1

MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS
STANDARD REFERENCE MATERIAL 1010a
(ANSI and ISO TEST CHART No. 2)

National Library
of Canada

Acquisitions and
Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale
du Canada

Direction des acquisitions et
des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Notice - Notice

Notice - Notice

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30, and subsequent amendments.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30, et ses amendements subséquents.

Canada

**Phototherapy in Bulimia Nervosa:
The Effects on Eating Behavior and Mood.**

Jacqueline Christine Carter

**A thesis submitted to
The Faculty of Graduate Studies and Research
in partial fulfilment of the requirements for the degree of**

Master of Arts

**Department of Psychology,
Carleton University
Ottawa, Ontario**

August, 1992

National Library
of Canada

Acquisitions and
Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale
du Canada

Direction des acquisitions et
des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Vous l'avez votre référence

Vous l'avez votre référence

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

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

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

ISBN 0-315-89732-5

Canada

UNIVERSITY OF OXFORD

DEPARTMENT OF PSYCHIATRY

WARNEFORD HOSPITAL
OXFORD
OX3 7JX

TEL. (0865) 741717 ext. 26-

TEL. direct line (0865) 2264-

FAX. (0865) 793101

November 8, 1993

Ms. Sneja Mouelhi
Carleton University
Library
Ottawa, Canada

Dear Ms. Mouelhi:

Thank you for your letter dated 29 October regarding the microfilming of my M.A. thesis.

I would like to have my thesis microfilmed without "Appendix B, ED1" (5 pages) and the "Structured Interview Guide..." (10 pages). I have enclosed a note to be included in the microfiche copy explaining why these pages are missing.

Thank you for your assistance.

Sincerely,

A handwritten signature in cursive script, appearing to read 'J Carter'.

Jacqueline Carter

NOTE

**The Eating Disorder Inventory and the Structured Interview
Guide for the Hamilton Depression Rating Scale are not
available because they are copyrighted material.**

The undersigned hereby recommend to
the Faculty of Graduate Studies and Research acceptance of the thesis:

Phototherapy in Bulimia Nervosa:
The Effects on Eating Behavior and Mood
submitted by
Jacqueline Christine Carter, B.A. (Hons.)
in partial fulfilment of the requirements
for the degree of Master of Arts

Chairman, Department of Psychology

Thesis Supervisor

Abstract

Recent research documenting marked seasonal variations in bulimic symptoms which are strikingly similarly to seasonal changes in photoperiod suggests that Bulimia Nervosa may be responsive to alterations in environmental light availability. The purpose of this study was to investigate the effects of phototherapy on eating behavior and mood in Bulimia Nervosa. Eighteen female subjects meeting DSM-III-R criteria for Bulimia Nervosa were randomly assigned to receive either 2500 lux of bright light therapy (experimental condition) or 500 lux of dim light therapy (placebo condition) in a blind fashion for seven consecutive days during the winter months. Bulimic symptomatology, mood and the macronutrient composition of binge meals were monitored for one week prior to, during, and for one week following the experimental manipulation. Mood was measured by means of the Structured Interview Guide for the Hamilton Depression Rating Scale - SAD Version (SIGH-SAD) and the Beck Depression Inventory (BDI) which were administered on a weekly basis. Subjects were also required to complete the Profile of Mood States (POMS) daily. Bulimic symptomatology was measured using a weekly Bulimic Symptoms Checklist (BSC) and a Daily Binge Record (DBR). The results indicated that subjects in the bright light condition exhibited a significant reduction in depressive symptoms one week after phototherapy and a significant relapse in depressive symptoms one week following withdrawal of the light. Subjects in the dim light condition showed no significant changes in affective symptoms. Phototherapy had no significant effect on bulimic behaviors including binge frequency, purge frequency, and the macronutrient content of binge foods. The results are discussed in terms of the physiological mechanisms which regulate affect and food intake in bulimia nervosa and seasonal affective disorder.

Table of Contents

	page
Abstract.....	i
Acknowledgements.....	ii
List of Tables.....	iii
List of Figures.....	iv
List of Appendices.....	v
Introduction.....	1
Bulimia Nervosa.....	1
The Prevalence of Bulimia Nervosa.....	7
Eating Behavior in Bulimia Nervosa.....	11
Bulimia Nervosa and Affective Disorder.....	15
Seasonal Variations in Mood and Behavior.....	22
Seasonal Affective Disorder.....	24
Serotonin Regulation in Bulimia Nervosa and Seasonal Affective Disorder.....	29
Seasonal Variations in Bulimia Nervosa.....	32
Animal Models of Human Seasonality.....	34
Phototherapy for Seasonal Affective Disorder.....	37
Specific Parameters of Light Therapy.....	45
The Mechanism of Action of Phototherapy.....	53
Phototherapy in Non-seasonal Depression and in Normals.....	55
The Effects of Phototherapy on Eating Behavior.....	58
Rationale.....	61
Method.....	64
(i) Subjects.....	64
(ii) Materials.....	65
(iii) Procedure.....	69
(iv) Statistical Analyses.....	71
Results.....	73

Discussion.....	89
References.....	98

Acknowledgements

I feel very indebted to my supervisor, Dr. Arthur Blouin, not only for his help with this thesis but for all of the opportunities he has provided for me over the course of the past several years. He has been very generous and I am truly grateful to him. I would also like to thank Dr. Jane Blouin for her statistical advice and helpful assistance with this project. Several other individuals made significant contributions to this study including Hilary Iversen, Gary Goldfield and Cathy Goldstein. Their assistance was greatly appreciated. Thanks also to Joel Ginsburg and Kim Schnare-Hayes for their support and encouragement. Finally, I would like to thank Frank Schiller for being a constant source of love and support.

List of Tables

<u>Table</u>	<u>page</u>
1 Basic Descriptive Characteristics of the Subjects.....	74
2 Analysis of the 21-item Hamilton Depression Rating Scale.....	84

List of Figures

<u>Figure</u>	<u>page</u>
1 Seasonal Variations in Binge Eating.....	77
2 Seasonal Variations in Purging.....	78
3 Seasonal Variations in Mood.....	79
4 The Effect of Phototherapy on the 21-item Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.....	81
5 The Effect of Phototherapy on the 17-item Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.....	82
6 The Effect of Phototherapy on the 8-item SAD total of the Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.....	83
7 The Effect of Phototherapy on the Beck Depression Inventory in Bulimia Nervosa.....	86
8 The Effect of Phototherapy on the Profile of Mood States (POMS) in Bulimia Nervosa.....	87

List of Appendices

	page
Appendix A: Informed Consent.....	114
Appendix B: Psychological Inventories.....	117

Introduction

Recent research has documented that the symptoms of bulimia nervosa evidence a distinct seasonal pattern with symptoms being significantly exacerbated during the winter months (Blouin et al., 1991). This research was prompted by several lines of evidence: first, there is a high co-morbidity between bulimia nervosa and affective disorder; second, there is a qualitative similarity between the symptoms of bulimia nervosa and seasonal affective disorder (SAD) including disturbances of food intake regulation; and third, seasonal variations in mood and behavior have been documented among the general population. Furthermore, bulimia nervosa and SAD have been shown to be responsive to treatments which activate central serotonin activity suggesting a common underlying pathophysiology (O'Rourke, Wurtman, Wurtman, Chebli & Gleason, 1989; Blouin, Blouin, Perez et al., 1988). In addition, serotonin levels have been shown to vary seasonally in a pattern similar to seasonal changes in photoperiod (Carlsson, Svennerholm & Winblad, 1980). Given the fact that phototherapy is known to be an effective treatment for SAD, it is likely that SAD symptoms are mediated by seasonal changes in light availability. Considering the strong association between bulimic symptoms and photoperiod (Blouin et al., 1991), it is reasonable to suggest that seasonal variations in bulimia nervosa may also be mediated by light availability. Thus, the purpose of this investigation was to study the effects of phototherapy on eating behavior and mood in bulimia nervosa.

Bulimia Nervosa

Bulimia nervosa is a psychiatric disorder characterized by three core

diagnostic features (American Psychological Association, APA, 1987). First, there are recurrent episodes of binge eating, the broadly defined term which is widely used to denote the cardinal symptom of this eating disorder. Binge eating is commonly defined as the rapid consumption of large amounts of food in a discrete period of time and is associated with a feeling of a loss of control over one's eating behavior.

The second key feature of bulimia nervosa involves pathological attempts to control shape and weight which may include self-induced vomiting, laxative or diuretic abuse, fasting or excessive exercise. Often, these behaviours occur in combination and like binge eating, their frequency varies among patients.

Finally, a disturbed set of attitudes involving excessive concerns about weight and shape is the third key feature of the disorder. These attitudes have been described as "a morbid fear of becoming fat" (Russell, 1979) or as a "persistent overconcern with weight and shape" (APA, 1987). Clearly, the psychological disturbance regarding weight and shape represents an extreme form of widely held social attitudes regarding the importance of bodily thinness to physical attractiveness. What distinguishes the attitudes characteristic of eating disorders as pathological is their intensity. It has been suggested that the other defining characteristics of bulimia nervosa may be seen as secondary to this "core psychopathology" (Fairburn & Garner, 1988). The DSM-III-R diagnostic criteria for bulimia nervosa are shown below:

- A. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time).
- B. A feeling of lack of control over eating behavior during the eating binges.

- C. The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.
- D. A minimum average of two binge eating episodes a week for at least three months.
- E. Persistent overconcern with body shape and weight.

In contrast to the diagnostic criteria presented in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, APA, 1980) which have been criticized for being overinclusive (Herzog & Gordon, 1985; Fairburn & Beglin, 1990), the revised criteria specify a minimum frequency of two binge eating episodes per week for at least three months and include purging as a required symptom (DSM-III-R, APA, 1987). In addition, the importance of attitudes to weight and shape are recognized. However, the criteria remain inadequate in clearly defining exactly what constitutes a binge episode such as a minimum amount of calories or food consumed during a binge or a specified length of a binge episode.

The onset of bulimia often occurs in association with a period of increased concern with body weight and with voluntary restrictive dieting. Several researchers have suggested that stringent dieting may actually cause binge eating since the deprivation associated with dieting may make the dieter vulnerable to give in to food cravings and to subsequently overeat (Polivy & Herman, 1985). Concerning the onset of bulimia in a group of female patients under investigation, Johnson, Stuckey, Lewis & Schwartz (1982, p.10) state: "Both the physiological and psychological deprivation involved in restrictive dieting reportedly triggered binge eating behavior that became increasingly

uncontrollable".

The average age at the onset of bulimia is approximately 18 years (Muuss, 1986; Pyle, Mitchell & Eckert, 1981). However, patients usually do not seek treatment until several years later. The average duration of the disorder is reported to be between four and seven years by the time initial treatment is sought (Mitchell, Pyle & Eckert, 1981; Mitchell, Hatsukami, Eckert & Pyle, 1985). Purging behaviours begin approximately one year later and are associated with more severe and frequent binge episodes (Johnson, Stuckey Lewis & Schwartz, 1983). However, the onset of vomiting has been found to precede the onset of bingeing (Abraham & Beumont, 1982).

Bulimic episodes typically occur on a daily basis. A descriptive survey of 275 patients who met DSM-III criteria for bulimia found that 82% were binge eating at least once daily and that 72% were vomiting daily (Mitchell et al., 1985). Binge episodes are often planned, highly secretive events with most bulimics preferring to binge at home alone during the late afternoon or evening (Pyle et al. 1981). For most, bingeing is a source of shame and self-disgust. Using self-report measures, investigators have identified several mood states which appear to be correlated with the onset of binge episodes. These include: tension, eating something, being alone, thinking about food, craving specific foods and feeling unhappy (Abraham & Beumont, 1982; Pyle et al., 1981).

A majority of bulimics will attempt to "undo" the effects of overeating and to prevent weight gain through some means of purging. Purging is believed to negatively reinforce bingeing behavior by providing a sense of relief from anxiety and discomfort (Rosen & Leitenberg, 1984). Self-induced vomiting, the

most common purging method, occurs on average once or twice daily (Johnson & Larson, 1982; Mitchell, Hatsutkami, Pyle and Eckert, 1986) with vomiting episodes lasting from five to thirty minutes (Abraham & Beumont, 1982).

In addition to the three key diagnostic symptoms of bulimia nervosa, a wide range of associated features may or may not be present. The clinical features of bulimia nervosa are characterized by heterogeneity. Most commonly, they include depression, anxiety, impaired social functioning, alcohol/drug abuse, self-injurious behavior and personality disorders (Fairburn, 1991).

Researchers have studied the personality traits associated with bulimia. Generally, it has been found that eating disordered individuals tend to be rigidly perfectionistic, high achieving, and academically and vocationally successful (Muuss, 1986). Their perfectionistic thinking may contribute to the maintenance of their eating disorder since they often set unrealistically high standards in terms of dieting and weight loss which inevitably lead to feelings of failure and desperation, feelings which can serve to perpetuate the disorder.

Weiss and Ebert (1983) investigated the psychological characteristics and behavioural patterns of a group of bulimic women in order to determine the extent and nature of psychopathology in bulimia. Compared to a control group, bulimics scored higher (i.e. indicated more pathology) on all nine symptom dimensions of the Hopkins Symptom Checklist-90 including somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, paranoid ideation and psychoticism (Derogatis, Lipman, Rickels et al., 1974). As well, bulimics reported engaging in significantly more impulsive behaviours including stealing, drug and alcohol abuse and suicide attempts. These results are consistent with those reported by other researchers who found higher levels of

depression, more frequent histories of drug or alcohol abuse and of stealing behavior among bulimics (Pyle et al., 1983; Mitchell et al., 1986).

A number of medical complications are associated with bulimia nervosa. The medical problems range from mild discomforts to serious life-threatening disturbances. Frequently reported health problems include tiredness, lethargy, bloating, stomach disorders, sore throat following vomiting, dental erosion from gastric acids, finger calluses from inducing the gag reflex and menstrual irregularities (Mitchell et al., 1985; Pyle et al., 1981; Johnson et al., 1982). The more serious medical consequences include electrolyte imbalances, gastrointestinal disturbances, kidney dysfunction, cardiovascular problems and renal dysfunction (Kaplan & Woodside, 1987; Gvirtzman, Roy-Byrne, Yager & Gerner, 1983).

The role of the family environment in the etiology of bulimia nervosa has been the focus of a considerable amount of research. Johnson and Flach (1985) examined the family characteristics of 105 patients with bulimia. According to their observations, families in which an individual with bulimia was present were characterized by low cohesion, a lack of independent behavior, a high degree of conflict and an absence of open expression. More recently, Blouin, Zuro and Blouin (1990) investigated family environment in 99 women meeting DSM-III criteria for bulimia and 33 normal control subjects using a self-report questionnaire. Overall, the bulimics perceived their families as less cohesive, less independent, more oriented toward achievement, less expressive, and less involved in recreational activities than did controls. However, this difference was only true for depressed bulimics while non-depressed bulimics did not differ significantly from controls. One difficulty with these types of studies is that it is

difficult to determine whether the disturbed family relations existed premorbidly or whether they developed as a consequence of the eating disorder.

Preliminary evidence that bulimia occurs more frequently in the biological relatives of patients with eating disorders and that the concordance rates for bulimia nervosa are significantly higher for monozygotic, as compared to dizygotic twins, suggests the possibility of genetic/biological predisposition mechanisms (Fichter & Noegel, 1990; Hsu, Chesler & Santhouse, 1990).

The Prevalence of Bulimia Nervosa

Based upon a recent review of research on the epidemiology of bulimia nervosa, Fairburn and Beglin (1990) observed that progressively lower estimates of the prevalence of the disorder have been obtained over the past decade. "The most sophisticated studies are impressively consistent in finding that bulimia nervosa appears to have a prevalence rate of about one percent among adolescent and young adult women" (Fairburn & Beglin, 1990, p.406). These authors point to two main reasons for the increasingly convergent prevalence estimates: improved methods of case detection and refinement of the diagnostic criteria for bulimia. Past estimates have ranged from under one percent to nineteen percent of females studied being classified as bulimic (Szmukler, 1985). Such inconsistencies are largely due to differences in the criteria used to define bulimia nervosa, to different methods of case detection, and to differences in the populations studied (Mitchell & Eckert, 1987).

Due to some of the problems with the broad nature of the DSM-III criteria discussed previously, studies employing these criteria have been criticized for classifying people as bulimic who do not resemble those in clinical

populations who are seeking treatment (Ben-Tovim, Subbiah, Scheutz & Morton, 1989). Consequently, many researchers have used additional constraints such as the requirement of purging behavior or a minimum frequency of binge eating. As a result, those studies employing the most stringent criteria report the lowest prevalence rates. The impact of the revisions seen in the DSM-III-R criteria for bulimia on prevalence rates is similar since they are more narrow.

Fairburn and Beglin (1990) criticize the epidemiologic work to date for its heavy reliance on self-report questionnaires. They recommend the inclusion of clinical diagnostic interviews as well as an increased emphasis on testing the efficacy of paper-and-pencil screening instruments. Definitional problems inherent in the diagnostic criteria (e.g. binge) make the ability of self-report questionnaires to detect cases in the community which resemble those in seen in clinics suspect (Ben-Tovim et al., 1989). Since bulimia is inherently secretive, prevalence studies are also complicated by the reluctance of some people to admit that they engage in disturbed eating behaviours as well as by the difficulty identifying individuals with bulimia since there is no apparent weight loss (George, Weiss & Jimerson, 1985). Another factor which may be contributing to discrepant findings is inadequate sample sizes.

As discussed previously, while most bulimic symptoms begin during adolescence, the majority do not seek treatment until several years later. Therefore, many studies have examined the prevalence of eating disorders among high school and college student populations. An investigation of the prevalence of bulimia among 1268 female high school students found that 8.3 percent met DSM-III criteria for bulimia. (Johnson, Lewis Love, Lewis & Stuckey, 1984). When the additional requirement of at least weekly bingeing and

purging was included, the prevalence rate fell to 0.9 percent.

A study of the prevalence of bulimia in a college population found that 13 percent of 355 summer students surveyed at an American university met DSM-III criteria for bulimia and that almost 10 percent reported using self-induced vomiting (Halmi, Falk & Schwartz, 1981). Strangler and Printz (1980) reviewed the psychiatric diagnoses of 500 students at a college psychiatric clinic and found that 3.8 percent of the sample fulfilled DSM-III criteria for bulimia. Using the same criteria, another study reported that 4.1 percent of a group of 1355 college freshmen were bulimic (7.8 percent of the females surveyed and 1.4 percent of the males) (Pyle et al., 1983). When the requirement of at least weekly bingeing was included in this study, the prevalence rate dropped to 2.1 percent and when the requirement of at least weekly vomiting was added it fell to 0.6 percent of the population studied.

A large-scale epidemiologic study found that between one and two percent of 5108 high school students surveyed met DSM-III criteria for bulimia and that less than one percent of the population engaged in self-induced vomiting or laxative use (Whitaker et al., 1989). Based upon these findings, it was concluded that "contrary to concern in the clinical literature adolescent eating disorders may not be epidemic" (Whitaker et al., 1989, p.156).

Since students may not be representative of the general population, several studies have used community samples. Using Russell's (1979) diagnostic criteria, described previously, Fairburn and Cooper (1983) reported a prevalence rate for 'bulimia nervosa' of 1.9 percent among 369 consecutive female clients at family planning clinic, 75 percent of whom were neither secondary school nor college students. Another community study distributed self-report questionnaires to a

random sample of 300 female shoppers. Results indicated that 10.3 percent met DSM-III criteria for bulimia (Pope, Hudson & Yurgelun-Todd, 1984). When bingeing and purging at least once a week were included for diagnosis, only 3 percent met the criteria. The authors note that a lifetime history of bulimia was most common among the youngest age group suggesting that the disorder is increasing in prevalence.

Finally, a recent Australian study investigated the incidence of bulimia in three subgroups of a community population: 387 female shoppers in a large shopping mall; 823 female high school students; and 220 female patients at a general practice medical clinic (Ben-Tovin et al., 1989). Among the combined community sample, 13 percent fulfilled DSM-III criteria for bulimia while only 1.4 percent met the DSM-III-R requirements for diagnosis.

Despite the well-known female predominance in bulimia, the disorder does not occur exclusively in females. Epidemiological work to date indicates that the prevalence of bulimia nervosa in males is between 0.2 percent and 1.1 percent (Carlat & Camargo, 1991). Based upon a review of all prevalence studies to date, Carlet and Camargo (1991) report that the female-to-male prevalence ratios range from 1.5 to 20.0 or that males make up approximately 10 percent to 15 percent of bulimic individuals in the community. It has been suggested that the male/female discrepancy in the prevalence of bulimia may be partly due to the fact that women may be more likely to seek treatment since men may be reluctant to admit to having a stereotypically feminine disorder (Mitchell & Goff, 1984).

Evidently, contradictory evidence exists regarding the prevalence of bulimia. The discrepancies illustrated above point to the importance of

considering the stringency of the criteria used to define bulimia, the method of case detection as well as the nature of the target population under study. Overall, it appears that between one and four percent of young women meet strict DSM-III-R diagnostic criteria for bulimia nervosa.

Eating Behavior in Bulimia Nervosa

Investigations aimed at studying the nature of eating behavior in bulimia nervosa have been approached from two methodological approaches: 1) self-monitoring in the natural environment and 2) experimental study of eating behavior in a laboratory setting. Until recently, standard descriptions of the eating behavior of bulimics were largely derived from the recollections of the patients. Descriptive studies based on self-report have found that patients typically report consuming several thousand calories during a binge usually in the form of high caloric easily ingested foods containing large amounts of carbohydrates, precisely the type of foods which they usually refrain from eating during non-binge times (Mitchell et al., 1981; Abraham & Beumont, 1982; Weiss & Ebert, 1983). A large degree of inter-individual and intra-individual variation in terms of the frequency and duration of binge eating as well as the amount and type of foods consumed has been reported (Johnson et al., 1982). Individual binges last an average of one hour and occur anywhere from two to twenty times per week (Pyle et al., 1981) during which time a range of approximately 1000 to 55,000 calories are consumed, the average intake being approximately 4800 calories (Johnson et al., 1982). Typical binge foods include ice cream, pastries, candy, doughnuts, cakes and cookies while non-binge meals are typically restricted to low-calorie fruits and vegetables (Abraham &

Beumont, 1982; Mitchell & Laine, 1985). Binge episodes are commonly alternated with periods of fasting or strict dieting at which time patients tend to avoid the “carbohydrate-rich” foods that they associate with binge eating (Weiss & Ebert, 1983).

Woell, Fichter, Pirke, and Wolfram (1989) analyzed the self-monitored food records (24 hour recall method) of 30 patients with bulimia. According to the results, the patients had an average frequency of 16 binge episodes per week (the range was from 0 to 54). The average energy intake during a binge was 1945 kilocalories. The nutrient composition of binge foods was found to be: carbohydrates 42%, fat 43%, and protein 12%. These values are very similar to those obtained by other researchers (Mitchell & Laine, 1985; Rosen, Leitenberg, Fisher, & Khazam, 1986).

More recently, researchers concerned with the validity and reliability of patient self-reports have developed methods that allow the objective measurement of eating behavior in the laboratory (Walsh, Kisseliff, Cassidy & Dantzic, 1989; Mitchell, Morley et al., 1989). Laboratory paradigms have made it possible to determine the answer to such questions as whether the amount of food consumed or the subjective experience of a bulimic during a binge is significantly different from that of a non-eating disordered individual during an episode of overeating. Other important questions include whether the disturbance in eating behavior is confined to binge episodes or if there is a more general abnormality in food intake regulation and whether binge episodes differ from non-binge episodes in terms of macronutrient composition.

Kaye and Gwirtsman (1985) attempted to verify the accuracy of patients self-reports concerning the pattern of food intake during bulimic episodes by

observing and documenting their behavior in the laboratory. Results indicated that self-report measures were generally accurate. However, the authors note that while patients report that binges consist primarily of high carbohydrate foods, large amounts of protein and fat are also consumed. Overall though, it was found that on average binge meals were composed of 52% carbohydrates.

Walsh et al. (1989) studied the eating behavior of twelve women with bulimia nervosa and ten non-eating disordered controls in an experimental paradigm. Subjects consumed a series of four test meals (either single course or multicourse) and were instructed either to binge or to eat as much as they would during a "normal" meal. Measures included the amount and macronutrient composition of food consumed, the rate of eating, as well as various subjective measures such as mood, hunger and satiety. In addition, subjects rated how typical each meal was of a binge episode.

