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THE EFFECT OF BRIGHT LIGHT VERSUS DIM LIGHT IN SEASONAL AFFECTIVE DISORDERS

by

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ABSTRACT

Seasonal affective disorders (SADS) is characterized by regularly occurring winter depression alternating with summer mental health. This study examines the effect of high intensity light (10,000 lux) versus low intensity light (500 lux) therapy on depression in SADS patients aged 33 to 54 years, as measured by the Hamilton Rating Scale for Depression-Seasonal Affective Disorder (SIGH-SADS).

Initially, 15 males and 28 females were referred to a research program for evaluation and treatment of winter depression. They were interviewed and the SIGH-SADS test for depression was administered. Of these patients, only 11 males and 25 females completed the study. The seven other patients dropped out of the study for a number of reasons. Patients were informed that light is known to alter the depressive mood; however, the intensity and duration were variables that needed to be examined. The patients were knowledgeable about the fact that they would be self-administering the two different intensities for 30 minutes every morning for one week of each light intensity. Subsequent to the treatment, 7 males and 10 females showed a 50% or greater improvement from the pretest SIGH-SADS to the post 10,000 lux SIGH SADS in the 29 items. The literature shows that 60% of the patients diagnosed clinically with SADS respond to light, whereas 40% do not respond.

The general hypothesis of this thesis is that there is a significant improvement ($p < .05$) in the mood of patients after they receive 10,000 lux light therapy, as measured by the SIGH-SADS.

Findings indicated that patients showed less depression on the SIGH SADS scale after they had received 10,000 lux as compared to post 500 lux. Since approximately four percent of the general population suffers from SADS, this study has implications for the non-medication treatment of a very large population of patients.

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CHAPTER I
INTRODUCTION

My interest in mood disorders that occur during different seasons was sparked by an Edmonton psychiatrist. He presented the topic to me and suggested that additional work needed to be completed. During the progress in my work, I realized that light had many other effects on the human body. As a manager of professional nurses for 18 years, I have noted certain changes occurring in some employees, which was directly related to the season and the reduction of light. Understanding this phenomenon has enabled me to provide greater flexibility in staff work schedules, for example, when a staff member requested to work permanent night shift because of an inability to develop a definite sleep pattern, I could understand that due to their circadian rhythm, night shift was easier for them. Other individuals had great difficulty performing during the night and, consequently, called in ill. Switching of their hours was done and this alleviated some of the problems.

Mood disorders have been a topic of considerable concern over the years. Seasonal variations in affective disorders were recorded as far back as Wong T'ai (2700 B.C.) and Hippocrates (400 B.C.), indicating that they had been present for a number of years. Today, there has been a growing interest in affective disorders relating to the winter season. Psychologists and psychiatrists have been treating mood disorders which seemed to be related to some externally related problem in the environment, such as personal losses, rejections, or great disappointments. Considering that the person in Edmonton, which is at a latitude of 53 N, experiences low levels of light for approximately six months, it is feasible to try to identify aggravating factors in the environment which may precipitate the mood disorder.

Purpose of the Study

The purpose of this study was to identify ways of helping people with the condition diagnosed as seasonal affective disorder (SADS). Light therapy is known to have altered mood in patients with seasonal affective disorders; however variables such as intensity, duration, time of day, and colour of light need to be studied. It is known that at least 2500 lux for a two-hour period can alter depressive states (Rosenthal et al., 1984).

This study was designed to assess the effect of a high intensity (10,000 lux) light therapy for a 30 minute duration as a treatment for patients depressed as a result of SADS. A comparison with 500 lux was made to determine the change in depression.

SADS is a mood disorder portraying some or all of the following characteristic symptoms: (a) decreased energy; (b) day-time drowsiness; (c) increased sleep; (d) interpersonal difficulties; (e) increased appetite; (f) carbohydrate craving; (g) weight gain; and (h) decreased libido. It has been reported that residents in high-latitudes are most affected (Rosenthal *et al.*, 1984, p. 33). Since SADS is a winter depression occurring from about October to April, it affects 50% of the victim's life. The effects on quality of life are considerable since job productivity decreases and the individual suffers a general loss of well being. Even day-to-day problem solving can become problematic. However, once the length of day increases and there are a greater number of hours of sunlight, symptoms subside and the individual reverts back to euthymic state.

Researchers have studied light effects and other therapeutic techniques on mood disorders. Medications, psychotherapy and electro-convulsive therapy are

presently used to treat depression; however, for specific mood disorders relating to the season, light therapy has been utilized successfully. A seasonal pattern must be examined by the patient and therapist (Rosenthal, 1984). It has been determined that in the United States, six percent of the population suffer from seasonal affective disorders and 14% suffer from a milder form of the condition. Patients are female by a ratio of 4:1 as compared to ratios of 2:1 typically reported in general surveys of depression (Lewy *et al.*, 1980). Of clinically identified SADS, 60% are light-responders and 40% are non-responders (Lewy *et al.*, 1980). The general demographics comprised a predominance of females in the age group 30 to 44 years (74-94% female subjects) (Lewy *et al.*, 1980, p. 35).

Although all types of people, regardless of gender, education, race or colour, may have seasonal affective disorders, the majority of reported cases are women from all backgrounds (Rosenthal, 1989, p. 46).

Revival of Interest

Lewy *et al.* (1980) reported that the research scientist, Hebert Kern, was instrumental in providing information that led to the first recorded case of phototherapy. He suffered from the illness and reported his experience to Lewy when he learned Lewy was doing research on light and human melatonin. Rosenthal *et al.* (1984) collected and reported the first series of patients using light therapy and began a major interest on SADS (p. 27). Today, Norman Rosenthal (M.D.) is chief at the Unit Outpatient Studies in the Clinical Psychobiology Branch, National Institute of Mental Health; Thomas A. Wehr is in the Clinical Psychobiology Branch, National Institute of Mental Health, Bethesda, Maryland.

In reference to ethnic groups, it has been considered that seasonal affective disorders has evolved as an adaptive mechanism. Especially in the far north, where winter conditions are harsh and little food is available, it was to one's advantage to be inactive, overeat, store body fat and withdraw during the winter. One is thus more likely to survive the elements. Consequently, this disease has occasionally been confused with hibernation. In fact, there are differences, as reported by Rosenthal *et al.* (1984):

> Seasonal affective disorder patients have no dramatic temperature changes and they actually sleep less deeply. Even though there are similarities of hypersomnia, hyperphagia, weight gain, and carbohydrate craving, in hibernation overeating precedes hibernation and during hibernation animals are anorexic. In seasonal affective disorders, overeating and carbohydrate craving persist in the winter. (p. 73)

Seasonal affective disorders are found in Scandinavian, fair, blond-haired, blue-eyed people, as well as Africans with dark hair and dark eyes. It is in the northern hemisphere that both manic and depressives show significant seasonal variation due to the changes in latitude and light deprivation (Wehr, 1983, p. 185).

Duration

Since Kripke (1981), Terman *et al.* (1988) and Wirz-Justice *et al.* (1986b) reported a positive correlation with duration of light and its effects, Terman *et al.* (1990) supplied data that a treatment of 10,000 lux for 30 minutes in the morning is just as successful as 2,500 lux for 120 minutes.

The advantage of only 30 minutes of administration was a major benefit. Levitt *et al.* (1993) discussed side effects of light therapy, but 10,000 lux for 30 minutes was not studied. Consequently, it may be that duration of light, as opposed to intensity of illumination, is the cause of headache, eyestrain and feeling "weird"

(Levitt *et al.*, 1993, p. 650). Light administered at night results in the patient's difficulty in falling asleep.

Spectrum

White light was found to be the colour of choice by most investigators. The known physiology of the retina reveals that light in the green region of the spectrum is best absorbed (Dartnall *et al.*, 1983).

Route

Even though Levitt *et al.* (1993) reported eye strain as a major side effect, light seems to have a therapeutic effect only through the eyes (Wehr *et al.*, 1987b).

Limitations of the Study

The light boxes were self-administered at home and no accurate check could be made as to actual compliance. Most of the patients were female.

Patients were informed that light has been known to improve the mood. They were also informed that approximately 60% respond to light. They were further informed that two light sources (10,000 lux and 500 lux) were being studied for comparison of effects.

None of the patients were in psychotherapy, however they could choose to see a psychologist and not notify the psychiatrist.

Subjects could not be randomly selected. They were referred to the psychiatrist by their family doctor, psychiatrist or psychologist for evaluation as to their diagnoses and response to light.

In addition, light boxes were assigned as they were available, rather than having all the high-intensity boxes given first. Light boxes were given in consecutive weeks, that is, one week the patient may have had 10,000 lux for seven days and on the eighth day be started on the 500 lux therapy and vice versa.

Definitions of Terms

In this study, the term *lux* refers to a measurement of light. It was noted that although 10,000 lux illumination is not high relative to light levels commonly experienced out of doors at midday, it represents an innovation in indoor lighting levels (Terman *et al.*, 1990, p. 8). The intensity of light commonly found in a home or in an office is approximately 400 to 800 lux.

The Hamilton Depression Rating Scale (21-item) is a standardized test used in studying patients with depression. The eight supplementary items were incorporated into a structured guide specifically for the purpose of seasonal affective disorder identification (see Appendices A and B).

The hormone *Melatonin* is a retinal pineal-mediated neurotransmitter normally secreted at night and suppressed with bright light. Winter depressions may be related to abnormalities in Melatonin secretion or cycling. Melatonin is secreted and synthesized by the pineal gland in response to stimulation of its beta-adrenergic receptors (Lewy *et al.*, 1982b, p. 127). Melatonin is known to have acute sleep-inducing properties which may exacerbate depression.

Mania refers to an elated and psychotic mood. *Hypomania* refers to an elevated but non-psychotic mood disorder. *Hypersomnia* refers to excessive sleep (18 hours per day).

Goldstein, Baker and Jamison (1980) provide a typical example of what an affective disorder patient experiences:

A Patient Speaks....

> *Hypomania*: "At first when I'm high it's tremendous... ideas are fast... like shooting stars you follow 'til brighter ones appear... all shyness disappears, the right words and gestures are suddenly there... uninteresting people, things become intensely interesting. Sensuality is pervasive, the desire to seduce and be seduced is irresistable. Your marrow is infused with unbelievable feelings of ease, power, well-being, omnipotence, euphoria... you can do anything.... But somewhere this changes...."
>
> *Mania*: "The fast ideas become too fast and there are far too many... overwhelming confusion replaces clarity... you stop keeping up with it -- memory goes. Infectious humor ceases to amuse -- your friends become frightened... everything is now against the grain... you are irritable, angry, frightened, uncontrollable and trapped in the blackest caves of the mind -- caves you never knew were there. It will never end. Madness carves its own reality."
>
> *After mania*: "Now there are only others' recollections of your behavior -- your bizarre, frenetic, aimless behavior -- at least mania has the grace to dim memories of itself... now it's over, but is it?... incredible feelings to sort through... Who is being too polite? Who knows what? What did I do? Why? and most hauntingly, will it, when will it, happen again. Medications to take, to resist, to resent, to forget... but always to take. Credit cards revoked... explanations at work... bad checks and apologies overdue... memory flashes of vague men (what did I do?)... friendships gone... a marriage ruined."
>
> *Depression*: "When I get depressed I feel like a drain and a burden on my friends... the guilt and resentment are overwhelming. Everything I see, say, or do seems extraordinarily flat and pointless; there is no color, there is no point of anything. Things drag on and on intermittently. I am exhausted, dead inside. I want to sleep, to escape somehow, but if I really could sleep, I must again wake to the dullness and apathy of it all. I doubt, completely, my ability to do anything well; my mind has slowed down and burned out... it's virtually useless. The wretched thing works only well enough to torment me with a dreary litany of my inaccuracies and to haunt me with the total desperate hopelessness of it all. What is the point of going on like this; it is crazy."

CHAPTER II
LITERATURE REVIEW

This chapter will be presented in sections representing different perspectives of seasonal affective disorders (SADS). The result is intertwining of the endocrinological, anthropological and psycho-sociological approaches that constitute in the mood disorder.

