of Affective Disorders, 80*, 209-219.

Meyer, B., Pilkonis, P., Krupnick, J., Egan, M., Simmens, S., & Sotsky, S. (2002). Treatment expectancies, patient alliance and outcome: Further analyses from the National Institute of Mental Health Treatment of Depression Collaborative Research Program *Journal of Consulting and Clinical Psychology, 70*(4), 1051-1055.

Michalak, E., & Lam, R. (2002). Evidence supports the validity of seasonal affective disorder. *The Canadian Journal of Psychiatry, 47*(8), 786.

Michalak, E., & Lam, R. (2002a). Seasonal affective disorder: The latitude hypothesis revisited. *The Canadian Journal of Psychiatry, 47*, 787-788.

Michalak, E., Wilkinson, C., Hood, K., & Dowrick, C. (2002). Seasonal and non-seasonal depression: How do they differ? Symptom profile, clinical and family history in a general population sample. *Journal of Affective Disorders, 69*, 185-192.

Michalak, E., Wilkinson, C., Hood, K., Dowrick, C., & Wilkinson, G. (2003). Seasonality, negative life events and social support in community sample. *British Journal of Psychiatry, 182*, 434-438.

Michalak, E., Murray, G., Wilkinson, C., Dowrick, C., Lasa, L., Lehtinen, V., Dalgard, O.S., Ayuso-Mateos, J., Vazquez-Barquero, J., & Casey, P. (2004). Estimating depression prevalence from the Beck Depression Inventory: Is season of administration a moderator? *Psychiatry Research, 129*, 99-106.

Michalak, E., Tam, E., Manjunath, C., Levitt, A., Levitan, R., & Lam, R. (2005). Quality of life in patients with seasonal affective disorder: Summer vs. winter scores. *The Canadian Journal of Psychiatry, 50*, 292-295.

Michalak, E., Murray, G., Levitt, A., Levitan, R., Enns, M., Morehouse, R., Tam, E., Cheung, A., & Lam, R. (2007). Quality of life as an outcome indicator in patients with seasonal affective disorder: Results from the Can-SAD study. *Psychological Medicine, 37*, 727-736.

Miller, B., & Keane, C. (1983). *Encyclopedia and dictionary of medicine, nursing and allied health* (3rd ed.). Philadelphia: W.B. Saunders Co.

Ministry of Health, (2008). Mood Disorders: *A portrait of health-key results of the 2006/07 New Zealand health survey* (pp. 170-173).

Moffitt, T., Caspi, A., Harrington, H., Milne, B., Melchior, M., Goldberg, D., & Poulton, R. (2007). Generalised anxiety disorder and depression: Childhood risk factors in a birth cohort followed to age 32. *Psychological Medicine, 37*, 441-452.

Moller, S. (1991). Carbohydrates, serotonin, and atypical depression. *Nordic Journal of Psychiatry, 45*, 363-366.

Morken, G., Sund, A., & Linaker, O. (2004). A help-line for children: Seasonal variations in issues. *Psychiatry Research, 128*(2), 191-197.

Morrissey, S., Raggatt, P., James, B., & Rogers, J. (1996). Seasonal affective disorder: Some epidemiological findings from a tropical climate. *Australian and New Zealand Journal of Psychiatry, 30*, 579-586.

Moscovici, L. (2006). Bright light therapy for seasonal affective disorder in Israel (latitude 32.6N): A single case placebo-controlled study. *Acta Psychiatrica Scandinavica, 114*, 216-219.

Murray, G., & Hay, D. (1997). Seasonal affective disorder in Australia: Is photoperiod critical? *Australian and New Zealand Journal of Psychiatry, 31*, 279-284.

Murray, G. (2001). Seasonality: the importance of longitudinal measurement. In T.

Partonen, & A. Magnusson (Eds.), *Seasonal affective disorder* (pp. 55-62). New York: Oxford University Press.

Murray, G., Allen, N., Rawlings, D., & Trinder, J. (2002). Seasonality and personality: A prospective investigation of five factor model correlates of mood seasonality. *European Journal of Personality, 16*(6), 457-468.

Murray, G. (2003). The seasonal pattern assessment questionnaire as a measure of mood seasonality: A prospective validation study. *Psychiatry Research, 120*(1), 53-59.

Murray, G., Allen, N., & Trinder, J. (2003). Seasonality and circadian phase delay: Prospective evidence that winter lowering of mood is associated with a shift towards Eveningness. *Journal of Affective Disorders, 76*, 15-22.

Murray, G. (2004). How common is seasonal affective disorder in temperate Australia? A comparison of BDI and SPAQ estimates. *Journal of Affective Disorders, 81*, 23-28.

Murray, G., Michalak, E., Levitt, A., Levitan, R., Enns, M., Morehouse, R., & Lam, R. (2005). Therapeutic mechanism in seasonal affective disorder: Do fluoxetine and light operate through advancing circadian phase. *Chronobiology International, 22*, 937-943.

Muscettola, G., Barbato, G., Ficca, G., Beatrice, M., Puca, M., Aguglia, E., & Amati, A. (1994). Seasonality of mood in Italy: Role of latitude and sociocultural factors. *Journal of Affective Disorders, 33*, 135-139.

Natale, V., Adan, A., & Scapellato, P. (2005). Are seasonality of mood and eveningness closely associated? *Psychiatry Research, 136*, 51-60.

National Institute of Water and Atmospheric research: NIWA (2009). *Daily temperature and sunshine hours for New Zealand*. Retrieved from: http://cliflo.niwa.co.nz/.

Neimeyer, R., Kazantzis, N., Kassler, D., Baker, K., & Fletcher, R. (2008). Group cognitive behavioural therapy for depression outcomes predicted by willingness to engage in homework, compliance with homework, and cognitive restructuring skill acquisition. *Cognitive Behaviour Therapy 37*(4), 199-215.

Neumeister, A., Willeit, M., Praschak-Rieder, N., Asenbaum, S., Stastny, J., Hilger, E., Pirker, W., Konstantinidis, A., & Kasper, S. (2001). Dopamine transporter availability in symptomatic depressed patients with seasonal affective disorder

and healthy controls. *Psychological Medicine, 31*, 1467-1473.

New Ethicals. (2008). *Fluoxetine hydrochloride*, [MIMS]. New Ethicals [2008].

Pan, B., Rowe, M., Singer, J., & Snow, C. (2005). Maternal correlates of growth in toddler vocabulary production in low-income families. *Child Development, 76*(4), 763-782.

Parslow, R., Jorm, A.F., Butterworth, P., Jacomb, P.A., & Rodgers, B. (2004). An examination of seasonality experienced by Australians living in a continental temperate climate zone. *Journal of Affective Disorders, 80*(2-3), 181-190.

Partonen, T., & Lonnqvist, J. (1996). Maclobermide and fluoxetine in treatment of seasonal affective disorder. *Journal of Affective Disorders, 41*, 93-99.

Partonen, T. (2001). Light Therapy. In T. Partonen & A. Magnusson (Eds.), *Seasonal affective disorder: Practice and research* (pp. 65-78). Oxford: Oxford University Press.

Partonen, T., & Rosenthal, N. (2001). Symptoms and course of illness. In T. Partonen, & A. Magnusson (Eds.), *Seasonal affective disorder: Practice and research* (pp. 11-18). Oxford: Oxford University Press.

Patterson, K. (2007). *SAD - Winter Blues*, Kiwi Families. Retrieved from http://www.kiwifamilies.co.nz/search.html?terms=SAD&source

Peiser, B. (2009). Seasonal affective disorder and exercise treatment: A review. *Biological Rhythm Research, 40* (1), 85-97.

Pinchasov, B., Shurgaja, A., Grischin, O., & Putilov, A. (2000). Mood and energy regulation in seasonal and non-seasonal depression before and after midday treatment with physical exercise or bright light. *Psychiatry Research, 94*(1), 29-42.

Pittendrigh, C. (1989). The photoperiodic phenomena: Seasonal modulation of the "day within". In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorder and phototherapy* (pp. 87-104). New York. The Guildford Press.

Pjrek, E., Winkler, D., Heiden, A., Praschak-Rieder, N., Willeit, M., Konstantinidis, A., Stastny, J., & Kasper, S. (2004). Seasonality of birth in seasonal affective disorder. *Journal of Clinical Psychiatry, 65*, 1389-1393.

Postolache, T., Hardin, T., Myers, F., Turner, E., Yi, L., Barnett, R., Matthews, J., &

Rosenthal, N. (1998). "Greater improvement in summer than with light treatment in winter in patients with seasonal affective disorder." *American Journal of Psychiatry 155*(11), 1614-1616.

Poston, J., & Hanson, W. (2010). Meta-analysis of psychological assessment as a therapeutic intervention. *Psychological Assessment, 22*(2), 203-212.

Praschak-Rieder, N., Willeit, M., Wilson, A., Houle, S., & Meyer, J. (2008). Seasonal variation in human brain serotonin transporter binding. *Archives of General Psychiatry, 65*(9), 1072-1078.

Putilov, A., & Danilenko, K. (2005). Antidepressant effects of light therapy and "natural" treatments for winter depression. *Biological Rhythm Research, 36*, 423-437.

Rastard, C., Sjödén, P., & Ulfburg, J. (2005). High prevalence of self-reported winter depression in a Swedish county. *Psychiatry and Clinical Neurosciences, 59*, 666-675.

Raudenbush, S. W. (2004). *HLM 6: Hierarchical linear and nonlinear modeling*: Scientific Software International.

Rawana, J. S. (2006). Integrating the cognitive-specificity and dual vulnerability hypotheses: Implications for vegetative and cognitive/affective differences in seasonal, non-seasonal, and sub-syndromal seasonal depression. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 67*(6-B), 3463.

Reardon, A. (2007). An exploratory study of seasonal affective disorder symptoms over the course of remission. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 68*(3-B), 1940.

Rock, D., Greenberg, D., & Hallmayer, J. (2003). Increasing seasonality of suicide in Australia 1970-1999. *Psychiatry Research, 120*(1), 43-51.

Rock, D., Judd, K., & Hallmayer, J. (2008). The seasonal relationship between assault and homicide in England and Wales. *International Journal of Care of the Injured, 39*, 1047-1053.