The results of this study indicated that although both groups exhibited a statistically significant increase in caloric intake when instructed to binge eat, bulimic patients increased their consumption significantly more than controls. When instructed to eat a normal meal, however, bulimics consumed less than controls in terms of total calories and fat intake (not statistically significant). Of note, patients rated themselves as hungrier than controls after both binge and non-binge meals despite the fact that their average caloric intakes were larger and their hunger ratings prior to eating were not different. Also, patients tended to increase their rate of consumption as the meal progressed while controls decreased their rate of eating.

In terms of nutrient content, the only statistically significant difference was for percent protein content with a trend for percent fat which indicated that

both groups obtained a smaller portion of energy from protein and a larger portion from fat during binge meals. Of note, the binge and non-binge meals were similar in terms of the proportion of carbohydrate consumed. These results were replicated in a study using a similar methodology (Hadigan, Kisseliff & Walsh, 1989).

There are several limitations inherent in laboratory studies of this type. First, the subjects' eating behavior is probably influenced by the controlled, unfamiliar environment of a laboratory making the generalizability of the results to their natural settings questionable. Perhaps only a select group of patients would be willing to be observed in this manner especially given the shame with which binge eating is usually associated for bulimics. In addition, patients may alter their behavior to conform with experimenter expectations. Also, patients are requested to eat at a specific time of day in the laboratory which may not conform to their usual eating patterns and may alter their behavior. Self-monitoring may be a better alternative for dealing with such confounds although patients may still distort their behavior since they know it will be evaluated by the researchers. Nonetheless, the results of laboratory studies and self-report studies have provided a consistent overall picture of eating behavior in bulimia nervosa which is consistent with patient descriptions of their eating disturbance.

In summary, research on the eating behavior of individuals with bulimia nervosa has found that bulimics consume significantly more than controls when asked to binge and that the foods consumed during a binge do not differ significantly from foods consumed during a normal meal. In addition, the increased hunger of bulimics compared to controls after eating and their progressively increased rate of eating during a meal are indicative of a

disturbance in satiety mechanisms.

The high co-morbidity between bulimia nervosa and major depression has promoted research investigating the possibility of a relationship between the eating disturbances in bulimia and disturbances of mood.

Bulimia Nervosa and Affective Disorder

Depression is a common associated feature of bulimia nervosa with the incidence of major affective disorder among bulimics being as high as 70% (Walsh, Roose & Glassman, 1985; Hudson, Pope, Jonas & Yurgelun-Todd, 1983; Blouin, McAffer, Blouin & Perez, 1986). Consequently, widespread research has attempted to define an association between bulimia and affective disorders. Traditionally, mood disturbances were viewed as being secondary to the eating disorder (Swift, Andrews & Barklage, 1986). However, disturbances of appetite and weight regulation are also primary features of clinical depression (Rosenthal & Heffernan, 1986). Evidence to suggest an overlap between bulimia and affective disorders in terms of symptomatology and underlying pathophysiology has lead researchers to suggest that bulimia may, in fact, be a variant of affective disorder (Hinz & Williamson, 1987). This theory has been investigated through descriptive, family history, neuroendocrine and pharmacological investigations.

Descriptive studies have suggested that depression is prominent in the clinical presentation of bulimia. Russell (1979) reported that 26 of the 30 bulimic patients in his investigation were at least moderately depressed, 37% of whom had made previous suicide attempts. Viessalman and Roig (1985) reported that 79% of 61 bulimic patients had histories of major depression. Based upon structured diagnostic interviews with 50 bulimic women, another study found

that 88% met lifetime criteria for an affective disorder (Walsh, Roose, Glassman, Gladis & Sadik, 1985).

Evidently, the lifetime prevalence of affective disorder among bulimic patients is quite high. However, the causal and temporal relationship between the disorders remains unclear. The depressive symptoms cannot be explained as simply a reaction to the direct effects of the eating disorder since depression may precede or follow the onset of bulimic symptoms (Pope, Hudson & Yurgelun-Todd, 1989). For example, Walsh et al. (1985) reported that 26% of subjects in their study had had an episode of major depression an average of three years before the onset of the eating disorder.

Family history studies revealing a high incidence of affective disorder among the first-degree relatives of bulimic patients have been cited as providing further evidence for the affective variant hypothesis. Hudson, Laffer and Pope (1982) examined the incidence of affective disorder among 46 first-degree relatives of 10 bulimic patients. Based upon psychiatric information obtained through semi-structured interviews with the patients and with available family members, 15% were given a probable diagnosis of major depression. The morbid risk for major depression among the family members of bulimics was similar to that of a reference group with bipolar affective disorder (11%) but significantly different from a comparison group of nondepressed psychiatric patients (1%). Hudson, Pope and Jonas (1983) later replicated these findings in an investigation of 420 first-degree relatives of 89 eating disorder patients, 55 of whom had bulimia. More recently, however, a controlled investigation failed to find positive results (Stern et al., 1984). The lifetime prevalence of affective disorder among first-and-second-degree relatives of 27 bulimic women (9%) was

similar to the prevalence among 27 women with no history of an eating disorder (10%).

The rationale behind family history studies is based on considerable evidence suggesting a genetic basis for affective disorder (Hinz & Williamson, 1987). Therefore, it is argued that a family history of depression among bulimic patients would suggest that bulimia is a form of affective disorder. However, according to Cooper and Fairburn (1983): "the only conclusion that can be drawn from this observation is that a family history of affective disorder predisposes individuals to develop an eating disorder" (p.97). Furthermore, this predisposition may not be due to genetic factors but rather to the effects of family environment (Swift, Andrews & Barklage, 1986).

The results of laboratory studies employing the dexamethasone suppression test (DST), which is considered to be a biological marker for endogenous depression, are believed to provide further evidence of an etiological link between bulimia and affective disorder. Approximately 50% of patients with major depression as opposed to only 4% of normals show abnormal DST results (Gwartzman, Roy-Byrne, Yager & Gerner, 1983). Several investigators have found similar rates of cortisol nonsuppression among subjects with bulimia (Hudson et al., 1982; Gwartzman et al., 1983). However, a recent investigation provided evidence that the high incidence of DST nonsuppression in bulimia is related to the presence weight fluctuation and suboptimal weight frequently observed in this population (Perez, Blouin & Blouin, 1988).

The apparent association between bulimia and depression has prompted researchers to investigate the effectiveness of antidepressant medication in the treatment of bulimia with generally successful results. Pope, Hudson, Jonas and

Yurgelun-Todd (1983) first reported that tricyclic antidepressants were effective in treating bulimia. In this placebo-controlled, double-blind study, eight out of nine imipramine treated subjects reported a significant (70%) reduction in binge eating as opposed to only one of ten placebo subjects. In addition, there was a significant decrease in depressive symptoms which was highly correlated with the reduction in bulimic symptoms. On the basis of these findings the authors concluded: "these results augment the growing evidence that bulimia may represent a form of affective disorder" (Pope et al., 1983, p. 558). However, antidepressants are effective for treating non-depressed bulimics as well. A recent double-blind cross-over study employing desipramine, a tricyclic antidepressant which inhibits noradrenalin re-uptake, found that although the drug was effective in reducing the frequency of bingeing and purging among 24 DSM-III bulimic patients, it had no significant effect on depressive symptoms (Barlow, Blouin, Blouin & Perez, 1988). The researchers concluded: "As desipramine's antibulimic effect was not accompanied by an alleviation of depressive symptoms, its effectiveness in relieving bulimic symptoms appears independent of desipramine's known antidepressant properties" (Barlow et al., 1988, p. 132). Blouin, Blouin, Perez & Barlow (1989) replicated these findings and suggested that the antibulimic effects of desipramine may be due to its effects on neurochemical processes known to control food intake rather than its antidepressant actions.

Typically, antidepressant medications are associated with increased appetite, carbohydrate craving and weight gain. However, among patients who report atypical depressive symptoms such as overeating, antidepressants have the opposite effect (Fernstrom, 1989). According to Kaye et al. (1987), it is possible

that anti-depressants work to enhance satiety through their effects on serotonin. Serotonin is a neurotransmitter which has been demonstrated, through both animal and human research, to have effects on both food intake and mood. Treatments which increase serotonin neurotransmission or stimulate serotonin receptors have been shown to cause a significant decrease in the size and length of meals (that is, to enhance satiety) as well as a reduction in the rate of eating (Samanin & Garattini, 1989). In addition, levels of brain serotonin have been found to be reduced in patients with depression and there is evidence that medications which increase serotonergic activity have an antidepressant effect. This is consistent with a hypothesis that bulimia involves a deficit in central nervous system (CNS) serotonin availability and that antidepressants may alleviate bulimic symptoms by increasing serotonin levels. This possibility will be discussed later in this paper.

In terms of eating behavior and weight regulation, two main patterns are observed in affective disorder patients: decreased appetite and weight loss (classical depression) or increased appetite and weight gain (atypical depression) (Rosenthal & Heffernan, 1986). Paykel (1977) examined patterns of appetite change among 208 depressed patients. It was found that 14% of the patients experienced increased appetite as part of their depressive episode. These atypical patients also differed from the others in that they were predominantly women (93%) and were more mildly depressed.

Harris, Young, and Hughes (1984) studied symptoms of appetite and weight change among 193 depressed outpatients. Weight was increased in 53.6% of patients, unchanged in 8.8% and decreased in 37.6%. Appetite was increased in 27% of patients, unchanged in 19% and decreased in 54%. It was

found that the nature of changes in appetite and weight were associated with previous history. Specifically, weight increase was associated with a history of variable weight while weight loss was associated with a steady weight history. Similarly, appetite increase was associated with previous overeating in times of stress. There was no overall association with endogenous features or gender which is inconsistent with the findings of Paykel (1977).

Investigations which have examined the nature of the depressive symptoms associated with bulimia have generally found that their symptoms are often qualitatively different from those observed in non-eating disordered patients with classical depression. Johnson and Larson (1982) described the clinical presentation of depression among fifteen bulimic women as "an agitated depressive state characterized by frequent weight fluctuations" (p.348). Similarly, Weiss and Ebert (1983) noted that symptoms characteristic of endogenous depression were generally not present among the fifteen bulimics in their study.

In the only investigation which attempted to systematically categorize the most recent episodes of major depression experienced by 31 female bulimic patients, it was reported that "only a minority of the episodes were of the 'classical' endogenous type" (Walsh et al., 1985, p.127). Over one-third were classified as having atypical depression. Viessalman and Roig (1985) found that 100% of 25 bulimics reported overeating in association with depression and that 52% reported hypersomnia (rather than insomnia which is characteristic of classical depression).

As discussed by Rosenthal and Heffernan (1986), researchers studying affective disorders often pay insufficient attention to appetite and weight

disturbances since many standard tests do not ask specifically about such atypical symptoms as overeating and weight gain. A recent study illustrates this problem (Pope, Hudson & Yurgelun-Todd, 1989). The Hamilton Rating Scale for Depression (HAM-D) was employed to compare the depressive symptoms of 45 bulimic subjects with those reported by 21 affective disorder patients and 27 non-psychiatric controls. Results indicated "virtually no difference between bulimic and depressed subjects in the character of their depressive symptoms" (p.97). However, the HAM-D rates only *decreased* appetite and weight *loss* as depressive symptoms and thus, would not rate atypical symptoms such as hyperphasia or weight gain as indicative of increased depression.

According to Rosenthal and Heffernan (1986): "The best explanation for the symptomatic overlap (between bulimia and affective disorder) is that eating behavior and mood regulation probably share neurobiological substrates which when disturbed, produce both types of symptoms" (p.148). Researchers have also speculated that manipulations of eating behavior in bulimia may represent an attempt to cope with mood disturbances and that binge eating may be perpetuated by the emotional relief it provides (Walsh, Gladis, & Roose, 1987). However, studies addressing the issue of the relationship between binge eating and mood have clearly established that bulimics experience a deterioration in mood following binge episodes (Johnson & Larson, 1982; Davis, Freeman & Solyom, 1985; Johnson-Sabine, Wood & Wakeling, 1984).

Recent evidence suggests that related mechanisms may be involved in the disturbances of appetite and mood seen in seasonal affective disorder (SAD), a special type of atypical depression characterized by recurrent periods of depression during the fall/winter accompanied by increased appetite,

carbohydrate-craving and weight gain (Rosenthal et al., 1984). Indeed, given the similarity of the defining symptoms of SAD to those seen in bulimia nervosa as well as the high prevalence of mood disorder in bulimia, it is possible that the mechanisms underlying seasonal changes in eating behavior and mood are related to the pathophysiology of bulimia (Rosenthal, Genhart, Sack et al., 1987).

Seasonal Variations in Mood and Behavior

Seasonal changes in sociological, psychological, physiological and biochemical variables in normal subjects have been well-documented (Lacoste & Wirz-Justice, 1989; Souetre et al., 1987). These factors include rates of birth, growth, suicide, and death from natural causes as well as sleeping behavior, thermoregulation, mood, appetite, weight gain and food choice (Lacoste & Wirz-Justice, 1989; Vanstaveren, Deurenberg, Burema, DeGroot & Hautvast, 1986).

Epidemiological research has revealed that a large proportion of the general population experiences seasonal changes in mood, energy and appetite and that among a sub-group, these changes reach such a level of severity in the winter months to warrant a diagnosis of Seasonal Affective Disorder. A telephone survey in the Washington, DC area found that within a random sample of 416 citizens, 92% reported seasonal variations in mood, energy level, sleep length, appetite and food preference similar to the pattern of changes seen in SAD patients but to a less severe degree (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989). For 4.3% to 10% of the subjects in this sample, seasonal changes were reported to cause a degree of impairment equal to that of patients with SAD. The effect of age and gender on degree of seasonality was also examined with results indicating that younger women reported the greatest

amount of seasonal changes. Thus, there appears to exist a continuum of seasonal variations ranging from people who report minimal seasonal changes to SAD patients who show "an exaggerated behavioural response to the seasons" (Rosenthal, Genhart, Jacobsen, et al., 1987, p. 216).

A second survey conducted in New York City confirmed the widespread experience of seasonal variations (Terman, 1988). Of note, seasonal changes in vegetative states (e.g., appetite) were more prominent than fluctuations in mood and occurred regardless of mood variations.

A longitudinal study of seasonal variations in food intake, physical activity and body weight among 114 young adult women failed to demonstrate a seasonal effect on mean energy intake (Vanstaveren et al., 1986). A 24-hour recall method was employed such that subjects were interviewed at one-month intervals for a period of 14 months and then at two-month intervals for a subsequent 12 months. There was however, a seasonal effect for the proportion of energy obtained by particular macronutrients: in summer significantly less fat and more carbohydrates were consumed compared to winter. In addition, mean body weight tended to increase in the winter and decrease in spring. This decrease in body weight was accompanied by a small increase in physical activity which could not alone account for changes in weight.

Scientific interest in seasonal variations of mood and behavior has peaked in the past decade with the recognition of seasonal affective disorder (SAD). It appears as though SAD represents an extreme point in a spectrum of seasonally changing phenomena which occur in the general population (Kasper, Wehr, Bartko, Gaist & Rosenthal, 1989).

Seasonal Affective Disorder

SAD was first defined by Rosenthal et al. (1984) on the basis of a descriptive investigation of 29 subjects who had experienced at least two consecutive years of depression during the fall /winter and remission during the following spring/summer. Research has subsequently uncovered two primary and opposite patterns of seasonal depression: fall/winter depression [usually accompanied by atypical depressive symptoms] and spring/summer depression [usually accompanied by classical depressive symptoms] (Wehr & Rosenthal, 1989).

A seasonal subtype of affective disorder is now included in the DSM-III-R (APA, 1987). The diagnostic criteria for the "seasonal pattern" of mood disorder are: A) A regular temporal relationship between the onset of an episode of Bipolar Disorder or Recurrent Major Depression and a particular 60-day period of the year; B) Full remissions (or change from depression to mania or hypomania) also within a particular 60-day period of the year; C) At least three periods of mood disturbance in three separate years that demonstrated the temporal relationship defined in A and B; at least two of the years were consecutive and ; D) Seasonal episodes of mood disturbance, as described above, outnumbered any nonseasonal episodes of such disturbance that may of occurred by more than three to one.

Most commonly, the winter depression accompanied by a distinct constellation of atypical symptoms including hypersomnia, daytime drowsiness, decreased libido, increased appetite, carbohydrate craving and weight gain (Rosenthal et al., 1984; Kasper, Wehr, et al., 1989). Interestingly, these atypical vegetative states often precede the onset of affective symptoms which include

feelings of sadness, irritability and anxiety (Hellekson, 1989). Frequently, SAD symptoms reach such a level of severity as to cause social withdrawal and work impairment (Hellekson, 1989). During the summer months, patients experience a remission in their depressive symptoms or hypomania. In fact, their energy and motivational levels are often higher than normal (Rosenthal, Genhart, Jacobsen et al., 1987). Phototherapy (exposure to bright artificial light) during the winter months has been shown to reverse the depressive symptoms of SAD suggesting that the seasonal effects are mediated by seasonal changes in light availability (Terman et al., 1989).

Other SAD patients experience an opposite pattern of summer depression and winter hypomania. Typically, these patients exhibit classical depressive symptoms such as insomnia, decreased appetite, and weight loss (Wehr, Sack & Rosenthal, 1987). Fall-winter SAD will be the basis of the present discussion since it appears most closely associated with bulimia nervosa.

The prevalence of SAD is unknown although researchers have reported that its prevalence varies with latitude and climate (Potkin, Zetin, Stamenkovic, Kripke & Bunney, 1986). Patients typically report an increase in the severity of their symptoms when they travel north and relief when they travel south. In addition, they report that any decrease in the amount of exposure to environmental light has a negative effect on their mood. Prevalence rates of 4.3% for winter SAD and 0.7% for summer SAD have been reported (Kasper et al., 1989). Although epidemiological information on SAD is limited, it is clear that there is a strong predominance of females. Approximately 74-94% of SAD patients are female (Hellekson, 1989). As noted by Rosenthal Genhart, Sack et al. (1987), this is a gender ratio more typical of the eating disorders than of the

affective disorders.

Descriptive investigations have shown that disturbances of eating behavior and weight regulation as well as changes in food preference are cardinal SAD symptoms (Rosenthal, Genhart, Jacobsen, et al., 1987). Like patients with bulimia, SAD patients report feeling 'out of control' during the binge-like episodes which characterize their winter depression (Rosenthal et al., 1984). Eating is not experienced as being pleasurable at this time. Rather, patients describe eating to satisfy "compulsions" and "cravings" (Rosenthal et al., 1984, p.73). Clearly, the disturbance in eating behavior typical of winter SAD resembles the binge eating behavior which is the defining feature of bulimia nervosa.

A study which examined food preference among SAD patients documented that they prefer to consume high carbohydrate foods during the fall/winter when they are depressed (O'Rourke, Wurtman, Abou-Nader et al., 1986 (unpublished data), as cited in Wurtman, 1987). Eight SAD patients were given equal access to high-carbohydrate and protein-rich snacks and meals during the fall months and again during the following spring. According to the results, only carbohydrate snacks were chosen during their fall depression. In contrast, during the spring, when their depression was in remission, they were just as likely to choose protein snacks. In fact, the patients consumed over 1000 calories a day in the form of carbohydrate-rich snacks during the fall but only 500 calories a day in carbohydrate snacks during spring. The carbohydrate content of their meals also increased in the fall but to a much less degree while protein intake remained constant. Unfortunately, seasonal effects on fat consumption was not reported.

As discussed previously, the macronutrient content of the binge meals of bulimic patients consists of approximately 50% carbohydrates, 40% fat and 10% protein which is similar to the nutritional content of non-binge meals as well as the meals chosen by normal controls who are asked to binge (Walsh, Kissileff, Cassidy & Dantzic, 1989; Woell, Fichter, Pirke & Wolfram, 1989; Hadigan, Kissileff & Walsh, 1989). According to Walsh et al. (1989): "This suggests that the essential abnormality in bulimia nervosa may be in the control of the amount of food consumed, not in the 'craving' for a specific macronutrient" (p. 58). However, given the widespread reports of bulimic patients that they experience carbohydrate cravings and that their binges consist of large amounts of carbohydrates (Mitchell, Pyle & Eckert, 1981; Abraham & Beumont, 1982; Mitchell, Hatsukami et al., 1985), it is possible that a subset of bulimics, like SAD patients, binge primarily on carbohydrates and that this tendency may exhibit seasonal variations .

Because disturbed eating behavior is such a prominent component of the SAD syndrome, researchers have suggested that the syndrome could, in fact, be viewed as a type of eating disorder (Rosenthal, Genhart, Sack et al., 1987). Indeed, it has been suggested that the factors underlying SAD may provide "a model for studying the reversible mechanisms that underlie the regulation of appetitive behavior and body weight" (Rosenthal, Genhart, Jacobsen et al., 1987, p. 217).

The pattern of consumption typical of SAD is remarkably similar to that seen in a sub-group of obese individuals who exhibit excessive carbohydrate snacking, a syndrome which has been called carbohydrate craving obesity. These individuals consume a moderate number of calories during their regular meals

but increase their daily intake by 800 calories or more through the consumption of high-carbohydrate, low-protein snack foods which they consume at a particular time of the day, usually late-afternoon or evening (Wurtman, 1987). Of note, binge episodes in bulimia nervosa are also most likely to occur in the late afternoon and early evening suggesting a similar tendency to be cyclic (Pyle et al., 1981; Abraham & Beumont, 1982).

A high rate of clinical depression has been found among obese carbohydrate cravers (OCC) who report improved mood following consumption of carbohydrates. Lieberman, Wurtman and Chew (1986) compared the effects of carbohydrate intake on mood in OCC and obese non-carbohydrate cravers using standardized self-report questionnaires administered immediately before and two hours after consumption of a high carbohydrate meal. OCC reported feeling less tired and less depressed than the obese individuals who did not crave carbohydrates. Thus, it appears that OCC consume carbohydrates as a means of improving their mood. Preliminary research suggests that subjects with SAD also experience improved mood and increased subjective energy levels following carbohydrate consumption (Wurtman, 1987).

Several areas of research suggest that central nervous system serotonergic dysregulation might play a role in the pathophysiology of bulimia nervosa, SAD and OCC. Since serotonin is known to be involved in the regulation of both food intake and mood, serotonin dysregulation could, theoretically, be related to disturbances in either system (Kaye, Gwirtsman, Brewerton et al., 1987).

Serotonin Regulation in Bulimia Nervosa and SAD

Animal research has shown that enhanced central serotonergic neurotransmission or direct activation of serotonin receptors causes a significant decrease in the size and duration of meals as well as in the rate of eating (Wurtman & Wurtman, 1979; Blundell, 1986; Leibowitz & Shor-Posner, 1986). This effect is specific to carbohydrate intake while protein consumption is spared. On the other hand, interventions which decrease serotonin levels increase food consumption and promote weight gain (Blundell, 1986; Leibowitz & Shor-Posner, 1986).

The rate of serotonin synthesis is determined by the availability of tryptophan, its precursor and the rate-limiting step in its production. Research evidence suggests that the release of serotonin is affected by food consumption (depending on the proportion of carbohydrate and protein) and that its release may affect subsequent food choice, particularly the consumption of carbohydrates (Wurtman, O'Rourke & Wurtman, 1989). In brief, research with animals has shown that the consumption of carbohydrates raises the ratio of tryptophan to the other large neutral amino acids (LNAAs) by stimulating insulin secretion and increased use of the other LNAAs in non-CNS tissues thereby producing increased 5-HT activity (Fernstrom & Faller, 1978). Dietary protein tends to block these effects by contributing large amounts of the other LNAAs to the blood stream thus lowering the tryptophan/LNAA ratio. Research with humans has confirmed that high carbohydrate, low protein diets elevate plasma tryptophan levels (Wurtman, 1983) and that furthermore, low tryptophan levels are associated with low mood (Young, Smith, & Pihl, 1985) and overeating (Kaye et al., 1988). Thus, theoretically, both bulimia nervosa and SAD may

involve a deficit in serotonergic activity which is related to disturbances in mood and food intake regulation. Indeed, the abnormal eating behavior may, in a sense, represent an attempt to correct the dysregulation of serotonin (Rosenthal & Heffernan, 1986).