General Overview

Temperature and Light Effect

Seasonal affective disorders were first observed in 400 B.C. by Hippocrates who noted that "melancholia occurs in the spring" (Lewy *et al.*, 1980, p. 34). Hippocrates stated that light or temperature could affect the patient (in Lewy *et al.*, 1980, p. 27). He contended that heat elevated the mood (Lewy *et al.*, 1980, p. 31). Galen (129-200 A.D.) compared the winter lethargy to hibernation and indicated that cold weakens mental activities.

Historical Information

Frederick Cook, an early polar explorer, used bright light to increase the well being of the crew. Years later, a research engineer presented his problem to Rosenthal, Sack and Wehr (1983) and agreed to receive light treatment. The engineer diligently kept a diary of his mood disturbances and reported very distinct patterns of depression during the winter and elation during the summer. Rosenthal

et al. (1983) then viewed the phenomenon of seasonal variables in unipolar and bipolar illnesses. He was the first to describe the condition (Rosenthal *et al.*, 1984). Lewy, Kern, Rosenthal and Wehr (1982) described the syndrome *Seasonal Affective Disorder* and indicated the therapeutic effects of exposure to bright artificial light. Rosenthal theorized about the underlying causes of the disorder.

Pinel, a French psychiatrist during the French Revolution, was one of the first psychiatrists in the 18th and 19th centuries who wrote about seasonal influences in his book *Treatise on Insanity*. This was also one of the first textbooks of psychiatry. Pinel described winter and summer mania (Lewy *et al.*, 1980, p. 14). Pinel treated his patients with the "usual routine of baths, bloodletting, and coercion, which were the standard treatment for mood disorders" (Rosenthal, 1983, p. 187). Rosenthal (1983) also stressed the importance of the physical environment in modulating mood (p. 188). During the late nineteenth and early twentieth centuries, bright artificial light was used therapeutically as it was known to have effects on the tuberculous bacillus as well as on depression (Lewy *et al.*, 1980). Consequently, the use of light to alter circadian rhythm became a common practice. Esquirol (1772-1840), who worked under Pinel, also made many contributions to seasonality of affective disorders (Lewy *et al.*, 1980, p. 16). He provided epidemiological data on variation of seasons and believed that the physical environment was a contributor to the disorder.

In 1923, Dr. Porter Phillips, a British psychiatrist, observed the benefits of light and went as far as to suggest that no structure is complete without a solarium: "The influence of sunlight on all living matter is well known" (in Rosenthal, 1983, p. 188). Therefore, light has been used therapeutically in previous situations for altering mood.

Landmark Study

In 1981-82, a research group at the American National Institute of Mental Health investigated a series of patients with winter depression and delineated clinical features of the syndrome (Rosenthal *et al.*, 1984, p. 21). This study was replicated by Wirz-Justice *et al.* (1986), Boyce and Parker (1988), Garvey *et al.* (1988) and Thompson and Isaacs (1988) (in Lewy *et al.*, 1980, p. 21) and the reults concurred that patients with winter depression possessed the same clinical features.

Seasonal affective disorder patients were identified as a subgroup of depression during the 1980s, after Rosenthal (1984) described a series of patients who experienced symptoms of decreased energy, day-time drowsiness, increased carbohydrate intake, and weight gain (p. 72). Lewy, Kern, Rosenthal and Wehr (1982a) also described a patient with a 20-year history of recurrent seasonal depression (p. 1496). Rosenthal indicated that seasonal affective disorders have been characterized by regularly occurring winter depression alternating with summer euthymic or hypomanic mood.

Duration of Light

In 1985, Lewy, Sack and Singer found that two hours of bright light therapy produced anti-depressant effects during a one-week study period (p. 295). They found that these patients had reported winter depression lasting three to four months for the three previous years.

In 1986, Wirz-Justice found that SADS was characterized by recurrent depression in autumn and winter, alternating with euthymia or hypomania during the spring and summer. These patients presented with atypical depressed symptoms of hypersomnia, fatigue, increased appetite and weight gain.

The landmark studies completed by Rosenthal *et al.* (1984) indicated that by extending the length of light during winter days, the depression decreased for those patients suffering from seasonal affective disorders. Bright light resulted in a marked anti-depressant effect on patients, whereas the dim light did not seem to have the same effect on patients (Rosenthal, 1985, p. 163).

Variables Altering Depression

Behaviour factors, such as sleep and a physical environment (light), can trigger or terminate episodes of seasonal affective disorders (Wehr, Sack & Rosenthal, 1985, p. 44). Patients relapse into their depression two to four days after the light treatment is discontinued.

Not only was light considered to contribute to the altered mood, but neurotransmitters were also being studied. The hormone Melatonin is known to have acute sleep inducing properties and may also exacerbate depression. Light suppresses the release of Melatonin in the body, thereby altering the sleepy stage. The incident of abnormal Melatonin concentrations also supports the hypothesis that depression can result from abnormalities of photoperiodic regulation (Kripke, 1985, p. 279).

A.M. Versus P.M. Treatments

According to Yerevanian, Anderson, Grota and Bray (1986), the time of day at which phototherapy is applied is related to circadian phenomena (p. 355). Once again, the critical factor is not when the phototherapy is applied but the fact that one can alter the Melatonin secretion. It seems that the time of day during which the circadian phase is most sensitive is in the morning (Wirz-Justice, Bucheli, Graw,

Kielholz, Fisch and Woggon, 1986, p. 574). Wehr, Skewer, Jacobson, Sack and Rosenthal (1987b) indicated that the effect of light on seasonal rhythms in animals is mediated by neural pathways leading from the eye to the hypothalamus and then to the pineal gland. Their study indicated that light was effective only when applied to the eyes (p. 753).

Michalon (1990) stated that light could be effective whether night or day (p. 106). This very important study showed how 14 researchers proved that for light to have significance in changing the scores of depression, morning light results had the greatest change. With early morning light, 53% of the patients improved; however, with evening light, only 38% showed improvement; and when light was administered during the midday, only 32% showed a change in their depression (Michalon, 1990, p. 107).

The amount of light required to suppress nocturnal melatonin is 2500 lux. Two hours (between 0600 hours and 0800 hours) seemed to be the selected administration time. Michalon (1990) also found that 30 minutes daily of 10,000 lux light therapy matches or exceeds the level of efficiency found with conventional light treatment. For positive results, a 2500 lux full-spectrum fluorescent light is used and the patient sits one metre from the light source, glancing approximately once every minute at a screen placed at eye level.

Wirz-Justice (1986) indicated that a high intensity light was required to suppress secretion of the pineal hormone Melatonin (p. 136) because a dimmer light was considered to be a control selection for evaluating the anti-depressant effect of light (p. 138). According to Rosenthal *et al.* (1985):

> Patients had an anti-depressant response to the bright light which occurred within two to four days and relapsed following withdrawal of light within the same time frame. (p. 263)

It seems that the anti-depressant effect could possibly be due to light having a placebo effect. It seems equally possible, however, that this is due to sleep deprivation which may occur under bright conditions.

Seasonality of Disorders

According to Wehr (in Rosenthal *et al.*, 1984a), there seem to be two seasonal influences on affective illness: one illness begins in the spring, peaking during the summer, whereas the other illness begins in the autumn and peaks during the winter (p. 11). Wehr also noted that changes in the physical environment can alter the sensitivity of the affective illness. The summer disease presents with anxiety, weight loss, anorexia and decreased sleep, which is opposite to winter depression (Rosenthal, 1989, p. 83).

In 1981-82, a research group at the National Institute of Mental Health delineated the clinical features of the syndrome (Rosenthal *et al.*, 1984, p.21). Original treatments were known to include change in temperature, latitude, light and darkness (Rosenthal *et al.*, 1984, p. 26).

Diagnostic Criteria

The revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R; American Psychiatric Association, 1987) included a *seasonal pattern* as a descriptor of recurrent major depression and bipolar disorder (p. 2).

In 1984, Rosenthal *et al.* defined the syndrome Seasonal Affective Disorder (SADS) as follows:

1. A history of major affective disorder, according to Research Diagnostic Criteria (Spitzer *et al.*, 1978);
2. At least two consecutive years of winter depressions that remitted the following spring-summer;

3. Absence of any other Diagnostic and Statistical Manual of Mental Disorders (3rd edition, DSM-III) Axis I psychiatric disorder;
4. Absence of seasonal psychosocial stressors. (pp. 34-35)

Incidence of SADS

Winter depression is a disorder affecting primarily women. Females predominate by a ratio 4:1, as compared to ratios of 2:1 typically reported in general surveys of depression (Rosenthal *et al.*, 1984, p. 3). Those suffering from SADS are considered to experience a milder type of depression since these patients rarely require hospitalization, do not become psychotic, and are not at serious risk of suicide.

Rosenthal *et al.* (1984) found that SADS patients were sensitive to short days of winter and the usual symptoms were exhibited by the patients. Patients usually begin to feel the difficulty with less than 10 hours of sunlight. The anti-depressant effect of artificial bright light begins within two to four days. The depression recurs at the same rate once artificial bright light is stopped.

Although a tendency to develop seasonal affective disorders seems to run in families, it is possible that the genetic vulnerability to seasonality may be expressed as an abnormality in visual information processing or in certain light-sensitive areas of the brain (Rosenthal, 1984, p. 42). This recurrent depression has a usual onset in September to early December and lasts into late March. The symptoms include fatigue, excessive sleep, carbohydrate cravings, and weight gain.

During the spring and summer, these patients feel elated, active and energetic, with generally well feelings reported. It was noted by Rosenthal *et al.* (1984) that the environmental variables played an important role in the pathogenesis of SADS.

Lewy and Sack (1986) indicated that 2500 lux of light was needed to change

the human circadian rhythms, or to suppress night-time Melatonin levels (p. 209). On the other hand, Zucher (1988) reported that animals and plants have not adopted hourglass mechanisms for measure of day length; instead, they make use of their circadian rhythms to distinguish long from short days (p. 220).

In 1987, Isaacs reported that the American National Institute Research Program group (NIMH) was the first to publish criteria for SADS (p. 17). An atypical depression picture was presented with a strong female preponderance (4:1). The mean age of onset was early to mid-twenties.

Wirz-Justice (1987) summarized the literature indicating that many biologic substrates vary seasonally in humans, raising the possibility that patients with SADS might fall at the extremes of both the behaviours and biologic spectral.

> Circadian phase-shift theory is perhaps the most currently actively studied theory of the mechanism of action of phototherapy. (p. 472)

Latitude Effects

In 1991, Lam discovered that the prevalence of SADS increases with higher latitude and is correlated with the shorter photoperiod in winter (p. 1926). Lam found that light therapy consisting of daily sustained exposure to intense bright light is an effective treatment of seasonal affective disorders. He also supported that the mechanism of action of light therapy is mediated through the eyes, presumably the retina (p. 1528). Changes in retinal sensitivity to light would be consistent with the abnormal phase-delayed circadian rhythms in seasonal affective disorders which normalize with light therapy (Lam, 1991, p. 1528).

Environmental Implications

Latitude and climate were first mentioned by Esquirol in the 18th century when he advised a patient to venture to Italy before the end of October and remain there until May. It was noted that the patient did not suffer another attack. Changes in the physical environment may initiate the affective disorder (Hellpach, 1911; in Lewy *et al.*, 1980, p. 24).

Anthropological Perspective

Seasonal variations in affective disorders were recorded as far back as 2700 B.C. by Wong T'ai who claimed that there were five humours -- fire, earth, air, water and wind. Later, the Hippocratic School Doctrine of Four Elements theory was formulated by Empedocles (490-430 B.C.) (Whybrow, Akiskal & McKinney, 1984, p. 23). He dropped wind and presented the elements as:

1. *Fire* - heat which was associated with blood and the function of the heart;
2. *Earth* - cold which was associated with (black bile) and a function of the spleen;
3. *Air* - moisture which was associated with (yellow bile) and a function of the liver; and,
4. *Water* - dryness which was associated with phlegm and a function of the brain.