Rohan, K. (1998). *Cognitive reactivity in seasonal affective disorder.* Unpublished Doctoral Thesis, Maine.

Rohan, K., Sigmon, S., & Dorhofer, D. (2003). Cognitive behavioral factors in seasonal affective disorder. *Journal of Consulting and Clinical Psychology, 71*, 22-30.

Rohan, K., Roecklein, K., Lindsey, K., Johnson, L., Lippy, R., Lacy, T., & Barton, F. (2007). A randomized controlled trial of cognitive-behavioral therapy, light therapy, and their combination for seasonal affective disorder. *Journal of Consulting and Clinical Psychology, 75*, 489-500.

Rosenthal, N. E., Sack, D., Gillin, J., Lewy, A., Goodwin, F., Mueller, P., Newsome, D., & Wehr, T. (1984). Seasonal affective disorder: A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry, 41*, 72-80.

Rosenthal, N. E., Sack, D., Skwerer, R., Jacobsen, F., & Wehr, T. (1989). Phototherapy for seasonal affective disorder. In N. E. Rosenthal, &M.C. Blehar (Eds.), *Seasonal affective disorder and phototherapy* (pp. 273-294). New York: The Guildford Press.

Rosenthal, N. E. (1998). *Winter blues: Seasonal affective disorder, what it is and how to overcome it*. New York: The Guildford Press.

Royal Astronomical Society of New Zealand: RASNZ (2011) *Sunrise and set times for New Zealand*. RASNZ. Retrieved from: http://www.rasnz.org.nz/

Ruby, N., Brennan, T., Xie, X., Cao, V., Franken, P., Heller, H.C., & O'Hara, B. (2002). Role of melanopsin in circadian responses to light. *Science, 298*, 2211-2213.

Ruhrmann, S., Kasper, S., Hawellek, B., Martinez, B., Hoflich, G., Nickelson, T., & Moller, H. (1998). Effects of fluoxetine versus bright light in the treatment of seasonal affective disorder. *Psychological Medicine, 28*, 923-933.

Russell, J., & Kazantzis, N. (2008). Medication beliefs and adherence to antidepressants in primary care. *New Zealand Medical Journal, 121*(1286), 14-20.

Ryan, N. (2001). Diagnosing pediatric depression. *Society of Biological Psychiatry, 49*, 1050-1054.

Sachs, G., Jain, U., Truman, C., Blais, M., Otto, M., & Hirschfeld. (1996). Seasonal Affective Disorder and personality characteristics: Assessing personality traits of pretreatment and post-treatment phases of seasonal depression. *Society for Light Treatment and Biological Rhythms, Abstract 8*, 36.

Sarafino, E. (1994). *Health psychology: Biopsychosocial interactions* (2nd ed.). New York: John Wiley & Sons Inc.

Sato, N. (1997). Seasonal affective disorder and phototherapy: A critical review. *Professional Psychology: Research and Practice, 28*, 167-169.

Sato, T., Bottlender, R., Sievers, M., & Moller, H. (2006). Distinct seasonality of depressive episodes differentiates unipolar depressive patients with and with out depressive mixed states. *Journal of Affective Disorders, 90*, 1-5.

Schneider, B., Weber, B., Frensch, A., Stein, J., & Fritz, J. (2000). Vitamin D in schizophrenia, major depression, and alcoholism. *Journal of Neural Transmission, 107*, 839-842.

Schwarz, G. (1978). Estimating the dimensions of a model. *Annals of Statistics, 6*, 461-464.

Scott, J. (2008 in press). Cognitive therapy. In N. Kazantzis, M. Reinecke, & A. Freeman (Eds.), *Cognitive behaviour therapies: Theoretical foundations*. New York: Guildford.

Shen, J., & Shapiro, C. (2002). Polysomnographic sleep effects of fluoxetine and nefazodone on a seasonal affective disorder patient. *Sleep Medicine, 3*, 5270528.

Sigmon, S., Whitcomb-Smith, S., Boulard, N., Pells, J., Hermann, B., Edenfield, T., LaMattina, S., & Schartel, J. (2007). Seasonal reactivity: Attentional bias and psychophysiological arousal in seasonal and non seasonal depression. *Cognitive Therapy and Research, 31*, 619-638.

Singer, J., & Willett, J. (2003). *Applied longitudinal data analysis: Modeling change and event occurrence*. New York: Oxford University Press.

Singer, J., & Willett, J. (2003a). Introduction to SAS: UCLA: Academic Technology Services, Statistical Consulting Group. Retrieved from http://www.ats.ucla.edu/stat/sas/notes2/.

Skwerer, R., Jacobsen, F., Duncan, C., Kelly, K., Sack, D., Tamarkin, P., Gaist, S., & Rosenthal, N. (1989). Neurobiology of seasonal affective disorder and phototherapy. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorder and phototherapy* (pp. 311-332). New York: The Guildford Press.

Sonis, W. A. (1989). Seasonal affective disorder of childhood and adolescence: A review. In N. E. Rosenthal, & M.C., Blehar (Eds.), *Seasonal affective disorders and phototherapy* (pp. 46-54). New York: The Guildford Press.

Spezzano, M. (2007). The efficacy of bright light treatment on the symptoms of seasonal affective disorder. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 67*(9-B), 5423.

SPSS, Inc. (2008). Statistical Program for the Social Sciences (Version 17): IBM.

Stewart, J., Quitkin, F., Terman, M., & Terman, J. (1990). Is seasonal affective disorder a variant of atypical depression? Differential response to light therapy. *Psychiatry Research, 33*(2), 121-128.

Stulz, N., Lutz, W., Leach, C., Lucock, M., & Barkham, M. (2007). Shapes of early change in psychotherapy under routine outpatient conditions. *Journal of Consulting and Clinical Psychology, 75*(6), 864-874.

Sturman, A., & Tapper, N. (2006). *The weather and climate of Australia and New Zealand* (2nd ed.). Melbourne: Oxford University Press.

Sullivan, B., & Payne, T. (2007). Affective disorders and cognitive failures: A comparison of seasonal and non-seasonal depression. *American Journal of Insanity, 164*, 1663-1667.

Sund, A., Morken, G., & Linaker, O. (2002). Seasonal variation in children's calls to a help-line: Implications for preventive service. *Journal of Affective Disorders, 70*(2), 175-180.

Sweedo, S. E., Pleeter, J.D., Richter, D.M., Hoffman, C.L., Allen, A.J., Hamburger, S.D., Turner, E.H., Yamada, E.M., & Rosenthal, N.E. (1995). Rates of seasonal affective disorder in children and adolescents. *American Journal of Psychiatry, 152*, 1016-1019.

Sweedo, S., Allen, A., Glod, C., Rosenthal, N., Teicher, M., Richter, D., Hoffman, C., Brown, C., & Clark, C. (1997). A controlled trial of light therapy for the treatment of pediatric seasonal affective disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(6), 816-821.

Swiecicki, L., & Szafranski, T. (2002). Side effects after phototherapy implementation in addition to fluoxetine or sertraline treatment: A report of two cases. *World Journal of Biological Psychiatry, 3*, 109-111.

Tabachnick, B., & Fidell, L. (2001). *Using multivariate statistics*. Boston: Allyn and Bacon.

Tam, E., Lam, R., & Levitt, A. (1995). Treatment of seasonal affective disorder. *The Canadian Journal of Psychiatry, 40*, 457-466.

Tapsell, R., & Mellsop, G. (2007). The contributions of culture and ethnicity to New Zealand mental health research findings. *International Journal of Social Psychiatry, 53*(7), 317-324.

Teicher, M., Glod, C., Magnus, E., Harper, D., Benson, G., Krueger, K., & McGreenery, E. (1997). Circadian rest-activity disturbances in seasonal affective disorder. *Archives of General Psychiatry, 54*, 124-130.

Terman, M. (1988). On the question of mechanism in phototherapy: Considerations of clinical efficacy and epidemiology. *Journal of Biological Rhythms, 3*, 155-172.

Terman, M. (1989). On the question of mechanism in phototherapy for seasonal affective disorder: Considerations of clinical efficacy and epidemiology. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders & phototherapy* (pp. 357-377). New York: The Guildford Press.

Terman, J., Terman, M., Schlager, D., Rafferty, B., Rosofsky, M., Link, M., Gallin, P., & Quitkin, F. (1990). Efficacy of brief, intense light exposure for treatment of winter depression: 29th annual meeting of the new clinical drug evaluation unit. *Psychopharmacology Bulletin, 26*, 3-11.

Terman, M., Amira, L., Terman, J., & Ross, D. (1996). Predictors of response and nonresponse to light treatment for winter depression. *American Journal of Psychiatry. , 153*, 1423-1429.

Terman, M., & Terman, J. (2005). Light therapy for seasonal and non-seasonal depression: Efficacy, protocol, safety and side-effects. *CNS Spectrums, 10*, 647-663.

Terman, M. (2007). Evolving applications of light therapy. *Sleep Medicine Reviews, 11*, 497-507.

Terman, M., & Terman, J. (2007). "Controlled trial of naturalistic dawn stimulation and negative air ionisation in seasonal affective disorder": Erratum. *American Journal of Psychiatry, 164*

Thase, M. (1989). Comparison between seasonal affective disorder and other forms of recurrent depression. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal*

affective disorder and phototherapy (pp. 64-79). New York: Guildford Press.

Thompson, C., & Cowan, A. (2001). The Seasonal Health Questionnaire: A preliminary validation of a new instrument to screen for seasonal affective disorder. *Journal of Affective Disorders, 64*(1), 89-98.

Velamoor, V. R. (1991). SAD chart revisited. *The Canadian Journal of Psychiatry, 36*(4), 310-311.

Volpe, F., Tavares, A., & Del Porto, J. (2008). Seasonality of three dimensions of mania: Psychosis, aggression and suicidality. *Journal of Affective Disorders, 108*, 95-100.

Wahl, T. (2001). Sustained attention and the effects of subsyndromal seasonal affective disorder and related depression. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 61*(12-B), 6389.