According to Wurtman and Wurtman (1989), serotonin is involved in a complex biochemical-behavioural feedback mechanism which controls carbohydrate consumption. Pharmacological investigations employing serotonin agonists such as fenfluramine or fluoxetine in both animal and human subjects, have found that carbohydrate consumption is selectively reduced following drug administration while protein intake is unaffected (Wurtman & Wurtman, 1989). This alteration of food intake is remarkably similar to the difference between summer and winter food consumption observed among SAD patients. Substantial evidence exists that serotonin levels vary seasonally and diurnally, with levels being highest during daylight hours and when photoperiod is longest (Rosenthal, Genhart, Jacobsen et al., 1987; Carisson, Svennerholm & Winbald, 1980). In addition, as discussed previously, serotonin levels have been found to be reduced in depressed patients. It is possible that sensitivity to seasonal variations in serotonin levels may be common to both bulimia nervosa and SAD.

The results of pharmacological studies employing fenfluramine in the treatment of OCC, SAD and bulimia nervosa provide evidence that the disturbances of appetite and mood in these disorders result from related biochemical disturbances. Fenfluramine selectively enhances release of serotonin, blocks reuptake, and stimulates serotonin receptors (Wurtman & Wurtman, 1989). In an initial study, the administration of fenfluramine significantly reduced carbohydrate snack consumption in six of nine OCC while

placebo administration had no effect on carbohydrate intake in any of the seven placebo subjects (Wurtman et al, 1981). More recently, fenfluramine significantly reduced bingeing and purging frequency among 12 DSM-III bulimic patients within one day of the first drug administration (Blouin et al.,1988). Of note, depressive symptomatology was also significantly reduced but not until the patients had received four weeks of drug treatment.

Preliminary investigations of the effects of fenfluramine on SAD symptoms, have reported positive results. In one study, four out of seven SAD patients responded to d-fenfluramine with a full remission of symptoms. (O'Rourke, Wurtman, Brzezinski, et al., 1989). In this study, the daily caloric intake was measured using a computerized food intake measurement system. After treatment, mean calorie intake had dropped significantly, almost reaching summertime levels. In another double-blind placebo-controlled study, 18 SAD patients were treated with d-fenfluramine and placebo in a cross-over fashion (O'Rourke, Wurtman, Wurtman, et al., , 1989). The results showed that 72% of subjects experienced a complete reversal of their symptoms (both affective and appetitive) while taking the active drug as measured by the HAM-D plus SAD addendum.

In summary, a co-morbidity of disturbances in eating behavior and mood are shared by several recently defined syndromes including OCC, SAD and bulimia nervosa. Research findings of seasonal variations in mood and behavior in normal subjects as well as the apparent overlap between SAD and bulimia in terms of comparable symptomatology and similar response to treatment with fenfluramine have lead to the investigation of seasonal patterns in bulimia nervosa.

Seasonal Variations in Bulimia Nervosa

In a preliminary study (Fornari et al., 1989), the presence of seasonal variations in mood and vegetative symptoms were assessed in 35 DSM-III-R bulimia nervosa patients and 27 DSM-III-R anorexia nervosa patients using the Seasonal Pattern Assessment Questionnaire (Rosenthal, Genhart, Sack, Skwerer, & Wehr, 1987), an 18-item retrospective self-report questionnaire. Results for the eating disorder subjects were compared to those of 149 patients with SAD and 46 normal controls. SAD patients indicated significantly higher seasonality scores than all others groups in terms of seasonal changes in sleep length, social activity, mood, weight, appetite and energy level. However, the bulimic group reported a significantly greater degree of seasonal change in appetite, weight and mood than did controls, indicating an exacerbation of these symptoms during the winter months. No such effect was found in the anorexic patients.

Kaplan and Levitan (1990) conducted a retrospective cross-sectional study of seasonality in 165 patients with bulimia nervosa. Subjects were assigned to one of four seasonal groups on the basis of date of admission (i.e., spring, summer, fall, winter) to a day hospital program for eating disorders. On the basis of an analysis of variance, no significant differences were found across seasons for mean binge frequency or depression scores. Although these results suggest that any seasonal effect in bulimia which may have been present was not as strong as might be expected, this analysis does not answer the question of whether there was a significant seasonal trend. Perhaps if these investigators had examined the association of binge frequency of the patients across the months of the year with a measure of seasonality such as photoperiod through a correlational analysis, they may have found a different result.

A recent study found that the retrospective self-reports of bulimia nervosa patients indicate a far greater likelihood of binge eating, purging and depressed mood during the winter months as compared to the summer months (Blouin et al. 1991). Thirty-one female subjects meeting DSM-III-R criteria for bulimia nervosa and thirty-one female normal controls were compared using a modified (to include bingeing and purging symptoms) version of the SPAQ. "Dark hours" (the opposite of photoperiod) significantly predicted the likelihood bingeing and purging in each month and showed a stable linear relationship over the entire twelve months of the year indicating that bingeing is strongly associated with reduced photoperiod. Photoperiod was also strongly correlated with the likelihood of feeling worst each month although these changes were less pronounced than the effects on food intake. Compared to the comparison group, bulimics reported a stronger degree of seasonal change in sleep, mood, weight and energy.

As part of the same investigation, a cross-sectional examination of bulimic and depressive symptomatology among 197 bulimia nervosa patients assessed at an eating disorder clinic during different months of the year over a four year period was conducted. The results indicated that actual binge frequency was strongly influenced by seasonal changes with greater binge frequency occurring during the winter months. Consistent with the results of the retrospective study, the mean binge frequency reported by each subject at the time of their initial assessment was significantly correlated with "dark hours" (or photoperiod). The number of patients meeting DSM-III criteria for bulimia also correlated significantly with "dark hours" which may reflect a seasonal variation in the severity of the disorder. However, mean purge frequency and mean depression

scores were not significantly correlated with photoperiod. These results point to the possibility that seasonal effects on food intake regulation may be independent of seasonal effects on mood in bulimia nervosa.

It is also possible that the types of foods consumed during a binge varies with the seasons in a pattern similar to that seen in SAD. As part of the larger investigation described above, seasonal changes in food preference was studied using the method described above. Overall, bulimics indicated significant seasonal changes in food preference for all food groups indicating increased food intake during the winter months but did not report an increased preference for any specific macronutrient during any particular season. In contrast, controls did not report a seasonal trend for food preference at all.

Overall, the findings on seasonal variations in bulimia nervosa point to the possibility that seasonal effects in bulimia are mediated by light availability given the strong correlation between binge frequency and photoperiod combined with the fact that seasonal symptoms in SAD, including appetitive symptoms, can be reversed by light therapy.

Animal Models of Human Seasonality

Knowledge of seasonal behavioural rhythms and photoperiodic effects in animals has been important in providing a conceptual framework for understanding human seasonality. Animal research has indicated tremendous diversity between species in terms of the nature of their circannual rhythms and the mechanisms or synchronizing agents involved in the regulation of these rhythms (Zucker, 1989). While caution must be exercised in attempting to extrapolate from animal to human systems in this area, knowledge of seasonal

rhythms in animals has been important in furthering our understanding of the constellation of symptoms in SAD. It has been cautioned, however, that it is more useful to examine seasonality of particular traits or processes in animals rather than attempt to find an animal model for the entire SAD syndrome (Mrosovsky, 1989).

Two physiological processes in animals have been studied in an attempt to understand the mechanisms by which environmental changes might influence human behavior: seasonal rhythms of reproduction and seasonal rhythms of energy conservation (Kasper & Rosenthal, 1989). In the case of reproductive functioning, this effect appears to be mediated by seasonal changes in melatonin secretion by the pineal gland which is regulated by the suprachiasmatic nuclei of the hypothalamus (Zucker, 1989). Photoperiod influences the timing and duration of melatonin secretion with light exposure having a direct suppressant effect on its secretion (Lewy, Sack, & Singer, 1985). Thus among Syrian hamsters, for example, the long duration of melatonin secretion which occurs during the winter inhibits reproductive behavior so that offspring are born during the spring when conditions are optimal for survival (Carter & Goldbloom, 1983). Pinealectomized hamsters remain reproductively active regardless of the photoperiod (Wade, 1989).

Decreased libido during their winter months is a commonly reported symptom in SAD. Sack, Rosenthal, Hobbs, and Wehr (unpublished observation, as cited in Mrosovsky, 1989) found large seasonal variations in conception rates among the offspring of 219 SAD patients with lowest rates of conception occurring during the winter months. However, decreased libido is not specific to *seasonal* depression. Moreover, as suggested by Mrosovsky (1989) "this is vastly

different from the total physiological shut-down in seasonal breeders" (p. 130).

While most animal research in the area of photoperiodic processes has examined seasonal reproductive function rather than energy balance, it has been found that several species have developed seasonal rhythms of energy conservation which function to enhance their chances of survival during the winter months. Syrian hamsters, for example, store energy in the form of body fat and gain weight when exposed to short photoperiods. This enhanced energy storage is often independent of an increase in food intake, suggesting that short photoperiods cause a decrease in energy expenditure (Wade, 1989).

Environmental temperature changes have also been shown to influence seasonal changes in body weight in animals (Hoffman, Davidson, & Steinberg, 1982). It has been speculated that changes in the macronutrient composition of their diet may play an important role in seasonal changes in energy balance in Syrian hamsters with the animals consuming more high-fat seeds and nuts in the autumn although the effects of photoperiod on macronutrient preferences of animals has yet to be empirically investigated (Wade, 1989).

Seasonal weight fluctuations in humans have been well-documented and also appear to occur in the absence of changes in total caloric intake suggesting important seasonal variations in human energy expenditure (Rosenthal, Genhart, Jacobsen et al., 1987; VanStaveren et al., 1986). Furthermore, many of the behavioural changes characteristic of SAD patients during the winter months (for example, overeating, oversleeping and weight gain) have been described as energy-conserving behaviours (Kasper & Rosenthal, 1989).

In summary, while animal models are of limited utility in the study of human seasonality, there are important similarities between the constellation of

symptoms that appear in SAD and seasonally occurring changes in animals. Photoperiod has been shown to be a critical seasonal time cue among many species and it was this finding which promoted the idea that SAD might be a photoperiodic disorder.

Phototherapy for SAD

The idea to use bright artificial light in the treatment of SAD came from the observation of Lewy, Wehr, Goodwin, Newsome and Markey (1980) that night-time melatonin production by the pineal gland in humans could be suppressed by high intensity (2500 lux) light whereas ordinary intensity artificial light could suppress nocturnal melatonin secretion in animals. As previously discussed, melatonin is known to be an important chemical mediator of the effects of light on seasonal rhythms in animals (Tamarkin et al., 1985). Before this time, there was widespread agreement among chronobiologists that light played at most a minor role in the regulation of human biological rhythms (Lewy & Sack, 1986). This finding had significant implications including the idea that humans have circadian and seasonal rhythms as well as the prospect that these rhythms were regulated by changes in photoperiod.

Since an initial case study in which a manic-depressive patient with seasonally recurring mood cycles was successfully treated by extending his winter photoperiod through three hours of bright light morning and evening (Lewy, Kern, Rosenthal & Wehr, 1982), numerous investigations have reported significant antidepressant responses of SAD patients to phototherapy (Rosenthal et al., 1984, 1985; James, Wehr, Sack, Parry & Rosenthal, 1985; Terman, 1988; Wehr, Jacobsen, et al., 1986; Hellekson, Kline & Rosenthal, 1986; Wirz-Justice

et al., 1986; Jacobsen, Wehr, Skwerer, Sack & Rosenthal, 1987; Kasper & Rosenthal, 1989; Sack, Lewy, White, et al., 1990). The antidepressant effect is believed to be mediated by the same neural pathways in humans but operates only when light of a much higher intensity is used. Most phototherapy studies conducted to date have been similar in terms of methodology and have largely followed the procedural framework developed at the National Institute of Mental Health (Rosenthal et al., 1984, 1985).

In an preliminary investigation, nine SAD patients were treated with bright, white, full-spectrum fluorescent light (2500 lux at 90 cm) and dim yellow fluorescent light (100 lux at 90 cm) in a single-blind study (Rosenthal et al., 1984). The light source was eight fluorescent tubes in a rectangular metal case with a plastic diffusing screen. Light was self-administered in the patients' homes for three hours before dawn (5 to 8 AM) and after dusk (5 to 8 PM) for a period of two weeks. Like most phototherapy studies to date, a cross-over design was employed such that all subjects received both light conditions. Inclusion criteria included a baseline score of at least 14 on the Hamilton Depression Rating Scale (HAM-D) (Hamilton, 1967) which has been the principle instrument used to measure depressive symptomatology in virtually all phototherapy studies of SAD. Subjects were randomly assigned to one of the above conditions. They were permitted to engage in light sedentary activities during light therapy but were instructed not to sleep. The initial condition was followed by a one week wash-out period if a significant clinical response (defined as a drop of at least four points on the HAM-D) had occurred. Subjects were then crossed over to the second type of light treatment which was administered according to the same conditions. The subjects were rated on a

weekly basis by raters blind to their condition with the 21-item HAM-D. Results indicated a significant antidepressant effect of bright light as evidenced by statistically significant decreases in HAM-D scores whereas no overall significant effect occurred in the dim light condition. One patient did show a significant response to the dim light followed by relapse after withdrawal, however.

This study raises a number of important methodological issues many of which have yet to be resolved in the literature. First, there are a number of problems inherent in the use of a cross-over design for research on phototherapy. Although this design offers the advantage of minimizing statistical noise due to inter-individual differences and maximizing statistical power from small sample sizes, subjects have often not been withdrawn from light for an appropriate wash-out period before being crossed-over to the second treatment condition or are withdrawn only if clinical improvement occurred as is the case with the above study (e.g. Lewy, Sack, Miller & Hoban, 1987; Wirz-Justice et al., 1986; Isaacs, Stainer, Sensky, Moor & Thompson, 1988). In such cases, the baseline for the second treatment is the post-treatment score of the first making evaluation of the response difficult. If the first treatment is more effective, this may create the impression that the patient is actually deteriorating with the second treatment. If the first treatment is only partially effective, this may also distort the effect of the second treatment.

This raises the possibility of ordering effects which may occur despite random ordering of conditions since the more effective treatment is likely to leave the patient at a lower level of depression than the less effective treatment. It has been suggested that in studies in which significant ordering effects did not

occur, this may be due to the small sample sizes employed (Rosenthal, Sack, Skwerer et al, 1989).

Third, placebo effects are particularly important to control for in phototherapy studies since the nature of the experimental intervention cannot generally be concealed from the patient. Thus, the use of an appropriate control condition that is both plausible and inactive is extremely important so that expectations of efficacy for the control treatment are equal to expectations for the active treatment (Kasper & Rosenthal, 1989). In the above study, light of different spectral qualities were employed between conditions which makes it likely that subjects were not blind to the type of light treatment thus contributing to the possibility of placebo effects. Since that time, dim fluorescent light (usually less than 400 lux) has generally been employed as a control condition although some researchers have reported an antidepressant effect for light of this intensity (Wirz-Justice, Bucheli, Graw et al., 1986; Isaacs et al., 1988).

Several researchers have used light of different spectral qualities as a control condition (Kripke, Risch & Janowsky, 1983; Rosenthal et al., 1984; Wirz-Justice et al., 1986; Magnusson & Kristbjarnarson, 1991). Researchers have since attempted to address this problem by asking patients to report their expectations either prospectively or retrospectively, making it possible to determine whether expectations are equal across conditions. However, retrospective reporting may be distorted by the patient's actual experience while prospective eliciting of expectations could actually influence response thereby enhancing the possibility of a placebo effect in either a negative or a positive direction. Fourth, there is the possibility of selection bias since participants in this study, as is the case in many phototherapy studies to date, were recruited by

means of a newspaper article which suggested that seasonal mood changes might be due to seasonal changes in light availability. This may have heightened subject expectations regarding the efficacy of light therapy thereby enhancing the likelihood of a placebo response. In addition, it has been questioned whether samples recruited via the media are similar to physician referrals in terms of symptom profiles. Thompson and Isaacs (1988) addressed this question and found that the only difference between these groups was in terms of the severity of symptoms: SAD patients referred by health care professionals tended to have more severe symptoms than those self-referred through the media.

Of particular note is the fact that the 21-item HAM-D employed in this study could not adequately assess the atypical symptoms of SAD including carbohydrate-craving, hypersomnia and weight gain. Thus, the actual antidepressant effect may in fact have been underestimated. Since the standard version of this scale is not effective for measuring many of the characteristic symptoms of SAD (e.g., CHO-craving, overeating, hypersomnia, social withdrawal), an addendum developed by Rosenthal and Heffernan (1986) is now employed in most studies. In the original version, changes such as decreased appetite or decreased length of sleep would be scored as indicating an exacerbation of depressive symptoms when in the case of SAD they would in fact indicate remission. Additional measures of depression should be employed in future research.

Finally, the possibility that sleep deprivation during the second half of the night contributed to the antidepressant response was not controlled for nor was the amount of environmental light exposure standardized across subjects (Schilgen & Tolle, 1980). Furthermore, as is the case with most phototherapy

studies conducted to present time, subjects were permitted to self-administer light therapy in their own homes making it difficult to keep the light administrations standardized across subjects.

In order to address the question of whether sleep deprivation plays a role in the antidepressant effect of phototherapy, this group replicated the above study and attempted to monitor and control sleeping behavior during light treatment (Rosenthal, Sack, Carpenter, et al., 1985). Inclusion criteria were somewhat more stringent since patients had to have an HAM-D score greater than 14 for two consecutive weeks before being included in this study. Thirteen SAD patients received two weeks of light therapy, separated by one week of no light. Light was administered according to the same schedule as the previous study so that the control treatment was essentially partial sleep deprivation plus the non-specific aspects of the intervention.

A statistically significant difference in hours of sleep during treatment compared to both baseline and withdrawal conditions was found. That is, there was a significant phase advance in time of waking under both bright and dim conditions. The overall results indicated a significant difference between the change in HAM-D scores comparing bright and dim treatments with bright light being superior to dim light. No overall statistically significant effect was found for the dim light treatment although five patients did show improvement with dim light. Since partial sleep deprivation was equally present under both bright and dim light conditions, it cannot sufficiently account for the antidepressant response. Because all patients were exposed to both light conditions, there is the possibility that subject expectations could have influenced the results, however. Interestingly, the only SAD patients who did not respond to bright light, were

also the only ones who did not report a history of carbohydrate craving.

Using a method similar to that of Rosenthal et al. (1984), a Swiss study did not replicate the difference between bright white light (2500 lux) and dim yellow light (250 lux) among 12 SAD patients according to conventional measures for "melancholia" or classical depressive symptomatology (Wirz-Justice et al., 1986). Subjects self-administered light therapy at home for at least five days with one week of withdrawal followed by transfer to the other light condition. The final sample size in this study was extremely small making the overall results questionable, however. Only four patients completed the entire crossover protocol although there was no difference in attrition rate between conditions. In addition to weekly Hamilton interviews, measures included weekly and daily self-ratings of depressive symptomatology as well as sleeping behavior. There were no significant differences between pre-and-post treatment depression scores for bright or dim light using the standard HAM-D. The only difference between the two conditions was in terms of relapse after withdrawal: after bright light, depression scores remained low for a longer period of time than after dim light. However, the failure to relapse after the light was withdrawn suggests that these patients may have exhibited a placebo response. When "atypical items for SAD" were examined however, a clear difference between bright and dim light was found. This corroborates the previous finding that atypical SAD patients who did not report carbohydrate craving did not respond to phototherapy (Rosenthal, Sack, Carpenter, et al., 1985) and suggests that the efficacy of light therapy is specific to the qualitatively unique symptomatology presented by this depressive syndrome.

Based upon a recent quantitative review of previous phototherapy studies,

criteria for distinguishing placebo responses from authentic treatment responses to light were proposed (Terman et al., 1989). Individual data for 332 SAD patients from 14 research centres were analysed using a clustering technique which allows a pooling of independent samples who have undergone a similar manipulation in order to obtain a statistically more powerful analysis of the overall effect size. A set of definitions of treatment efficacy, varying in strictness, was applied to the pooled data.

Using the criterion of a statistically significant difference between pre- and-post treatment scores alone, all treatment conditions (both active and control) would be considered effective across centres. However, the authors suggest that the usefulness of significance tests in these studies is limited by low statistical power due to small sample sizes. Instead, they propose that in future effect sizes should be central to evaluating treatment responses to phototherapy. In their analysis, clinical remission was operationally defined as a post-treatment Hamilton score of less than eight points and a final score which is 50 percent of the baseline score. Based upon this criteria, it was found that the ratio across studies of responders to the total number of participants ranged from 0.31 to 0.51. For dim light, the mean ratio was 0.11. A potential reason for the low number of subjects meeting these strict criteria is the small sample sizes employed in the individual studies. A problem with the above criteria is the fact that it is less difficult for a patient whose baseline score is relatively low to meet this definition of remission compared to those who are more severely depressed. In fact, Terman et al. (1989) notes that the magnitude of the treatment response varies as a function of the baseline severity of SAD symptoms with those experiencing the least severe depression responding best. Of note, the cross-

centre analysis was based on the 21-item HAM-D and may, therefore, have been artificially inflated such that actual therapeutic responses were underestimated.

In summary, the clinical efficacy of phototherapy as a treatment for SAD appears to have been established at this time although a number of methodological issues remain to be resolved. Problems include small samples, unconvincing placebo controls, reliance on the HAM-D as the sole measure of depression, and possible confounds introduced by non-standardized sleep schedules and variable environmental light exposure. Furthermore, there are two areas of considerable controversy in the phototherapy literature. First, there is disagreement as to the specific parameters of light treatment which are necessary to achieve a therapeutic response. These parameters include the time of day of light exposure, optimal intensity and duration and more recently the wavelength (or spectrum) of light responsible for the therapeutic effect. A second and related area of controversy concerns the therapeutic mechanism of action of light therapy which is important both in terms of understanding the underlying pathophysiology of SAD and the neuroanatomical pathways involved in the antidepressant response to light.

Specific Parameters of Light Therapy

Numerous investigators have studied the importance of the intensity of light to the antidepressant effectiveness of phototherapy. Intensity is generally measured as illuminance. That is, the amount of light incident on the patient rather than the amount of light leaving the light source or the perceived brightness of the light source. Typically, illuminance is reported in lux which is defined as "a unit of illumination equal to the direct light of a candle on a surface

one meter away" (Wegscheider-Hyman, 1990, p. 216). Intensity of the light source was the first parameter to be studied systematically since the intensity of ordinary lighting was obviously not strong enough to suppress winter depressive symptoms. Many studies have compared the effectiveness of bright-light treatment (typically 2500 lux) to dim-light treatment (typically 400 lux or less). Most have reported that bright light is significantly more antidepressant than dim light which is frequently used as a control treatment. Since some researchers have reported an antidepressant effect of dim light, there are probably individual differences in the exact optimal threshold (Wirz-Justice et al., 1986, Isaacs et al., 1988).

Preliminary data shows that 10,000 lux is significantly superior to 2500 lux when a brief exposure time of 30 minutes is used (Terman, 1988). Magnusson and Kristbjarnarson (1991) administered either 10,000 lux of bright full-spectrum fluorescent light or 400 lux of red light (placebo) to ten patients who met DSM-III-R criteria for seasonal pattern of depression over a period of eight days in Iceland. Ten subjects self-administered light either in the morning or in the afternoon. Patients were crossed over to the other condition after a wash-out period of eight days. Subjects were rated by blind raters on the HAM-D and SAD addendum. They also completed the Beck Depression Inventory (Beck et al., 1961) at the beginning and end of each treatment. The results indicated that phototherapy was significantly superior to placebo according to all measures employed. There was a non-significant trend in favour of morning light. All but two subjects had equal expectations for both conditions. In addition, all participants in this study were contacted and rated on the same measures the following summer. It was found that the depression

scores were somewhat lower during the summer than after phototherapy but the difference did not attain statistical significance. These findings suggest a reciprocal relationship between intensity and duration.