Hippocrates indicated that the balance of these humours determined health and illness. In Whybrow *et al.* (1984) it is also stated that melancholics were born under the sign of Saturn and dominated by black bile secreted by the spleen (p. 23).

It was also believed that the divine played an important role in determining who would suffer insanity. For example, "The Lord shall smite thee with madness and blindness and astonishment of the heart" (Deuteronomy, Chapter 28, Verse 28).

The Greeks purported that depression was a dark mood, a black bilious humour. Another postulate stated by Hippocrates (460-355 B.C.) asserts that mental disorders were due to brain dysfunction.

> And men ought to know that from nothing else but thence [the brain] come joys, delight, laughter and sports, and sorrows, griefs, dependence, and lamentations.
>
> And by the same organ, we become mad and delirious, and fears and terrors assail us, some by night, and some by day, and dreams and untimely wanderings, and cares that are not suitable and ignorance of present circumstances .... All these things we endure from the brain when it is not healthy. (cited in Whybrow et al., *1984*, p. 24)

In the 1st century A.D., the Roman Catholic church mounted the Inquisition to destroy the threat of paganism. Madness was considered to be evil. Men and women were killed in what was seen as the exorcism of a devil's army, intent upon human perversion (Whybrow *et al.*, 1984, p. 24).

Descartes, in the 17th century, was a product of Jesuit education and he described everything in terms of reductionism. The four rules of logic included:

1. To maintain a posture of doubt,
2. To divide every problem into as many parts as possible, and only then
3. By reflection upon these elements could one
4. Commence the analytical task. (Whybrow *et al.*, 1984, p. 25)

Reductionist cast a problem in man's relationship with God since it presented the idea that MIND -- the soul of man -- was not to be considered dependent for survival upon the earthly body (Whybrow *et al.*, 1984, p. 26). This was known as Cartesian Dualism.

Models of depression or ideas about mood disorders were thus placed into frames of reference: intrapsychic, behavioural and biological perspectives (Whybrow *et al.*, 1984, p. 32).

The intrapsychic perspective was depicted by the psychoanalytical, existential and cognitive schools. One traces the experience of depression to past, perhaps remote, events that impinge on the cognitive, emotional and experiential world of the patient.

The behavioural perspective used the depression cyclic impact of interpersonal or other social events, external to the patient. These two perspectives used psychological constructs to look at origins of depression.

The biological approach looked at physiochemical events that may underlie or actually cause mood difficulties.

It has also been noted that the risk for mood disorders can be linked genetically and subsequently increases with the proportion of genes shared with a mood-disordered patient. The risk to relatives is presumably greater than the risk to the general population.

Women are at greater risk for unipolar disease; a ratio of 4:1 women to men suffer SADS. It has been suggested that a dominant locus on the X chromosome is involved in bipolar-related mood disorders (Tsuang & Farone, 1990, p. 167). It was also determined that the risk for mood disorders could be a result of a cultural transmission (Tsuang & Farone, 1990).

Psychological and Sociological Perspective

Depression can be caused by anger turned inward. This condition is also known as retroflexed anger. In addition, one can experience depression as a response to object loss. This includes traumatic separations from significant objects of attachment, for example, a person or a job. Occasionally one cannot live up to one's ego ideals and consequently suffers depression due to a loss of self-esteem. In this case, the concept of ego is determined by social reality. In addition, helplessness and hopelessness are experiences of depressed individuals. Such negative thinking associated with depression encourages the helplessness. Seligman (in Whybrow *et al.*, 1984, p. 37) discussed learned helplessness and he stated that lack of assertiveness, passivity and resignation are learned. According to this view, absence of a reinforcement for a positive interaction results in depression as noncontingent reinforcement (Whybrow *et al.*, 1984, p. 37). In other words, a bad case of mood disturbance can be due to something one hates.

Relationship of Art and Literature in Mood Disorders

Disturbance of mood is very persistent in our culture. Whether one wants to consider it due to causality or merely by ultimate purpose, depressive phenomena remain. Experience of sadness is interwoven with everyday life and with art and literature. Reviewing surrealist paintings provides a sense of dissociation with the real object. The experience of illness and its relationship to creativity and psychiatry disorder is exemplified in Sylvia Plath or Anne Sexton's biography. During investigations of families of creative writers, nearly two-thirds of the sample suffered from affective illness (in Whybrow *et al.*, 1984, p. 17). Melancholia will be

suffered by one in twenty people, but it is when sadness become destructive that it is considered a severe illness. Aristotle even wrote that "those who have become eminent in philosophy, poetry, politics and the arts all have tendencies toward melancholia" (Whybrow *et al.*, 1984, p. 17).

It is possible that the feminist movement has contributed to the re-examination of SADS. In 1984, when Rosenthal reviewed the syndromes, it was certainly due to the fact that people were no longer willing to be content and wait the depression out. Since more women suffer from the disease and generally more women seek medical attention, a label was given to the debilitative condition. The American National Institute of Mental Health in Bethesda, Maryland, included the following criteria as a framework for Seasonal Affective Disorders:

1. At least one lifetime episode of major depression;
2. History of recurrent episodes of depression (major or minor episodes occurring in at least two consecutive winters), with onset in fall or winter and recovery by spring or summer; and,
3. Absence of any other major psychiatric disorder (Rosenthal *et al.*, 1984).

The syndrome consists of a sad, anxious and irritable mood that remains reactive to social events. There is a pronounced reduction in socialization and physical activity, along with hypersomnia. Appetite is increased with carbohydrate craving and weight gain.

Environmental events can create a reactive depression characterized by late-night insomnia and feelings of self-pity and anxiety. The change in season from summer to fall with a resulting marked decrease of sunlight seemed to alter the mood. The confusion associated with endogenous depression was examined.

Characteristics of endogenous depression were somewhat different from the seasonal disorder. According to Rosenthal *et al.* (1984), these characteristics are as follows:

1. Early morning awakening;
2. Psychomotor retardation;
3. Severely depressed mood;
4. Feelings of guilt, remorse, and worthlessness;
5. Difficulty concentrating;
6. Loss of interest; and,
7. Weight change.

Environmental factors were once considered influential factors in suicide; however, social and hereditary factors play a greater role in determining suicide (Hillman, 1964). The anniversary dates must be eliminated since they offset feelings and emotions which may cause depression.

Suicide as an illness was studied to determine its place in the order of depression. Although the spring and summer were the highest seasons reported, it was not significant since the months between October and April were the months of depression for the seasonal affective disorder (Hillman, 1964). He also noted that hospital admissions were higher in the summer; however, it was not known if that was mainly due to bed availability or to an increase in disease.

Endocrinological or Biological Perspective

During the 1960s, the disturbances in biogenic amine metabolism was hypothesized as the predisposing factor in mood disorders. Pharmacology was thus utilized to improve depression by increasing the presence of amines in the synapse.

In 1972, new concepts included cholinergic mechanisms suggesting a noradrenergic-cholinergic imbalance in affective illness (Whybrow *et al.*, 1984).

According to Wehr (1984), adrenocortical and thyroid dysfunctions were found to initiate mood disturbances. As seen in Cushing's syndrome, the hypersecretion of steroids seemed to play a role in depression. Studies also revealed that depressed persons had elevated cortisol levels during their active illness. Sodium retention, which is intracellular and therefore found in intraneuronal space, is also noted in women with episodes of depression.

Tsuang and Farone (1990) reported that neurobiological genetic studies have implicated measures of cholinergic supersensitivity and the transport of lithium into red blood cells as biological markers that cosegregate with mood disorders within pedigrees (p. 167). These studies also found platelet monoamine oxidate to be decreased among those suffering with mood disorders (Tsuang & Farone, 1990, p. 164).

Melatonin is an indoleamine hormone secreted by the pineal gland. The pineal gland acts as an interface between an individual and his or her environment. Melatonin production occurs during the night in all species (Tsuang & Farone, 1990, p. 103). Bright light of sufficient intensity evidently suppresses nocturnal melatonin. Melatonin is synthesized from tryptophan and serotonin within the pineal gland.

The suprachiasmatic nucleus has a direct and unique connection to the pineal gland (Lewy *et al.*, 1979). Even though the total function of the pineal gland remains a mystery, it is thought that the synthesis of melatonin is triggered by darkness and its molecular structure strongly resembles the neurotransmitted serotonin. According to Lewy *et al.* (1979), light suppresses melatonin synthesis, which varies with the external light-dark cycle (p. 44).

Seasonal melatonin lows occur in the spring and fall and highs occur in January and July (Lewy *et al.*, 1979, p. 42). It is thought that seasonal melatonin variances may relate to seasonal variations in mood and recurring episodes of mania and depression.

One primary pathway carrying external light-dark information is probably via the retinal ganglion cells that form the retinohypothalamic tract, which leads from the retina to the suprachiasmatic nucleus of the hypothalamus, also known as the primary internal circadian pacemaker. The suprachiasmatic nucleus comprises a pair of small egg-shaped clusters of neuron cell bodies located at the front of the hypothalamus above the optic chiasma (Rosenthal *et al.*, 1985).

Rosenthal *et al.* (1985) also believed that the neuroanatomical pathways along which light exerts its seasonal effects in animals consists of light acting on the retina, and the nerve impulses thus generated are conducted along the retinohypothalamic tract to the suprachiasmatic nuclei (SCN) of the hypothalamus. From there the information is conveyed to the pineal gland by way of the paraventricular nuclei of the hypothalamus, the intermediolateral cell columns of the spinal cord, and the superior cervical ganglia (p. 265). The rhythm of nocturnal pineal melatonin secretion is generated by the SCN and the duration and pattern of secretion are modified by the timing of dawn and dusk. Serotonin deficiency has been hypothesized to be related to depression and carbohydrate craving (Rosenthal *et al.*, 1985, p. 267).

General depression and seasonal affective disorder related depression must thus be differentiated.

Depression

In 1896, Emil Kraeplin (in Corfman, 1984), the founding father of modern psychiatry, created an early diagnostic classification scheme of types of depression in which he noted that a generic class of mental illness which he called "manic depressive insanity" had a periodic or cyclic nature. One class of serious mental illnesses made up of affective psychosis included depression (unipolar) and manic depression (bipolar).

Becher (1974) included in his definition that depression is a period of one's life which may be marked by worry, pessimism, low energy and a sense of futility. These mood variations are not readily attributable to external circumstances (p. 199). Becher (1974) also stated that major affective disorders included a group of psychoses characterized by a single disorder mood -- either extreme depression or elation -- that dominates the mental life of a patient and is responsible for whatever loss of contact he/she has with the environment. Onset does not seem to relate directly to a particular life experience and, therefore, is distinguishable from psychotic depressive reaction and depressive neurosis (Becher, 1974, p. 200).

Involutional melancholic is a disorder that occurs in the involutional period and is characterized by worry, anxiety, agitation, and severe insomnia. One may also experience feelings of guilt, and somatic preoccupation is often present, even to the point of delusional proportions (Becher, 1974).

Manic-depressive disorders are marked by severe mood swings and a tendency to remission and recurrence. The manic phase symptoms include flight of ideas, irritability, talkativeness, excessive elation, and accelerated speech and motor activity. The depressive phase includes symptoms of severe depressive mood by

mental and motor retardation progressing occasionally to stupor, uneasiness, apprehension and delusions which usually consist of guilt or of hypochondriacal or paranoid ideas (Becher, 1974).

Causes of mild depression from the blues and grief are mostly environmental. They are usually of short duration. Normal functioning persists and recovery comes spontaneously. For excessive and prolonged grief reaction and "reactive" or "neurotic" depressions, the symptoms are not so easily sloughed. Some biological or genetic predispositions may be a factor; treatment usually involves psychotherapy and medication. The generic words "*affective*," "*depressed*" and "*manic*" describe the most noticeable or distinctive aspects of these illnesses and their powerful influence on mood (NIMH, 1984, p. 11).