Wehr, T., Skwerer, R., Jacobsen, F., Sack, D., & Rosenthal, N. (1987). Eye versus skin phototherapy of seasonal affective disorder. *American Journal of Psychiatry, 144*, 753-757.

Wehr, T. (1989). Seasonal affective disorders: A historical overview. In N. E. Rosenthal, & M.C. Blehar (Eds.), *Seasonal affective disorders & phototherapy* (pp. 11-33). New York: The Guildford Press.

Wells, J. E., & Horwood, L.J. (2004). How accurate is recall of key symptoms of depression? A comparison of recall and longitudinal reports. *Psychological Medicine, 34*, 1001-1011.

Westrin, A., & Lam, R. (2007). Seasonal affective disorder: A clinical update. *Annals of Clinical Psychiatry, 19*, 239-246.

Westrin, A., & Lam, R. (2007a). Long-term and preventative treatment for seasonal affective disorder. *CNS Drugs, 21*, 901-909.

Whitcomb-Smith, S. (2004). The role of cognitive factors in the development of seasonal affective disorder episodes. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 65*(2-B), 1042.

Whitley, B. E. (2002). *Principles of research in behavioral science* (2nd ed.). Boston: McGraw Hill.

Wilkins, C., Sheline, Y., Roe, C., Birge, S., & Morris, J. (2006). Vitamin D deficiency is

associated with low mood and worse cognitive performance in older adults. *American Journal of Geriatric Psychiatry, 14*, 1032-1040.

Willett, J., Singer, J., & Martin, N. (1998). The design and analysis of longitudinal studies of development and psychopathology in context: Statistical models and methodological recommendations. *Development and Psychopathology, 10*, 395-426.

Wills, F., & Sanders, D. (1997). *Cognitive therapy: Transforming the image*. London: Sage

Wilson, J., & Read, J. (2001). What prevents GPs from using out side resources for women experiencing depression? A New Zealand study. *Family Practice, 18*, 84-86.

Winkler, D., Pjrek, E., Konstantinidis, A., Praschak-Rieder, N., Willeit, M., Stastny, J., & Kasper, S. (2006). Anger attacks in seasonal affective disorder. *International Journal of Neuropsychopharmacology, 9*, 215-219.

Woodson, H. (2004). Winter seasonal affective disorder: Epidemiological evidence for the light-deprivation hypothesis. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 64*(12-B), 6346.

World, Health Organisation (1994). *ICD-10 classification of mental and behavioral disorders*. Edinburgh: Churchill Livingston.

World Meteorological Organisation, (2008, 2010). *WMO Guide to meteorological instruments and methods of observation. Part one: Measurement of sunshine duration* (7th), World Meteorological Organisation: WMO. Retrieved from: http://www.wmo.int/pages/prog/www/IMOP/publications/CIMO-Guide/CIMO%20Guide%207th%20Edition,%202008/Part%20I/Chapter%208.pdf.

Wurtman, J., Wurtman, R., Berry, E., Gleason, R., Goldberg, H., McDermott, J., Kahne, M., & Tsay, R. (1993). Dexfenfluramine, fluoxetine, and weight loss among female carbohydrate cravers. *Neuropsychopharmacology, 9*, 201-210.

Yip, P., Tang, K., & Qin, P. (2006). Seasonality of suicides with and without psychiatric illness in Denmark. *Journal of Affective Disorders, 96*, 117-121.

Young, M. A., Watel, L.G., Lahmeyer, H.W., Eastman, C.I. (1991). The temporal onset

of individual symptoms in winter depression: Differentiating underlying mechanisms. *Journal of Affective Disorders, 22*, 191-197.

Young, M. A., Meaden, P.M., Fogg, L.F., Cherin, E.A., & Eastman, C.I. (1997). Which environmental variables are related to the onset of seasonal affective disorder? *Journal of Abnormal Psychology, 106*, 554-562.

Young, M. A. (1999). Integrating psychological and physiological mechanisms of SAD: The dual vulnerability model. *Biological Rhythms Bulletin, 1*(3), 4-6.

Zimmerman, M. (1994). *Interview guide for evaluating DSM-IV psychiatric disorders and the mental status examination*. East Greenwich: Psych Products Press.

Appendix A

Study to find out how to beat the blues

A team of specially trained Massey psychologists is offering free therapy to first-time depression sufferers in Auckland as part of a collaborative international study involving Harvard University and the London Institute of Psychiatry.

The University's Centre for Psychology will provide data from therapy sessions with volunteers, so that the team of international researchers can better understand the dynamics of Cognitive Behavioural Therapy (CBT), how it alleviates depression symptoms and how it equips sufferers to avoid repeat bouts of depression.

Findings from the study could offer hope for depression sufferers, many of whom do not have access to affordable, effective treatment, says Dr Nik Kazantzis, senior lecturer and practitioner who heads the team.

Depression sufferers typically experience low mood, poor appetite, lack of energy, disturbed sleep, feelings of helplessness and guilt. They may find decision-making difficult, feel miserable when they make even the smallest mistake and generally feel life has become overwhelming.

CBT teaches people how to become their own therapists by teaching them skills so they can deal better with difficult situations and the painful emotions they trigger, says Dr Kazantzis. Volunteers are being offered 20 hour-long individual sessions to learn strategies for changing problem thoughts and behaviours.

He says CBT is a widely used, mainstream therapy developed by American-born psychiatrist Dr Aaron Beck in the 1960s. Although it has been endorsed by more than 400 studies internationally as an effective, low-cost treatment for a range of disorders, including depression little is known about why it works.

Dr Kazantzis, who trained under Dr Beck two years ago, believes the therapy is particularly suited to New Zealanders as it offers immediate, practical help in coping with the present and does not necessarily require clients to embark on in-depth analysis of their pasts to be effective.

People can volunteer for the therapy if they have not been previously diagnosed with depression and are not taking medication that affects the brain.

About 121 million people world wide suffer from depression but fewer than 25 per cent have access to effective treatment, according to the World Health Organisation. Depression is the fourth-highest contributor to the global burden of disease, and is expected to become the second highest by 2020.

For more info about participating in the study call Nicole

Taken from: http://www.massey.ac.nz/massey/about-us/news/article.cfm?mnarticle=study-to-find-out-how-to-beat-the-blues-21-05-2008

Appendix B

Participant Information Sheet

Depression Study

You are invited to take part in a research study involving a brief psychological treatment for depression called Cognitive Behaviour Therapy (CBT). The purpose of the study is to examine certain processes of therapy which may increase its positive benefits. The study will involve 70 individuals between the age of 18 and 65 years, recruited within the greater Auckland area. Like yourself, these individuals will currently be experiencing a major depressive episode for the first time. Before you consent to be part of this study, please read the following. Ask as many questions as you need to be sure that you understand what taking part will involve. The decision to take part is entirely your choice.

If you provide written consent to be involved, you will receive a comprehensive psychological assessment, then a 20 session protocol of CBT for depression over a 16 week period. Treatment will be individualised based on your specific needs and goals, and provided by advanced clinical psychology trainees under close supervision. Consistent with prior research on CBT for depression, sessions will be scheduled twice a week for the first 4 weeks and then weekly for the next 12 weeks. Follow-up sessions will occur at 2 months and 6 months after treatment has ended. Participants will be asked to complete some assessment questionnaires to determine treatment gains, and also asked to provide informal feedback on the CBT they received. Your total time commitment (assessment, therapy sessions, questionnaires, and follow-up) is estimated to be about 30 hours, plus travel to and from the Centre for Psychology. Therapy will be provided by clinical psychology doctoral/masters students trained in delivering this protocol.

How will the study benefit you? It is expected that new information, which may benefit you or others, will be obtained by this study. Furthermore, it is very likely that the comprehensive psychological assessment and therapy offered as part of this study will improve your condition, although this cannot be guaranteed. These services will be provided free of charge. Due to funding limitations, you will be responsible for your own travel costs to and from the Centre for Psychology in Albany. Parking will be provided free of charge.

Who is unable to take part? Participants will need to be proficient in reading, writing, and conversing in English. They must be free from taking drugs which act on the central

nervous system. They must not meet diagnostic criteria for substance abuse, psychosis, or borderline personality disorder. Lastly, they must be able to be managed safely with outpatient psychotherapy.

If you do agree to take part, you are free to withdraw from the study at any time without having to give a reason. This will in no way affect your continuing health care, as you will be referred to an appropriate provider to further assist your specific needs. Participation in this study will be stopped should any harmful effects appear or if an appropriate medical professional feels it is not in your best interest to continue. You may be taken out of the study if you need treatment that is not allowed during this study, or if the study is cancelled. You will be asked to check with your study therapist before taking any other treatment; this includes anything from the supermarket, pharmacy or health shop.

Will my information remain confidential? Participating in this study will involve having your therapy sessions videotaped (and transferred to DVD discs) in order for the researchers to monitor the therapy protocol. All information collected about you during the study, including the recorded sessions, will be kept strictly confidential and only accessed by those researchers and clinical supervisors directly involved in the study. The only time in which confidentiality is breached is in the event that you express an intention to harm either yourself or somebody else, in which case a crisis team would become involved. No material which could personally identify you will be used in any reports on this study. All assessment information and clinical notes will be kept in individual files stored in a locked clinical records room, with files coded with anonymous identification numbers. Files will be stored in a separate location from both the identifying information and the DVD archive.

The information collected will be used for the research project and for publication in academic journals. All participants will be offered a summary of the findings at the conclusion of the study. This will include details of any publication arrangements that have been made. Please note that there is likely to be a delay between data collection and publication.

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover; generally this will affect your right to sue the investigators. If you have any questions about ACC, contact your nearest ACC office or the investigator.

If at any time you have questions or concerns about this study, you are welcome to contact: Dr. Nikolaos Kazantzis (who now has an academic office at La Trobe University), phone: Auckland (09) 8898292, or email: N.Kazantzis@latrobe.edu.au

If you have any questions about any issues pertaining to Maori in this study, regardless of your own ethnicity, you are welcome to contact Kaumatua koro Turoa, via the School of Psychology, phone Auckland (09) 414 0800 extension 2040.