Duration of light therapy appears to be an important parameter. Terman et al. (1989) evaluated the effectiveness of one, two, and four hour durations of bright light therapy (2500 lux) and concluded that improvement increased with duration. This finding has been replicated in several studies. Wirz-Justice et al. (1987) found that two hours of phototherapy were superior to half an hour. The exact optimal duration probably varies on an individual basis and is likely to be a function of other variables such as the intensity of the light source. It is likely that beyond a certain threshold, increasing the duration would not have an added effect (Rosenthal, Skwerer, Hardin et al., 1990)

Recently, researchers have become interested in studying the spectral qualities of the light which are necessary to achieve an antidepressant effect. Specifically, investigators are attempting to determine which wavelengths of light are responsible for the effect. This information would be important for developing an optimal light source for the treatment of SAD and would also enable researchers to determine which photoreceptors mediate the effects of light. Most investigators have used full-spectrum fluorescent light which resembles other fluorescent lights but have more blue and near-ultraviolet light and less yellow and green light (Wehr & Rosenthal, 1989). Brainard et al. (1990) employed a balanced incomplete block crossover design in which eighteen SAD subjects were randomly assigned to two out of three conditions: white, red, and blue light. Intensity was controlled by equating the photon densities of the light treatments. That is, the light conditions consisted of an equal number of

photons/second per centimetre squared. Each light treatment was administered for one week followed by at least one week of no light before being crossed over to another condition. The data were analyzed using the criteria suggested by Terman et al. (1989). According to the results, significantly more patients in the white light treatment exhibited a 50% decrease in their HAM-D scores compared to the other types of light. No other statistically significant differences were found between the different conditions in terms of the number of patients who had post-treatment Hamilton scores of less than eight or both a 50% decrease in HAM-D scores and a final score below eight. There was no statistically significant difference in subjects' expectations for the different treatment conditions. The authors conclude that broad spectrum white light is superior to restricted wavelengths of light in treating SAD and that therefore, the light sources currently in use could not be improved by shifting the wavelengths towards either end of the visible spectrum.

A recent investigation compared the efficacy of green versus red light therapy (Oren et al., 1991). Green light was chosen because retinal photoreceptors are more sensitive to green wavelengths than to any others providing a basis for making conclusions as to which photoreceptors mediate the effects of light therapy. Twenty SAD patients were entered into a cross-over study in which they received one week of daily two-hour red or green light treatment in the early morning followed by one week of no light after which they were crossed over to the other condition. Ultra-violet light was excluded from both conditions. The photon-densities of both treatments were similar to those of studies using 2500 lux of full-spectrum white light. Statistically significant decreases in 21-item Hamilton depression scale scores were found for

green light but not for red light. When changes in the SAD addendum were examined however, a significant decrease was apparent for both conditions. Of note is that a statistically significant order by colour interaction occurred. Specifically, patients who received red light first showed equal improvement under both conditions while patients treated with green light first showed significantly greater improvement after green light compared to red. This suggests a strong placebo response to red light in the previous group. The authors view the effectiveness of green light as indicative that retinal photoreceptors mediate the antidepressant response in SAD. However, considering both lights were equally effective in reducing the atypical symptoms which are particularly characteristic of SAD, their conclusion would appear unwarranted. As no ultraviolet radiation was present in either condition, the authors conclude that ultraviolet light is not critical to the therapeutic efficacy of phototherapy.

To date, only one controlled study has examined the anatomical route through which light exerts its therapeutic effects. Wehr, Skwerer, Jacobsen, Sack, and Rosenthal (1987) compared eye versus skin exposure to light therapy among ten SAD patients and found that eye exposure was significantly more effective.

The question of whether the timing of phototherapy is important for achieving an antidepressant response has been a topic of considerable interest among phototherapy researchers since it is an issue with both practical and theoretical implications. It has been approached from two separate theoretical orientations. The first approach came from studies of photoperiodic control of seasonal rhythms in animals in which it was found that the effects of light on

behavior depended on whether light exposure takes place at certain critical times of the day outside of the usual winter photoperiod thus extending the winter photoperiod (Hoffman, 1981; Wehr, Jacobsen, Sack, et al., 1986). This theory has become known as the photoperiodic model of phototherapy. The second approach is the "phase-shift hypothesis" which is based on the theory that the timing of SAD patients' circadian rhythms is abnormally delayed in the winter and that light exposure at certain critical times of the day may correct this abnormality by advancing circadian rhythms thereby exerting an antidepressant effect (Lewy et al, 1987). Thus, if the mechanism of action of light therapy and/or the underlying pathophysiology in SAD involves an effect on circadian rhythms, the timing of phototherapy would be critical since light can have opposite effects depending on the time of day exposure occurs. Light in the morning advances circadian rhythms while light in the evening delays them. However, light during mid-day would have no effect.

The findings concerning the importance of the time of day of exposure to phototherapy are conflicting. Lewy et al. (1987) randomly assigned seven normal controls and eight patients with 'winter depression' to either morning bright light exposure or evening bright light exposure for one week. Winter depression was defined as meeting the criteria for a major depression (Research Diagnostic Criteria, Feighner et al., 1972) that developed during the fall or winter and remitted the following spring or summer for the last two consecutive years prior to the study. Subjects were then crossed over to the other condition. Environmental light exposure was held constant. Depression was measured using the 21-item HAM-D which as previously mentioned does not represent an adequate measure of SAD symptoms. Results indicated that depression ratings

were significantly lower after morning but not evening light exposure. In addition, using the onset of melatonin secretion as a marker for the timing of circadian rhythms, it was found that the SAD patients were significantly phase-delayed compared to controls and that morning light therapy advanced the timing of melatonin onset while evening light delayed it.

More recently, these researchers replicated the above study with 10 SAD patients but scheduled morning light an hour earlier and measured melatonin onset twice during each treatment week instead of once in order to examine the rate of phase-shifting due to light exposure. The study was conducted on an out-patient basis but all subjects were required to comply with a standardized light and sleep schedule. Treatment expectations were assessed prospectively on the first day of the study. Several measures of depressive symptomatology were employed: the 21-item HAM-D, the Beck Depression Inventory (BDI) (Beck et al., 1961) and a seven-point global depression rating scale. Analysis of treatment expectation data showed that treatment expectations were significantly higher for morning light. However, there was no significant interaction between treatment expectations and treatment outcome. HAM-D ratings were significantly reduced in both morning and evening conditions while the BDI and global ratings dropped significantly only after morning light. Since the evening light essentially served as a control for the effects of light other than phase-shifting, the moderate response to evening light suggest a mechanism of action other than phase advance. Morning light exposure caused melatonin onset to shift significantly earlier in the patients but not in the control subjects while evening light shifted melatonin secretion significantly later in the patients but not in the controls. However, it is important to note that the issue is not whether bright

light has circadian phase-shifting effects but rather whether these effects account for its therapeutic efficacy.

Other studies have shown no difference between light treatment in the morning and at other times of the day (Hellekson, Kline & Rosenthal, 1986; Wehr et al., 1986; Wirz-Justice et al., 1986; Jacobsen et al., 1987). Wehr et al. (1986) examined whether phototherapy acts via a photoperiodic mechanism by comparing long and short photoperiods. Two different treatment schedules were administered to seven patients in a balanced, randomized cross-over design: two hours of bright, full-spectrum light therapy in the early morning and late evening (photoperiodic extension) versus the same treatment administered in the late morning and early evening. HAM-D scores and the SAD addendum scores were significantly reduced in both conditions and there was no statistically significant difference between the antidepressant effects of the two treatments suggesting that the amount of light rather than the timing was the critical factor. However, the sample size used was very small and in addition there was no control group for the non-specific aspects of the intervention.

Another group of researchers administered bright (2500 lux) full-spectrum light and dim (300 lux) full-spectrum light for four hours in the evening to nine patients with SAD in a balanced-order crossover design (James et al., 1985). HAM-D scores were significantly reduced after bright light treatment compared to baseline but not after dim light treatment. No significant ordering effect was found. The authors compared the results of this study to their earlier results with morning only and morning plus evening light therapy and found no significant difference in the antidepressant response to evening light alone. A study which compared two-versus-four-hour evening phototherapy of SAD

found that both schedules were equally and significantly effective in reducing the symptoms of SAD (Doghramji, Gaddy, Stewart, Rosenthal, & Brainard, 1990).

Hellekson, Kline and Rosenthal (1986) compared three different two-hour, one-week schedules of bright (2500 lux) full-spectrum light treatment using a random order crossover design with a one week wash-out between conditions. The treatments were: 1) one hour in the morning and one hour in the evening; 2) two hours in the evening; and 3) two hours in the morning. The subjects were ten patients with SAD according to the criteria of Rosenthal et al. (1984). Results indicated a significant difference between baseline and treatment Hamilton scores for patients in all conditions. No difference in Hamilton change scores between conditions was found.

Time of day of light exposure does not appear to be a critical parameter for most SAD patients. It is possible that these inconsistent findings are an artifact of methodological differences between these studies. Overall, it appears that certain patients are differentially sensitive to phototherapy in the morning but that for most time of day is not critical (Terman et al., 1989).

The Mechanism of Action of Phototherapy

Several hypotheses have been advanced to explain the therapeutic mechanism of action of phototherapy although neither of them have been fully supported by existing research. The "phase-shift" hypothesis is based on the theory that a circadian rhythm mechanism is responsible for light's therapeutic effects (Lewy, Sack, Miller & Hoban, 1987). Specifically, morning light exposure provides a corrective phase-advance of the circadian rhythms of SAD patients. In support of this hypothesis, nocturnal melatonin secretion rhythm

(Lewy et al., 1987) and body temperature rhythm (Terman et al., 1989) have shown phase advances in association with positive therapeutic responses to morning light. Since the phase-shift theory was discussed previously in the context of the importance of the time of day of light exposure, it will not be elaborated on here.

According to the melatonin hypothesis, light therapy exerts its effects by modifying the pattern of melatonin secretion (Rosenthal, Sack, Jacobsen et al., 1986). However, research has demonstrated that suppression of melatonin is not critical to effective phototherapy (Wehr, Jacobsen, Sack et al., 1986). Oral administration of melatonin was found to exacerbate the atypical but not the typical depressive symptoms of SAD in one study (Rosenthal, Sack, Jacobsen, et al., 1986) while in another investigation it was found to have no effect (Wirz-Justice, Graw, et al., 1990). Rosenthal, Jacobsen, et al. (1988) administered the beta-adrenergic blocking agent, atenolol, which has been shown to suppress melatonin, to nineteen patients with SAD and found it was no more effective than placebo.

The photon-counting hypothesis of Rosenthal, Sack, James, et al. (1985) proposes that it is the total number of photons absorbed rather than the timing of light exposure which is critical to the antidepressant effect. According to these researchers, light therapy satisfies an inflated need for light during the winter months. Related to this idea, Reme, Terman and Wirz-Justice (1990) have suggested that SAD could be caused by a deficiency in the retinal photoreceptor renewal mechanisms. Normally, a process called "photostasis" causes an adjustment of visual pigment availability so that enhanced photon absorption occurs during the winter. SAD patients may suffer from a deficient adaptation

of their photoreceptor systems to reduced light availability thereby decreasing light sensitivity. Bright light is theorized to stimulate "retinal synthetic activity" leading to a normalization of light sensitivity which ultimately triggers CNS mechanisms.

Finally, Wurtman and Wurtman (1989) have hypothesized that a dysregulation of central serotonergic functioning may produce the atypical depressive symptoms characteristic of SAD. Although d-fenfluramine, a direct serotonin agonist has been used successfully to treat SAD patients, it is not yet understood how light could affect this system (O'Rourke, Wurtman, Brzezinski, et al., 1989). Support for the hypothesis that serotonin availability is involved in the etiology of SAD was obtained from a study which compared the effects of the serotonergic precursor L-tryptophan, placebo and evening phototherapy on the depressive symptoms of 13 SAD patients. The results indicated that, on average, the atypical SAD symptoms improved more during the L-tryptophan condition than either alternative although the effects of L-tryptophan and light were not significantly different. It is possible that the time of day of light therapy played a role in its somewhat lower effectiveness.

In summary, although several theories have been put forth to explain phototherapy's mechanism of action, the question of how light exerts its antidepressant effects in SAD remains unresolved.

Phototherapy in Non-seasonal Depression and in Normals

Several investigators have studied the effect of phototherapy on non-seasonal depressive disorders reporting inconsistent results. Kasper et al. (1989) studied twenty individuals with mild SAD-type symptoms (S-SAD; subsyndromal

SAD) and twenty individuals with no history of seasonal difficulties. Degree of seasonal difficulties was measured with the SPAQ. The winter difficulties experienced by the subsyndromal group had not caused them functional disruption and were regarded as a variant of normal behavior for which they had not previously sought help. The subjects were divided into sex-and-age matched subgroups of ten who received either five or two hours of bright (2500 lux) full-spectrum light per day (divided between the morning and evening) for a duration of one week preceded by one week of baseline observation and followed by one week of withdrawal. Measures included the HAM-D plus SAD addendum. Results showed that S-SAD subjects show a significantly better response to phototherapy than control subjects and that five hours of phototherapy was superior to two hours. Of note is the authors' observation that in terms of their symptom profiles S-SAD patients are less bothered by depressed mood in the winter than individuals with SAD. Instead, their winter symptoms consist primarily of the atypical vegetative symptoms.

These results were not corroborated by the findings of Kasper, Rogers, Madden, Joseph-Vanderpool, and Rosenthal (1990) who compared the effects of phototherapy on forty individuals with varying degrees of seasonal difficulties as measured by the SPAQ. Participants received two hours of bright or dim light daily in the morning for two weeks with twenty subjects in each group. There were no significant differences between the outcome measures for bright and dim light conditions. In addition, there were no significant correlations between treatment responses and seasonality scores on the SPAQ in either condition.

Volz, Mackert, Stieglitz and Muller-Oerlinghausen (1990) treated thirty inpatients with non-seasonal major affective disorder with either bright (2500

lux) fluorescent light or dim (50 lux) incandescent light in the morning for two hours over a period of seven days. Depression was measured using the HAM-D administered by a blind rater and several self-rating scales. While a non-significant reduction in depressive symptoms during the course of light therapy was observed for both conditions, no difference in the effects of bright and dim light was found. These results were consistent among the various instruments used to assess depression. It was concluded that bright light is not effective for non-seasonal depression.

Another study compared the effectiveness of phototherapy in treating non-seasonal atypical depression and SAD (Stewart, Quitkin, Terman, & Terman, 1990). The authors rationalized that if the disorders are manifestations of the same underlying pathophysiology they should show similar treatment responses. Patients meeting criteria for atypical depression report symptoms qualitatively similar to those characteristic of SAD such as hypersomnia and hyperphasia. Eight patients with atypical depression and twenty-five SAD patients were treated with 2500 lux of full-spectrum fluorescent light for two hours twice daily (morning and evening) for two weeks. Changes in depressive symptomatology were measured using the "Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorder Version "(SIGH-SAD; Williams, Link, Rosenthal & Terman, 1988) which was administered by a clinician who was not blind to the diagnoses although he was unaware of whether the patient was receiving light therapy at the time of the interview. Patients also completed several self-rating scales. Severity of symptoms was not significantly different between the groups at baseline. Both HAM-D and atypical vegetative symptoms scores showed highly significant reductions among the SAD patients

following phototherapy but not among the atypical non-seasonal depressives. Individual item analysis revealed that for subjects with atypical depression no item was significantly reduced by phototherapy. Self-ratings were in agreement with clinician ratings. It was concluded that the two disorders are separate entities although this conclusion is based on a relatively small sample of atypical depressives.

The Effects of Phototherapy on Eating Behavior

Appetitive symptoms are a prominent component of the SAD syndrome. It is therefore surprising that phototherapy researchers have paid so little attention to the effects of phototherapy on eating behavior. According to Rosenthal Genhart, Sack, et al. (1987) 'carbohydrate craving' is the item which responds most dramatically and rapidly to light treatment as measured by the HAM-D with SAD addendum. However, it is questionable whether changes in eating behavior can be adequately assessed with this instrument. For example, to assess the symptom of carbohydrate craving, patients are asked whether they have been craving "starches or sugars" over the previous week. Subjects are probably just as likely to recall craving high fat foods (e.g potato chips or pastries) as they are to report craving carbohydrates. Thus, even when changes in eating symptoms are measured, phototherapy researchers have typically relied solely on the SAD version of the HAM-D making these results of questionable validity. It is possible that the notion that "carbohydrate-craving" is a prominent feature of both SAD and bulimia may have arisen from patient's misevaluations of the nutrient content of their binge foods. As discussed previously, recent research which has attempted to describe objectively the eating

behavior of bulimics has found that the most pronounced difference between binge and non-binge foods is in fat content (Kales, 1990; Walsh, Kissileff, Cassidy & Dantzie, 1989; Woell, Fitcher, Pirke, & Wolfram, 1989) This may also be the case for SAD patients although this possibility has not yet been investigated.

In the only study to date which focussed specifically on the effects of phototherapy on eating symptoms in SAD, Krauchi, Wirz-Justice and Graw (1990) randomly assigned 36 SAD patients to one week of light therapy (2500 lux, full-spectrum) for one hour daily either morning or evening after one week of baseline and followed by one week of withdrawal. The purpose of this study was to compare the effects of light therapy on eating behavior to the natural changes that occur in the summer. Subjects were rated weekly with the HAM-D and SAD addendum as well as being required to keep daily self-ratings of food intake using the Food and Drink Frequency Questionnaire (FDFQ). This instrument is designed to assess macronutrient composition of food consumed. In addition, 26 of the 36 subjects completed the self-ratings for one week during the following summer in order to be able to compare the effects of light with the effects of a change in season..

A significant reduction in HAM-D ratings for depression was found with no relapse after withdrawal for both morning and evening schedules. In terms of the SAD addendum, significant improvements were found for social withdrawal and the sleep symptoms after one week of light therapy. However, a significant decrease in the food-related items (CHO-craving, overeating) was not achieved until one week after withdrawal. Analysis of the macronutrient data indicated that light therapy selectively reduced carbohydrate intake during the

second half of the day but did not affect any other food category. However, since this reduction did not reach statistical significance until one week after light therapy, it is questionable whether the obtained changes in eating behavior were actually due to the effects of the light, whether they were secondary to a primary effect on mood or finally, whether they reflected a placebo effect. The same selective effect on carbohydrate intake was observed during the summer compared to winter eating behavior. Of note, there was a significant positive correlation between total carbohydrate intake during baseline and its degree of suppression by light. This suggests that only those subjects for whom CHO-craving is a prominent feature of their winter depression will be CHO suppressors by light or by season. These results also raise the possibility that food intake and mood regulation may be controlled by separate mechanisms.

There are several problems with this study which could have affected the results. First, while the FDFQ was designed to differentiate various macronutrients, a serious flaw in this instrument is the fact that it does not assess the frequency of consumption of high fat foods. Instead, it includes only high carbohydrate, high fibre, high protein, dairy, alcoholic, and high caffeine foods. Thus, it is possible that important effects of light on the consumption of high fat foods may have been overlooked. In addition, the duration of the light treatment employed in this study was very short combined with the fact that there was no relapse after withdrawal of the lights which points to the possibility of a placebo effect.

Rationale

Up to 70% of individuals with bulimia nervosa have a history major affective disorder (Walsh et al., 1985; Hudson et al., 1983; Blouin et al., 1986). In addition, depressed non-bulimic individuals exhibit disturbances of food intake and weight regulation. While a majority of depressed patients present with a loss of appetite, a sub-group report overeating and weight gain in association with depressed mood. This atypical profile is most pronounced among seasonal affective disorder patients (Garvey, Wesner & Godes, 1988). A co-morbidity between disturbances of food intake regulation and mood is shared by another recently defined syndrome, carbohydrate craving obesity (CCO) which also exhibits a tendency to be cyclic.

Research on eating behavior in SAD and CCO has indicated that the eating disturbance which is characteristic of these disorders is specific to a particular macronutrient: SAD and CCO patients tend to crave and to binge on high carbohydrate foods (Wurtman & Wurtman, 1989). Furthermore, it has been found that consumption of high carbohydrate foods leads to an improvement in mood among these patients. Animal research indicating that CNS serotonin availability is related to satiety mechanisms lead researchers to speculate that a deficit in CNS serotonin neurotransmission may be responsible for these symptoms (Fernstrom & Faller, 1978). In addition, serotonin levels have been found to vary seasonally and serotonin dysregulation has been implicated in major depression. In support of this hypothesis, both CCO and SAD have been successfully treated with fenfluramine, a serotonin agonist. However, limitations of the methods employed to study the nutrient composition of food intake in these types of investigations make it unclear at this time whether the

satiety-inducing effects of fenfluramine in SAD and CCO are macronutrient specific.

Evidence of a satiety deficit in bulimia nervosa comes from descriptive studies of the eating behavior of bulimic individuals which have indicated that these patients tend exhibit an increased rate of eating during a meal and report being more hungry after a meal than do non-eating disordered controls. Researchers have hypothesized that the apparent satiety deficit in bulimia may be due to decreased availability of serotonin. In support of this hypothesis, bulimia nervosa has also successfully been treated with fenfluramine suggesting that sensitivity to variations (and perhaps seasonal variations) in serotonin levels may be common to both SAD and bulimia. Increasing evidence suggests that the satiety deficit in bulimia may not be specific to carbohydrates since these patients also binge on large amounts of fat and protein. This finding is probably the result of the more objective methods used to study the macronutrient content of binge meals in bulimia. In terms of the relationship between binge eating and mood in bulimia nervosa, it has been well-established that mood deteriorates following binge eating episodes (Johnson & Larson, 1982; Davis et al., 1985; Johnson-Sabine et al., 1984).

Studies of seasonal patterns in bulimia nervosa have been prompted by recent findings of seasonal variations in eating behavior and mood in the general population as well as the similarity between the symptoms of bulimia nervosa and SAD. Bulimics retrospectively report that their symptoms (binging, purging, and mood) are worse during the winter months. Furthermore, actual binge frequency is highly correlated with photoperiod indicating that as photoperiod decreases binge frequency increases. Seasonal effects on mood are

less pronounced.

Overall, there appears to exist a complex association between neurophysiological mechanisms which regulate food intake and those which regulate mood. The possibility that serotonergic mechanisms may be involved in both disorders of satiety and disturbances of mood has lead researchers to speculate that "abnormal regulation of CNS serotonin may act as a shared biological vulnerability for eating disorders and depression"(Jimerson, Lesem, Kaye, Hegg & Brewerton, 1990, p.444).

Given the fact that phototherapy is an effective treatment for SAD, it is likely that SAD symptoms are mediated by seasonal changes in light availability although the mechanism of this effect is not as yet understood. It has been hypothesized that light therapy is effective in SAD through an effect on serotonin (Wurtman & Wurtman, 1989). Considering the strong association between binge eating and photoperiod, it seems reasonable to suggest that seasonal variations in bulimic symptomatology may also be mediated by seasonal changes in light availability. To date, one published case report study has reported positive results with phototherapy in "seasonal bulimia" (Lam, 1989).

The purpose of the present study was to investigate the effects of phototherapy on eating behavior and mood in bulimia nervosa. The following specific hypotheses were examined: First, bulimia nervosa patients who receive the bright light treatment (experimental condition) will respond with a greater reduction in bulimic symptoms (binging and purging) than those who receive the dim light treatment (placebo condition). Second, following phototherapy, bulimia nervosa patients exposed to bright light will respond with a greater improvement in mood than those exposed to dim light. Since measures of bulimic

symptoms and mood will be conducted on a daily basis, this study will provide an opportunity to track the time course of changes in mood and eating symptoms. Third, phototherapy will have a specific effect on the carbohydrate composition of binge foods with those patients in bright light exhibiting a proportionally greater reduction in foods high in carbohydrate content than patients in the dim light condition. It was believed that the results would bear indirectly on issues related to a further understanding of the pathophysiology of bulimia nervosa.

Method

Subjects. Eighteen female volunteers who met DSM-III-R criteria for bulimia nervosa completed the study. The subjects were recruited primarily from patients presenting to the Eating Disorder Clinic of the Ottawa Civic Hospital (n=10) and from patients on a waiting list for an assessment at the clinic (n=4). Subjects were also recruited by means of the news media (n=4). An article was submitted to a local newspaper which explained that seasonal variations in bulimia may be mediated by light availability. Persons who believed they were suffering from bulimia nervosa and who wished to volunteer for the study were invited to contact the Eating Disorder Clinic. In addition, three patients agreed to participate in the study but decided not to continue beyond the preliminary assessment for the following reasons: time constraints (n=2) and insomnia due to termination of a medication (n=1). Exclusion criteria included current substance abuse, serious suicidal risk, acute psychoticism and physical illness.