Biologic or genetic predispositions are more likely to be influential and the syndrome is expressed in a cluster of symptoms involving many of the body's systems, affecting mood, beliefs, sleep, activity, energy, appetite, and physiological function (NIMH, 1984, p. 9). Such full-blown depressions are characteristic of what we call the major affective disorders and are clinically distinguishable into two categories (NIMH, 1984):

1. The *Unipolar* category correlates comprise little mania in the family history; its late onset (40s); moderate to high levels of wakefulness, e.g., activated or retarded psychomotor activity, or both; more anxiety and physical complaints; abnormal personality profile during an episode; and lower frequency of post-partum depression.
2. The *Bipolar* category includes in its pattern periods of mania (or less severe hypomania), and its correlates comprise higher incidence of mania in the family history; early age onset (20s); low activity levels

during depression episodes; psychomotor retardation; less anxiety and fewer physical complaints; normal personality during a depressed episode as scored on a widely used measurement scale (*Minnesota Multi-phasic Personality Inventory* [MMPI]); and higher frequency of post-partum depression.

Thematic Summary

The different perspectives of depression, whether one attributes it to psycho-social, endocrinological, or anthropological etiologies is a debilitating and disturbing occurrence. Treatment for the illness can occur utilizing several methods, including pharmacology, light therapy and psychotherapy. The cost of light treatment is considerably less than the other treatments. This study examined the effect of high intensity light versus a low intensity light each administered for 30 minutes for seven days.

The purpose of this study was to determine the number of SADS patients who respond to bright light therapy. Responders must have a 50% or greater reduction in the SIGH- SAD score from pretest.

CHAPTER III
METHODOLOGY

Subjects

The subjects presented in this study were consecutive patients referred by a mental health worker to a private psychiatrist. A total of 28 females and 15 males were accepted into the study after a clinical interview and administration of the *Hamilton Rating Scale for Depression - Seasonal Affective Disorder Tool.* A commonality among the subjects was that in their family history, at least one member suffered from depression. Most patients had taken some type of anti-depressant in the past.

Seven subjects withdrew from the research study for the following reasons:

- Patient #9 (M) cannot get up in the morning, before school to use the light
- Patient #10 (F) had flu and wanted two weeks of 10,000 lux
- Patient #11 (F) wore sunglasses with light, experienced eye irritation
- Patient #16 (F) refused low-level light, thought it would be useless
- Patient #21 (M) demanded that he be given therapy, and did not fit into SADS group
- Patient #35 (M) on medication
- Patient #37 (M), major change in patient's life and could not evaluate patient with regard to light variable

Eleven males and 25 females were subsequently entered into the study. The results are based on these 36 patients. Only 7 males and 10 females (47.2%) showed a 50% or greater improvement in the 29 items on SIGH-SADS. Another

five patients (13.8%) displayed a good response to light, that is, they had scores which were greater than 50% by 0.5 to 1.5 points in the SIGH- SAD. In other words, if they were scored by 0.5 to 1.5 lower on the post-test SIGH-SAD, they would have fulfilled the criteria for a positive response. If the near- responders are added to the responders, the response rate is 13.8 + 47.2 = 61.0%.

Procedure

The standardized test was used in determining the level of depression, carefully screening the patients for SADS. Once these patients met the criteria that they were depressed at one time in their lives, had a history of depression that recurred in the winter for at least two consecutive years with an experienced remission during the spring and had no ongoing drug/alcohol abuse, they entered the study.

Each patient acted as his/her own control by using 10,000 lux for 30 minutes one week and 500 lux for 30 minutes the next (or vice versa). Each patient was assessed and interviewed with the SIGH-SADS structured interview guide for the pre-test, post-test 10,000 lux and post-test 500 lux. The patients were randomly assigned 10,000 lux or 500 lux for the first week and then assigned the opposite light for the second week. Patients did not receive any other treatment modality during the study.

Method of Treatment

The type of light box used was a rectangular box placed horizontally on a table approximately 14 inches from the patient. The box delivered 10,000 or 500 lux at the patients' eye level. The light boxes are portable and patients were asked to self-administer the light treatment. Patients could proceed with reading or completing other tasks providing that the intensity of light remained 10,000 lux or 500 lux for the 30 minutes of treatment. Patients were asked to glance at the light every minute for one second.

The Instrument

Hamilton Depression Rating Scale - Modified for SADS

Williams (1988) referred to the Hamilton Depression Rating Scale (HDRS) as the most widely used scale for patient selection and follow-up in research studies of treatments of depression (p. 742). The scale, developed in the late 1950s, was a standardized scale for measurement of the severity of depressive symptoms (Hamilton, 1960). As outlined by Williams (1980), the symptoms are defined by anchor-point descriptions that increase in intensity. The clinicians must consider both the intensity and frequency of a symptom when assigning a rating value (Williams, 1988, p. 742). Initially, the scale was designed to have a score based on 17 of the 21 items. According to Magnusson *et al.* (1991), the Hamilton Rating Scale did not reflect the severity of depression (p. 146), and Isaacs *et al.* (1988) stated that the eight additional items in the SIGH-SADS rating scale almost doubled the patient's depression score.

The scale remains successful in determining depression because of its comprehensible coverage of depressive symptoms and related psychopathology, as well as its psychometric properties (Hidlund *et al.*, 1979). Carroll *et al.* (1973) reported that the total SIGH-SAD score has proved reliable, having a high degree of concurrent and differential validity (p. 742).

There is a general lack of item reliability of the SIGH-SADS due to varying interpretations of the meanings of the anchor-point descriptions and variability in the way in which the information is obtained to make the various rating distinctions (Williams, 1988, p. 743). In this study, two different clinicians completed the interviews and scored the SIGH-SADS. The structured interview guide increases the reliability. All inquiries begin with open-ended questions to encourage patients.

Because patients with SADS frequently had added complaints, eight supplementary atypical-vegetative items were incorporated into the structured interview guide for the *Hamilton Depression Scale - Seasonal Affective Disorder Version* (SIGH-SAD; Williams *et al.*, 1988). These supplementary items included:

1. Social withdrawal;
2. Weight gain;
3. Appetite increase;
4. Increased eating;
5. Carbohydrate craving;
6. Hypersomnia;
7. Fatigability; and
8. Diurnal variation Type B.

CHAPTER IV
RESULTS

The raw scores were tabulated and then transferred to a computer statistical analysis program: SPSS-X and the descriptive statistics, t-tests for pairs and Pearson-Product Moment. A summary of the pre- and post-test bright light and post-test dim light depression scores for the SIGH-SADS scale is found in Table 1. The order of light administration was tested with a 2 x 2 (order of administration of light treatment) fixed effects ANOVA with repeated measures on the second factor. These results are summarized in Table 2.

Inclusion criteria were a score of at least 12 on the 21-item Ham-D plus at least 10 points or more on the eight supplementary atypical-vegetative items incorporated into the *Structured Interview Guide for the Hamilton Depression Scale - Seasonal Affective Disorder Version* (SIGH-SAD; Williams *et al.*, 1988). A patient was identified as a light responder if there was a 50% or greater improvement on the 29-scale SIGH-SAD.

The total group mean and standard deviation of the pretest for questions 1 to 29 were M = 36.14 and SD = 7.01. The total group mean and standard deviation of the post-10,000 lux for questions 1 to 29 were M = 17.64 and SD = 10.24. The mean difference and standard deviation of the pretest and post-10,000 lux for questions 1 to 29 were M = 18.50 and SD = 12.13. The means were significantly different for questions 1 to 29 ($t(35) = 9.15$, $p < .05$).

The total group mean and standard deviation of the post-500 lux for questions 1 to 29 were M = 29.52 and SD = 9.16. The mean difference and standard

deviation of the pretest and post 500-lux for questions 1 to 29 were M = 6.61 and SD = 8.21. The means were significantly different ($t(35) = 4.83$, $p < .05$).

The mean difference and standard deviation of the post-10,000 lux and post 500-lux for questions 1 to 29 were M = -11.89 and SD = 12.87). The means were significantly different ($t(35) = -5.54$, $p < .05$).

Post 10,000 lux treatment, the patients were less depressed; however, all people do not respond similarly to light, that is, people who are more depressed may respond better to light.

The total group mean and standard deviation of the pretest for questions 22 to 29 were M = 15.03 and SD = 3.66. The total group mean and standard deviation of the post-10,000 lux for questions 22 to 29 were M = 7.86 and SD = 4.47. The mean difference and standard deviation of the pretest and post-10,000 lux for questions 22 to 29 were M = 7.17 and SD = 5.31. The means were significantly different for questions 22 to 29 ($t(35) = 8.11$; $p < .05$).

The total group mean and standard deviation of the post-500 lux for questions 22 to 29 were M = 12.81 and SD = 4.87. The mean difference and standard deviation of the pretest and post 500 lux for questions 22 to 29 were M = 2.22 and SD = 3.84. The means were significantly different ($t(35) = 3.47$, $p < .05$).

The mean difference and standard deviation of the post-10,000 lux and post-500 lux for questions 22 to 29 were M = -4.94 and SD = 5.81. The means were significantly ($t(35) = -5.11$; $p < .05$).

The total group mean and standard deviation of the pretest for questions 1 to 21 were M = 21.11 and SD = 5.56. The total group mean and standard deviation of the post-10,000 lux for questions 1 to 21 were M = 9.78 and SD = 7.08. The mean

difference and standard deviation of the pretest and post-10,000 lux were M = 11.33 and SD = 7.97. The means were significantly different ($t(35) = 8.53$; $p < .05$).

The toal group mean and standard deviation of the post-500 lux for questions 1 to 21 were M = 16.72 and SD = 5.79. The mean difference and standard deviation of the pretest and post-500 lux for questions 1 to 21 were M = 4.39 and SD = 5.53. The means were significantly different ($t(35) = 4.76$, $p = .05$).

The mean difference and standard deviation of the post-10,000 lux and post-500 lux for questions 1 to 21 were M = -6.94 and SD = 7.79. The means were significantly different ($t(35) = -5.35$, $p < .05$).

Frequent symptoms reported on the SIGH-SADS were as follows:

Question Numbers:	28.	Fatigability
	7.	Work and activities
	22.	Social withdrawal
	1.	Depressed mood
	27.	Hypersomnia

The most frequently rated symptom was fatigability with a pretest mean of 2.95 (SD = .54); post-10,000 lux mean of 1.36 (SD = .80); and post-500 lux mean of 2.68 (SD = .89). Table 1 includes the mean and standard deviation for each question.

Conclusion

The results indicate that there is a change in scores as reported by the instrument when the high-intensity 10,000 lux is given. When patients received the 10,000 lux intensity, they showed less depression as reported by the scores than when they received 500-lux intensity. In this study the female ratio was 25:11. Only one patient experienced side effects from the light therapy.

Graph 1 depicts that before light, all patients responded with a score indicating depression. After the 10,000 lux treatment, the patients' scores indicated less depression. Subsequent to the 500 lux treatment, their scores were more varied. Patients 1, 2, 12, and 36 could be considered placebo responders or they could belong to a sub-group who respond to low-level light.

For follow-up, the people who had a good response to light and good affordability purchased a light box. The people who were not reponsive to light were prescribed selective-serotonin re-uptake inhibitors (i.e. prozac, luvox, zoloft) by their family physicians or psychiatrists.