If you have any queries or concerns regarding your rights as a participant in this research study, you can contact an independent Health and Disability Advocate. This is a free service provided under the Health & Disability Commissioner Act:

Telephone (NZ wide): 0800 555 050
Free Fax (NZ wide): 0800 2787 7678 (0800 2 SUPPORT)
Email: advocacy@hdc.org.nz
This study has received ethical approval from the Northern X Regional Ethics Committee.

Massey University
COLLEGE OF HUMANITIES AND SOCIAL SCIENCES
Te Kura Pūkenga Tangata

Consent Form
Depression Study

This consent form will be held for a period of five (5) years

• I have read and I understand the Information Sheet dated 20 February, 2009, for volunteers taking part in the Depression Study

• I have had the details of the study explained to me.

• I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study.

• My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

• I have been given contact details to use in case I have future questions about the study.

• I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time.

• I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.

• I agree to my sessions in this study being videotaped.

• I understand that I will not receive any compensation for travel costs or for the time I spend as a participant in this study.

• I have had adequate time to consider whether or not to take part in this study. I agree to participate in this study under the conditions set out in the Information Sheet.

Signature:_____ **Date:**_____

Full Name – printed_____

Appendix C

List of Hypotheses

Hypothesis 1: More people will present in the winter months (May- August) than over the rest of the year.

Hypothesis 2: People who present in the winter period (May – August) will exhibit higher depressive and vegetative symptom scores than those who present over the rest of the year.

Hypothesis 3: Vegetative symptoms will be positively associated with depression scores

Hypothesis 4: Irritability scores will be positively associated with vegetative symptom scores

Hypothesis 5: The age of persons presenting in the winter months will be lower than those applying at other times of the year.

Hypothesis 6: Age at presentation will be negatively correlated with: a) vegetative symptoms; b) depression scores and; c) irritability.

Hypothesis 7: Higher numbers of women will present in the winter months compared to men.

Hypothesis 8: Women will have higher vegetative and depressive scores compared to men at presentation and on days with lower temperatures

Hypothesis 9: Higher numbers of men will present during days of lower sunshine hours compared to women

Hypothesis 10: Higher numbers of women will indicate increased appetite as a symptom compared to men

Hypothesis 11: Men will have higher irritability scores compared to women

Hypothesis 12: Hours of bright sunshine will be negatively correlated with a) depression scores and b) vegetative scores.

Specific multilevel modeling hypotheses for Study 3:

Hypothesis 13: BDI-II scores will show higher rates of change than vegetative symptom scores over time

Hypothesis 14: Higher vegetative symptom expression will be associated with lower sunshine hours over time.

Hypothesis 15: Sunshine hours will show a significant effect on rate of change in depressive and vegetative symptom scores.

Hypothesis 16: Temperature will show a significant effect on rate of change in depressive and vegetative symptom scores.

Appendix D

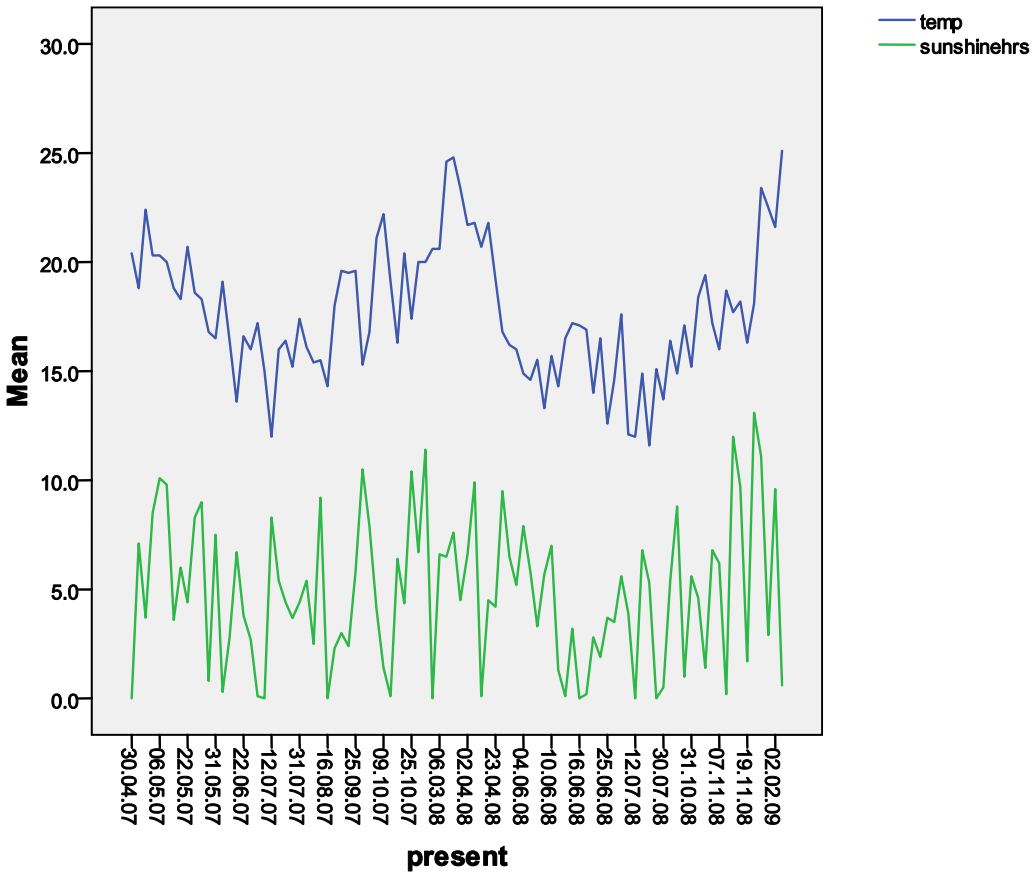

Figure D1. Graphical representation of recorded bright sunshine hours and daily temperature readings for session days over the course of the study.

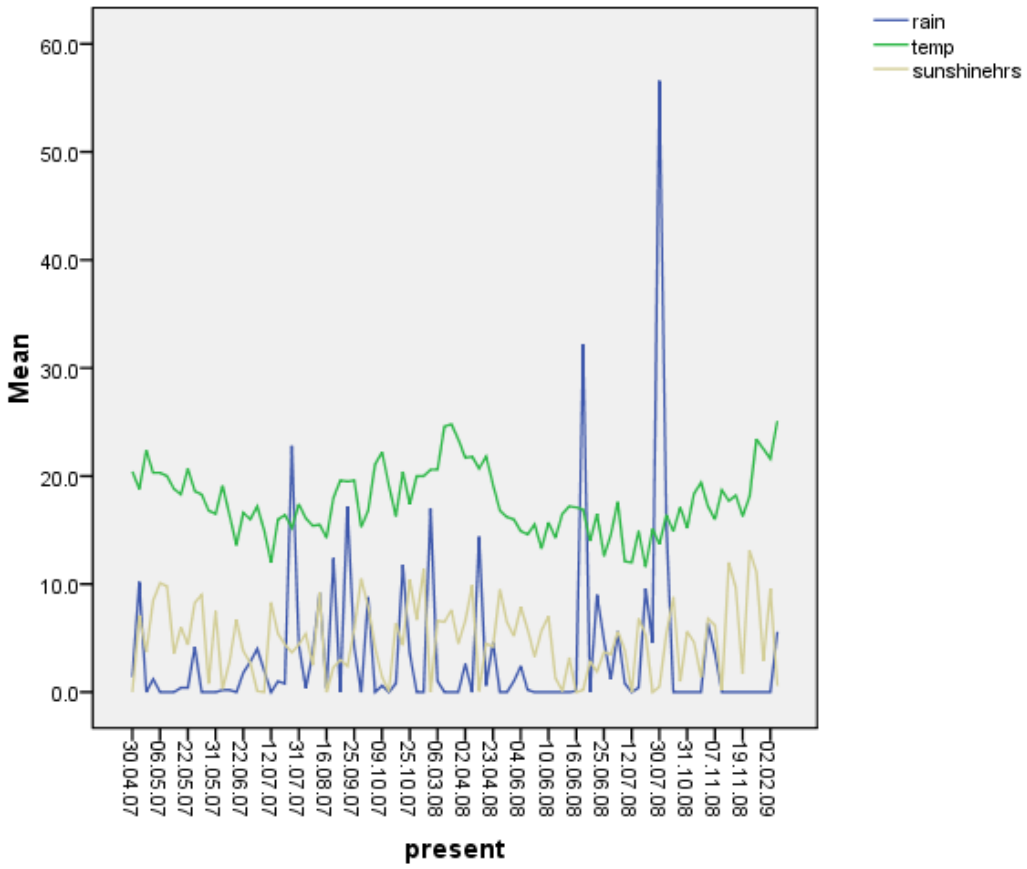

Figure D2. Graphical representation of recorded bright sunshine hours, daily low temperature and rainfall readings for session days over the course of the study.

Appendix E

COGNITIVE BEHAVIOR THERAPY HOMEWORK PROJECT

PHONE SCREENING INTERVIEW PROTOCOL

Introduction

When person answers say something like:

Hello, may I speak to Jane Smith (who presumably has left a message somewhere saying she is interested in taking part).

If yes, then say, *Jane* or *Ms Smith* (depending on age) *this is Mary Brown, I'm a researcher from Massey University. You left a message on (*say when or can't remember say recently) *about possibly taking part in our research study. Is this a convenient time to talk about it?*

If yes then listen to what they spontaneously say to be polite and if they don't say *the easiest way to start is by just outlining again the what the study is about and if you're still ok with it, I'll ask you some questions to help work out whether you fit within the group of people we are looking for this time.*

If no, then arrange a more convenient time to call back if they are still interested

So, basically what we are wanting to do is work out how treatment for depression, in particular Cognitive Behavioural Therapy can be improved to help people with depression. It is already a proven treatment, but we think there are ways that it could be improved. We are looking for 70 people between the age of 18-65 years from the greater Auckland area that are experiencing an episode of major depression for the first time in their lives. People can only be included in this study if they meet a certain set of criteria, therefore I will have to ask you several questions, this may take 20-30 minutes of your time.

But before going any further, you should know that every you say will be confidential. But there are two exceptions to this – and that is if I think there is any chance that you may cause harm to yourself. In that case I have break confidentiality for the sake of yours and others safety.