The diagnosis of bulimia nervosa was based on the subjects responses to the bulimia nervosa section of the National Institute of Mental Health Diagnostic

Interview Schedule-Revised [NIMH-DIS-R] (Robins, Helzer, Croughan & Ratcliffe, 1981). This is a structured diagnostic interview which provides Axis I DSM-III-R diagnoses. That this instrument has acceptable reliability and validity has been well-established (Robins, Helzer, Ratcliffe & Seyfried, 1982). A computerized version of this interview which has been established to have equivalent reliability was employed in the present study (Blouin, Perez, & Blouin, 1988).

Two subjects were taking antidepressant medication and one subject was taking a very mild dose of an anxiolytic medication during the study. However, no subject began or ended medication while participating in the study. When the statistical analyses were re-run excluding these three patients, the results were identical.

All subjects signed an informed consent (see Appendix A). This study received approval from the Research Ethics Committees of the Ottawa Civic Hospital and Carleton University.

Materials. The light sources used in this study were identical to those used by previous phototherapy researchers (e.g., Rosenthal et al., 1984). Artificial bright light was provided by a "light box": a rectangular metal fixture (2 feet by 4 feet) which contained 6 fluorescent tubes behind a plastic diffusing screen (Durotest Inc.). There were two conditions. In the first, this device emitted an intensity of 2500 lux of full-spectrum fluorescent light (bright condition). In the second, the same fixture was used to administer an intensity of not greater than 500 lux (dim condition). This was accomplished by having only 3 of the 6 light tubes illuminated. The intensity of the light emitted from these

devices was measured and controlled using a light meter and by adjusting the distance patients sat from the light boxes until the exact desired intensity was attained.

The psychological inventories employed in this study included the following (see Appendix B):

(1) Symptom Checklist-90 Revised (SCL-90-R). The SCL-90-R is a 90-item self-report inventory which assesses 9 symptom dimensions including somatization, obsessive-compulsiveness, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism (Derogatis, 1977). The validity and reliability of this instrument have been studied extensively. High levels of internal consistency and test-retest reliability have been reported (Derogatis, Rickels & Rock, 1976). In addition, the convergent and concurrent validity of this inventory has been well-established (Derogatis & Cleary, 1977).

(2) Eating Disorder Inventory (EDI). The EDI is a 64-item self report measure which assesses a number of psychological and behavioural traits common in anorexia nervosa and bulimia nervosa (Garner & Olmsted, 1984). It consists of eight subscales including drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears. The validity and reliability of the EDI has been established (Garner, Olmsted & Polivy, 1983).

(3) Seasonal Pattern Assessment Questionnaire (SPAQ). The SPAQ is an instrument designed to assess the extent to which one has previously been affected by seasonal changes in a variety of areas and the months in which subjects are most likely to experience certain behaviours (Rosenthal et al., 1984).

These areas include mood, eating behavior, weight, sleep, energy level and social activity. This instrument was modified for the purposes of the present study to include seasonal changes in bingeing and purging. The SPAQ has been demonstrated to have acceptable test-retest reliability and discriminate validity (Thompson, Stinson, Fernandez, Fine & Isaacs, 1988).

(4) Beck Depression Inventory (BDI) The BDI is a 21-item self-report inventory which assesses cognitive and neurovegetative symptoms of depression (Beck, Ward , & Mendelson, 1961). It has been shown to have high levels of internal consistency and convergent validity (Beck et al., 1961; Schwab, Bialow & Holzer, 1967).

(5) Bulimic Symptoms Checklist (BSC). The BSC is a self-report measure which assesses the severity of bulimic symptoms (Blouin et al., 1988). It consists of two parts. First, it measures average weekly frequency of bingeing, vomiting and purging (laxatives/diuretics). Second, it presents a 5-point severity rating scale for the above three symptoms plus three additional items covering the same time period: persistent preoccupation with food; lack of control with relation to food; and global severity of bulimic symptoms. This instrument has been demonstrated to be a sensitive measure of changes in bulimic symptoms in pharmacological research (Blouin, Blouin, Perez, et al., 1988; Blouin et al., 1989).

(6) Structured Interview Guide for the Hamilton Depression Rating Scale-Seasonal Affective Disorders Version. (SIGH-SAD). The 21-item Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960) is the most widely used measure of depression severity in research studies of treatments for depression (Williams, 1988). It has been shown to have a high degree of scale

reliability and its concurrent, discriminant and construct validity have been well-established (Knesevich, Biggs, Clayton, & Ziegler, 1977; Zimmerman, Coryell, Pfohl & Stangl, 1986; Maier, Heuser, Philipp, Frommberger & Demuth, 1988). With the goal of improving test-retest reliability of the HAM-D at the item level, Williams (1988) developed the Structured Interview Guide (SIGH-D) in order to standardize the administration of the scale. The SIGH-D has since been demonstrated to have highly acceptable internal consistency and inter-rater reliability (Potts, Daniels, Burnam & Wells, 1990). The SIGH-SAD version was developed in order to provide a standard method of assessing the atypical symptoms of SAD including social withdrawal, weight gain, appetite increase, increased eating and carbohydrate craving (Williams, Link, Rosenthal & Terman, 1988). It consists of the original 21 items plus eight additional ones. The SIGH-SAD has been demonstrated to be sensitive to changes in the depressive symptoms of SAD patients in previous phototherapy research (Magnusson & Kristbjarnarson, 1991).

(7) Profile of Mood States (POMS). The POMS is a self-report mood inventory which consists of six factors: tension-anxiety, depression, anger-hostility, vigour, fatigue and bewilderment (McNair, Lorr & Droppleman, 1971). It has 65 adjectives to be rated on a 5 point scale and will be used to measure daily mood fluctuations. The POMS has been found to be a sensitive measure of mood changes in pharmacological research (File, Bond & Lister, 1982). Values of between .65 to .74 have been observed in examinations of test-retest reliability in treatment studies (McNair et al., 1971). These lower than expected values were explained as being attributable to treatment effects. The predictive validity of the POMS has been well-established (McNair et al., 1971).

(8) Daily Binge Record (DBR). The DBR was constructed for the purposes of this study. It is a food diary to be completed on a daily basis which asks for the following information: number of binges, the type and amount of food consumed during a binge, the time any binges occurred, and the number and type of purging episodes. The macronutrient content of binge foods was analysed using a computerized nutrient analysis program based on the 1988 Canadian Nutrient Files (Warwick, 1989).

Procedure. The study was conducted during the months of January through March inclusive. There were seven groups of subjects (consisting of between one and five subjects per group) who received phototherapy during the following time periods in 1991: Jan. 14-20, Jan. 23-29, Feb. 4-10, Feb. 13-19, Feb.27-Mar. 5 and Mar. 6-12. The average daily photoperiod for these weeks was 9.2, 9.5, 10.0, 10.5, 11.2, 11.6, and 11.6 respectively. Data on daily photoperiod was obtained from the Weather Office of Environment Canada.

All subjects underwent an extensive psychological assessment as part of their initial intake appointment at the Eating Disorder Clinic (including any subjects obtained through the media) which included an examination of their medical and psychiatric histories.

The duration of each subject's participation was three weeks. Subjects were randomly assigned to receive either the bright or dim light condition (single blind condition). The subjects were not told which specific condition they were assigned but they were aware of the fact that there was a less effective condition and a more effective condition and that they had a fifty percent chance of receiving either one. All assessment procedures took place at the Ottawa

Civic Hospital.

The first week of the study constituted a baseline period. On the first day of the baseline week, the subjects received a preliminary assessment which consisted of the following instruments: the SCL-90-R, the EDI, the SPAQ, the BDI, the BSC, and the SIGH-SAD. The SIGH-SAD was always administered by a trained interviewer who was blind to the light conditions of the subjects. In addition, ten of the SIGH-SAD administrations were rated by two blind interviewers in order to assess inter-rater reliability. The second week of the study was the week of phototherapy. On the first day and last day of the treatment week, subjects were administered the BDI, the BSC and the SIGH-SAD either immediately prior to or immediately following phototherapy. The third week of the study was a post-treatment period. Subjects returned for a final assessment on the last day of this week and were again administered the BDI, the BSC, the SIGH-SAD as well as a brief interview based on the DSM-III-R criteria for seasonal mood pattern. In order to assess whether blind conditions were maintained throughout the study, subjects were asked to report whether they believed they were in the bright or dim light condition at this time. Throughout their entire 21-day participation, subjects were required to complete the DBR and POMS on a daily basis and were instructed to complete these inventories at the end of each day. They returned these inventories once weekly and the measures were scanned by the examiner to ensure accuracy and clarity. All assessment procedures took place at the same time of day: late afternoon/early evening. In addition, patients were asked to keep their sleep schedules and environmental light exposure constant and unchanged from their usual routine for the entire duration of the study.

During the treatment week, subjects attended the Ottawa Civic Hospital daily between the hours of 5 and 7 pm for seven consecutive days. The treatment consisted of two hours of phototherapy. Due to the blind nature of the study, subjects in the bright and dim light conditions were tested on separate days or in separate rooms. Subjects were instructed not to stare into the light but to glance at the light box approximately once a minute for a few seconds. They were permitted to engage in light sedentary activities (e.g. knitting or reading) during the light administrations but were not permitted to sleep. An experimenter was present at all times during all light sessions to ensure compliance to these instructions. All subjects were treated in a standardized manner and were asked not to converse with other subjects during the study. They were also asked to save any questions they may have had regarding phototherapy until a feedback session (debriefing) following the last treatment interval.

After the entire study was complete, subjects were contacted and thoroughly debriefed regarding the purpose and theoretical basis of the study. At this time any questions the patients had about phototherapy were answered. Subjects received two articles which discussed practical aspects of phototherapy and explained research findings on the effects of light on human behavior and mood. Those participants in the dim condition were offered one week of daily bright light treatments if they so desired.

Statistical Analyses. The distributional properties of the data were examined separately for each group in order to determine whether the assumptions underlying multivariate analysis of variance (MANOVA) were met.

Frequency distributions as well as the index of skewness indicated that the binge/purge variables as well as the macronutrient variables were positively skewed. Square root transformations were conducted on these variables to normalize their distribution. MANOVA is robust to modest violation of normality as long as the violation is created by skewness rather than outliers (Tabachnick & Fidell, 1989). To identify outliers, standardized scores (z-scores) were examined in order to identify cases with standard scores in excess of plus or minus three (Tabachnick & Fidell, 1989). One outlier, who had extreme values for the DBR (i.e., total calories, total carbohydrates, total fat and total protein) was identified using this criteria and excluded from the analyses of this instrument.

All analyses included the Bartlett test for homogeneity of variance. This assumption was violated for the DBR until the outlier described above was excluded.

The results were analyzed using repeated measures MANOVA which was conducted using SYSTAT (Wilkinson, 1989). This study constitutes a split-plot factorial design. The independent variables were the within-subjects factor of time (weeks 1-3) and the between-subjects factor of light condition (bright versus dim). A separate MANOVA was conducted for each dependent variable. The dependent measures were binge frequency, purge frequency, food preoccupation, lack of control with relation to food, BDI, SIGH-SAD, POMS, and caloric and macronutrient (protein, carbohydrate, fat) content of binge foods. Significant multivariate results were followed by the appropriate pairwise comparisons using the Newman-Keuls procedure to control the Type 1 error rate (Howell, 1987).

In order to assess whether the experimental and placebo subjects were equivalent at baseline, discriminant function analyses were conducted on information collected at the preliminary assessment. One employed the following cluster of descriptive predictor variables: age, present weight, highest past weight, lowest past weight, binge frequency and purge frequency. A second assessed differences on the EDI subscales and a third focussed on the SCL-90-R subscales.

Data on photoperiod for Ottawa were obtained from the Herzberg Institute of Astrophysics of the National Research Council of Canada. Pearson correlations were calculated between the average photoperiod for each month and the proportion of patients who reported that a given month was the month during which they were most likely to binge according to the SPAQ. Pearson correlations were also calculated between binge frequency over the 21 days of the study and daily photoperiod as well as between binge frequency and average daily temperature.

To examine the relationship between eating behavior and mood, Kendall tau-B correlation coefficients were calculated between daily binge frequency on the DBR and daily POMS depression scores. In addition, the correlation between weekly binge frequency as assessed by the BSC and weekly Beck Depression Inventory scores across subjects were examined.

Results

The basic descriptive characteristics of the 9 experimental and 9 placebo subjects appear in Table 1. Averaged across conditions, the group (n=18) had a mean age of 28 years, an average weight of 140 lb, and an average height of 5

Table 1**Basic Descriptive Characteristics of the Subjects**

Variable	Bright (n=9)		Dim (n=9)	
	Mean (SD)	N (%)	Mean (SD)	N (%)
Age	29 (9)		26 (7)	
Hollingshead Status (mode)	3		3	
Marital Status (% single)	89%		67%	
Weight (lbs.)	148 (50)		132 (46)	
Height (ins.)	64 (3)		65 (3)	
Binge/week	6.8 (4)		4.9 (5)	
Vomit/week	4 (6)		4 (6)	
Purge/week	0.4 (1)		2.3 (5)	
Major Depression-Single		7 (78%)		6 (67%)
Major Depression-Recurrent		0 (0%)		1 (11%)
Dysthymia		3 (33%)		2 (22%)
Seasonal Mood Pattern (DSM-III-R)		1 (11%)		2 (22%)
SPAQ SAD		6 (66%)		4 (44%)
SPAQ Subsyndromal SAD*		1 (11%)		0 (0%)

* includes only those subjects who did not meet SPAQ criteria for SAD

feet 4 inches. At baseline, they reported binge eating an average of 5.8 times per week, self-induced vomiting an average of 4.1 times per week, and an average of 1.4 other purge episodes (including laxative and diuretic use) per week.

Seventy-two percent ($n=13$) of the subjects met DSM-III-R criteria for a lifetime diagnosis of Major Depressive Disorder (single episode or recurrent) among whom three (1 experimental and 2 placebo) met DSM-III-R criteria for Seasonal Mood Pattern. Ten subjects (6 experimental and 4 placebo) met the SPAQ criteria for Seasonal Affective Disorder. This difference was not statistically significant ($\text{Chi-square}=.90$, $df=1$, ns). There were too few observations to statistically analyze the response of patients with SAD versus those without SAD. However, an examination of the means revealed a similar pattern of results for both of these groups except that there was a tendency for depression scores to continue declining upon withdrawal of phototherapy among SAD subjects in the dim condition. The discriminant function analyses showed that the experimental and placebo groups could not be distinguished on the basis of the descriptive variables ($F(6,9)=0.38$, ns), the EDI scores ($F(3,14)=0.69$, ns) or the SCL-90-R scores ($F(9,8)=1.02$, ns) at baseline.

Only four of the subjects correctly guessed which light condition they had been assigned (below chance levels) which indicates that the blind conditions were successfully maintained ($\text{Chi-square}=4.267$, $df=2$, ns).

The random assignment of subjects to the bright versus dim light conditions resulted in the experimental group being tested when there was an average of 10.02 hours per day of environmental light while the placebo group was tested when there was an average of 11.24 hours per day of environmental light. This difference attained statistical significance ($t=3.71$; $df(1, 16)$; $p<.05$).

The mean inter-rater reliability coefficient for the 21-item SIGH-SAD was very high (Kendall tau-B=0.91).

Seasonality of Bulimic Symptoms. Seasonal variations in binge eating, purging and mood, as assessed by the SPAQ, are presented in figures 1, 2 and 3, respectively. Also plotted are the average number of dark hours for each month (24 hours minus the average photoperiod for each month). Photoperiod was converted to dark hours in order to enhance the graphical display of the association between bulimic symptoms and photoperiod. The pattern was virtually identical for the experimental and placebo groups and therefore, the data were combined. Dark hours was significantly correlated with the likelihood of binge eating ($r = 0.85$, $df = 1$, $p < .0001$), purging ($r = 0.89$, $df = 1$, $p < .0001$) and feeling worst ($r=0.88$, $df = 1$, $p<.0001$) each month. A quadratic equation significantly explained the monthly variation in photoperiod ($R^2=0.99$, $df=12$, $p < .001$), binge eating ($R^2=0.89$, $df=12$, $p < .001$), purging ($R^2=0.94$, $df=12$, $p < .001$) and mood ($R^2=0.85$, $df=12$, $p < .01$).

Correlational analyses indicated that the daily binge frequency of the 18 subjects over the 21 days was not significantly correlated with daily photoperiod over the 21 days ($r = -0.17$, $df = 1$, ns) or with average daily temperature over the 21 days ($r = -0.04$, $df = 1$, ns).

Affective Symptoms. Phototherapy had a significant impact on depressed mood. As predicted, the MANOVA revealed a significant group (bright vs. dim) by time (baseline vs. treatment vs. withdrawal) interaction for the 21-item Hamilton depression score [SIGH-SAD] ($F(2,15)=4.14$, $p<.01$). Post hoc comparisons using the Newman-Keul's test indicated that depression scores

Figure 1. Plotted is the percent of 18 Bulimia Nervosa patients who report that a given month is a month during which they are most likely to binge eat along with the average number of dark hours per month.

Seasonal Variations in Binge Eating

Figure 2. Plotted is the percent of 18 Bulimia Nervosa patients who report that a given month is a month during which they are most likely to purge along with the average number of dark hours per month.

Seasonal Variations in Purging

Figure 3. Plotted is the percent of 18 Bulimia Nervosa patients who report that a given month is a month during which they are most likely to feel worst along with the average number of dark hours per month.

Seasonal Variations in Mood

declined significantly from baseline to treatment and rose significantly from treatment to withdrawal for those in the bright light condition. For subjects in the dim condition, there were no differences in 21-item Hamilton depression scores across time. These results are shown in figure 4.

The group by time interaction obtained borderline levels of statistical significance for the 17-item total Hamilton (SIGH-SAD) score ($F(2,15)=3.01$, $p<.08$). The effect of time was statistically significant ($F(2,32)=3.64$, $p<.05$). Newman-Keuls comparisons indicated that depression scores declined significantly from baseline to treatment and rose from treatment to withdrawal (not statistically significant) among subjects in both conditions. These results are illustrated in figure 5.

Table 2 illustrates the analysis of the 21-item SIGH-SAD according to the method of Terman et al. (1989). Using these criteria for clinical remission, 44 percent of those in the bright condition as compared to only 11 percent of those in the dim condition exhibited a 50 percent reduction in Hamilton depression scores. Among subjects in the bright condition, 67 percent had a post-treatment score which was less than or equal to eight as compared to only 22 percent of subjects in the dim condition. There were too few observations to conduct a formal statistical analysis of this data.

The group by time interaction for the 8-item SAD score of the SIGH-SAD interview failed to reach statistical significance ($F(2,15)=1.606$, ns). However, the effect of time was statistically significant ($F(2,32)=3.42$, $p<.05$). The 8-item SAD scores declined from baseline to treatment and rose from treatment to withdrawal (not statistically significant) among subjects in both conditions. These results are shown in figure 6.

Figure 4. The effect of phototherapy on the 21-item Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.

2 of/de 2

MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS
STANDARD REFERENCE MATERIAL 1010a
(ANSI and ISO TEST CHART No. 2)

Figure 5. The effect of phototherapy on the 17-item Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.

17-item SIGH-SAD

Figure 6. The effect of phototherapy on the 8-item SAD total of the Hamilton Depression Rating Scale (SIGH-SAD) in Bulimia Nervosa.

8-item SIGH-SAD

Table 2**Analysis of the 21-item Hamilton Depression Rating Scale**

	<u>Bright (n=9)</u>	<u>Dim (n=9)</u>
50% drop	4/9 (44%)	1/9 (11%)
post < 8	6/9 (67%)	2/9 (22%)
50% drop and post < 8	4/9 (44%)	1/9 (11%)

Numbers in columns labelled bright and dim represent number of responding to non-responding patients.

There was a significant group by time interaction for the Beck Depression Inventory ($F(2,14)=7.16, p<.01$). The Newman-Keuls comparisons indicated that BDI depression scores decreased significantly from baseline to treatment and increased significantly from treatment to withdrawal among subjects in the bright condition. For subjects in the dim condition, there were no differences in BDI depression scores across time. The results of the BDI are presented in figure 7.

Analysis of the daily POMS revealed no significant group by time interaction effects for any subscale. Figure 8 illustrates the results for the POMS depression subscale. It is apparent that there was a trend towards reduced depression during the treatment week in both groups but only the bright group appeared to relapse following withdrawal.

There was a significant effect of time on the POMS tension-anxiety subscale ($F(2,28)=3.38, p<.05$) indicating that tension-anxiety scores declined across time for subjects in both conditions. There were no significant effects of group on any POMS subscale.

Bulimic Symptoms. Contrary to predictions, phototherapy had no effect on bulimic symptoms. There were no significant effects of group (bright vs. dim), time (baseline vs. treatment vs. withdrawal) or their interaction on binge frequency ($F(2,15)=0.55, ns$), purge frequency ($F(2,13)=0.05, ns$), food preoccupation ($F(2,14)=2.32, ns$), or perceived lack of control over eating ($F(2,14)=0.64, ns$) as measured by the BSC. This was confirmed by the results of the DBR which also showed no effect of group, time or their interaction on binge frequency ($F(2,13)=.84, ns$), vomit/purge frequency ($F(2,12)=.33, ns$),

Figure 7. The effect of phototherapy on the Beck Depression Inventory (BDI) in Bulimia Nervosa.

Figure 8. The effect of phototherapy on the Profile of Mood States (POMS) in Bulimia Nervosa.

POMS Depression

mean daily caloric intake ($F(2,13)=1.46$, ns) or macronutrient content of binges including carbohydrate ($F(2,13)=1.08$, ns), fat ($F(2,13)=.88$, ns) or protein ($F(2,13)=2.08$, ns). At baseline, the average binge consisted of 1384 calories which was composed of 68 percent carbohydrates, 21 percent protein and 11 percent fat. There were no baseline group differences in the size or composition of binges.

Depressed mood as measured by the daily POMS was not significantly correlated with the frequency of binge eating according to the DBR for the experimental group (Kendall tau-B=0.23, ns) or the placebo group (Kendall tau-B= -0.05, ns). Furthermore, weekly binge frequency was not found to be significantly correlated with weekly Beck Depression Inventory Scores at baseline (Kendall tau-B= 0.08, ns), treatment (Kendall tau-B= -0.01, ns), or withdrawal (Kendall tau-B= 0.11, ns).

Summary. The 9 bulimia nervosa subjects who received the bright light treatment exhibited a significant reduction in depressive symptomatology as measured by the 21-item Hamilton depression interview (SIGH-SAD) and the BDI following treatment. In addition, their depressive symptoms demonstrated a significant relapse upon withdrawal of the treatment. The 8-item SAD total of the SIGH-SAD inventory decreased following treatment and increased following withdrawal for both groups. The dim light treatment had no effect on depressed mood as measured by the 21-item SIGH-SAD and the BDI among the 9 bulimia nervosa patients in the placebo condition. Both experimental and placebo subjects exhibited a trend towards decreased depression during the treatment according to the POMS depression scores but only subjects in the bright

condition appeared to relapse after withdrawal. Phototherapy had no significant effects on bulimic symptoms including binge frequency, purge frequency or the composition of binge meals.

Discussion

Recent research has documented a marked seasonal pattern in the symptoms of bulimia nervosa, a pattern which is strongly correlated with photoperiod. The present study used an experimental model to test the hypothesis that environmental light influences bulimic symptomatology. The results indicate that a standard one-week application of phototherapy for two hours in the early evening caused a significant improvement in depressive symptoms among bulimia nervosa patients in comparison to a dim light placebo treatment. Contrary to predictions, phototherapy had no effect on bulimic symptoms or on the macronutrient composition of binge foods.

In the present study, several independent measures of depression were employed. The Beck Depression Inventory and the 21-item Hamilton scores converged to indicate that the bright light treatment induced a significant remission in depressive symptoms while the dim light had no effect on depression. The 17-item Hamilton scores reached only borderline levels of significance. It is possible that this may reflect increased reliability of the longer version. The POMS depression subscale showed trends which were consistent with the direction of change indicated by the other measures of depression. The fact that the POMS depression scores did not reach strict levels of significance may be related the time-frames of these instruments. The Hamilton and the Beck both measure affective symptoms over the course of a week while the POMS

measures daily mood fluctuations.