Table 1
Means and Standard Deviations for Individual Questions 1 to 29
n = 36

Question	Pretest	SD	Post 10,000 lux	SD	Post 500 lux	SD
1	2.31	.58	1.00	.91	1.97	.88
2	1.25	.81	.50	.61	1.06	.73
3	.17	.51	.08	.50	.11	.40
4	.75	.81	.17	.45	.33	.59
5	.86	.80	.14	.35	.53	.77
6	.67	.75	.31	.53	.64	.80
7	2.64	.49	1.36	.95	2.28	.73
8	1.33	.93	.58	.73	1.28	.91
9	.94	.91	.42	.65	.72	.97
10	1.81	.67	1.08	.81	1.72	.74
11	1.11	.94	.56	.74	.89	.85
12	.39	.60	.22	.49	.39	.55
13	1.36	.54	.89	.67	1.25	.55
14	1.34	.83	.56	.70	.89	.79
15	.47	.70	.29	.62	.29	.45
16	0.00	0.00	0.00	0.00	0.00	0.00
17	.19	.56	.14	.49	.22	.64
18	1.52	.74	.58	.77	1.26	.82
19	.25	.65	.03	.17	.22	.54
20	.11	.40	.06	.33	.17	.45
21	.39	.60	.06	.24	.42	.60
22	2.50	.66	1.31	.86	2.02	.81
23	1.00	.90	.47	.77	.78	.87
24	1.25	1.03	.72	.82	1.22	1.17
25	1.31	.95	.75	.77	1.14	.99
26	1.81	.75	.92	.81	1.53	1.00
27	2.14	1.22	.94	.92	1.78	1.15
28	2.94	.53	1.75	1.05	2.58	.91
29	2.08	.87	1.00	.86	1.75	1.00

Table 2
Summary ANOVA

Source	df	MS	F	p
Variable 10,000 lux				
Between Groups	1	3.83	.034	.25
Within Groups	34	108.01		
Variable 500 lux				
Between Groups	1	15.98	.1860	.25
Within Groups	34	85.9117		

As revealed in this table, there is no significant light order of administration effect ($F = 0.18$, $p < .25$), but there is a significant main effect ($F = 0.02$, $p < .25$).

Table 3
Symptoms of Winter Depression
Ranked in Order from Most Troublesome to Least Troublesome
n = 36

1. Fatigability
2. Work and activities
3. Social withdrawal
4. Depressed mood
5. Hypersomnia
6 Diurnal Variation Type B
7. Anxiety psychic
8. Carbohydrate craving
9. Diurnal Variation Type A
10. Retardation
11. Feelings of guilt
12. Somatic symptoms general
13. Increased eating
14. Appetite increase
15. Genital symptoms
16. Anxiety somatic
17. Weight gain
18. Agitation
19. Insomnia middle
20. Insomnia early
21. Insomnia late
22. Hypochondriasis
23. Obsessional symptoms
24. Somatic symptoms - gastro-intestinal
25. Derealization and depersonalization
26. Loss of weight
27. Paranoid symptoms
28. Suicide
29. Insight

Table 4
Raw Scores of the Hamilton Rating Depression Test - Seasonal Affective Disorders
n = 36

Patient No.	Response	Sex	Questions	Pretest	Post 10,000 lux	Post 500 lux
1*		M	1-21	26	5	5
			22-29	15	6	3
			1-29	41	11	8
2*		F	1-21	18	2	7
			22-29	9	3	4
			1-29	27	5	11
3	GR	F	1-21	19	6	12
			22-29	12	10	13
			1-29	31	16	25
4	R	M	1-21	22	11	21
			22-29	16	8	14
			1-29	38	19	35
5*	R	F	1-21	21	9	24
			22-29	19	11	16
			1-29	40	20	40
6	R	F	1-21	15	10	14
			22-29	22	9	15
			1-29	37	19	29
7	NR	F	1-21	24	26	20
			22-29	7	9	8
			1-29	31	35	28
8*	NR	F	1-21	19	23	21
			22-29	12	12	11
			1-29	31	35	32
12		M	1-21	14	8	7
			22-29	15	8	6
			1-29	29	16	13
13*	NR	F	1-21	18	11	14
			22-29	17	15	15
			1-29	35	26	29

(cont'd)

Table 4 (cont'd)

Patient No.	Response	Sex	Questions	Pretest	Post 10,000 lux	Post 500 lux
14*	R	F	1-21	14	0	17
			22-29	14	0	15
			1-29	28	0	32
15*	NR	F	1-21	25	25	24
			22-29	18	19	19
			1-29	43	44	43
17	R	F	1-21	23	5	25
			22-29	17	2	17
			1-29	40	7	42
18*	R	M	1-21	33	9	30
			22-29	18	4	18
			1-29	51	13	48
19	R	F	1-21	29	10	25
			22-29	22	11	17
			1-29	51	21	42
20	R	M	1-21	19	0	18
			22-29	16	0	18
			1-29	35	0	36
22*	R	M	1-21	14	5	17
			22-29	22	6	21
			1-29	36	11	38
23*	R	M	1-21	23	5	20
			22-29	13	5	16
			1-29	36	10	36
24	GR	F	1-21	13	5	10
			22-29	16	11	16
			1-29	29	16	26
25	R	F	1-21	16	8	15
			22-29	15	5	14
			1-29	31	13	29

(cont'd)

Table 4 (cont'd)

Patient No.	Response	Sex	Questions	Pretest	Post 10,000 lux	Post 500 lux
26*	R	M	1-21	19	8	21
			22-29	14	7	7
			1-29	33	15	28
27	GR	F	1-21	19	13	19
			22-29	13	4	11
			1-29	32	17	30
28	GR	M	1-21	21	11	14
			22-29	19	10	14
			1-29	40	21	28
29	NR	F	1-21	15	7	18
			22-29	15	11	17
			1-29	30	18	35
30*	NR	F	1-21	14	12	15
			22-29	14	19	21
			1-29	28	31	36
31	R	F	1-21	26	3	18
			22-29	14	4	9
			1-29	40	7	27
32*	NR	F	1-21	18	9	21
			22-29	11	9	11
			1-29	29	18	32
33	NR	M	1-21	21	18	15
			22-29	6	3	3
			1-29	27	21	18
34	R	F	1-21	17	1	9
			22-29	15	9	15
			1-29	32	10	24
36*		F	1-21	17	10	7
			22-29	15	10	6
			1-29	32	20	13

(cont'd)

Table 4 (cont'd)

Patient No.	Response	Sex	Questions	Pretest	Post 10,000 lux	Post 500 lux
38	R	M	1-21	20	4	16
			22-29	17	2	14
			1-29	37	6	30
39*	NR	F	1-21	20	18	12
			22-29	16	10	8
			1-29	36	28	20
40*	NR	F	1-21	10	6	14
			22-29	10	9	8
			1-29	20	15	22
41*	R	F	1-21	21	8	16
			22-29	17	7	17
			1-29	38	15	33
42*	R	F	1-21	19	2	19
			22-29	15	4	12
			1-29	34	6	31
43*	GR	F	1-21	25	10	17
			22-29	15	11	13
			1-29	40	21	30

NOTES:

* Patient given 10,000 lux intensity light box first
NR Non-responder
GR Good responder, that is 0.5-1.5 score higher than 50% reduction from pre-test
R Responder (i.e. 50% or greater than 50% reduction in post-10,000 lux score)

Table 5
Means, Standard Deviations and Standard Error for Patients Pretest, Post-10,000 Lux and Post-500 Lux
Questions 1 - 29

	Pretest			Post 10,000 lux			Post 500 Lux		
	Mean	SD	SE	Mean	SD	SE	Mean	SD	SE
n = 36	36.14	7.01	1.17	17.64	10.25	1.71	29.53	9.16	1.52

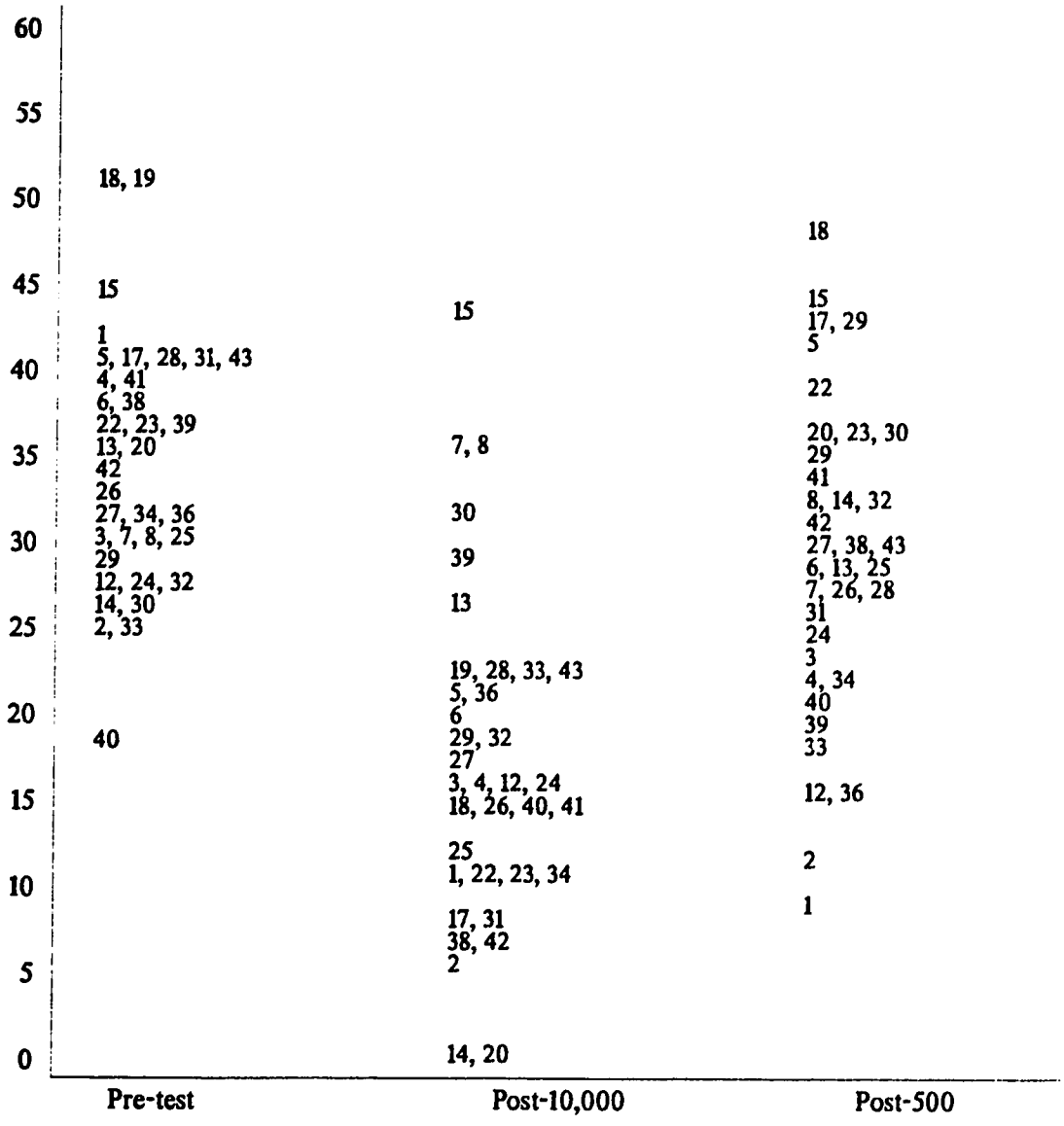
Table 6
Means, Standard Deviations and Standard Error for Patients Pretest, Post-10,000 Lux and Post-500 Lux
Questions 1 - 21

	Pretest			Post 10,000 lux			Post 500 Lux		
	Mean	SD	SE	Mean	SD	SE	Mean	SD	SE
n = 36	21.11	5.56	.93	9.78	7.08	1.18	16.72	5.79	.97

Table 7
Means, Standard Deviations and Standard Error for Patients Pretest, Post-10,000 Lux and Post-500 Lux
Questions 22 - 29

	Pretest			Post 10,000 lux			Post 500 Lux		
	Mean	SD	SE	Mean	SD	SE	Mean	SD	SE
n = 36	15.03	3.66	.61	7.86	4.47	.74	12.81	4.87	.81

Graph 1
SIGH-SADS Scores - Comparison of the Pre-test with the Post 10,000 Lux and Post 500 Lux
n = 36



* The numbers indicate the patient number and the subsequent raw score

CHAPTER V
DISCUSSION AND CONCLUSIONS

This study demonstrates that high intensity light therapy does have an effect on the mood of SADS patients. The results are statistically significant. The patients receiving 10,000 lux of light rated a score on the *Hamilton Rating Scale for Depression - Seasonal Affective Disorders* were less depressed.