OK?

If yes, then proceed with, *right, then let's start with the questions*

If not ok, then listen to reservations, reassure and proceed or otherwise terminate.

Initial Questions

a) *What is your DOB? How old are you?* – needs to be between ages of 18 and 65 but since age already discussed at outset, won't be too many problems with this.

b) *Can you read, English OK? Write? Hold a conversation?*

c) *Are you currently taking any medication prescribed by your doctor?* If yes, *What is it*? This could include the contraceptive pill or sleeping medication. Google. Exclude occasional hypnotic and oral contraception.

d) *What help are you getting if any for your depression*?

e) *Is this the first time you have felt like this?*

I now have to ask you some questions about the way you have been feeling lately, the reason I am asking these questions is to assess whether you would benefit from participating in the study. We ask these questions so we are sure that the people participating in this study are likely to benefit from the treatment we provide.

DSM-IV-TR MAJOR DEPRESSION

At least 5 of the following symptoms have been present during the same two week period and represent a change from previous functioning; at least one of the symptoms is depressed mood or loss of interest.

1. Do you have a depressed mood (feel "sad", "down", "angry" or "empty") most of the day, every day. How is your mood?
   - How long have you been feeling…..
   - Do you feel that way nearly every day?
   - How much of the day does it last?
   - How bad is the feeling?
   - SUD – how would you rate this feeling on a scale of 0-100 (0 being not at all sad and 100 being the most sad you have ever been)?

   Criteria – depressed mood most of the day, nearly everyday, as indicated by subjective report or observation by others

2. Have you lost interest in or do you get less pleasure from the things that you used to enjoy?
   a. What do you normally enjoy doing? (TV, reading, sports, shopping, socialising, eating, hobbies?)
   b. What do you still enjoy?
   c. What have you lost interest in?
   d. For how long have you not enjoyed these things like you used to?
   e. Is it like that nearly everyday?

   Criteria – Markedly diminished interest or pleasure in all or almost all activities most of the day nearly everyday

3. Have there been any changes in your appetite for food?
   a. Increased? Decreased?
   b. How much more/less have you been eating?
   c. Is it like that nearly everyday?
   d. For how long has it been this way?
   e. Have you gained or lost any weight? How much? Since when?

   Criteria - Significant weight loss/gain when not dieting (change of more than 5% in month) or a decrease or increase in appetite nearly every day.

4. How has your sleeping been?
   a. How many hours per night have you been sleeping?
   b. How does this compare to normal?
   c. Increased? Decreased?
      i. Is it a problem nearly everyday?
      ii. How long have you had these sleep problems?
      iii. If decreased – do you have any problems falling asleep, staying asleep, or waking up to early in the morning?

Criteria – insomnia or hypersomnia nearly everyday.

5. Listen for slowed speech, long pauses before answering questions or between words.
   a. Agitation: Have you been feeling more fidgety lately? Are you having problems sitting still?
      i. IF YES: Do you pace back and forth?
      ii. Have others noticed your restlessness
   b. Retardation: Have you felt slowed down, like you are moving in slow motion
      i. IF YES: have others noticed this?

   Criteria – psychomotor agitation or retardation nearly everyday (has to be observable by others)

6. How has your energy levels been?
   a. Have you been feeling tired or worn out?
   b. IF YES: Duration? (For how long have you…) Persistence? (Do you feel like this nearly everyday?)

   Criteria – Fatigue or loss of energy nearly everyday?

7. How have you been feeling about yourself?
   a. What has your self-esteem been like?
      i. IF LOW: What types of thoughts do you have about yourself?
      ii. Do you feel like you are worthless or a failure?
         1. If yes: Tell me about it
   b. Have you been blaming yourself for things?
      i. Like what?
   c. Do you feel guilty
      i. IF YES: what about?
      ii. How hard is it to get your mind off this?
      iii. Do you think about things from the past and feel guilty about them?
      iv. IF YES: Like what?
      v. Is the patient's guilt or worthlessness on the patients mind everyday?
      vi. What has your **self esteem** been like?
         1. Has there been any change?

   Criteria – feelings of worthlessness or excessive or inappropriate guilt nearly everyday (not merely self-reproach or guilt about feeling sick)

8. Have you been having problems thinking or concentrating?
   a. IF YES: what does this interfere with?
   b. Are you able to watch TV? Read? Follow a conversation?
   c. How long have you noticed this happening?
   d. Does it happen nearly everyday?

   Is it harder to make decisions than before?
   IF YES: What kind of decisions is harder to make?
   a. What about every day decisions?
   b. How long have you had this problem?

c. Does it happen nearly everyday?

Criteria – Diminished ability to think or concentrate, or indecisiveness, nearly everyday.

9. **SUICIDE SCREEN -** Sometimes when a person feels down or depressed they may think about dying, this is quite common. It is very typical and common for people to have thoughts about harming or killing themselves. Have you been having any thoughts like that?
   a. IF YES: Tell me about it. Have you thought about taking your life?
      i. IF YES: Did you think of ways to do it?
      ii. Do you currently have a plan?
      iii. Do you have the means to carry out this plan?
      iv. How close have you come to doing it?
      v. IF you have not attempted, why not? What stops you from doing it? What are the protective factors?
      vi. IF NO: Do you wish you were dead? Do you have thoughts of death or dying?
      vii. When you go to sleep do you often wish that you would not wake up?

Criteria – Recurrent thoughts of death, not just of dying, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

10. What difficulties in your life has the depression caused? OR: How have these difficulties affected your life?
    a. Does it bother you a lot that you feel this way?
    b. Has it caused problem in your job? Study? Relationships? Friends? Family? Social life? Doing household chores?

Criteria – The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

11. Do you have any medical conditions?
    a. YES:
       i. When did the conditions start?
       ii. Has there been any change to this condition? Lately?
12. Have you lost a loved one in the last two months? Exclude: unless – if associated with marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.
13. During this time have you been experiencing delusions or hallucinations? At the same time as depressive symptoms.
14. IF PSYCHOTIC
    a. Was there a time when you had the (hallucinations/delusions) but did not feel sad or depressed?

b. IF YES: How long did you have [psychotic symptoms] only?
c. When did the depression begin in relation to this?

15. Has there been a major event in your life lately that may have came before you felt like this? E.g. a death of a close relative or friend (bereavement)?

16. When you started experiencing these symptoms have you ever felt this way?
    a. Have there been times lasting at least a few days when you felt the opposite of depressed, that is when you were very cheerful or high and this felt different from your normal self?

    IF YES or UNCLEAR:

    1. Did you feel hyper, or like you were high on drugs, even though you had not taken anything?
    2. Did anything cause your good mood?
    3. How long did it last?
    4. So, was this more than just feeling good?
    5. When did this occur?
    6. How many periods like this have you had?

    IF NO:

    7. What about a period lasting at least a few days when you were unusually irritable, and quick to argue or fight?

    IF YES: Describe what that was like.

    i. Were you using drugs or alcohol?
    ii. Did you get into many arguments or fights?
    iii. How long did this period last?
    iv. Was there a reason you felt this way?
    v. When did it occur?
    vi. How many times have you felt this way?

If the clients report manic symptoms ask these questions:

- Did you find during this period you needed less sleep than usual?
- Did you notice you had racing thoughts and ideas?
- Were you easily distracted?
- Did you find you were overly occupied with pleasurable activities (or goal oriented)?
- Did you find during this period you were involved in a lot of risky and potentially self-damaging behaviour e.g. gambling, drinking, stealing, speeding?
- During this time did you feel more irritated than normal?
- During this time did you find it hard to relax?
- Did you find you felt like this constantly over a period of a week? (apply to all questions).

DRUG ABUSE/DEPENDENCE

Alcohol:

Pre-screen:

Frequency: How often do you have a drink containing alcohol?
Daily? Weekly?
How many drinks containing alcohol do you have on a typical day when drinking?
How often do you have six or more drinks in one occasion?

Now I am going to ask you some questions about your use of alcohol
What are your drinking habits like? – for example how much alcohol do you consume a night? How much would you consume in a week?
Was there ever a time in your life when you drank too much?
IF YES: How old were you?
Has anyone in your family said that you were an excessive drinker?
Have friends, a doctor, or anyone else ever said that you drank too much?
Has alcohol ever caused problems for you?
IF YES: What kind of problems?
How old were you when you had these problems?
If all questions are answered NO unlikely to meet diagnosis of Alcohol abuse

Street drugs:
Have you ever used street/recreational drugs?
*if used less that 10 times go to prescribed medicines
Did you ever think you used drugs too much?
IF YES: how old were you?

Has anyone in your family said that you use drugs too much?
Have friends, a doctor, or anyone else said that you use drugs too much?
Have drugs ever caused a problem for you?
IF YES: What kinds of problems?
How old were you when you had these problems?

Prescribed medication:

Have you ever used sleeping pills, tranquilisers, weight loss medicines or pain killers?
IF YES:
How long did you take the drug?
Did you get hooked or addicted to it?
Did you take much more than was prescribed?

IF all the above are answered NO, diagnosis of drug dependence unlikely

A1:

Because of drinking or taking drugs how often have you

- Missed work or school?
- Have trouble at work or school?
- Got fired etc?
- Not taken care of children?
- No cook, clean house, go grocery shopping?

A2:

Do you drive while intoxicated?

How often?

Did you ever drink (take drugs) and them do something that was potentially dangerous (e.g. operating machinery)

A3:

Were you ever arrested for driving under the influence, or disorderly conduct?

Were you ever busted for selling/buying drugs?

IF YES: How many times

Recurrent drug related legal problems

A4:

Because of your drug taking/drinking did you….

- frequently have problems or arguments with friends or family?
- Spend less time with family or friends?
- Get separated or divorced?
- Get into physical fights?
- Get violent?
- IF YES TO ANY: do you still drink/take drugs despite these problems?

**Have you taken any drugs/consumed any alcohol today? Do you plan to drive anywhere? (e.g. do your children need to be picked up etc – follow up.)

Drug Abuse = A maladaptive pattern of drug use leading to clinically significant impairment or distress, as manifested by one or more of the following occurring within a 12 month period.

