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I am submitting herewith a dissertation written by Andrew Roy Getzfeld entitled "Characteristics of Female Bulimics and the Effectiveness of Desipramine Treatment." I have examined the final copy of this dissertation for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Doctor of Philosophy, with a major in Education.

R. Steve McCallum,
Major Professor

We have read this dissertation
and recommend its acceptance:

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CHARACTERISTICS OF FEMALE BULIMICS AND THE EFFECTIVENESS OF DESIPRAMINE TREATMENT

A Dissertation

Presented for the

Doctor of Philosophy

Degree

The University of Tennessee, Knoxville

Andrew Roy Getzfeld

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DEDICATION

This dissertation is dedicated to the memory of my grandfather, Henry Getzfeld, who taught me the true meaning of perseverance during difficult times in one's life.

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ABSTRACT

Relationships between bulimia (an eating disorder) and depression, bulimia and perceived sex role beliefs, and bulimia and dysfunctional body image perceptions were examined. In addition, the effectiveness of desipramine (an antidepressant) as a treatment modality for bulimia was examined. Eighteen women randomly assigned to either a placebo control group or an experimental group completed pretest Eating Disorder Inventory, Beck Depression Inventory and Bem Sex Role Inventory questionnaires. Women in the experimental group initially went through a two week placebo washout phase and then received desipramine during the six week active phase of the study. A t-test revealed a significant difference between the experimental and control group; the experimental group showed a significant reduction of the severity of bulimic symptoms ($t = 4.09$, $p < .001$). For the experimental group, a correlational analysis showed that desipramine blood levels displayed a low inverse trend with the reduction of bulimic symptoms. Additional correlational analyses from pretreatment data revealed a moderately strong positive relationship between the severity of bulimic symptoms and depression ($r = .60$); a negative relationship was also established between the severity of bulimic symptoms and perceived female sex role beliefs. Finally, no relationship was found between the severity of bulimic symptoms and dysfunctional body image perceptions.

The results indicate that bulimia may be related to depression, that the relationship between the severity of bulimic symptoms and perceived female sex role beliefs is unclear and requires further clarification, and that desipramine seems to be effective in reducing the severity of bulimic symptoms. Evidence was also provided for the need to establish an effective therapeutic range for desipramine in bulimic women.

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Introduction

Eating disorders are a serious problem in the United States, and their prevalence seems to be increasing. They have recently received much media exposure; for example, Princess Diana of England has often been mentioned on television as having the eating disorder bulimia nervosa. Other examples of the desire to obtain a perfect body can be found by picking up fashion magazines, where the supermodels who dominate their pages possess "ideal" bodies.

As our society becomes more and more obsessed with obtaining perfect bodies, more and more women can be expected to manifest eating disorders. Due to the increasing prevalence of eating disorders in the United States (Mitchell & Eckert, 1987), I decided to undertake the following study.

CHAPTER ONE

OVERVIEW

Purpose

The primary purpose of this study was to examine the effectiveness of desipramine (an antidepressant) on the treatment of bulimia, an eating disorder. In addition, the design of this study allowed an investigation of the hypothesis that a relationship exists between bulimia and major depression (Mitchell, 1990) by comparing the results of the Eating Disorder Inventory (Garner et al., 1983) with the results of the Beck Depression Inventory (Beck et al., 1961). Finally, the Bem Sex Role Inventory (Bem, 1978) was used to examine the relationship between sex roles and bulimia, a relationship that has only rarely been empirically assessed in female bulimics (e.g. Steiger, Fraenkel & Leichner, 1989).

Background Information

Bulimia and anorexia nervosa are major health problems in the United States, especially in high schools, colleges, and universities. Published studies on the prevalence of bulimia, depending on the operational definition, include 26% to 79% of women assessed who report binge eating (Mitchell & Eckert, 1987).

Some authors (e.g. Mintz & Betz, 1988) have noted that fully developed cases of bulimia do not appear to be as prevalent on college campuses as many researchers believe.

Mintz and Betz (1988) found that only 20 out of 643 (3%) of the subjects in their study could be classified as being bulimic; however, only 211 out of 643 (33%) subjects reported "normal" eating habits. Drewnowski, Hopkins and Kessler (1988), using a national sample of 1,007 male and female college and university students in the United States and using Diagnostic and Statistical Manual of Mental Disorders (Third Edition-Revised) (DSM-III-R; American Psychiatric Association, 1987) criteria, found that 1% of the women and 0.2% of the men in their sample were classified as bulimic. The DSM-III-R (American Psychiatric Association, 1987) states that in a study of college freshman, 4.5% of the females and 0.4% of the males had a history of bulimia. When a criterion of at least one binge and purge episode per week was applied, Shisslak, Crago, Neal, and Swain (1987) examined various studies and found that the prevalence rate of bulimia in high school and college students varied from 1% to 3%. Halmi, Falk, and Schwartz (1981) found that of 355 college students they examined, 13% reported experiencing all of the major symptoms of bulimia.