These findings have implications for the treatment of SADS patients. Phototherapy is inexpensive and can be carried out in the patient's own home. The cost of a light box is approximately $700.00.

Further research needs to be carried out to determine alternative treatments for patients who experience this lower quality of life. It is recommended that future studies include:

- Color of eye and the effect on light therapy
- Changes in altitude and light therapy
- Changes in latitude in light therapy
- Gender differences in light therapy
- Hormonal effects and light therapy
- Effect of light in blind people

Since it is difficult to obtain a global concentration of light beyond a certain brightness, it would be impossible to attain a therapeutic lux level in the work environment.

The point that the patients must keep in mind is that the intensity of light, i.e. 10,000 lux, for a period of 30 minutes daily was used as their treatment. This study indicated that there were four placebo or low level responders.

There are no widely recognized standardized schedules or protocols for treatments; therefore, a need also exists to conduct research to define therapeutic thresholds and to define the dosage relationships of timing, intensity, and duration of light treatment. Moreover, patients may have been experiencing other situations in their lives which could be a confounding variable. The effect of light on melatonin has implications for administering the light during the morning, since light administered at night could cause sleeplessness.

With regard to the findings, patients must commit themselves to making significant alterations in winter-time lifestyle. Patients must commit as much as 30 minutes per day for their therapy throughout their risk period, generally, October to April.

This information has to reach as many of the population as possible. Sources for transmitting the information may include newspaper and television advertising. Once these people have been identified, treatment can commence. An alternate, however impractical, form of treatment would be that the patient relocate to a sunny climate for the duration of the winter depression.

With a decrease in health care dollars, it is a significant cost saving measure to treat people with seasonal affective disorders outside the hospital setting with light rather than with medications that are costly and may have significant side effects..

Conclusion

Beginning with the classical study by Rosenthal (1984), other researchers identified the predominant symptoms in people with Seasonal Affective Disorders and the effect of bright light therapy to alter the depressed mood. Rosenthal (1984)

indicated that by extending the length of light during the winter, the depression decreased.

Light therapy seems to be the treatment of choice for winter depressives and recently it was found to be used in the treatment of circadian rhythm sleep disorders, for jet lag, and possibly other types of depression.

Since my first introduction to people with seasonal affective disorders, I have concluded that this syndrome has indeed been present throughout history. Since the renewal or revival of the feminist movement, perhaps women felt less intimidated in discussing their feelings to the predominantly male psychiatric profession. Women decided no longer to suffer another "winter blues" because someone or something could assist them.

The patient excerpts mentioned in Chapter I are very typical of a person with seasonal affective disorders. During a group session with SADS patients, I realized the incapabilities which these people really experience for half of their lives. The group consisted of people involved in a range of jobs from housewife to university student. Regardless of education, the gloom of the disease was the same. There was some reluctance to continue to share their difficulties with outsiders, such as myself, and this could be a result of the social stigma still present when one has a "psychological" problem. A better understanding of the physiological effects would assist these people in handling their problem.

In conclusion, the present study indicates that high intensity light therapy will decrease depression in people with seasonal affective disorders.

BIBLIOGRAPHY

Adams, F. (ed.)(1979). *The genuine work of Hippocrates.* Baltimore: Williams and Wilkins.

Arendt, J., Borbely, A.A., Franey, C., & Wright, J. (1984). The effects of chronic, small doses of melatonin given in the late afternoon on fatigue in man: A preliminary study. *Neuroscience Letters, 45,* 317-321.

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, D.C.: Author.

Avery, D., Bolte, M.G., Dager, S., Wilson, L., Weyer, M., Cox, G., & Dunner, D. (1993). Dawn simulation treatment of winter depression: A controlled study. *American Journal of Psychiatry, 150,* 113-117.

Avery, D.H., Khan, A., Dager, T.R., Cox, G.B., & Dunner, D.L. (1990). Bright light treatment of winter depression: Morning versus evening light. *Acta Psychiat. Scand., 82,* 335-338.

Bassi, C., & Powers, M. (1985). Daily fluctuations in the detectability of dim lights in humans. *Physiology and Behavior, 38,* 871-877.

Beck-Friis, J., Kjellman, B.F., Aperia, B., Undén, F., von Rosen, D., Ljunggren, J.-G., & Wetterberg, L. (1985). Serum melatonin in relation to clinical variables in patients with major depressive disorder and a hypothesis of a low melatonin syndrome. *Acta Psychiatrist Scana.,* 319-330.

Becher, J. (1974). *Depression: Theory and research.* Washington, D.C.: Winston & Sons.

Bick, P. (1986). Seasonal major affective disorder. *American Journal of Psychiatry, 143,* 90-91.

Blehar, M., & Rosenthal, N. (1989). Seasonal affective disorders and phototherapy. *Archives of General Psychiatry, 46,* 469-474.

Blehar, M., Rosenthal, N., Terman, M., & Wehr, T. (1990). Identification, assessment and treatment of seasonality in mood disorders. *Psychopharmacology Bulletin, 26*(1).

Boyce, P., & Kennaway, D.J. (1987). Effects of light on melatonin production. *Biological Psych., 22,* 473-478.

Boyce, P., & Parker, G. (1988). Seasonal affective disorder in the southern hemisphere. *American Journal of Psychiatry, 141*(1), 92-99.

Burrows, J. (ed.)(1977). *Handbook of studies on depression.* Amsterdam; London; New York: Excerpta Medica.

Byerley, W., Brown, J., & Lebeque, B. (1987). Treatment of seasonal affective disorder with morning light. *Journal of Clinical Psychiatry, 48*(11), 447-448.

Carroll, B.J., Fielding, J.M., & Blashki, T.G. (1973). Depression rating scales: A critical review. *Archive General Psychiatry, 28*, 361-366.

Corfman, E. (1984). *Depression, manic - depressive illness and biological rhythms.* Rockville, Maryland: National Institute of Mental Health, Division of Scientific and Public Information, U.S. Department of Health Education and Welfare.

Dartnall, H.J.A., Bowmaker, J.K., & Mellon, J.D. (1983). Human visual pigments: Microspectrophotometric results from the eyes of seven persons. *Proceedings from the Royal Society of London B., 220*, 115-130.

Deltito, J.A. (1993). Effects of phototherapy on non-seasonal unipolar and bipolar depressive spectrum disorders. *Journal of Affective Disorders, 23*(4), 231-33.

Dilsaver, S.C. (1990). Onset of winter depression earlier than generally thought? *Journal of Clinical Psychiatry, 51*(6), 258.

Dublin, L.I. (1963). *Suicide: A sociological and statistical study.* New York: Ronald Press Co.

Easton, M.S. (1990). Seasonal affective disorders. *Current Opinion in Psychiatry, 3*, 54-57.

Eastwood, M.R., & Peacock, J. (1976). Seasonal patterns of suicide, depression and electroconvulsive therapy. *British Journal of Psychiatry, 129*, 472-475.

Garvey, M.J., Wesner, R., & Godes, M. (1988). Comparison of seasonal and nonseasonal affective disorders. *American Journal of Psychiatry, 145*, 100-102.

Haggerty, J., Evans, D., Golden, R., Pederson, C., Simon, J., & Nemeroff, C. (1990). The presence of antithyroid antibodies in patients with affective and nonaffective psychiatric disorder. *Biological Psychiatry, 27*, 51-60.

Halbreich, U. (ed.)(1987). *Hormones and depression.* New York: Raven Press.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neural Neurosurgi. Psychiatry, 23,* 56-62.

Hamilton, M. (1967). Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology, 6,* 278-296.

Haug, H.J., & Fähndrich, E. (1990). Diurnal variations of mood in depressed patients in relation to severity of depression. *Journal of Affective Disorders, 19,* 37-41.

Hedlund, J.L., & Vieweg, B. (1979). The Hamilton rating scale for depression: A comprehensive review. *Journal of Operational Psychiatry, 10,* 149-165.

Hellakson, C.J., Kline, J.A., & Rosenthal, N.E. (1986). Phototherapy for seasonal affective disorders in Alaska. *American Journal of Psychiatry, 143,* 1035-1037.

Hillman, J. (1964). *Suicide and the soul.* Dallas, TX: Spring Publications, Inc.

Isaacs, G., Stainer, D.S., & Sensky, T.E. (1988). Phototherapy and its mechanisms of action in seasonal affective disorders. *Journal of Affective Disorders, 14,* 13-19.

James, S., Wehr, T., Sack, D., Perry, B., & Rosenthal, N. (1985). Treatment of seasonal affective disorder with light in the evening. *British Journal of Psychiatry, 147,* 424-428.

Kasper, S., Rogers, S., Yancey, A., Schulz, 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., Wehr, T.A., Bartko, J.J., Gaist, P.A., & Rosenthal, N.E. (1989). Epidemiological findings of seasonal changesin mood and behavior. *Archives of General Psychiatry, 46,* 823-833.

Kraepelin, E. (1921). *Manic-depressive illness and paranoia,* Translated by R.M. Barclay; G.M. Robertson, E. Livingstone & M. Livingstone (eds.). Edinburgh, Scotland: Livingstone.

Kripke, D.(1981). Photoperiodic mechanisms for depression and its treatment. In C. Perris, G. Struwe, B. Jansson (eds), *Biological psychiatry* (pp. 1249-52). North Holland: Biomedical Press, Elsevier.

Kripke, D. (1985). Therapeutic effects of bright light in depressed patients. *Annals of the New York Academy of Sciences, 453*(270), 270-281.

Kripke, D., Risch, C., & Janowsky, D. (1983). Lighting up depression. *Psychopharmacology Bulletin, 19*, 526-530.

Lam, R.W., Beattie, C., Buchanan, A., Remick, R., & Zio, A. (1991). Low electrooculographic ratios in patients with seasonal affective disorders. *American Journal of Psychiatry, 148*(11), 1526-1529.

Lam, R.W., Buchanan, A., & Remiek, R.A. (1989a). Seasonal affective disorder: A Canadian sample. *Annals of Clinical Psychiatry, 1*, 241-145.

Lam, R.W., Kripke, D.F., & Gillin, J.C. (1989b). Phototherapy for depressive disorders: A review. *Canadian Journal of Psychiatry, 34*, 140-147.

Levitt, J.A., Joffe, R.T., Move, D., Lam, R., Teicher, M., Lebeque, B., Murray, M., Oren, D., Schwarz, P., Buchanan, A., Glod, C., & Brown, J. (1993). Side effects of light therapy in seasonal affective disorders. *American Journal of Psychiatry, 150*, 650-652.

Lewy, A.L. (1990). Reply to . . . Healy, D. and Waterhouse, J.M. *Chronobiology - International, 7*(1), 15-21.

Lewy, A., Kern, H.A., Rosenthal, N.E., & Wehr, I.A. (1982a). Bright artificial light treatment of a manic-depressive patient with seasonal mood cycle. *American Journal of Psychiatry, 139*, 1496-1498.

Lewy, A., Rosenthal, N., Sleva, L., Wehr, T., Numberger, J., Uhate, T., Newsane, D., Kopin, I., Becker, L., Goodwin, F., & Marhey, S. (1982b). Melatonin secretion as a neurobiological "marker" and effects of light in humans. *Psychopharmacology Bulletin, 18*(4), 127-129.

Lewy, A.J., Sack, R.L., & Singer, C.M. (1985). Treating phase-typed chronobiologic sleep and mood disorders using appropriately timed bright artificial light. *Psychopharmacology Bulletin, 21*, 268-372.

Lewy, A.J., Wehr, T.A., Goodwin, C.K., Newsonae, P.A., & Markey, S.P. (1980). Light suppresses melatonin secretion in humans. *Science, 210*, 1267-1269.

Lindjaerdi, O., Bratlin, T. Hansen, T., & Gotestam, K.G. (1986). Seasonal affective disorder and midwinter insomnia in the far north: Studies on two related chronobiological disorders in Norway. *Clinical Neuropharmacology, 9*(Suppl 4), 187-189.

Magnusson, A. (1991). Treatment of seasonal affective disorder with high-intensity light. *Journal of Affective Disorders, 21*(2), 141-147.