Screen for psychosis: *Now I will ask you some questions about how you perceive or view the world around you.*

DELUSIONS AND HALLUCINATIONS

DELUSION OF REFERENCE

When watching TV, listening to the radio, or reading the paper do you notice that they are referring to you, or that there are special message intended for you?

- What have you noticed?

Does it seem like **strangers on the street are taking special notice** of you or talking to you? Is it a feeling you have, or **are you pretty sure** that they are talking about / referring to you? How do you know? Does it seem like things are especially arranged for you? In what way? **DELUSIONS OF PERSECUTION** Is anybody against you, following you, giving you a hard time, or trying to hurt you? Do you feel like there's a plot to hurt you? Who's involved? Why would they want to hurt you? **THOUGHT BROADCASTING** Do you ever think of something so strongly that people could hear your thoughts? So, people can hear what you are thinking even when you are not talking? How do you know? **DELUSIONS OF MIND READING** Are people able to read your mind and know what you're thinking? How can they do this? Do they literally read your thoughts, or do they read your facial expression to know what you're thinking? **THOUGHT WITHDRAWAL** Are your thoughts ever taken out of your head? Does someone or some force reach into your head and steal or remove your thoughts? **THOUGHT INSERTION** Are there ever thoughts in your head that have been put there from the outside? **DELUSION OF GUILT** Do you think you've done something so terrible and deserve to be punished?	

I know it will be hard to talk about, but what do you feel so guilty about?

Do you blame yourself for bad things going on in the world, like wars, crime, and starvation?

DELUSION OF GRANDIOSITY

What is your self-esteem like?

Do you feel more self-confident than usual?

Do you think you have special talents, abilities, or powers?

When some people feel (HIGH, EUPHORIC, etc) they may think they're going to become famous or do great things. Did you have any thoughts like that?

DELUSION OF CONTROL

Do you ever get the feeling that you're being controlled by some force or power from the outside?

At times, does it seem like you're not in control of your body, almost like you're a puppet and something from the outside pulls the strings?

So, at times your body does certain things without your willing it?

SOMATIC DELUSION

Are you concerned that you have a serious physical illness that a doctor hasn't found, or that something is wrong with your body?

HALLUCINATIONS

VISUAL HALLUCINATIONS

Have you seen visions or other things that other people didn't see?

What did you see?
What time of the day did this occur?

How long ago did it start?
Do you see it everyday?
How often do you see it?

AUDITORY HALLUCINATIONS

Have you heard noises, or sounds, or voices that other people didn't hear?

What did you hear?

Do the voices seem to come from inside or outside your head?

IF INSIDE: But you hear it with your ears? How many voices do you hear? Are they male or female? Do you recognize them? Do you ever hear two or more voices talking to each other? Do the voices ever talk about what you're doing or thinking? IF YES: Do they keep a running commentary on what you are doing or thinking just like a sports commentator? How long ago did the voices start? Do you hear them everyday? How often during the day do you hear them? Do they influence your behaviour? Do they tell you to do things? **TACTILE HALLUCINATIONS** Do you ever notice strange sensations in your body or on your skin? Do you ever feel something creeping or crawling on your body, or something push or punch you but no one is there? IF YES: Like what? When did it happen for the first time? How often has it happened? **OLFACTORY AND GUSTATORY HALLUCINATIONS** What about smells that other people don't notice, or strange tastes in your mouth? IF YES: Like what? When did it happen for the first time? How often has it happened? Are they associated with any other physical symptoms like an upset stomach, numbness, tingling, or brief memory loss? Tell me about that?	

Screen for Borderline PD

I am now going to ask you some questions about your relationships with other people. I will specifically ask you questions about your feelings in your relationships

- **Inclusion = at least 5 criteria**

- **Pervasive pattern of instability of interpersonal relationships, self-image, and marked impulsivity beginning at early adulthood and present in a variety of contexts:**

1) Have there been times when you've been very upset, almost to the point of being distraught, because you thought someone you loved or needed, might leave you?
a. How often has this happened?
b. What did you do to stop them from leaving?

Do you spend a lot of time thinking of ways to keep people from leaving you?

Frantic efforts to avoid real or imagined abandonment (do not include self mutilating behaviour.

2) Do your relationships with friends and lovers tend to be intense and stormy with lots of ups and downs?
a. IF YES: can you tell me about some of them
With some people do you switch from loving, respecting, and admiring them at one time, to despising them at another time?
If YES: Tell me about that

A pattern of unstable and intense interpersonal relationships characterised by alternating between extremes of idealisation and devaluation.

3) Does the way you think about yourself change so often that you don't know who you are?
a. IF YES: tell me about this
Do you ever feel like you are something else, or that you're evil, or maybe that you don't even exist? Tell me about that.

Identity disturbance: markedly and persistently unstable self-image or sense of self.

4) I am going to read you a list of behaviours that sometimes causes problems for people. How many times in the last 5 years have you:
a. Gambled more money than you could afford to lose
b. Spent money on things you could not afford
c. Been high on drugs
d. One night stands/ sexual affairs
e. Intoxicated by alcohol
f. Charged with reckless, driving, speeding ticket etc
g. Driving while intoxicated or high

h. Gone on eating binges **i.** Done anything impulsive where you could have gotten hurt *Impulsivity in at least two areas that are potentially self-damaging (do not include suicidal or self-mutilating behaviour.* 5) Have you ever been so upset that you told someone that you wanted to kill or hurt yourself? a. IF YES: Tell me about it b. How often have you done this? Have you ever made a suicide attempt, even one that wasn't very serious? IF YES: What did you do? How many attempts have you made? Have you ever been so upset or tense that you deliberately hurt yourself by cutting your skin, putting your hand through a glass window, burning yourself or anything like this? IF YES: What have you done? How often? *Recurrent suicidal behaviour, gestures or threats, or self-mutilating behaviour.* 6) Has anyone ever told you that you are irritable *(touchy, short-tempered, cross, ill-tempered)* or that your moods seem to change lots? a. IF YES: tell me about it Do you often have days when your mood is constantly changing—days when you shift back and forth from feeling your usual self, to feeling angry or depressed or anxious? IF present: Are the mood swings mild or very intense? How often does this happen in a typical week? How long do the moods last? *Affective instability due to marked reactivity of mood – lasting from a few hours and only rarely a few days.* 7) Do you feel empty most of the time? a. IF YES: What percent of the time do you feel that way? *Chronic feelings of emptiness* 8) How easily do you lose you temper? How often do you lose your temper? Do you feel angry much of the time? What kinds of things get you really angry? Are you sometimes angry with knowing why you feel that way? Tell me what you are like when you are angry. How long do you usually stay angry? Do you ever throw or break things? Have you ever hit anyone? Do you get into physical fights? IF YES: tell me about it. When you are angry do you ever give someone the silent treatment?	

IF yes: how long can you keep it up? Is that a common reaction for you? *Inappropriate, intense anger or difficulty controlling anger* 9) When some people are under stress, they have experiences that are very hard to explain to other people. Have you ever felt like things around you were somehow strange, or changed in size or shape? IF YES: describe what that is like. When you've been under stress, have you ever felt your body of part of it was somehow changed or not real? Have you ever felt you were watching yourself from outside your body? IF YES: describe what that was like? Do you ever have brief blackouts and forget what has happened? When you are feeling stressed, do you ever get paranoid or suspicious of people you usually trust? IF NO: what about being afraid that someone is spying on you or trying to hurt you? IF YES: Does this happen even when you are stressed? IF YES to any of above - were you using any drugs or alcohol when these experiences happened? - IF YES: does this only happen when you are taking drugs and alcohol? - IF NOT: How long do the experiences last? - DO they go away when you are not under stress? *Transient, stress related paranoid ideation or severe dissociative symptoms.* **Acceptance and Referral** ACCEPTANCE *a) We would like to offer you the opportunity to be involved as a participant in this study.* *b) We are optimistic about this therapy, there has been significant scientific support to demonstrate CBT as an effective and beneficial first line treatment for first episode depression.* *c) Due to limited resources in this study it is important for you to keep in mind that this study is based in western/Pakeha culture. We do not offer a culturally specific service. However, if you identify as Maori and you are concerned, we will have a Kaumatua available for consultation during the therapy process – he is available to consult us on any cultural issues that may arise during the process.* - Would you prefer a culturally specific service? Yes? Refer to WDHB.	

Please note that if a participant identifies culturally-related concerns during the course of their participation in assessment or treatment, clinical supervisors should be consulted in the first instance. The School of Psychology Kaumatua koro Turoa will also be available for consultation regarding any cultural issues that may arise in the process of this study. Turoa may be contacted in emergencies at 027-2888-135, and less urgent enquiries should be directed through Robyn Knuth, Secretary to the Head of School, Turitea campus. Set up time for interview and meeting with therapist… REFFERAL Make sure safety checks have been carried out. (Suicide screen, present use of alcohol drugs) – contacted acute services if needed. Make a referral to the best alternative mental health service. Provide contact details. Contact referral source and contact study co-coordinator.	

Appendix F

NAME: _____

ID NUMBER: _____

DEPRESSION STUDY
Personal Data Form

1. **Age** years

2. **Gender** *(tick one)* Female ☐ Male ☐

3. **Marital Status** *(tick one)* Never Married ☐ Divorced ☐ Married ☐ Widow/ Widower ☐

4. **Ethnicity** *(tick one)* Asian ☐ European/ Caucasian ☐ Maori ☐ Pacific Islander ☐ Other ☐

5. **Occupation** _____

6. **Education** *(tick one)* Some primary school ☐ Completed primary school ☐ Some high school ☐ Completed high school ☐ Technical training beyond high school ☐ Some university ☐ Graduated from university ☐

7. **Have you received any mental health treatment in the past?**

(psychotherapy, counselling, or medication, such as prozac)

Yes No

If yes, what kind of treatment and for approximately how long?

Type of treatment:	How long (approx.)?	Months
Type of treatment:	How long (approx.)?	Months
Type of treatment:	How long (approx.)?	Months
Type of treatment:	How long (approx.)?	Months

DEPRESSION STUDY

Personal Data Form

Part II: Extra Information

Marital Status

At beginning of therapy (Circle one)

Single
Dating
De Facto
Married
Divorced
Widow / Widower

Occupation

- At beginning of therapy you told us your occupation at that time. What was it?