Diagnosis is often difficult due to reliance on self-reports and the often clandestine nature of bulimics' behaviors; in addition, the treatment picture with this population is even more obscure (Mitchell & Eckert, 1987; Walsh, Gladis, Roose, Stewart, Stetner & Glassman, 1988).

Traditional long-term psychotherapy has often been difficult to maintain with bulimics; this may be due to a lack of client motivation, a lack of success, or to other unknown factors (Mitchell & Eckert, 1987). Fortunately, recent advances in pharmacological therapy, particularly in the area of antidepressant medications, have increased the efficacy of some treatment models. Pharmacological therapy seems to be an alternative worthy of consideration; it requires less intrinsic motivation than psychotherapy; it may be used to control the bulimic behavior when used in conjunction with other therapies and, in a sense, can be more cost-effective. Some studies have suggested that the use of antidepressant medications alone can be successful in the treatment of bulimia (e.g. Hughes et al., 1986; Walsh et al., 1988). Some studies have also hypothesized that a physiological basis exists for bulimia (e.g. Kaplan & Woodside, 1987). Other studies have hypothesized about the sex role confusions of bulimic women (e.g. Striegel-Moore, Silberstein & Rodin, 1986). Finally, some studies have attempted to link bulimia with affective disorders, usually major depression (Kaplan & Woodside, 1987; Mitchell & Eckert, 1987).

The Operational Definition of Bulimia

Most studies of bulimia use the DSM-III-R's or the Diagnostic and Statistical Manual of Mental Disorders (Third Edition)'s (DSM-III; American Psychiatric Association, 1987;

1980) definition of bulimia. The DSM-III-R criteria which must be met in order for someone to be diagnosed as bulimic include:

1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time. Eating binges may be planned. The food that is consumed during these binges may have a high caloric content, a sweet taste, and a texture that facilitates rapid eating. The food is usually eaten as inconspicuously as possible, or secretly. A binge is usually terminated by abdominal discomfort, sleep, social interruption, or induced vomiting. The food eaten during a binge may be gobbled down rather rapidly, with little chewing;
2. A feeling of lack of control over eating behavior during the eating binges;
3. Self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain. Vomiting decreases the physical pain of abdominal distention, allowing either continued eating or termination of the binge, and often reduces post-binge anguish. In some cases vomiting itself may be desired, so that the person may binge in order to vomit or will vomit after eating a small amount of food. Although eating binges

may be pleasurable, disparaging self-criticism and a depressed mood often follow;

4. Persistent overconcern with body weight and shape. Bulimics invariably exhibit great concern about their weight and make repeated attempts to control it by dieting, vomiting, or the use of diuretics. Frequent weight fluctuations due to alternating binges and fasts are common. Bulimics may feel that their lives are dominated by conflicts surrounding eating;

5. The subject must have had, on the average, a minimum of two binge eating episodes a week for at least three months in order to qualify for the diagnosis of bulimia nervosa. Some bulimics may be slightly underweight and some may be overweight, but most people with bulimia are within a normal weight range. A depressed mood that might be part of a Depressive Disorder is often seen. Some bulimics may have Psychoactive Substance Abuse or Dependence, most often involving sedatives, amphetamines, cocaine or alcohol (American Psychiatric Association, 1987, pp. 67-69).

The parents of bulimic individuals are often obese (American Psychiatric Association, 1987). A higher than expected frequency of Major Depression in first degree biologic relatives of bulimic people has been reported in several studies (e.g. Geracioti & Liddle, 1988; Keck, Pope,

Hudson, McElroy, Yurgelun-Todd & Hundert, 1990). The course of the illness is chronic and intermittent, lasting a period of many years (American Psychiatric Association, 1987, p. 68).

Physiological Mechanisms

Neurology

The diagnosis of bulimia is generally based on behavioral evidence (American Psychiatric Association, 1987); however, its etiology and underlying physiological mechanisms are unclear. Neurologically, Wermuth, Davis, Hollister and Stunkard (1977) found that only six out of twenty binge eaters had abnormal EEG's and these EEG's were not correlated with a treatment response. Johnson and Brief (1983) addressed the relationship between EEG abnormalities and bulimia and stated that bulimia may be related to epileptic-style seizures. It has been suggested that the relation between seizure disorders and bulimia may be traced to a single hypothalamic dysfunction or to an unknown dysfunction that increases the person's arousal state (Johnson & Brief, 1983).