Magnusson, A., & Kristbjarnarson, H. (1991). *Journal of Affective Disorders, 21*, 141-147.

Michalon, M. (1990). Shedding light on seasonal affective disorders. *The Canadian Journal of Diagnosis*, (December), 101-109.

Morin, G.D. (1990). Seasonal affective disorders, the depression of winter: A literature review and description from a nursing perspective. *Archives of Psychiatric Nursing, IV*(3, June), 182-187.

Oren, D.A. (1991). Usage paterns of phototherapy in seasonal affective disorder. *Comprehensive Psychiatry, 32*(2), 147-152.

Paykel, E.S., & Copen, A. (eds.)(1979). *Psychopharma-cology of affective disorders*. Oxford University Press.

Potkin, S.G., Zetin, M., Stamenkovic, V., Kripke, D.F., & Bunney, w.E. (1986). Seasonal affective disorder: Prevalence varies with latitude and climate. *Clinical Neuropharmacol., 9*(Suppl 4), 181-183.

Roccatagliata, G. (1986). *A history of ancient psychiatry*. New York: Greenwood Press.

Rosen, L.N., Targum, S.D., & Terman, M. (1990). Prevalence of seasonal affective disorders at four latitudes. *Psychiatry Research, 31*, 131-144.

Rosenthal, N.E. (1992). Towards understanding athe mechanism of action of light in seasonal affective disorders. *Pharmacopsychiatry, 25*(1), 56-60.

Rosenthal, N. (1990). *Seasons of the mind*. New York: Bantam Books.

Rosenthal, N., & Blehar, M. (eds.)(1989). *Seasonal affective disorders and phototherapy: NIMH*. New York & London: The Guilford Press.

Rosenthal, N.E., DellaBella, P., Hahn, L., & Skwerer, R.G. (1989b). Seasonal affective disorder and visual impairment: Two case studies. *Journal of Clinical Psychiatry, 50*, 469-472.

Rosenthal, N.E., Jacobsen, F.M., & Sack, D.M. et al(1988a). Atenolol in seasonal affective disorder: A test of the melatonin hypothesis. *American Journal of Psychiatry, 145*, 52-56.

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

Rosenthal, N., Sack, D., Carpenter, C., Party, B., Mendelson, W., & Wehr, T. (1985a). Antidepressant effects of light in seasonal affective disorders. *American Journal of Psychiatry, 142*(2, February), 163-170.

Rosenthal, N., Sack, D., Christian, G., Lewy, A., Goodwin, F., Davenport, Y., Meuller, P., Newsome, D., & Wehr, T. (1984b). Seasonal affective disorders: A description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry, 41*(January), 72-80.

Rosenthal, N., Sack, D., James, S., Parry, B., Mendelson, W., Tamarlin, L., & Wehr, T. (1985b). Seasonal affective disorder and phototherapy. *Annals of the New York Academy of Sciences, 45*(3), 260-269.

Rosenthal, N.E., Sack, D.A., Skwerer, R.G., Jacobsen, F.M., & Wehr, T.A. (1988b). Phototherapy for seasonal affective disorder. *Journal of Biological Rhythms, 3*, 101-120.

Rosenthal, N., Sack, D., & Wehr, T. (1983). Seasonal variation in affective disorders. In A.A. Wehr & F.K. Goodwin (eds.), *Circadian rhythms in psychiatry* (pp. 185-201). New York: The Boxwood Press.

Rosenthal, N., & Wehr, T. (1987). Seasonal affective disorders. *Psychiatric Annals, 17*(10, October), 670-674.

Sack, R., Lewy, A., White, D., Singer, C., Fireman, M., & Vandiver, R. (1990). Morning versus evening light treatment for winter depression. *Archives General Psychiatry, 47*, 343-351.

Seligman, M.E.P. (1975). *Helplessness: On depression, development, and death.* San Francisco, CA: W.H. Freema.

Shafi, M., & Shafi, S.L. (eds.)(1990). *Biological rhythms, mood disorders, light therapy, and the pineal gland.* Washington, D.C.: American Psychiatric Press.

Shneidman, E., & Ortega, M. (1969). *Aspects of depression.* Boston: Little & Brown.

Sonis, W. (n.d.). Seasonal affective disorder of childhood and adolescence: A review. In N. Rosenthal & M. Blehar (eds.), *Seasonal affective disorders and phototherapy* (pp. 46-54). New York: Guilford Press.

Sonis, W., Yellin, A., Garfinkel, B., & Haberman, H. (1987). The antidepressant effect of light in seasonal affective disorder of childhood and adolescence. *Psychopharmacology Bulletin, 23*(3), 360-363.

Spitzer, R.L., Endicott, J., & Robbins, E. (1978). Research diagnostic criteria: Rationale and reliability. *Arch. Gen. Psychiatry, 35*, 773-782.

Stinson, D., & Thompson, C. (1990). Clinical experience with phototherapy. *Journal of Affective Disorders, 18*, 129-135.

Takahashi, K. (1991). Multi-center study of seasonal affective disorders in Japan. *Journal of Affective Disorders, 21*(1), 57-65.

Terman, J.S. (1990). Efficacy of brief, intense light exposure for treatment of winter depression. *Psychopharmacology, 26*(1), 3-11.

Terman, J.S., Schlager, D., Fairhurst, S., & Perlman, B. (1989). Dawn and dusk simulation as a therapeutic intervention. *Biological Psychiatry, 25*, 966-970.

Terman, J.S., Schlager, D., Terman, M., Rafferty, B., Rosofshy, M., Link, M., Gallin, P., & Quitkin, F. (1990). Identification, assessment and treatment of seasonality in mood disorders: Efficacy of brief, intense light exposure for treatment of winter depression. *Psychopharmacology Bulletin, 26*(1), 3-11.

Terman, M. (1991). The visual input stage of the mammalian circadian pacemaking system. *Journal of Biological Rhythms, 6*, 31-48.

Terman, M., Remé, C.E., Rafferty, B., Gallin, P.F., & Terman, J.S. (1990). Bright light therapy for winter depression: Potential ocular effects and theoretical implications. *Photochemistry and Photobiology, 51*, 781-792.

Terman, M., Terman, J.S., Quitkin, F.M., Cooper, T.B., Lo, E.S., Gorman, J.M., Stewart, J.W., & McGrath, P.J. (1988). Response of the melatonin cycle to phototherapy for seasonal affective disorder. *Journal of Neural Transmission, 72*, 147-165.

Terman, M., Terman, J.S., Quitkin, F.M., [illegible], Stewart, J.W., & Rafferty, B. (1989). Light therapy for seasonal [illegible]isorder: A review of efficacy. *Neuropsychopharmacology, 2*, 1-22.

Thompson, C. (1986). Seasonal affective disorder and phototherapy: Experience in Britain. Clinical *Neuropharmacology, 9*(Suppl 4), 190-192.

Thompson, C., & Isaacs, G. (1988). Seasonal affective disorder - a British sample. Symptom atology in relation to mode of referral and diagnostic subtype. *Journal of Affective Disorders, 14*, 1-11.

Thompson, C., & Silverstone, T. (eds.)(1989). *Seasonal affective disorder.* London: Clinical Neuroscience Publishers.

Tsuang, M., & Farone, S. (1990). *The genetics of mood disorders.* Baltimore & London: The John Hopkins University Press.

Val, E., Gariria, M., & Flaherty, J. (1982). *Affective disorders: Psychopathology and treatment.* Yearbook Medical Publishers.

Walter, S.D. (1977). Seasonality of mania: A reappraisal. *British Journal of Psychiatry, 131*, 345-350.

Watts, C.A. (1966). *Depressive disorders in the community.* Bristol: John Wright & Sons Ltd.

Watts, C.A. (1973). *Depression: The blue plague.* Bristol: Priority Press Ltd.

Wehr, T., Jacobsen, F., Sack, D., Arendt, J., Tamarkin, L., & Rosenthal, N. (1986). Phototherapy of seasonal affective disorder. *Clinical Psychobiology Branch*, (January).

Wehr, T.A., & Rosenthal, N.E. (1989). Seasonality and affective illness. *American Journal Psychiatry, 146*, 829-839.

Wehr, T.A., Sack, D., & Rosenthal, N. (1985). Antidepressant effects of sleep deprivation and phototherapy. *Acta Psychiatrist Belg., 85*, 593-602.

Wehr, T.A., Sack, D., & Rosenthal, N. (1987a). Seasonal affective disorder: Disorder with summer depression and winter hypomania. *American Journal of Psychiatry, 144*(12), 1602-1603.

Wehr, T.A., Skwerer, R., Jacobsen, F.M., Sack, D., & Rosenthal, N. (1987b). Eye versus skin: Phototherapy of seasonal affective disorder. *American Journal of Psychiatry, 14*(6), 753-757.

Wehr, T.A., & Wirz-Justice, A. (1982). Circadian rhythm mechanisms in affective illness and in antidepressant drug action. *Pharmacopsychiatrist, 15*, 31-39.

Wever, R.A. (1983). Organization of the human circadian system: Internal interactions. In *Circadian Rhythms in Psychiatry*. The Boxwood Press.

Wever, R.A. (1986). Characteristics of circadian rhythms in human functions. *Journal of Neural Transmission, 21*(Suppl.), 323-373.

Wever, R.A., Polasek, J., & Wildgruber, C. (1983). Bright light affects human circadian rhythms. *Pflü Archives, European Journal of Physiology, 396*, 85-87.

White, D.M. (1990). Is winter depression a bipolar disorder? *Comprehensive Psychiatry, 31*(3), 196-204.

Whybrow, P., Akiskal, H., & McKinney, W. (1984). *Mood disorders: Toward a new psychobiology*. New York & London: Plenum Press.

Williams, J.B.W. (1988). A structured intensive guide for the Hamilton depression rating scale. *Archives of General Psychiatry, 45*, 742-747.

Williams, J.B.W., Link, M.J., Rosenthal, N.E., & Terman, M. (1988). *Structured intensive guide for the Hamilton depression scale - seasonal affective disorder version 3*. New York: New York State Psychiatric Institute.

Wirz-Justice, A. (1986a). Light therapy for depression: Present status, problems, and perspectives. *Psychopathology, 19*(Suppl. 2), 136-141.

Wirz-Justice, A. (1987). Light and dark as a drug. In E. Jucker (ed.), *Progress in drug research* (Vol. 31). Switzerland: Birk Hauser Verlag, Psychiatric University Clinic.

Wirz-Justice, A., Bucheli, C., Graw, P., Kielholz, P., Fisch, H.U., & Woggon, B. (1986b). Light treatment of seasonal affective disorder in Switzerland. *Acta Psychiatric Scandinavia, 74*, 193-204.

Wirz-Justice, A., Grain, P., Kräuchi, K., Gesin, B., Arendt, J., Aldhaes, M., & Pödinger, W. (1990). Morning or night-time melatonin is ineffective in seasonal affective disorder. *Journal of Psychiatric Res., 24*(2), 129-137.

Wirz-Justice, A., Schmid, A.C., Graw, P., Kräuchi, K., Kleilholz, P., Pöldinger, W., Fisch, H., & Buddeberg, C. (1987). Dose relationships of morning bright white light in seasonal affective disorders. *Experiencia, 43*, 574-576.

Wurtman, J. (1986). Introduction: Melatonin in humans. *Journal of Neural Transmission, 21*(Suppl.), 1-8.

Wurtman, J. (1990). Carbohydrate craving: Relationship between carbohydrate intake and disorders of mood. *Drugs*, *39*(Suppl. 3), 49-52.

Yerevanian, B., Anderson, J., Grota, L., & Bray, M. (1986). Effects of bright incandescent light on seasonal and nonseasonal depressive disorder. *Psychiatry Research*, *18*, 355-364.