_____

- Were you actively engaged in that occupation at the time or was this a past occupation? (Circle one)

Current
Past

- If it was current, were you working: (Circle one)

Full time (35+ hours per week)
Part time

Family

Do you have Children?

Yes
No

If yes, how many children do you have?

_____

Appendix G

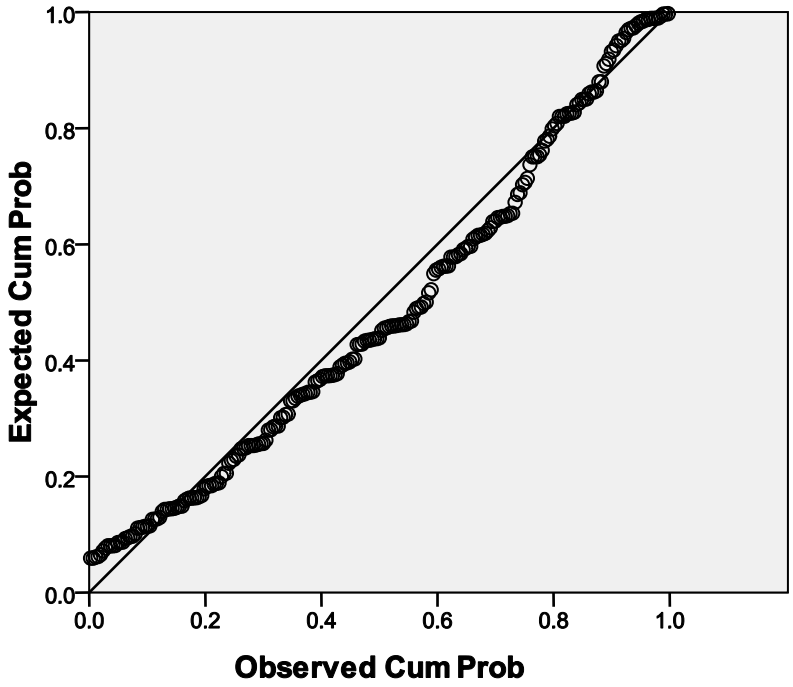

Figure G1. Normal P-Plot standardised residual plot for daily bright sunshine hours recorded on session days over the course of the study.

Note. The analyses depicted in this figure, and in all other figures in this appendix, involved regression analyses against BDI-II scores as the dependent variable.

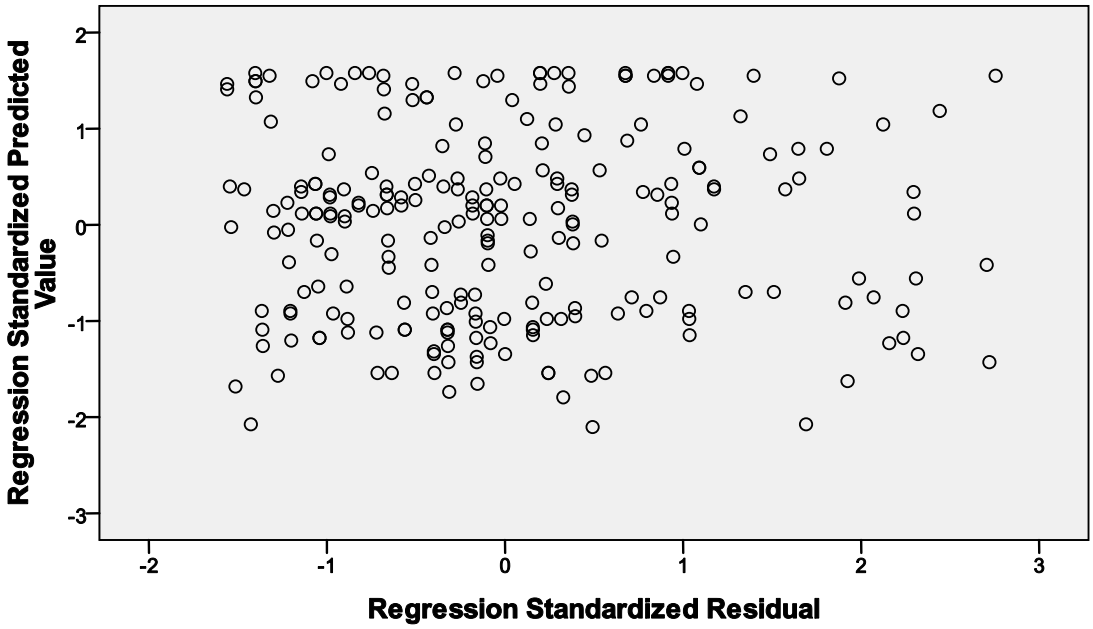

Figure G2. Standardised residual scatterplot for daily bright sunshine hours recorded on session days over the course of the study

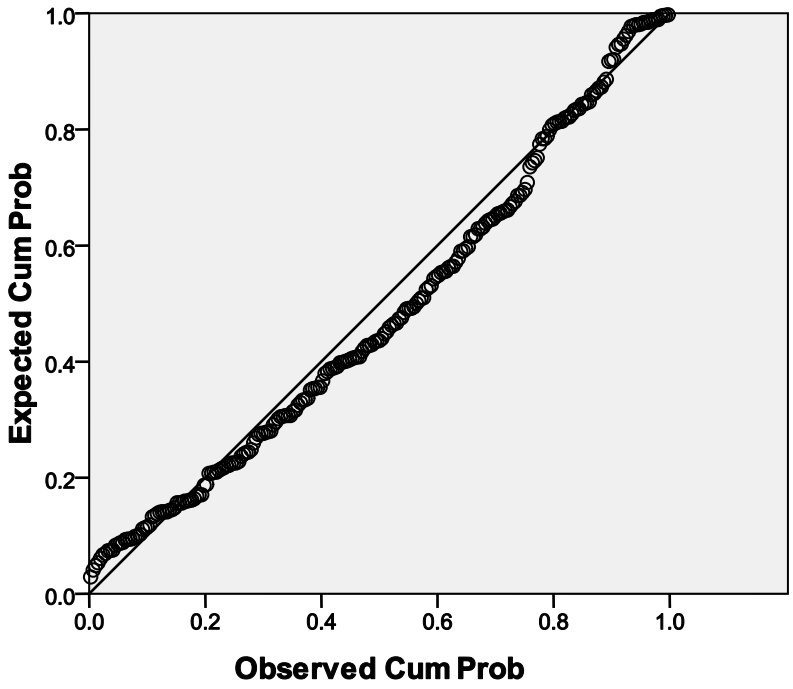

Figure G3. Normal P-Plot standardised residual plot for daily low temperatures recorded on session days over the course of the study.

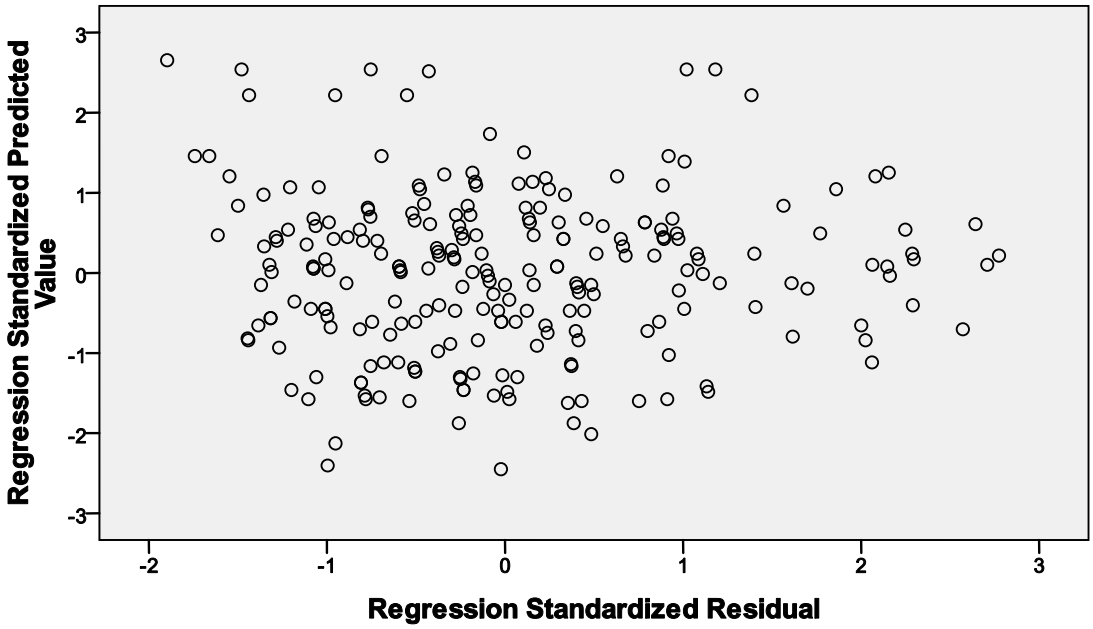

Figure G4. Standardised residual scatterplot for daily low temperatures recorded on session days over the course of the study

Appendix H

Table H1

Reliability Analysis for BDI-II Across all Sessions.