The impact of phototherapy in bulimia nervosa appears to be similar to its mood-elevating effects in SAD. Several studies have evaluated the effectiveness of phototherapy as a treatment for SAD (Rosenthal et al., 1984, 1985; Wirz-Justice et al., 1987; Terman, 1988; Kasper & Rosenthal, 1989; Sack et al., 1990). Most researchers have used similar methodologies and most have reported statistically significant improvements in depressive symptoms following treatment. On the basis of these findings, there appears to be a general agreement in the literature that "this simple nonpharmacologic intervention can induce complete clinical remissions within a few days" (Terman et al., 1989). Thus, it is widely believed that the seasonally recurring affective and appetitive disturbances which are characteristic of SAD are mediated by light availability.

However, phototherapy research has been characterized by several important methodological limitations which make unequivocal conclusions about the effectiveness of phototherapy as a treatment for SAD seem unwarranted at this time. First, all light therapy studies conducted to date have employed a cross-over design in which all subjects have received both the experimental and placebo light conditions. This design has been chosen in order to maximize statistical power with the small number of SAD subjects available for research. However, this type of design makes it difficult to ensure that treatment expectations are equivalent across conditions thereby contributing to the possibility of placebo effects and/or ordering effects. An important achievement of the present study was the maintenance of blind conditions by employing a between-subjects design in which subjects received only one light condition. The intensity of the dim light condition in this study (500 lux) was higher than

levels commonly employed by previous researchers (400 lux or less). However, it was shown to be a plausible placebo since subjects were unable to guess which condition they had been assigned with better than chance accuracy.

Furthermore, the effects of phototherapy on depression as measured by the SIGH-SAD was ascertained under double-blind conditions, a major strength in comparison to past research.

A second methodological weakness of past research has been the reliance on the HAM-D (or in a few cases the SIGH-SAD) as the sole measure of depression. The employment of several independent measures of mood in the present study represents a significant methodological improvement particularly in light of the converging results for the self-report measure (BDI) and the interview (SIGH-SAD).

A third area of concern is the specific design parameters necessary to achieve a maximal effect including time of day, intensity, and duration. The issue of the optimal time of day for phototherapy administration remains unresolved. It has been argued by several authors that evening administration is less effective than light delivered in the morning (Lewy et al., 1987) while others have demonstrated no difference between the effectiveness of morning versus evening light therapy (Hellekson et al., 1986; Wehr et al., 1986). In terms of intensity, studies have shown that high-intensity light (e.g. 10,000 lux) has a rapid anti-depressant effect and that there is a reciprocal relationship between duration and intensity (Magnusson & Kristbjarnarson, 1991). In the past, subjects have been permitted to self-administer light in their own homes making it difficult to ensure that the treatment is standardized across subjects but making it feasible to increase duration.

For several reasons, the present experiment could be considered to be a relatively conservative test. The intensity and duration of the light treatment represented the minimum levels shown to be effective by past research. In addition, light was administered in the evening. In order to ensure compliance, the subjects were tested in the laboratory. Under these conditions, a longer exposure time or a different time of day would have been problematic. Also, the random assignment of subjects to conditions resulted in there being significantly more environmental light available while subjects in the placebo condition were being tested. This may have reduced the impact of phototherapy. However, the results clearly indicate that light at this intensity, duration and time of day was psychoactive in bulimia nervosa without influencing food intake. Thus, the hypothesis that seasonal variations in bulimic behavior is mediated by light availability was not supported. What the results suggest is that binge behavior and mood can be manipulated independently in bulimia nervosa and that furthermore, these symptoms may be differentially sensitive to environmental light.

The present results lend themselves to the interpretation that mood and eating behavior in bulimia nervosa are regulated by separate mechanisms. Previous pharmacological studies, have pointed to a similar conclusion (Brotman, Herzog & Woods, 1984; Hughes, Wells, Cunningham et al., 1986; Barlow et al., 1988; Blouin et al., 1988; Blouin et al., 1989). Brotman et al. (1984) demonstrated that the use of antidepressant medication in bulimia often eliminated bulimic symptoms without a simultaneous remission in depression. Amongst bulimic subjects without depression, Hughes et al. (1986) found that desipramine effectively reduced bulimic symptoms. Similarly, Barlow et al.

(1988) demonstrated that desipramine's antibulimic effects were not associated with an alleviation of depressive symptoms in bulimia. Finally, Blouin et al. (1988) determined that fenfluramine, which is not an antidepressant, induced a remission in depressive symptoms among a group of bulimic women which was secondary to a primary effect on the core bulimic symptoms. In a study which sought to identify variables useful for predicting a positive response to antidepressant treatment in bulimia, it was concluded that "the predictors of the antibulimic effect of desipramine in bulimia are independent of its antidepressant properties" (Blouin et al., 1989, p. 24). Although desipramine did not exert an overall significant antidepressant effect in that study, there was some evidence that a small subgroup of depressed bulimics with evidence of a biological basis for the depression (e.g., abnormal DST response) may be depression "responders". Factors associated with bulimic symptomatology alone discriminated bulimia responders from non-responders. Specifically, patients who responded with a significant decrease in their bulimic symptomatology were characterized by a lower urge to binge eat and a higher frequency of bulimic behaviors than patients who were not responsive. In the present study, a qualitatively unique psychoactive manipulation was shown to produce a similar dissociation between appetitive and affective symptoms in bulimia nervosa.

Of note, the participants were not chosen on the basis of seasonal variations in their symptoms nor were they recruited for the presence of SAD. Consistent with previous findings (Blouin et al., 1991), they reported, retrospectively, a degree of seasonal change in their bulimic symptoms which was highly correlated with photoperiod. Seventy-two percent of the subjects met the DSM-III-R criteria for a lifetime diagnosis of major depressive disorder,

a rate which closely approximates that reported by previous researchers (Hudson et al., 1983; Walsh et al., 1895; Blouin et al., 1986). Six experimental and four placebo subjects met the SPAQ criteria for SAD. However, these criteria are much less conservative than the DSM-III-R criteria which more closely resemble the definitions used by previous phototherapy researchers to recruit SAD patients. Only one experimental subject and two placebo subjects met the more conservative criteria for a "seasonal mood pattern" suggesting that the presence of SAD cannot alone account for these findings. Future research in this area should attempt to define a subgroup of bulimia nervosa patients who experience "seasonal bulimia".

It is interesting that the atypical symptoms of depression, as measured by the SIGH-SAD were not found to be significantly responsive to phototherapy in the present study. Like SAD patients, the depressive symptoms associated with bulimia nervosa have also been found to be atypical. However, phototherapy has not been demonstrated to be effective for non-seasonal atypical depression (Stewart et al., 1990). Nor has it been found to have any effects in normal subjects or in non-seasonal classical depression suggesting a unique neurophysiological effect in bulimia (Volz et al., 1990; Kasper et al., 1989). It has been demonstrated that individuals with subsyndromal SAD benefit from light therapy especially in terms of the atypical vegetative symptoms but that they respond better to light of a longer duration than the standard two hours (Kasper et al., 1989). The results of the present study are not consistent with these findings since phototherapy had no impact on the vegetative depressive symptoms as measured by the 8-item SAD score of the SIGH-SAD. Future research should manipulate the parameters of the study design by using a longer

duration time or light of a higher intensity.

Descriptive analyses of the caloric and macronutrient composition of binge meals as measured by a self-report daily binge diary indicated that the present findings were consistent with those reported by previous researchers (Mitchell & Laine, 1985; Rosen et al., 1986; Woell et al., 1989). Binge meals consisted primarily of carbohydrates with moderate amount of fat and protein being consumed. However, the proportion of calories obtained through fat was somewhat lower in this study. Qualitatively, the binge meals of subjects in the present study were not different from those reported for non-bulimic control subjects (Walsh et al., 1989).

The results of this study have important theoretical significance. Overall, the evidence points to the conclusion that there is an independence between food intake regulation and mood regulation in bulimia nervosa. The positive effects of antidepressants in bulimia appear to be independent of the presence of depression. Reductions in bulimic symptoms through pharmacological interventions have been achieved with no change in depression or without a simultaneous change in depression. Furthermore, as the results of this study indicate, depressive symptoms can be manipulated independent of bulimic behaviors. In support of this hypothesis, depressed mood was not significantly correlated with the frequency of binge eating over the course of the 21 days of this study. Similarly, it is also possible that seasonal variations in eating behavior and mood may be regulated by distinct neurochemical processes or by distinct environmental factors. For example, researchers studying the thermoregulatory effects of food have found that abnormalities in thermoregulation may be associated with abnormal food intake patterns

(Levitsky, Schuster, Stallone & Strupp, 1986). The possibility that seasonal variations in food intake may be related to thermoregulatory effects has not yet been investigated. The very preliminary examination of the relationship between binge eating and environmental temperature in the present study produced negative results, although the range in temperature exposure was quite limited and was not controlled in this study.

In the only study to date which focussed specifically on the effects of phototherapy on eating symptoms in SAD, no change in eating behavior was found until one week after withdrawal of light (Krauchi et al., 1990). In all other previous phototherapy studies, changes in eating behavior have not been adequately assessed. Researchers have relied solely upon the Hamilton SAD addendum items. Thus, it is possible that a similar independence may exist in SAD although this question has not as yet been satisfactorily addressed.

Taken together, the results of this study strongly support the conclusion that light influences depressive symptomatology in bulimia nervosa. The psychoactive properties of phototherapy in bulimia are apparent within seven days of light administration and a relapse in depressed mood is apparent within one week following withdrawal of light. These results are consistent with previous research showing that light has antidepressant effects in seasonal affective disorder and suggest the possibility of an overlap in terms of underlying pathophysiological mechanisms in SAD and bulimia nervosa.

Serotonin dysregulation has been implicated in the pathophysiology of both bulimia nervosa and SAD. Both disorders have been treated successfully with fenfluramine, a serotonin agonist. In bulimia, fenfluramine appears to work through a primary effect on food intake and a secondary effect on

depressed mood (Blouin et al., 1988). Preliminary research on the treatment of SAD with fenfluramine indicates that the drug is effective for both the appetitive and affective symptoms (O'Rourke, Wurtman, Brzezinski, et al., 1989; O'Rourke, Wurtman, Wurtman, et al., 1989). Recent evidence that serotonin levels exhibit seasonal fluctuations in a pattern which is remarkably similar to seasonal changes in SAD and bulimia, further implicate serotonin in the etiology and treatment of these disorders (Carlsson et al., 1980). It is possible that the mechanism by which phototherapy exerted a beneficial effect on mood in the present study was unrelated to serotonin availability. Perhaps the mechanism by which serotonin affects food intake can be manipulated independently from the mechanism which modulates mood.

Clinically, it is not possible to rule out the idea that phototherapy may play a role in the treatment of bulimia nervosa on the basis of the present study. There appear to be at least a sub-group of bulimics for whom seasonal variations in their symptomatology is a cardinal feature of their disorder. Although light therapy did not affect bulimic behavior, the fact that it induced a remission in depressive symptoms suggests an underlying pathophysiology which is unique from non-seasonal depression. Further research focussing on this seasonal sub-group is needed before discarding the idea that environmental light may impact bulimic symptoms.

References

- Abraham, S. F. & Beumont, P. J. V. (1982). How patients describe bulimia or binge eating. Psychological Medicine, 12, 625-635.
- American Psychiatric Association. (1980). Diagnostic and Statistical Manual of Mental Disorders (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1987). Diagnostic and Statistical Manual of Mental Disorders (3rd ed. Revised). Washington, DC: Author.
- Barlow, J., Blouin, J., Blouin, A. & Perez, E. (1988). Treatment of bulimia with desipramine: A double-blind crossover study. Canadian Journal of Psychiatry, 33, 129-133.
- Beck, A.T., Ward, C.H. and Mendelson, M. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 561-571.
- Ben-Tovin, D.I., Subbiah, N., Scheutz, B. & Morton, J. (1989). Bulimia: Symptoms and syndromes in an urban population. Australia and New Zealand Journal of Psychiatry, 23, 73-80.
- Blehar, M.C. and Rosenthal, N.E. (1989) Seasonal affective disorders and phototherapy. Archives of General Psychiatry, 46, 469-474.
- Blouin, A.G., Blouin, J. H., Aubin, P., Carter, J., Goldstein, C., Boyer, H. and Perez, E. (1991). Seasonal patterns of bulimia nervosa. American Journal of Psychiatry 149, (1), 73-81.
- Blouin, A.G., Blouin, J. H., Perez, E. L., Bushnik, T., Zuro, C. & Mulder, E. (1988). Treatment of bulimia with fenfluramine and desipramine. Journal of Clinical Psychopharmacology, 8 (4), 261-269.
- Blouin, A.G., McAffer, V., Blouin, J. and Perez, E. (1986). The incidence of depression in bulimia. Presented at the Second International Conference on Eating Disorders, New York.
- Blouin, A.G., Perez, E.L., Blouin, J.H. (1988). Computerized administration of the Diagnostic Interview Schedule. Psychiatry Research 23, 335-344.
- Blouin, J.H., Blouin, A.G., Perez, E. & Barlow, J. (1989). Bulimia: Independence of antibulimic and antidepressant properties of desipramine. Canadian Journal of Psychiatry, 34, 24-29.

- Blundell, J.E. (1986). Serotonin and appetite. Neuropharmacology, 23, 1537-1551.
- Brainard, G.C., Sherry, D., Skwerer, R.G., Waxler, M., Kelly, K. and Rosenthal, N.E. (1990). Effects of different wavelengths in seasonal affective disorder. Journal of Affective Disorders, 20, 209-216.
- Brotman, A.W., Herzog, D.B. and Woods, S.W. (1984). Antidepressant treatment of bulimia: the relationship between bingeing and depressive symptomatology. Journal of Clinical Psychiatry, 45, 7-9.
- Carlet, D.J. and Camargo, C.A. (1991). Review of bulimia nervosa in males. American Journal of Psychiatry, 148 (7), 831-843.
- Carlsson, A., Svennerholm, L. and Winblad, B. (1980). Seasonal and circadian monoamine variations in human brains examined post-mortem. Acta Psychiatrica Scandinavica, 61(Suppl 280), 75-85.
- Carter, D.S. and Goldbloom, B.D. (1983). Antigonal effects of timed melatonin infusion in pinealectomized male Djungarian hamsters (*Phodopus sungorus*): Duration is the critical parameter. Endocrinology, 112, 1257-1261.
- Cooper, P. J. & Fairburn, C. G. (1986). The depressive symptoms of bulimia nervosa. British Journal of Psychiatry, 148, 268-274.
- Cooper, P. J. & Fairburn, C. G. (1983). Are eating disorders forms of affective disorders? British Journal of Psychiatry, 143, 96-97.
- Cooper, P.J. and Fairburn, C.G. (1983). Binge-eating and self-induced vomiting in the community: a preliminary study. British Journal of Psychiatry, 142, 139-144.
- Czeisler, C.A., Kronauer, R.E., Mooney, J.J., Anderson, J.L. and Allan, J.S. (1987). Biologic rhythm disorders, depression and phototherapy: A new hypothesis. Psychiatric Clinics of North America, 10, 687-709.
- Davis, R., Freeman, R. & Solyom, L. (1985). Mood and food: An analysis of bulimic episodes. Journal of Psychiatric Research, 19 (2/3), 331-335.
- Derogatis, L.R. (1977). SCL-90-R: Administration, Scoring and Procedures Manual - II. Towson, MD: Clinical Psychometric Research.

- Derogatis, L.R. and Cleary, P. (1977). Confirmation of the dimensional structure of the SCL-90: A construct validation. Journal of Clinical Psychology, 33 (4), 981-989.
- Derogatis, L.R., Rickels, K. and Rock, A. (1976). The SCL-90 and the MMPI: A step in the validation of a new self-report scale. British Journal of Psychiatry, 128, 280-289.
- Doghramji, K., Gaddy, J.R., Stewart, K.T., Rosenthal, N.E. and Brainard, G.C. (1990). 2-versus 4-hour evening phototherapy of seasonal affective disorder. The Journal of Nervous and Mental, 178(4), 257-260.
- Fairburn, C.G. (1991). The heterogeneity of bulimia nervosa and its implications for treatment. Journal of Psychosomatic Research, 35 (Suppl. 1), 3-9.
- Fairburn, C.G. and Beglin, S.J. (1990). Studies of the epidemiology of bulimia nervosa. American Journal of Psychiatry, 147 (4), 401-408.
- Fairburn, C.G. and Garner, D.M. (1988). Diagnostic criteria for anorexia nervosa and bulimia nervosa: The importance of attitudes to shape and weight. In D.M. Garner and P.E. Garfinkle (Eds.), Diagnostic Issues in Anorexia Nervosa and Bulimia Nervosa, New York: Brunner and Mazel.
- Feighner, J.P., Robins, E., Guze, S.B., Woodruff, R.A., Winokur, R.A. and Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. Archives of General Psychiatry, 26, 57-63.
- Fernstrom, M. (1989). Depression, antidepressants and body weight change. Annals of the New York Academy of Sciences, 575, 31-40.
- Fernstrom, J.D. and Faller, D.V. (1978). Neutral amino acids in the brain: Changes in response to food ingestion. Journal of Neurochemistry, 30, 1531-1538.
- Fichter, M.M. and Noegel, R. (1990). Concordance for bulimia in twins. International Journal of Eating Disorders, 9 (3), 255-263.
- File, S., Bond, A. and Lister, R. (1982). Interaction between effects of caffeine and lorazepam in performance tests and self-ratings. Journal of Clinical Psychopharmacology, 2, 102-106.

- Fornari, V.M., Sandberg, D.E., Lachenmeyer, J., Cohen, D., Matthews and Montero, G. (1989). Seasonal variations in bulimia nervosa. Annals of the New York Academy of Science, 575, 509-511.
- Garner, D.M. and Olmstead, M.P. (1984). Manual for the Eating Disorder Inventory. Psychological Assessment Resources, Inc: U.S.A.
- Garner, D.M., Olmstead, M.P. and Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. International Journal of Eating Disorders, 2, 15-34.
- Garvey, M.J. Wesner, R. & Godes, M. (1988). Comparison of seasonal and non-seasonal affective disorders. American Journal of Psychiatry, 145 (1), 100-102.
- George, D.T., Weiss, S.R. & Jimerson, D.C. (1985). Clinical characteristics of normal weight bulimia. In Kaye, W. H. & Gwirtzman, H. E. (Eds.). The Treatment of Normal Weight Bulimia. (pp. 1-18). Washington: American Psychiatric Press.
- Gladis, M. M. & Walsh T. W. (1987). Premenstrual exacerbation of bulimia. American Journal of Psychiatry, 144 (12), 1592-1595.
- Gwirtsman, H. E., Roy-Byrne, P., Yager, J. & Gerner, R. H. (1983). Neuroendocrine abnormalities in bulimia. American Journal of Psychiatry, 140 (5), 559-563.
- Hadigan, C. M., Kissileff, H. R. & Walsh, B. T. (1989). Patterns of food selection during meals in women with bulimia. American Journal of Clinical Nutrition, 50, 759-766.
- Halmi, K. A. Falk, J. R. & Schwartz, D. M. (1981). Binge-eating and vomiting: A survey of a college population. Psychological Medicine, 11, 697-706.
- Hamilton, M. (1967). Development of a rating scale for primary depressive illness. British Journal of Social and Clinical Psychology, 6, 278-296.
- Harris, B., Young, J. and Hughes, B. (1984). Appetite and weight change in patients presenting with depressive illness. Journal of Affective Disorders, 6, 331-339.
- Hellekson, C. (1989). Phenomenology of seasonal affective disorder: An Alaskan perspective. In Rosenthal, N. E. & Blehar, M. (eds.). Seasonal Affective Disorder and Phototherapy. (pp. 33-45). New York: Guilford.

- Hellekson, C., Kline, J.A. and Rosenthal, N.E. (1986). Phototherapy for seasonal affective disorder in Alaska. American Journal of Psychiatry, 143(8), 1035-1037.
- Herzog, D. & Gordon, C. (1985). Are the DSM-III criteria for bulimia too broad? American Journal of Psychiatry, 142 (4), 393.
- Hinz, L. D. & Williamson, D.A. (1987). Bulimia and depression: A review of the affective variant hypothesis. Psychological Bulletin, 102 (1), 150-158.
- Hoffman, R.A., Davidson, R and Steinberg, K (1982). Influence of photoperiod and temperature on weight gain, food consumption, fat pads and thyroxine in male golden hamsters. Growth, 46, 150-162.
- Hoffman, R. A. (1981). Photoperiodism in vertebrates. In J. Aschoff (Ed.), Handbook of Behavioral Neurobiology, (pp. 449-473). New York: Plenum.
- Howell, D.C. (1987). Statistical Methods for Psychology. (2nd edition). Boston: PWS Publishers.
- Hudson, J. I., Laffer, P. S. & Pope, H. G. (1982). Bulimia related to affective disorder by family history and response to the dexamethasone suppression test. American Journal of Psychiatry, 139 (5), 685-687.
- Hudson, J. I., Pope, H. G., Jonas, J. M. & Yurgelun-Todd, D. (1983). Family history study of anorexia nervosa and bulimia. British Journal of Psychiatry, 142, 133-138.
- Hudson, J. I., Pope, H. G. and Jonas, J. M. (1983). Phenomenological relationship of eating disorders to major affective disorder. Psychiatry Research, 9, 345-354.
- Hughes, P., Wells, L.A., and Cunningham, M.S. ((1986). Treating bulimia with desipramine: A double-blind cross-over study. Archives of General Psychiatry, 43, 182-186.
- Hsu, L.K.G., Chesler, B.E. and Santhouse, R. (1990). Bulimia nervosa in eleven sets of twins: A clinical report. International Journal of Eating Disorders, 9 (3), 275-282
- Isaacs, G., Stainer, D.S., Sensky, T.E., Moor, S. and Thompson, C. (1988). Phototherapy and its mechanisms of action in seasonal affective disorder. Journal of Affective Disorders, 14, 13-19.

- Jacobsen, F.M., Wehr, T. A., Skwerer, R.A., Sack, D.A. and Rosenthal, N.E. (1987). Morning versus midday phototherapy of seasonal affective disorder. American Journal of Psychiatry, 144, 1301-1305.
- James, S.P., Wehr, T.A., Sack, D.A., Parry, B.L. and Rosenthal, N.E. (1985). Treatment of seasonal affective disorder with light in the evening. British Journal of Psychiatry, 147, 424-428.
- Jimerson, D.C., Lessem, M.D., Kaye, W.H., Hegg, A.P. and Brewerton, T.D. (1990). Eating disorders and depression: Is there a serotonin connection? Biological Psychiatry, 28, 443-454.
- Johnson, C. & Flach, A. (1985). Family characteristics of 105 patients with bulimia. American Journal of Psychiatry, 142 (11), 1321-1324.
- Johnson, C. L. & Larson, R. (1982). Bulimia: An analysis of moods and behavior. Psychosomatic Medicine, 44 (4), 341-350.
- Johnson, C. L., Lewis, C., Love, S., Lewis, L. & Stuckey, M. (1984). Incidence and correlates of bulimic behavior in a female high school population. Journal of Youth Adolescence, 13 (1), 15-26.
- Johnson, C. L., Stuckey, M. K., Lewis, L. D. Schwartz, D. M. (1982). Bulimia: A descriptive survey of 316 cases. International Journal of Eating Disorders, 2 (1), 3-16.
- Johnson, C. L., Stuckey, M. K., Lewis, L. D. Schwartz, D. M. (1983). A survey of 509 cases of self-reported bulimia. In Darby, P.L., Garfinkel, P. E., Garner, D. M. & Coscina, D. V. (Eds.). Anorexia Nervosa: Recent Developments in Research, (pp. 159-171). New York: Alan R. Liss.
- Johnson-Sabine, E.C., Wood, K.H., and Wakeling, A. (1984). Mood changes in bulimia nervosa. British Journal of Psychiatry, 145, 512-516.
- Kales, E.F. (1990)., Macronutrient analysis of binge eating in bulimia. Physiology and Behavior, 48, 837-840.
- Kaplan, A. S. and Levitan, R.D. (1990). Seasonal variation in bulimic symptoms. American Journal of Psychiatry, 147(11), 1579.
- Kaplan, A. S. and Woodside, D. B. (1987). Biological aspects of anorexia nervosa and bulimia nervosa. Journal of Consulting and Clinical Psychology, 55 (5), 628-634.