APPENDIX A

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

STRUCTURED INTERVIEW GUIDE FOR
THE HAMILTON DEPRESSION RATING SCALE

SEASONAL AFFECTIVE DISORDERS VERSION
(SIGH-SAD)

STRUCTURED INTERVIEW GUIDE FOR COMPLETION OF THE HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION - SEASONAL AFFECTIVE DISORDER (SIGH-SAD)

INTERVIEWER:
The questions in bold type are tc be read exactly as written. The additional questions are provided for further exploration or clarification as needed. Finally, you may add your own follow-up questions if necessary. Enter each score in the relevant page in the case report form.

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

What's your mood been like this past week (compared to when you feel OK)?

Having you been feeling down or depressed?

Sad? Hopeless? Helpless? Worthless?

In the last week, how often have you felt (OWN EQUIVALENT)? Every day? All day?

Have you been crying at all?

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

DEPRESSED MOOD (sadness, hopeless, helpless, worthless):

0 - absent
1 - indicated only on questioning
2 - spontaneously reported verbally
3 - communicated non-verbally, i.e., facial expression, posture, voice, tendency to weep
4 - VIRTUALLY ONLY; this in spontaneous verbal and non- verbal communication

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

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

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

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

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

Is there anything you look forward to?

WORK AND ACTIVITIES:

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

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

*SOCIAL WITHDRAWAL:

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

How has your interest in sex been this week? (I'm not asking about actual sexual activity, but about your interest in sex – how much you think about it.)

Has there been any change in your interest in sex (from when you were not depressed)?

Is it something you've thought much about? IF NO: Is that unusual for you?

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

0 - absent
1 - mild
2 - severe

How has your appetite been this past week? (What about compared to your usual appetite?)

Have you had to force yourself to eat?

Have other people had to urge you to eat?

Have you had any stomach or intestinal problems? (Have you needed to take anything for that?)

SOMATIC SYMPTOMS GASTROINTESTINAL:

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

Have you lost any weight in the past week? IF YES: Was it because of this depression? How much?

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

LOSS OF WEIGHT (Rate either A or B):

A. When rating by history:

0 - no weight loss
1 - probable weight loss due to current depression
2 - definite (according to patient) weight loss due to depression
3 - not assessed

B. When actual weight changes are measured:

0 - less than 1 lb. loss in week
1 - greater than 1 lb. loss in week
2 - greater than 2 lb. loss in week
3 - not assessed

Have you gained any weight in the last week? IF YES: How much?

*WEIGHT GAIN:

0 - no weight gain
1 - probable weight gain due to current depression
2 - definite (according to patient) weight gain due to depression

In the past week, has your appetite been greater than when you feel well or OK? IF YES: Do you want to eat a little more, somewhat more, or much more than when you feel well or OK?

*APPETITE INCREASE:

0 - no increase in appetite
1 - wants to eat a little more than usual
2 - wants to eat somewhat more than usual
3 - wants to eat much more than usual

In the past week, have you actually been eating more than when you feel well or OK? IF YES: A little more, somewhat more, or much more than when you feel well or OK?

*INCREASED EATING:

0 - is not eating more than usual
1 - is eating a little more than usual
2 - is eating somewhat more than usual
3 - is eating more than normal

In the last week, have you been craving or eating more starches or sugars? IF YES: Have you been eating more starches or sugars than when you feel well or OK, much more, or irresistibly craving them?

*CARBOHYDRATE CRAVING (in relation to total amount of food desired or eaten):

0 - no change in food preference
1 - craving more carbohydrates (starches or sugars) than before
2 - craving much more carbohydrates than before
3 - irresistable craving for sweets or starches

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

Have you had any trouble falling asleep at the beginning of the night? (Right after you go to bed, how long has it been taking you to fall asleep?)

How many nights this week have you had trouble falling asleep?

INSOMNIA EARLY:

0 - no difficult falling asleep
1 - complains of occasional difficulty falling asleep – i.e., more than 1/2 hour
2 - complains of nightly difficulty falling asleep

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

When you get back in bed, are you able to fall right back asleep?

Have you felt your sleeping has been restless or disturbed some nights?

INSOMNIA MIDDLE:

0 - no difficulty
1 - complains of being restless and disturbed during the night
2 - waking during the night – any getting out of bed (except to void)

What time have you been waking up in the morning for the last time, this past week?

IF EARLY: Is that with an alarm clock, or do you just wake up yourself? What time do you usually wake up (that is, before you got depressed?)

INSOMNIA LATE:

0 - no difficulty
1 - waking in early hours of morning but goes back to sleep
2 - unable to fall asleep again if gets out of bed

Have you been sleeping more than usual this past week? IF YES: How much more? IF NO: What about weekends?

***HYPERSOMNIA (Compare sleep length to euthymic and NOT to hypomanic sleep length. If this cannot be established, use 8 hours):**

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

Sleep length used (circle one):

euthymic 8 hour

How has your energy been this past week?

If LOW ENERGY: Have you felt tired? (How much of the time? How bad has it been?)

This week, have you had any aches or pains? (What about backaches, headaches, or muscle aches?)

Have you felt any heaviness in your limbs, back or head?

SOMATIC SYMPTOMS GENERAL:

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

RATING BASED ON QUESTIONS ABOVE

***FATIGUABILITY (or low energy, or feelings of being heavy, leaden, weighted down):**

0 - does not feel more fatigued than usual
1 - feels more fatigued than usual but this has not impared function significantly; less frequent than in (2)
2 - more fatigued than usual; at least one hour a day; at least three days a week
3 - fatigued much of the time most days
4 - fatigued almost all the time

Have you been especially critical of yourself this past week, feeling you've done things wrong, or let others down? IF YES: What have your thoughts been?

Have you been feeling guilty about anything that you've done or not done? What about things that happened a long time ago?

Have you thought that you've brought (THIS DEPRESSION) on yourself in some way?

Do you feel you're punished by being sick?

FEELINGS OF GUILT:

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

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

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

SUICIDE:

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

Have you been feeling especially tense or irritable this past week? IF YES: Is this more than usual for you?

Have you been worrying a lot about little things, things you don't ordinarily worry about? IF YES: Like what, for example?

ANXIETY PSYCHIC:

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

In this past week, have you had any physical symptoms that sometimes go along with being nervous, like (READ LIST, PAUSING AFTER EACH SX FOR REPLY)?

How much have these things been bothering you this past week? (How bad have they gotten? How much of the time, or how often, have you had them?)

NOTE: DON'T RATE IF CLEARLY DUE TO MEDICATION (E.G. DRY MOUTH)

ANXIETY SOMATIC (physiologic concomitants of anxiety, such as

GI - dry mouth, gas, indigestion, diarrhea, cramps, belching
C-V - heart palpitations, headaches
Resp - hyperventilating, sighing

Having to urinate frequently (Sweating):

0 - absent
1 - mild
2 - moderate
3 - severe
4 - incapacitating

In the last week, how much have your thoughts been focused on your physical health or how your body is working (compared to your normal thinking)? (Have you really be preoccupied with this?)

Do you complain much about how you feel physically?

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

HYPOCHONDRIASIS:

0 - not present
1 - self-absorption (bodily)
2 - preoccupation with health
3 - frequent complaints, requests for help, etc.
4 - hypochondriacal delusions

RATING BASED ON OBSERVATION DURING INTERVIEW.

INSIGHT:

0 - acknowledgement being depressed and ill or not currently depressed
1 - acknowledges illness but attributes cause to bad food, overwork, virus, need for rest, etc.
2 - denies being ill at all

RATING BASED ON OBSERVATION DURING INTERVIEW.

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

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

RATING BASED ON OBSERVATION DURING INTERVIEW.

AGITATION:

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

This past week, have you been feeling better or worse in the first few hours after waking up, compared to the last few hours before you go to sleep?

DIURNAL VARIATION TYPE A:

A. Note whether symptoms are worse after awakening or before sleeping. If NO diurnal variation, mark none:

0 - no variation OR not currently depressed
1 - worse after sleeping
2 - worse before going to sleep

RATER NOTE: DO NOT COUNT ABOVE SCORE IN HAMILTON TOTAL

IF VARIATION: How much worse do you feel in the (MORNING or EVENING)? IF UNSURE: A little bit worse or a lot worse?

B. When present, mark the severity of the variation:

0 - none
1 - mild
2 - severe

This week, have you regularly had a slump in your mood or energy in the afternoon or evening?

IF YES: Is that every day? At what time has the slump usually occurred? (———— o'clock). How big a slump -- would you say it's generally mild, moderate, or severe?

*DIURNAL VARIATION TYPE B:

0 - no
1 - yes, of mild intensity
2 - yes, of moderate intensity
3 - yes, of severe intensity

In the past week, have you ever suddenly had the feeling that everything is unreal, or you're in a dream, or cut off from other people in some strange way? Any spacey feelings? IF YES: How bad has that been? How often this week has that happened?

DEPERSONALIZATION AND DEREALIZATION (such as feelings of unreality and nihilistic ideas):

0 - absent
1 - mild
2 - moderate
3 - severe
4 - incapacitating

This past week, have you felt that anyone was trying to give you a hard time or hurt you?

What about talking about you behind your back?

IF YES: Tell me about that.

PARANOID SYMPTOMS:

0 - none
1 - suspicious
2 - ideas of reference
3 - delusions of reference and persecution

In the past week, have there been things you've had to do over and over again, like checking the locks on the doors several times? IF YES: Can you give me an example?

Have you had any thoughts that don't make any sense to you, but that keep running over and over in your mind? IF YES: Can you give me an example?

OBSESSIONAL AND COMPULSIVE SYMPTOMS:

0 - absent
1 - mild
2 - severe

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

TOTAL 8-ITEM S.A.D. SCORE (starred items only):

TOTAL 29-ITEM HAMILTON DEPRESSION SEASONAL AFFECTIVE DISORDER SCORE (including starred items):

APPENDIX B

THE HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION SEASONAL AFFECTIVE DISORDER

INVESTIGATOR _____ PATIENT NO. _____

DATE OF VISIT _____

HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION - SEASONAL AFFECTIVE DISORDER

Please use the structured interview guidelines provided when completing this form. Please complete all 29 observations.

1. DEPRESSED MOOD	☐	0-4
2. FEELINGS OF GUILT	☐	0-4
3. SUICIDE	☐	0-4
4. INSOMNIA EARLY	☐	0-2
5. INSOMNIA MIDDLE	☐	0-2
6. INSOMNIA LATE	☐	0-2
7. WORK AND ACTIVITIES	☐	0-4
8. RETARDATION	☐	0-4
9. AGITATION	☐	0-4
10. ANXIETY PSYCHIC	☐	0-4
11. ANXIETY SOMATIC	☐	0-4
12. SOMATIC SYMPTOMS, GASTRO-INTESTINAL	☐	0-2
13. SOMATIC SYMPTOMS, GENERAL	☐	0-2
14. GENITAL SYMPTOMS	☐	0-2
15. HYPOCHONDRIASIS	☐	0-4
16. INSIGHT (When, in fact, ill)	☐	0-2
17. LOSS OF WEIGHT	☐	0-2
18. DIURNAL VARIATION		
TYPE A	☐	0-2
If present, please indicate whether symptoms are worse in the morning or evening:		
Worse in morning	☐	1
Worse in evening	☐	2

19. DEREALIZATION AND DEPERSONALIZATION	☐	0-4
20. PARANOID SYMPTOMS	☐	0-4
21. OBSESSIONAL SYMPTOMS	☐	0-2
SCORE OF FIRST 21 ITEMS	☐	
22. SOCIAL WITHDRAWAL	☐	0-4
23. WEIGHT GAIN	☐	0-2
24. APPETITE INCREASE	☐	0-3
25. INCREASED EATING	☐	0-3
26. CARBOHYDRATE CRAVING	☐	0-3
27. HYPERSOMNIA	☐	0-4
EUTHYMIC SLEEP (8 HOURS)		
YES	☐	
NO	☐	
28. FATIGABILITY	☐	0-4
29. DIURNAL VARIATION		
TYPE B	☐	0-3
Time of Day Slump Occurs		
_____		
SCORE FOR THE LAST EIGHT ITEMS:	☐	

INVESTIGATOR'S SIGNATURE:

_____