Session	*N*	α	*M*	*SD*
0	28	0.91	32.69	12.28
1	28	0.93	29.69	13.95
2	28	0.93	24.89	11.12
3	28	0.95	22.1	12.04
4	28	0.95	24.11	11.98
5	28	0.96	21.30	13.40
6	28	0.94	19.14	11.13
7	27	0.96	19.33	11.84
8	27	0.95	18.60	12.0
9	27	0.94	18.50	11.80
10	26	0.94	17.42	11.93
11	26	0.94	16.61	10.85
12	25	0.94	18.92	10.97
13	25	0.93	15.73	10.50
14	23	0.94	15.65	10.71
15	22	0.94	15.82	11.09
16	22	0.93	15.30	10.72
17	22	0.91	14.97	9.58
18	21	0.91	14.33	8.77
19	20	0.94	12.65	10.68
20	19	0.92	10.89	8.62
2 month	15	0.95	10.20	8.84
6 month	6	0.92	4.83	8.64

Appendix I

Table I1

Correlations between BDI-II Scores, Age and the Number of Sessions Attended

	Initial BDI-II	age	sessions	Mid BDI-II	End BDI-II
Initial BDI-II		-.005	.070	**.589****	**.405***
age			-.257	- .248	-.314
sessions				.266	-.005
Mid BDI-II					**.772****
M	30.68	45.11	8.36	16.89	9.32
SD	10.548	11.555	1.062	11.695	8.092

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

Appendix J

```
title "Model A".
mixed BDI
  /print=solution
  /method=ml
  /fixed=intercept
  /random intercept | subject(client).
title "Model B".
mixed BDI with session
  /print=solution
  /method=ml
  /fixed = time
  /random intercept time | subject(client) covtype(un).
title "Model C".
mixed BDI with sun session
  /print=solution
  /method=ml
  /fixed = sun session sun*session
  /random intercept session | subject(client) covtype(un).
title "Model D".
mixed BDI with age sun session
  /print=solution
  /method=ml
  /fixed = age sun session age*session sun*session
  /random intercept time | subject(client) covtype(un).
```

Note. These variables shown are the ones used in the final "depression" model. The variables were dependent on the model fitted, for example with the vegetative symptoms model BDI was not used.

Appendix K

Table K1

The Uncontrolled Effects of Bright Sunshine Hours and Daily Low Temperatures in Model C

		Parameter	A	B	Temperature	Sunshine
Fixed Effects						
Initial status						
π_{0i}	Intercept	γ_{oo}	19.11***	27.50***	29.63***	27.14***
			(1.68)	(2.07)	(2.62)	(2.26)
		γ_{01}			-0.21	0.06
					(0.16)	(0.17)
Rate of Change						
	Intercept	γ_{10}		- 0.95***	-1.08***	-1.02***
π_{1i}	session			(0.097)	(0.16)	(0.12)
	Temperature	γ_{11}			0.01	0.01
					(0.01)	(0.01)
Variance Components						
Level 1	Within person	σ^2_e	83.02	24.55	24.46	24.19
	resid est		(8.15)	(2.62)	(2.62)	(2.58)
Level 2	In initial status	σ^2_0	69.37	111.55	109.68	112.54
			(21.1)	(31.89)	(31.42)	(32.14)
	In rate of change	σ^2_1		0.22	0.18	0.19
				(0.09)	(0.07)	(0.07)
	covariance	σ_e		-2.38	-2.32	-2.43
				(1.16)	(1.14)	(1.16)
Pseudo R^2 statistics						
	R^2_ε			.70	.70	.70
	R^2_0				.017	#
	R^2_1				.18	.22
Goodness of Fit						
	Deviance		1763.67	1551.79	1550.91	1548.36
	AIC		1769.46	1563.79	1566.09	1564.36
	BIC		1779.85	1584.55	1593.76	1592.03

***p<0.001, **p<.05, *p<.10

Note. # indicated negative statistics found. The implications of this are discussed in Study 3.

Table K2

Alternative Versions of Model C using Age and Gender and Season of Presentation

		Parameter	*A*	*B*	*Gender*	*Age*	*Season*
	Fixed Effects						
Initial status	Intercept	γ_{oo}	19.11***	27.50***	29.34***	33.38***	40.45***
π_{0i}				(1.68)	(2.07)	(2.51)	(11.45)
	Factor	γ_{01}			-5.27	-0.13	-4.16
					(4.2)	(0.18)	(3.63)
Rate of Change	Intercept	γ_{10}		- 0.95***	-0.97**	-0.56	-1.29*
π_{1i}	session			(0.097)	(0.12)	(0.38)	(0.55)
	Factor	γ_{11}			0.05	-0.01	0.11
					(0.2)	(0.01)	(0.18)
	Variance Components						
Level 1	Within person	σ^2_e	83.02	24.55	24.34	24.45	24.56
			(8.15)	(2.62)	(2.62)	(2.61)	(2.62)
Level 2	Initial status	σ^2_0	69.37	111.55	105.14	109.11	106.19
			(21.1)	(31.89)	(30.16)	(31.22)	(30.45)
	Rate of change	σ^2_1		0.22	0.19	0.18	0.19
				(0.09)	(0.07)	(0.07)	(0.07)
	covariance	σ_e		-2.38	-2.32	-2.5	-2.24
				(1.16)	(1.13)	(1.15)	(1.12)
	Pseudo R^2 statistics						
	R^2_ε			.70	.70	.70	.70
	R^2_0				.05	.02	.04
	R^2_1				.14	.18	.14
Goodness of Fit	*Deviance*		1763.67	1551.79	1550.03	1548.24	1550.50
	AIC		1769.46	1563.79	1566.03	1564.24	1566.50
	BIC		1779.85	1584.55	1593.71	1591.92	1594.18

***p<0.001, **p<.05, *p<.10

Table K3

Alternative Versions of Model D using Gender and Age as Control Variables for Sunshine Hours

Level	Variable	*A*	*B*	*C*	*gender*	*age*
	Fixed Effects					
Initial status	Intercept	19.11***	27.50***	27.14***	29.08***	33.38**
π_{0i}		(1.68)	(2.07)	(2.26)	(2.66)	(8.42)
	Sunshine			0.06	0.07	0.07
				(0.17)	(0.17)	(0.17)
	Factor				-5.47	-0.14
					(4.21)	(0.18)
Rate of Change	Intercept		- 0.95***	-1.02***	-1.05***	-0.67*
π_{1i}	session		(0.097)	(0.12)	(0.15)	(0.39)
	Sunshine			0.01	0.01	0.01
				(0.01)	(0.01)	(0.01)
	Variance Components					
Level 1	Within person	83.02	24.55	24.19	24.18	24.08
	resid est	(8.15)	(2.62)	(2.58)	(2.58)	(2.57)
Level 2	Initial status	69.37	111.55	112.54	105.68	109.72
		(21.1)	(31.89)	(32.14)	(30.29)	(31.36)
	Rate of		0.22	0.19	0.18	0.18
	change		(0.09)	(0.07)	(0.07)	(0.07)
	covariance		-2.38	-2.43	-2.33	-2.55
			(1.16)	(1.16)	(1.12)	(1.15)
	Pseudo R^2 statistics					
	R^2_ε		.70	.70	.70	.70
	R^2_0			#	.05	.02
	R^2_1			.22	.18	.18
	Goodness of Fit					
	Deviance	1763.67	1551.79	1548.36	1546.98	1544.91
AIC		1769.46	1563.79	1564.36	1566.59	1564.91
BIC		1779.85	1584.55	1592.03	1601.19	1599.5

****p< .001, ***p < .05, *p <.10*

Appendix L

Table L1

Alternative Variables for use in Model C of Vegetative Symptom Model

.

Level	Variable	Parameter	A	B	Sunshine	Temperature
	Fixed Effects					
Initial status π_{0i}	Intercept	γ_{oo}	1.22*** (0.12)	1.95*** (0.22)	2.32*** (0.3)	1.54*** (0.77)
	Factor	γ_{01}			-0.08* (0.04)	0.02 (0.05)
Rate of π_{1i}	Intercept	γ_{10}		-0.07** (0.02)	-0.11** (0.03)	-0.06 (0.06)
		γ_{11}			0.008 (0.003)	-0.0004 (0.003)
	Variance Components					
Level 1	Within person	σ^2_e	0.44 (0.04)	0.36 (0.04)	0.35 (0.04)	0.35 (0.04)
Level 2	Initial status	σ^2_0		0.57 (0.38)	0.53 (0.37)	0.68 (0.41)
	Rate of change	σ^2_1		0.004 (0.003)	0.004 (0.003)	0.005 (0.003)
	covariance	σ_e		-0.03 (0.03)	-0.03 (0.03)	-0.04 (0.04)
	Pseudo R^2 and Goodness of Fit Statistics					
	R^2_ε			.18	.18	.18
	R^2_0				.07	#
	R^2_1				0	0
	Deviance		530.59	507.81	501.05	504.76
	AIC		536.59	519.81	517.05	520.76
	BIC		546.93	540.57	544.72	548.43

****p< .001, ***p < .05, *p <.10*

Table L2

Alternative Variations for Model D using Gender and Age to Control for the Effects of Sunshine on Change in Vegetative Symptoms.

Level	Variable	Parameter	A	B	C	Gender	Age
	Fixed Effects						
Initial status π_{0i}	Intercept	γ_{oo}	1.22*** (0.12)	1.95*** (0.22)	2.32*** (0.3)	2.66*** (0.32)	2.52** (0.89)
	Sunshine	γ_{01}			-0.08* (0.04)	-0.09** (0.04)	-0.07* (0.04)
	Factor	γ_{02}				-0.92** (0.43)	0.004 (0.02)
Rate of π_{1i}	Intercept	γ_{10}		-0.07** (0.02)	-0.11** (0.03)	-0.13*** (0.03)	0.07 (0.08)
	Sunshine	γ_{11}			0.008 (0.003)	0.01** (0.003)	0.008** (0.003)
	Factor	γ_{12}				0.06 (0.04)	-0.00 (0.002)
	Variance Components						
Level 1	Within person	σ^2_e	0.44 (0.04)	0.36 (0.04)	0.35 (0.04)	0.35 (0.04)	0.35 (0.04)
Level 2	Initial status	σ^2_0		0.57 (0.38)	0.53 (0.37)	0.34 (0.31)	0.54 (0.37)
	Rate of change	σ^2_1		0.004 (0.003)	0.004 (0.003)	0.004 (0.003)	0.004 (0.003)
	covariance	σ_e		-0.03 (0.03)	-0.03 (0.03)	-0.02 (0.02)	-0.03 (0.03)
	Pseudo R^2 and Goodness of Fit Statistics						
	R^2_ε			.18	.18	.18	.18
	R^2_0				.07	.40	.07
	R^2_1				0	0	0
	Deviance		530.59	507.81	501.05	496.41	499.37
	AIC		536.59	519.81	517.05	516.41	519.37
	BIC		546.93	540.57	544.72	551.01	553.97

****p< .001, ***p < .05, *p <.10*