- Kasper, S., Rogers, S.L.B., Madden, P.A., Joseph-Vanderpool, J.R. and Rosenthal, N.E. (1990). The effects of phototherapy in the general population. Journal of Affective Disorders, 18, 211-219.
- Kasper, S., Rogers, S.L.B., Yancey, A., Schulz, P.M., Skwerer, R.G. and Rosenthal, N.E. (1989). Phototherapy in individuals with and without subsyndromal seasonal affective disorder. Archives General Psychiatry, 46, 837-844.
- Kasper, S. and Rosenthal, N.E. (1989). Anxiety and depression in seasonal affective disorders. In P.C. Kendall and D. Watson (Eds.), Anxiety and Depression: Distinctive and Overlapping Features. (pp. 341-375). New York: Academic Press.
- Kasper, S., Wehr, T. A., Bartko, J. J., Gaist, P. A. & Rosenthal, N. E. (1989). Epidemiological findings of seasonal changes in mood and behavior. Archives of General Psychiatry, 46, 823-833.
- Kaye, W. H. & Gwirtsman, H. E. (1985). Mood changes and patterns of food consumption during bingeing and purging: Are there underlying neurobiologic relationships? In W. H. Kaye & H. E. Gwirtsman (Eds.), The Treatment of Normal Weight Bulimia. (pp. 19-36). Washington: American Psychiatric Press.
- Kaye, W. H., Gwirtsman, H. E., Brewerton, T. D., George, D.T., Jimerson, D. C. & Ebert, M. H. (1987). Serotonin regulation in bulimia. In J. I. Hudson & H. G. Pope (Eds.), The Psychobiology of Bulimia. (pp. 158-174). Washington: American Psychiatric Press.
- Knesevich, J.W., Biggs, J.T., Clayton, P.J. and Ziegler, V.E. (1977). Validity of the Hamilton Rating Scale for Depression. British Journal of Psychiatry, 131, 49-52.
- Krauchi, K. and Wirz-Justice, A. (1988). The four seasons: Food intake frequency in seasonal affective disorder in the course of a year. Psychiatry Research 25, 323-338.
- Krauchi, K., Wirz-Justice, A. and Graw, P. (1990). The relationship of affective state to dietary preference: Winter depression and light therapy as a model. Journal of Affective Disorders, 20, 43-53
- Kripke, D.F., Risch, S.C. and Janowsky, D. (1983). Bright white light alleviates depression. Psychiatry Research, 10, 105-112.

- Lacoste, V. and Wirz-Justice, A. (1989). Seasonal variation in normal subjects: An update of variables current in depression research. In N.E. Rosenthal and M.C. Blehar, (Eds.), Seasonal Affective Disorders and Phototherapy. (pp.167-229). New York: Guildford Press.
- Lam, R.W. (1989). Light therapy for seasonal bulimia. American Journal of Psychiatry, 146, 1640-1641.
- Leibowitz, S.F. and Shor-Posner, G. (1986). Brain serotonin and eating behavior. Appetite, 7, 1-13.
- Levitsky, D.A., Schuster, J.A., Stallone, D. and Strupp, B.J. (1986). Modulation of the thermic effect of food by fenfluramine. International Journal of Obesity, 10, 169-173.
- Lewy, A. J., Kern H. A., Rosenthal, N. E. et al. (1982). Bright artificial light treatment of a manic depressive patient with a seasonal mood cycle. American Journal of Psychiatry, 139, 1496-1498.
- Lewy, A.J. and Sack, R.L. (1986). Light therapy and psychiatry. Proceedings of the Society for Experimental Biology and Medicine, 183, 11-18.
- Lewy, A.J., Sack, R.L., Miller, S. and Hoban, T.M. (1987). Anti-depressant and circadian phase-shifting effects of light. Science, 235, 352-354.
- Lewy, A.J., Sack, R.L. and Singer, C.M. (1985). Melatonin, light and chronobiological disorders. In D. Evered and S. Clark (Eds.), Photoperiodism, Melatonin and the Pineal, (pp. 231-252). London: Pitman.
- Lewy, A. J., Wehr, t. A., Goodwin, F. K., Newsome, D.A. and Markey, S.P. (1980). Light suppresses melatonin secretion in humans. Science, 210, 1267-1269.
- Magnusson, A. and Kristbjarson, H. (1991). Treatment of seasonal affective disorder with high-intensity light: A phototherapy study with an Icelandic group of patients. Journal of Affective Disorders, 21, 141-147.
- Maier, W., Heuser, I., Philipp, M., Frommberger, U. and Demuth, W. (1988). Improving depression severity assessment. II. Content, concurrent and external validity of three observer depression scales. Journal of Psychiatric Research, 22, 13-19.
- McGrath, R.E., Buckwald, B. and Resnick, E.V. (1990). The effect of l-tryptophan on seasonal affective disorder. Journal of Clinical Psychiatry, 51(4), 162-163.

- McNair, D.M., Lorr, M. and Doppleman, L.F. (1971). POMS Manual. San Diego, CA: Educational and Industrial Testing Service.
- Mitchell, J. E. & Eckert, E. D. (1987). Scope and significance of eating disorders. Journal of Consulting and Clinical Psychology, 55 (5), 628-634.
- Mitchell, J. E. & Goff, G. (1984). Bulimia in male patients. Psychosomatics, 25 (12), 909-913.
- Mitchell, J. E., Hatsukami, D., Eckert, E. D. & Pyle, R. L. (1985). Characteristics of 275 patients with bulimia. American Journal of Psychiatry, 142 (4), 482-485.
- Mitchell, J.E., Hatsutkami, D., Pyle, R. L. & Eckert, E. D. (1986). The bulimia syndrome: Course of the illness and associated problems. Comprehensive Psychiatry, 27 (2), 165-170.
- Mitchell, J.E. and Laine, D.C. (1985). Monitored binge-eating behavior in patients with bulimia. International Journal of Eating Disorders, 4, 177-183.
- Mitchell, J. E., Pyle, R. L. & Eckert, E. D. (1981). Frequency and duration of binge eating episodes in patients with bulimia. American Journal of Psychiatry, 138(6), 835-836.
- Mocre, R.Y. (1977). Central neural control of circadian rhythms. In W.F. Ganong and L. Martini (Eds.), Frontiers in Neuroendocrinology, Vol 5, New York: Raven Press.
- Mrosovsky (1989). Seasonal affective disorder, hibernation, and annual cycles in animals: Chipmunks in the sky. In N.E. Rosenthal and M.C. Blehar, (Eds.), Seasonal Affective Disorders and Phototherapy, (pp. 127-148). New York: Guildford Press.
- Muuss, R. E. (1986). Adolescent eating disorder: Bulimia. Adolescence, 21 (82), 257-267.
- Oren, D.A., Brainard, G.C., Johnston, S.H., Joseph-Vanderpool, J.R., Sorek, E. and Rosenthal, N.E. (1991). Treatment of seasonal affective disorder with green and red light. American Journal of Psychiatry, 148(4), 509-511.

- O'Rourke, D., Wurtman, J. J., Brzezinski, A., Abou-Nader, P. Marchant, & Wurtman, R. J. (1989). Treatment of seasonal affective disorder with d-fenfluramine. Annals of the New York Academy of Sciences, 575, 329-330.
- O'Rourke, D., Wurtman, J. J., Wurtman, R. J., Chebli, R. & Gleason, R. (1989). Treatment of seasonal depression with d-fenfluramine. Journal of Clinical Psychiatry, 50 (9), 343-347.
- Paykel, E. S. (1977). Depression and appetite. Journal of Psychosomatic Research, 21, 401-407.
- Perez, E. L., Blouin, J. & Blouin, A. (1988). The dexamethasone suppression test in bulimia: Non suppression associated with depression and suboptimal weight. Journal of Clinical Psychiatry, 49 (3), 94-96.
- Polivy, J. & Herman, P. (1985). Dieting and bingeing: A causal analysis. American Psychologist, 40 (2), 193-201.
- Pope, H. G., Hudson, J. I., Jonas, J. M. & Yurgelun-Todd, D. (1983). Bulimia treated with imipramine: A placebo controlled, double-blind study. American Journal of Psychiatry, 140 (5), 554-558.
- Pope, H. G., Hudson, J. I. & Yurgelun-Todd, D. (1984). Anorexia nervosa and bulimia among 300 suburban women shoppers. American Journal of Psychiatry, 141 (2), 292-294.
- Pope, H. G., Hudson, J. I. & Yurgelun-Todd, D. (1989). Depressive symptoms in bulimic depressed and non-psychiatric control subjects. Journal of Affective Disorders, 16, 93-99.
- Potkin, S., Zetin, M., Stamenkovic, Kripke, D. F. & Bunney, W. E. (1986). Seasonal affective disorder: Prevalence varies with latitude and climate. Clinical Neuropharmacology, 9, 181-183.
- Potts, M.K., Daniels, M., Burnam, A. and Wells, K.B. (1990). A structured interview version of the Hamilton Depression Rating Scale: Evidence of reliability and versatility of administration. Journal of Psychiatric Research, 24, 335-350.
- Pyle, R. L., Mitchell, J. E. & Eckert, E. D. (1981). Bulimia: A report of 34 cases. Journal of Clinical Psychiatry, 42 (2), 60-64.

- Pyle, R. L., Mitchell, J. E., Eckert, E. D., Halvorson, P. A., Neuman, P. A. & Goff, G. M. (1983). The incidence of bulimia in freshman college students. International Journal of Eating Disorders, 2, 75-85.
- Reme, C., Terman, M. and Wirz-Justice, A. (1990). Are deficient retinal renewal mechanisms involved in the pathogenesis of winter depression? Archives of General Psychiatry, 47, 878-879.
- Robins, L. N., Helzer, J. E., Croughon, J. and Ratcliffe, K. (1981). The National Institute of Mental Health Diagnostic Interview Schedule (NIMH- DIS). Archives of General Psychiatry, 38, 381-389.
- Robins, L. N., Helzer, J. E., Ratcliffe, K. and Seyfried, W. (1982). Validity of the Diagnostic Interview Schedule: DSM III diagnoses. Psychological Medicine, 12, 855-870.
- Rosen, J. & Leitenberg, H. (1984). Bulimia nervosa: Treatment with exposure plus response prevention. Behavioral Therapy, 13, 117-124.
- Rosen, J. Leitenberg, H., Fisher, C. and Khazam, C. (1986). Binge-eating episodes in bulimia nervosa. The amount and type of food consumed. International Journal of Eating Disorders, 5, 255-267.
- Rosenthal, N. E., Genhart, M., Jacobsen, F. M., Skwerer, R. G. & Wehr, T. A. (1987). Disturbances of appetite and weight regulation in seasonal affective disorder. Annals of the New York Academy of Sciences, 499, 216-230.
- Rosenthal, N. E., Genhart, M., Sack, D. A., Skwerer, R. G. & Wehr, T. A. (1987). Seasonal affective disorder and its relevance for the understanding and treatment of bulimia. In J. I. Hudson & H. G. Pope, Jr. (Eds.), The Psychobiology of Bulimia (pp. 205-228). Washington, DC: APA.
- Rosenthal, N. E. & Heffernan, M. M. (1986). Bulimia, carbohydrate craving, and depression: A central connection? In R. J. Wurtman & J. J. Wurtman (Eds.), Nutrition and the brain. Volume 7 (pp. 139-166).
- Rosenthal, N.E., Jacobsen, F.M., Sack, D.A., Arendt, J., Parry, B.L. and Wehr, T.A. (1988). Atenolol in seasonal affective disorder: A test of the melatonin hypothesis. American Journal of Psychiatry, 145, 52-56.
- Rosenthal, N.E., Sack, D. A., Carpenter, C.J., Parry, B.L., Mendelson, W.B. and Wehr, T.A. (1985). Antidepressant effects of light in seasonal affective disorder. American Journal of Psychiatry, 142(2), 163-170.

- Rosenthal, N. E., Sack, D. A., Gillin, J. C., Lewy, A. J., Goodwin, F. K., Davenport, Y., Mueller, P. S. & Wehr, T. A. (1984). Seasonal affective disorder: A description of the syndrome and preliminary findings with light therapy. Archives of General Psychiatry, 41, 72-80.
- Rosenthal, N.E., Sack, D.A., Jacobsen, F.M., James, S.P., Parry, B.L., Arendt, J., Tamarkin, L. and Wehr, T.A. (1986). Melatonin in seasonal affective disorder and phototherapy. Journal of Neurotransmission, 21 (Suppl.), 257-267.
- Rosenthal, N.E., Sack, D.A., James, S.P., Parry, B.L., Mendelson, W.B., Tamarkin, L. and Wehr, T.A. (1985). Seasonal affective disorder and phototherapy. Annals of the New York Academy of Sciences, 453, 260-269.
- Rosenthal, N.E., Sack, D.A., Skwerer, R.G., Jacobsen, F.M. and Wehr, T.A. (1989). Phototherapy for seasonal affective disorder. In N.E. Rosenthal and M. Blehar, M. (eds.), Seasonal Affective Disorders and Phototherapy. (pp. 273-294). New York: Guildford.
- Russel, G. (1979). Bulimia nervosa: An ominous variant of anorexia nervosa. Psychological Medicine, 9, 429-448.
- Sack, R.L., Lewy, A.J., White, D.M., Singer, C.M., Fireman, M.J. and Vandiver, R. (1990). Morning versus evening light treatment for winter depression. Archives of General Psychiatry, 47, 343-351.
- Samanin, R. & Garattini, S. (1989). Serotonin and the pharmacology of eating disorders. Annals of the New York Academy of Sciences, 575, 194-207.
- Schwab, J.J., Bialow, M.R. and Holzer, C.E. (1967). A comparison of two rating scales for depression. Journal of Clinical Psychology, 23, 94-96.
- Schilgen, B. and Tolle, R. (1980). Partial sleep deprivation as therapy for depression. Archives of General Psychiatry, 37, 267.
- Skwerer, R.G., Jacobsen, F.M., Duncan, C.C., Kelly, K.A., Sack, D.A., Tamarkin, L., Gaist, P.A., Kasper, S. and Rosenthal, N.E. (1989). Neurobiology of seasonal affective disorder and phototherapy. In N.E. Rosenthal and M.C. Blehar (Eds.), Seasonal Affective Disorders and Phototherapy. (pp.311-332). New York: Guildford Press.

- Souetre, E., Salvati, E., Belugou, J.L., Douillet, P, Braccinni, T. and Darcourt, G. (1987). Seasonality of suicides: environmental, sociological and biological covariations. Journal of Affective Disorders, 13, 215-225
- Steiger, H. (1989). Anorexia nervosa and bulimia in males: Lessons from a low-risk population. Canadian Journal of Psychiatry, 34, 419-424.
- Stern, S. L., Dixon, K. N., Nemzer, E. Lake, M.D., Sansone, R.A., Smeltzer, D.J., Lantz, S. and Schrier, S.S. (1984). Affective disorder in the families of women with normal weight bulimia. American Journal Psychiatry, 141, 1224-1227.
- Stewart, J.W., Quitkin, F.M., Terman, M. and Terman, J.S. (1990). Is seasonal affective disorder a variant of atypical depression? Differential response to light therapy. Psychiatry Research, 33, 121-128
- Strangler, R. S. & Printz, A. M. (1980). DSM-III: Psychiatric diagnosis in a university population. American Journal of Psychiatry, 137, 937-940.
- Swift, W. J., Andrews, D. & Barklage, N. E. (1986). The relationship between affective disorders and eating disorders: A review of the literature. American Journal of Psychiatry, 143 (3), 290-299.
- Szmukler, G. I. (1985). The epidemiology of anorexia nervosa and bulimia. Journal of Psychiatric Research, 19, 143-153.
- Terman, M. (1988). On the question of mechanism in phototherapy for seasonal affective disorder: Considerations of clinical efficacy and epidemiology. Journal of Biological Rhythms, 3 (2), 155-172.
- Terman, M., Terman, J.S., Quitkin, F.M., Stewart, J.W., McGrath, P.J., Nunes, E.V., Wager, S.G. and Tricamo, E. (1989). Dosing dimensions of light therapy: Duration and time of day. In C. Thompson and T. Silverstone (Eds.), Seasonal Affective Disorder, London: CRC Clinical Neuroscience.
- Terman, M., Terman, J.S., Quitkin, F.M., McGrath, P.J., Stewart, J.W. and Rafferty, B. (1989). Light therapy for seasonal affective disorder: A review of efficacy. Neuropsychopharmacology, 2(1), 1-21.
- Thompson, C., Stinson, D., Fernandez, M., Fine, J. & Isaacs, G. (1988). A comparison of normal, bipolar and seasonal affective disorder subjects using the Seasonal Pattern Assessment Questionnaire. Journal of Affective Disorders, 14, 257-264.

- VanStaveren, W.A., Deurenberg, P., Beurema, J., DeGroot, L.C.P and Hautvast, J.G.A. (1986). Seasonal variation in food intake, pattern of physical activity and change in body weight in a group of young adult Dutch women consuming self-selected diets. International Journal of Obesity, **10**, 133-145
- Viesselman, J. O. & Roig, M. (1985). Depression and suicidality in eating disorders. Journal of Clinical Psychiatry, **46**, 118-124.
- Volz, H.P., Mackert, A., Stieglitz, R.D. and Muller-Oerlinghausen, B. (1990). Effect of bright white light therapy on non-seasonal depressive disorder: Preliminary results. Journal of Affective Disorder, **19**, 15-21.
- Wade, (1989). Seasonal variations in body weight and metabolism in hamsters. In N.E. Rosenthal and M.C. Blehar, (Eds.), Seasonal Affective Disorders and Phototherapy, (pp. 105-126). New York: Guildford Press
- Walsh, B.T., Gladis, M. and Roose, S.P. (1987). Food intake and mood in anorexia nervosa and bulimia. Annals of the New York Academy of Sciences, **499**, 231-236.
- Walsh, B. T., Kissileff, H. R., Cassidy, S. M. & Dantzic, S. (1989). Eating behavior of women with bulimia. Archives of General Psychiatry, **46**, 54-58.
- Walsh, B. T., Roose, S. P., Glassman, A. H., Gladis, M. & Sadik, C. (1985). Bulimia and depression. Psychosomatic Medicine, **47** (2), 123-131.
- Warwick, E. (1989). Nutrient Analysis Program. Home Economics Department, University of PEI.
- Wegscheider-Hyman (1990). The Light Book: How Natural and Artificial Light Affect our Health, Mood and Behaviour. Los Angeles: J.P. Tarcher, Inc.
- Wehr, T. A., Jacobsen, F. M., Sack, D. A., Arendt, J., Tamarkin, L. and Rosenthal, N.E. (1986). Phototherapy of seasonal affective disorder: Time of day and suppression of melatonin are not critical for antidepressant effects. Archives of General Psychiatry, **43**, 870-875.
- Wehr, T. A. & Rosenthal, N. E. (1989). Seasonality and affective illness. American Journal of Psychiatry, **146** (7), 829-839.
- Wehr, T. A., Sack, D. A. & Rosenthal, N. E. (1987). Seasonal affective disorder with summer depression and winter hypomania. American Journal of Psychiatry, **144**, 1602-1603.

- Wehr, T.A., Skwerer, R.G., Jacobsen, F.M., Sack, D.A. and Rosenthal, N.E. (1987). Eye versus skin phototherapy of seasonal affective disorder. American Journal of Psychiatry, 144(6), 753-757.
- Weiss, S. R. & Ebert, M. H. (1983). Psychological and behavioral characteristics of normal-weight bulimics and normal-weight controls. Psychosomatic Medicine, 45 (4), 293-303.
- Whitaker, A., Davies, M., Shaffer, D., Johnson, J., Abrams, S., Walsh, B. T. & Kalikow, K. (1989). The struggle to be thin: a survey of anorexic and bulimic symptoms in a non-referred adolescent. Psychological Medicine, 19, 143-163.
- Williams, J.B. (1988). A structured interview guide for the Hamilton Depression Rating Scale. Archives of General Psychiatry, 45, 742-747.
- Williams, J.B., Link, M.J., Rosenthal, N.E. and Terman, M. (1988). Structured Interview Guide for the Hamilton Depression Rating Scale- Seasonal Affective Disorders Version. Unpublished document, New York State Psychiatric Institute, New York, New York.
- Wilkinson, L. (1989). SYSTAT : The System for Statistics. Evanston, IL: SYSTAT, Inc.
- Wirz-Justice, A., Bucheli, C., Graw, P., Kielholz, P., Fisch, H.U. and Woggon, (1986). Light treatment of seasonal affective disorder in Switzerland. Acta Psychiatrica Scandinavia, 74, 193-204.
- Wirz-Justice, A., Graw, P., Krauchi, K., Gisler, B., Arendt, J., Aldhous, M. and Poldinger, W. (1990). Morning or night-time melatonin is ineffective in seasonal affective disorder. Psychiatry Research, 24 (2), 129-137
- Wirz-Justice, A., Schmid, A.C., Graw, P., Krauchi, K., Kielholz, P., Fisch, H.U. and Bubbeberg, C. (1987). Dose relationships of morning bright white light in seasonal affective disorders. Experientia, 43, 574.
- Woell, C., Fichter, M. M., Pirke, K. & Wolfram, G. (1989). Eating behavior of patients with bulimia nervosa. International Journal of Eating Disorders, 8 (5), 557-568.
- Wurtman, J. J. (1984). The involvement of brain serotonin in excessive carbohydrate snacking by obese carbohydrate cravers. Journal of the American Dietetic Association, 84 (9), 1004-1007.

- Wurtman, J. J. (1987). Carbohydrate craving: A disorder of food intake and mood. In J. I. Hudson & H. G. Pope, Jr. (Eds.), The Psychobiology of Bulimia (pp.229-239). Washington, DC: APA.
- Wurtman, J. J. and Wurtman, R. J. (1979). Drugs that enhance central serotonergic transmission diminish elective carbohydrate consumption by rats. Life Sciences, 24, 895-903.
- Wurtman, J. J., Wurtman, R. J., Growdon, J. H., Henry, P., Lipsomb, M. Zeisel, S. H. (1981). Carbohydrate craving in obese people: Suppression by treatments affecting serotonergic transmission. International Journal of Eating Disorders, 1, 2-11.
- Wurtman, R.J. (1983). Behavioral effects of nutrients. Lancet, 1, 1145-1147.
- Wurtman, R. J., O'Rourke, D. & Wurtman, J. J. (1989). Nutrient imbalances in depressive disorders: Possible brain mechanisms. Annals of the New York Academy of Sciences, 575, 75-82.
- Wurtman, R. J. & Wurtman, J. J. (1989). Carbohydrates and depression. Scientific American, 260 (1), 68-75.
- Young, S.N., Smith, S.E. and Pihl, R.O. (1985). Tryptophan depletion causes a rapid lowering of mood in normal males. Psychopharmacology, 87, 173-177.
- Zimmerman, M., Coryell, W., Pfohl, B. and Stangl, D. (1986). Validity of the Hamilton endogenous subscale: independent replication. Psychiatry Research, 18, 209-215.
- Zucker (1989). Seasonal affective disorders: Animal Models non fingo. In N.E. Rosenthal and M.C. Blehar, (Eds.), Seasonal Affective Disorders and Phototherapy, (pp. 149-164). New York: Guildford Press.

Appendix A

Informed Consent

TITLE OF RESEARCH: PHOTOTHERAPY IN BULIMIA NERVOSA.

INVESTIGATOR: DR. A. BLOUIN

FOR INFORMATION CONTACT: DR. A. BLOUIN (613) 761-4746

INFORMED CONSENT

This study titled, PHOTOTHERAPY IN BULIMIA NERVOSA, is being conducted by researchers from the Eating Disorder Clinic of the Ottawa Civic Hospital. This study is designed to investigate the effect of light therapy (phototherapy) on symptoms of bulimia nervosa.

My participation will mean that for one week prior to receiving the light therapy, I will fill out some questionnaires pertaining to my symptoms (self-monitoring), on a daily basis. Filling out these questionnaires will take approximately 20 minutes daily. Following this, I will come for one week on seven consecutive days, to receive two hours of light exposure each evening. During each light exposure session, I will sit in front of a light fixture (rectangular light box measuring approximately 2 ft. by 4 ft.) and glance intermittently for a few seconds at a time, at the light. I may read or engage in light sedentary activity (such as knitting or crocheting) while undergoing the light treatment.

I agree to fill out a few brief forms pertaining to my symptoms on a daily basis for one week prior to light therapy, again during the week of light therapy, and for one week following the light therapy. There are four assessment sessions, one each week of the study beginning at the start of the first week, with the last one being at the end of the last week of my participation. Each assessment will take approximately one to one and a half hours and will involve filling out some questionnaires and receiving a brief interview.

Informed Consent....Phototherapy cont'd....2

During the week of light treatment, I agree to attend these sessions at the Ottawa Civic Hospital from 5:00 p.m. until 7:00 p.m. for seven consecutive days. I understand that the study involves giving some participants a higher intensity of light than others. I will not be told until the end of the study which light intensity I am receiving. Nor will the investigator conducting my interviews know which intensity I am assigned to until the end of my participation. At that time, I may decide to try the other light intensity, if I wish.

I understand that there is a small risk that I may feel somewhat irritable or that my eyes may initially feel uncomfortable during the light exposure. These effects are not long lasting and usually dissipate if the exposure to and/or distance from the light is adjusted. If the feeling persists, I will notify the investigator. I may or may not notice improvement in my bulimic symptoms and my moods.

While there is no permanent benefit from the light therapy, I may be identified as someone who's symptoms may be improved by regular light exposure during the winter months.

I understand that my participation is completely voluntary and that I may withdraw at any time, without prejudice, to my treatment in the Eating Disorder Clinic, or my relationship with anyone or any service at the Ottawa Civic Hospital. The overall results of this study may be published in scientific journals, or presented at scientific meetings, but my identity will remain strictly confidential.

I voluntarily agree to participate in this study titled "Phototherapy in Bulimia Nervosa". I may have a copy of this form if I wish.

SIGNED:_____ DATE:_____ AGE:_____

WITNESS:_____

Appendix B

Psychological Inventories

Eating Disorder Inventory (EDI)

Symptom Checklist-90 Revised (SCL-90-R)

Seasonal Pattern Assessment Questionnaire (SPAQ)

Bulimic Symptoms Checklist (BSC)

Beck Depression Inventory (BDI)

Structured Interview Guide for the Hamilton Depression Rating Scale

Seasonal Affective Disorders Version (SIGH-SAD)

Daily Binge Record (DBR)

Profile of Mood States (POMS)

THE FOLLOWING MATERIAL HAS BEEN REMOVED DUE TO COPYRIGHT
RESTRICTIONS.

PLEASE CONTACT THE UNIVERSITY LIBRARY.

LE MATERIEL SUIVANT A ETE ENLEVE DUE AU DROIT D'AUTEUR.

S. V. P. CONTACTER LA BIBLIOTHEQUE DE L'UNIVERSITE.

NATIONAL LIBRARY OF CANADA
CANADIAN THESES SERVICE

BIBLIOTHEQUE NATIONALE DU CANADA
LE SERVICE DES THESES CANADIENNES

Eating Disorder Inventory (EDI)

David M. Garner, Ph.D.
Marion P. Olmstead, M.A.
Janet Poilvy, Ph.D.

SCL 90

NAME _____

DATE (D/M/Y) ____/____/____

INSTRUCTIONS

BELOW IS A LIST OF PROBLEMS AND COMPLAINTS THAT PEOPLE SOMETIMES HAVE. PLEASE READ EACH ONE CAREFULLY. AFTER YOU HAVE DONE SO PLEASE FILL IN ONE OF THE NUMBERED SPACES TO THE RIGHT THAT BEST DESCRIBES HOW MUCH THAT PROBLEM HAS BOTHERED OR DISTURBED YOU DURING THE PAST INCLUDING TODAY. MARK ONLY ONE NUMBERED SPACE FOR EACH PROBLEM AND DO NOT SKIP ANY ITEM. PLEASE DO NOT MAKE ANY EXTRA MARKS ON THE SHEET. PLEASE USE THE EXAMPLE BELOW BEFORE BEGINNING

EXAMPLE

HOW MUCH WERE YOU BOTHERED BY:

not at a little moderately quite a extremely
all bit bit

1. BACKACHE (PAST WEEK) ☐ ☒ ☐ ☐ ☐

HOW MUCH WERE YOU BOTHERED BY:

not at a little moderately quite a extremely
all bit bit

1. HEADACHES	☐	☐	☐	☐	☐
2. NERVOUSNESS OR SHAKINESS INSIDE	☐	☐	☐	☐	☐
3. UNWANTED THOUGHTS, WORDS, OR IDEAS
THAT WON'T LEAVE YOUR MIND	☐	☐	☐	☐	☐
4. FAINTNESS, OR DIZZINESS	☐	☐	☐	☐	☐
5. LOSS OF SEXUAL INTEREST OR PLEASURE	☐	☐	☐	☐	☐
6. FEELING CRITICAL OF OTHERS	☐	☐	☐	☐	☐
7. THE IDEA THAT SOMEONE ELSE CAN CONTROL
YOUR THOUGHTS	☐	☐	☐	☐	☐
8. FEELING OTHERS ARE TO BLAME FOR MOST
OF YOUR TROUBLES	☐	☐	☐	☐	☐
9. TROUBLE REMEMBERING THINGS	☐	☐	☐	☐	☐
10. WORRIED ABOUT SLOPPINESS OR
CARELESSNESS	☐	☐	☐	☐	☐
11. FEELING EASILY ANNOYED OR IRRITATED	☐	☐	☐	☐	☐

HOW MUCH WERE YOU BOTHERED BY:

	not at all	a little bit	moderately	quite a bit	extremely
12. PAINS IN HEART OR CHEST	☐	☐	☐	☐	☐
13. FEELING AFRAID OF OPEN SPACES OR ON THE STREET	☐	☐	☐	☐	☐
14. FEELING LOW IN ENERGY OR SLOWED DOWN	☐	☐	☐	☐	☐
15. THOUGHTS OF ENDING YOUR LIFE	☐	☐	☐	☐	☐
16. HEARING VOICES THAT OTHER PEOPLE DO NOT HEAR	☐	☐	☐	☐	☐
17. TREMBLING	☐	☐	☐	☐	☐
18. FEELING THAT MOST PEOPLE CANNOT BE TRUSTED	☐	☐	☐	☐	☐
19. POOR APPETITE	☐	☐	☐	☐	☐
20. CRYING EASILY	☐	☐	☐	☐	☐
21. FEELING SHY OR UNEASY WITH THE OPPOSITE SEX	☐	☐	☐	☐	☐
22. FEELING OF BEING TRAPPED OR CAUGHT	☐	☐	☐	☐	☐
23. SUDDENLY SCARED FOR NO REASON	☐	☐	☐	☐	☐
24. TEMPER OUTBURSTS THAT YOU COULD NOT CONTROL	☐	☐	☐	☐	☐
25. FEELING AFRAID TO GO OUT OF YOUR HOUSE ALONE	☐	☐	☐	☐	☐
26. BLAMING YOURSELF FOR THINGS	☐	☐	☐	☐	☐
27. PAIN IN LOWER BACK	☐	☐	☐	☐	☐
28. FEELING BLOCKED IN GETTING THINGS DONE	☐	☐	☐	☐	☐
29. FEELING LONELY	☐	☐	☐	☐	☐
30. FEELING BLUE	☐	☐	☐	☐	☐
31. WORRYING TOO MUCH ABOUT THINGS	☐	☐	☐	☐	☐
32. FEELING NO INTEREST IN THINGS	☐	☐	☐	☐	☐
33. FEELING FEARFUL	☐	☐	☐	☐	☐

HOW MUCH WERE YOU BOTHERED BY:

	not at all	a little bit	moderately	quite a bit	extremely
34. YOUR FEELINGS BEING EASILY HURT	☐	☐	☐	☐	☐
35. OTHER PEOPLE BEING AWARE OF YOUR THOUGHTS	☐	☐	☐	☐	☐
36. FEELING OTHERS DO NOT UNDERSTAND YOU OR ARE UNSYMPATHETIC	☐	☐	☐	☐	☐
37. FEELING THAT PEOPLE ARE UNFRIENDLY OR DISLIKE YOU	☐	☐	☐	☐	☐
38. HAVING TO DO THINGS VERY SLOWLY TO INSURE CORRECTNESS	☐	☐	☐	☐	☐
39. HEART POUNDING OR RACING	☐	☐	☐	☐	☐
40. NAUSEA OR UPSET STOMACH	☐	☐	☐	☐	☐
41. FEELING INFERIOR TO OTHERS	☐	☐	☐	☐	☐
42. SORENESS OF YOUR MUSCLES	☐	☐	☐	☐	☐
43. FEELING THAT YOU ARE WATCHED OR TALKED ABOUT BY OTHERS	☐	☐	☐	☐	☐
44. TROUBLE FALLING ASLEEP	☐	☐	☐	☐	☐
45. HAVING TO CHECK AND DOUBLE CHECK WHAT YOU DO	☐	☐	☐	☐	☐
46. DIFFICULTY MAKING DECISIONS	☐	☐	☐	☐	☐
47. FEELING AFRAID TO TRAVEL ON BUSES, SUBWAYS, OR TRAINS	☐	☐	☐	☐	☐
48. TROUBLE GETTING YOUR BREATH	☐	☐	☐	☐	☐
49. HOT OR COLD SPELLS	☐	☐	☐	☐	☐
50. HAVING TO AVOID CERTAIN THINGS, PLACES OR ACTIVITIES BECAUSE THEY FRIGHTEN YOU	☐	☐	☐	☐	☐
51. YOUR MIND GOING BLANK	☐	☐	☐	☐	☐
52. NUMBNESS OR TINGLING IN PARTS OF YOUR BODY	☐	☐	☐	☐	☐
53. A LUMP IN YOUR THROAT	☐	☐	☐	☐	☐

HOW MUCH WERE YOU BOTHERED BY:

	not at all	a little bit	moderately	quite a bit	extremely
54. FEELING HOPELESS ABOUT THE FUTURE	☐	☐	☐	☐	☐
55. TROUBLE CONCENTRATING	☐	☐	☐	☐	☐
56. FEELING WEAK IN PARTS OF YOUR BODY	☐	☐	☐	☐	☐
57. FEELING TENSE OR KEYED UP	☐	☐	☐	☐	☐
58. HEAVY FEELINGS IN YOUR ARMS OR LEGS	☐	☐	☐	☐	☐
59. THOUGHTS OF DEATH OR DYING	☐	☐	☐	☐	☐
60. OVEREATING	☐	☐	☐	☐	☐
61. FEELING UNEASY WHEN PEOPLE ARE WATCHING OR TALKING ABOUT YOU	☐	☐	☐	☐	☐
62. HAVING THOUGHTS THAT ARE NOT YOUR OWN	☐	☐	☐	☐	☐
63. HAVING URGES TO BEAT, INJURE, OR HARM SOMEONE	☐	☐	☐	☐	☐
64. AWAKENING IN THE EARLY MORNING	☐	☐	☐	☐	☐
65. HAVING TO REPEAT THE SAME ACTION SUCH AS TOUCHING, COUNTING, OR WASHING	☐	☐	☐	☐	☐
66. SLEEP THAT IS RESTLESS OR DISTURBED	☐	☐	☐	☐	☐
67. HAVING URGES TO BREAK OR SMASH THINGS	☐	☐	☐	☐	☐
68. HAVING IDEAS OR BELIEFS THAT OTHERS DO NOT SHARE	☐	☐	☐	☐	☐
69. FEELING VERY SELF CONSCIOUS WITH OTHERS ...	☐	☐	☐	☐	☐
70. FEELING UNEASY IN CROWDS SUCH AS SHOPPING OR AT THE MOVIES	☐	☐	☐	☐	☐
71. FEELING EVERYTHING IS AN EFFORT	☐	☐	☐	☐	☐
72. SPELLS OF TERROR OR PANIC	☐	☐	☐	☐	☐
73. FEELING UNCOMFORTABLE ABOUT EATING OR DRINKING	☐	☐	☐	☐	☐
74. GETTING INTO FREQUENT ARGUMENTS	☐	☐	☐	☐	☐

HOW MUCH WERE YOU BOTHERED BY:

	not at all	a little bit	moderately	quite a bit	extremely
75. FEELING NERVOUS WHEN YOU ARE LEFT ALONE	☐	☐	☐	☐	☐
76. OTHERS NOT GIVING YOU PROPER CREDIT FOR YOUR ACHIEVEMENTS	☐	☐	☐	☐	☐
77. FEELING LONELY EVEN WHEN YOU ARE WITH PEOPLE	☐	☐	☐	☐	☐
78. FEELING SO RESTLESS YOU COULDN'T SIT STILL	☐	☐	☐	☐	☐
79. FEELINGS OF WORTHLESSNESS	☐	☐	☐	☐	☐
80. FEELING THAT FAMILIAR THINGS ARE STRANGE AND UNREAL	☐	☐	☐	☐	☐
81. SHOUTING OR THROWING THINGS	☐	☐	☐	☐	☐
82. FEELING AFRAID THAT YOU WILL FAINT IN PUBLIC	☐	☐	☐	☐	☐
83. FEELING THAT PEOPLE WILL TAKE ADVANTAGE OF YOU IF YOU LET THEM	☐	☐	☐	☐	☐
84. HAVING THOUGHTS ABOUT SEX THAT BOTHER YOU A LOT	☐	☐	☐	☐	☐
85. THE IDEA THAT YOU SHOULD BE PUNISHED FOR YOUR SINS	☐	☐	☐	☐	☐
86. FEELING PUSHED TO GET THINGS DONE	☐	☐	☐	☐	☐
87. THE IDEA THAT SOMETHING SERIOUS IS WRONG WITH YOUR BODY	☐	☐	☐	☐	☐
88. NEVER FEELING CLOSE TO ANOTHER PERSON	☐	☐	☐	☐	☐
89. FEELINGS OF GUILT	☐	☐	☐	☐	☐
90. THE IDEA THAT SOMETHING IS WRONG WITH YOUR MIND	☐	☐	☐	☐	☐

THE FOLLOWING MATERIAL HAS BEEN REMOVED DUE TO COPYRIGHT
RESTRICTIONS.

PLEASE CONTACT THE UNIVERSITY LIBRARY.

LE MATERIEL SUIVANT A ETE ENLEVE DUE AU DROIT D'AUTEUR.

S.V.P. CONTACTER LA BIBLIOTHEQUE DE L'UNIVERSITE.

NATIONAL LIBRARY OF CANADA
CANADIAN THESES SERVICE

BIBLIOTHEQUE NATIONALE DU CANADA
LE SERVICE DES THESES CANADIENNES

Structured Interview Guide for the Hamilton Depression Rating Scale

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

Seasonal Pattern Assessment Questionnaire

1. ID#: _____	2. Date: Month: _____ Day: _____ Year: _____
3. Birth place: City & Prov: _____ <div style="text-align: center; margin-top: 10px;">Country: _____</div>	4. Current weight (lbs.): _____ 5. How many years have you lived in this climate: _____

Complete this section only if willing to be contacted regarding a follow-up study to this one

Current Address: Street & Apt: _____ City & Prov: _____ Postal Code: _____ Telephone: (____) _____-____	Summer Address (if different from current): Street & Apt: _____ City & Prov: _____ Postal Code: _____ Telephone: (____) _____-____
--	---

The purpose of this questionnaire is to find out how your mood and behaviour change over time. Please fill-in the relevant blanks or boxes. Note: We are interested in your experience, not others you may have observed.

6. To what degree do the following change with the seasons?
 (MARK AN 'X' IN ONE SQUARE ONLY FOR EACH QUESTION)

	NO CHANGE	SLIGHT CHANGE	MODERATE CHANGE	MARKED CHANGE	EXTREMELY MARKED CHANGE
A. Sleep length	☐	☐	☐	☐	☐
B. Social activity	☐	☐	☐	☐	☐
C. Mood (overall feeling of well-being)	☐	☐	☐	☐	☐
D. Weight	☐	☐	☐	☐	☐
E. Appetite	☐	☐	☐	☐	☐
F. Energy level	☐	☐	☐	☐	☐

8. Using the scale below, indicate how the following weather changes make you feel.
(MARK AN X IN ONE SQUARE ONLY FOR EACH QUESTION)

-3	In very low spirits or markedly slowed down
-2	Moderately low/slowed down
-1	Mildly low/slowed down
0	No effect
+1	Slightly improves your mood or energy level
+2	Moderately improves your mood or energy level
+3	Markedly improves your mood or energy level

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

9. By how much does your weight fluctuate during the course of the year?

0-3 pounds.....Δ	12-15 pounds.....Δ
4-7 pounds.....Δ	16-20 pounds.....Δ
8-11 pounds.....Δ	Over 20 pounds.....Δ

SPAQ ID#: ____

10. Approximately how many hours of each 24-hour day do you sleep during each season?
(Include naps) Circle the appropriate number.

	Hours slept per day																		Over 18hrs	
WINTER	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	☐
SPRING	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	☐
SUMMER	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	☐
FALL	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	☐

11. Do you notice a change in food preference during the different seasons? ☐ Yes ☐ No

If yes, please specify: _____

12. If you experience changes with the seasons, do you feel these are a problem for you?

☐ Yes ☐ No

If yes, is this problem

MILD.....Δ

MODERATE.....Δ

MARKED.....Δ

SEVERE.....Δ

DISABLING.....Δ

15. Do you engage in or have you ever engaged in bingeing i.e consuming a large amount of food in a short period of time?

1. No
2. Yes.....

Mark an 'X' in the box(es) which corresponds to the month(s) of the year when you binge or cinged the most

[illegible]

16. Do you engage in or have ever engaged in purging (i.e. vomiting, laxative use, diuretic use, exercise, etc.) following these bingeing episodes?

1. No
2. Yes...

Mark an "X" in the box(es) which corresponds to the month(s) of the year when you purge or purged the most

[illegible]

17. In the following question mark an 'X' in as many boxes as necessary to indicate, in general, at what time of day you are most likely to ____

[illegible]

BSC

WEEKLY SYMPTOM CHECK LIST **ID#** **DATE:** .

1. Please indicate the number of episodes or times in the past week you experienced the following symptoms:

SYMPTOM **NUMBER OF EPISODES LAST WEEK**

- a) Binging _____
b) Vomiting _____
c) Purging _____
(Laxative and/or diuretic use)

2. Please rate the severity of the following symptoms using the corresponding rate scale. The rating of severity should correspond to the degree to which you have been experiencing these symptoms in the past week. Circle your choice for each item.

SYMPTOM

LEVEL OF SEVERITY

1. Binge eating.	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely
2. Vomiting.	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely
3. Other purging (i.e. laxative, and/or diuretic use etc).	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely
4. Pre-occupation with food.	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely
5. Lack of control over eating.	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely
6. Overall severity of these symptoms.	a) Not at all
b) A little
c) Moderately
d) Quite a bit
e) Extremely

BECK INVENTORY

INSTRUCTIONS:

Below there are groups of statements. Please read each group of statements carefully. Then pick the statement in each group that best describes the way you have been feeling in the past week, including today.

If several statements apply equally well, select each one. Be sure to read all the statements in each group before making your choice.

NAME: _____

DATE: _____

1. () 0 I do not feel sad
1 I feel sad
2 I am sad all the time and I can't snap out of it
3 I am so sad or unhappy that I can't stand it
2. () 0 I am not particularly discouraged about the future
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to
3 I feel that the future is hopeless and that things cannot improve
3. () 0 I do not feel like a failure
1 I feel I have failed more than the average person
2 As I look back on my life, all I can see is a lot of failures
3 I feel I am a complete failure as a person
4. () 0 I get as much satisfaction out of things as I used to
1 I don't enjoy things the way I used to
2 I don't get real satisfaction out of anything anymore
3 I am dissatisfied or bored with everything
5. () 0 I don't feel particularly guilty
1 I feel guilty a good part of the time
2 I feel quite guilty most of the time
3 I feel guilty all of the time
6. () 0 I don't feel I am being punished
1 I feel I may be punished
2 I expect to be punished
3 I feel I am being punished
7. () 0 I don't feel disappointed in myself
1 I am disappointed in myself
2 I am disgusted with myself
3 I hate myself
8. () 0 I don't feel I am any worse than anybody else
1 I am critical of myself for my weaknesses or mistakes
2 I blame myself all the time for my faults
3 I blame myself for everything bad that happens
9. () 0 I don't have any thoughts of killing myself
1 I have thoughts of killing myself, but I would not carry them out
2 I would like to kill myself
3 I would kill myself if I had the chance
10. () 0 I don't cry anymore than usual
1 I cry more now than I used to
2 I cry all the time now
3 I used to be able to cry, but now I can't even though I want to

NAME:

DATE:

- 11 () 0 I am no more irritated now than I ever am
1 I get annoyed or irritated more easily than I used to
2 I feel irritated all the time now
3 I don't get irritated at all by the things that used to irritate me
- 12 () 0 I have not lost interest in other people
1 I am less interested in other people than I used to be
2 I have lost most of my interest in other people
3 I have lost all of my interest in other people
- 13 () 0 I make decisions about as well as I ever could
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.
- 14 () 0 I don't feel I look any worse than I used to
1 I am worried that I am looking old or unattractive
2 I feel that there are permanent changes in my appearance that make me look unattractive
3 I believe that I look ugly
- 15 () 0 I can work about as well as before
1 It takes an extra effort to get started at doing something
2 I have to push myself very hard to do anything
3 I can't do any work at all
- 16 () 0 I can sleep as well as usual
1 I don't sleep as well as I used to
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep
3 I wake up several hours earlier than I used to and cannot get back to sleep
- 17 () 0 I don't get more tired than usual
1 I get tired more easily than I used to
2 I get tired from doing almost anything
3 I am too tired to do anything
- 18 () 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be
2 My appetite is much worse now
3 I have no appetite at all anymore
- 19 () 0 I haven't lost much weight, if any, lately
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds
3 I have lost more than 15 pounds
- I am purposely trying to lose weight by eating less Yes _____ No _____
- 20 () 0 I am no more worried about my health than usual
1 I am worried about physical problems such as aches and pains; or upset stomach, or constipation
2 I am very worried about physical problems and it's hard to think of much else
3 I am so worried about my physical problems, that I cannot think about anything else
- 21 () 0 I have not noticed any recent change in my interest in sex
1 I am less interested in sex than I used to be
2 I am much less interested in sex now
3 I have lost interest in sex completely

DAILY POMS DATE: _____

PARTICIPANT ID # _____

Below is a list of words that describe feelings people have. Please read each one carefully, then circle ONE of the numbers to the right which best describes how you have been feeling during the past 24 hours.

The numbers refer to these phrases:

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

1)	Friendly	0	1	2	3	4
2)	Tense	0	1	2	3	4
3)	Angry	0	1	2	3	4
4)	Worn out	0	1	2	3	4
5)	Unhappy	0	1	2	3	4
6)	Clear-headed	0	1	2	3	4
7)	Lively	0	1	2	3	4
8)	Confused	0	1	2	3	4
9)	Sorry for things done	0	1	2	3	4
10)	Shaky	0	1	2	3	4
11)	Listless	0	1	2	3	4
12)	Peeved	0	1	2	3	4
13)	Considerate	0	1	2	3	4
14)	Sad	0	1	2	3	4
15)	Active	0	1	2	3	4
16)	On edge	0	1	2	3	4
17)	Grouchy	0	1	2	3	4
18)	Blue	0	1	2	3	4
19)	Energetic	0	1	2	3	4
20)	Panicky	0	1	2	3	4
21)	Hopeless	0	1	2	3	4
22)	Relaxed	0	1	2	3	4
23)	Unworthy	0	1	2	3	4
24)	Spiteful	0	1	2	3	4
25)	Sympathetic	0	1	2	3	4
26)	Uneasy	0	1	2	3	4
27)	Restless	0	1	2	3	4
28)	Unable to concentrate	0	1	2	3	4
29)	Fatigued	0	1	2	3	4
30)	Helpful	0	1	2	3	4
31)	Annoyed	0	1	2	3	4
32)	Discouraged	0	1	2	3	4
33)	Resentful	0	1	2	3	4
34)	Nervous	0	1	2	3	4
35)	Lonely	0	1	2	3	4
36)	Miserable	0	1	2	3	4
37)	Muddled	0	1	2	3	4
38)	Cheerful	0	1	2	3	4
39)	Bitter	0	1	2	3	4
40)	Exhausted	0	1	2	3	4
41)	Anxious	0	1	2	3	4

END

1 | 8-07-94

FIN