Hypothalamus

Gandour (1984) and Herzog (1986) proposed that bulimia may be caused by a hypothalamic dysfunction, but Gandour (1984) concluded that the notion of a dysfunctional hypothalamus in bulimics should remain at the hypothetical stage. Irregular eating patterns may, in some way, alter

neurotransmitter levels to change the release of hypothalamic hormones. Kaplan and Woodside (1987) noted that the hypothalamic-pituitary function is indeed disturbed in people who have anorexia nervosa or bulimia; the disturbance may be of either the unique contributions of the pituitary or the hypothalamus or an interaction of the two. However, at least some of these disturbances are generally mediated by mechanisms that are secondary to the effects of disordered eating. For example, abnormalities in thermoregulation and in gonadotropic hormone levels relate directly to weight loss and are seen in other states of emaciation, regardless of the cause. These abnormalities, like amenorrhoea, may reflect the effects of weight loss, disordered eating, or other factors that are not known. Hsu (1990) noted that amenorrhoea can occur in response to nonspecific emotional stress, malnutrition, and weight loss. In a recent study of normal college-age women, two to five percent reported having amenorrhoea and ten percent reported having menstrual abnormalities (Hsu, 1990).

Menstrual abnormalities have also been shown to occur in female bulimics of normal weight. In one study of 34 bulimic women, 26 had at least one episode of amenorrhoea lasting at least three months (Pyle, Mitchell & Eckert, 1981). In a later study of 35 bulimic women, Fairburn & Cooper (1984) found that 38.6% of the bulimic patients had extremely erratic menstruation, 21.1% had amenorrhoea, 5.3%

(one patient) reported normal menstruation, and the other half of the sample used oral contraceptives which tend to artificially regulate the menstrual cycle. Thus, the percentages of bulimic women with menstrual abnormalities cited by Pyle et al. (1981) and by Fairburn and Cooper (1984) seem high when contrasted with those cited by Hsu (1990) for "normal" college-age women. In contrast, Geracioti and Liddle (1988) found that 12 out of 14 of the normal-weight bulimic patients in their study reported regular menstrual periods. The cause and relationship of bulimia with irregular menstruation or with amenorrhoea remains unclear.

Neurotransmitters

Other physiological mechanisms involving the hypothalamus may have neurotransmitter bases. Kaplan and Woodside (1987) also support the view, found most often in the animal literature, that neurotransmitters are directly implicated in the control of feeding behavior. Serotonin inhibits feeding by inducing satiety; norepinephrine stimulates feeding through its activity in the medial hypothalamus and paraventricular nucleus. Norepinephrine can also inhibit feeding through its action on the lateral hypothalamus. Both of these neurotransmitters may be affected by the availability of certain nutrients; for example, serotonin appears to be regulated by the availability of tryptophan. Infusion of norepinephrine into

the medial hypothalamus causes a specific preference for carbohydrates (Hsu, 1990) and seems to be produced when food is ingested (Kaplan & Woodside, 1987). Kaplan and Woodside (1987) also noted that serotonin may be involved in the regulation of mood, pain, sleep, appetite, and in the control of the hypothalamic and pituitary hormones. The key here is that all of these functions have been found to be disturbed in people with eating disorders.

Mitchell and Eckert (1987) noted that, in animal studies, the release of norepinephrine into the hypothalamus stimulated the animals to eat, even if the animal was sated from previous exposure to food. The amount of carbohydrates ingested also increased. Carbohydrate consumption produces an increase in the precursor for serotonin production. As serotonin increases, the rising level produces the opposite effect by decreasing eating behavior, specifically decreasing the desire to ingest carbohydrates (Mitchell & Eckert, 1987). Results of human studies showed that serotonin dysfunction and disregulation may occur in bulimics, thus possibly leading to the disinhibition of carbohydrate intake. Davis, Freeman and Garner (1988) reported that bulimics tended to binge more on grains and cereals during binging episodes, one of the diagnostic criteria of the DSM-III-R (American Psychiatric Association, 1987). Neurotransmitters, such as norepinephrine and serotonin, may be involved in the biological bases of eating

disorders, in the behavioral symptomatology as well as in the dynamic chemical state of the individual.

Hormones

Hormone levels have also been shown to vary between groups of bulimic and normal individuals. Geracioti and Liddle (1988) found that the hormone cholecystokinin was significantly impaired in bulimic patients. This was determined by comparing the bulimics' blood plasma levels of cholecystokinin with those blood plasma levels of normal women.

Cholecystokinin, located in the brain and in the gut, has been found to be involved in satiety regulation in rats, sheep, monkeys, and in humans (Bennett & McMartin, 1979; Geracioti & Liddle, 1988). Cholecystokinin, an amino acid polypeptide hormone secreted by the membrane of the upper intestine after eating, is also secreted by the hypothalamus. Cholecystokinin stimulates gallbladder contractions and pancreatic secretions and may impact on gut motility. That expansion or contraction of the gut, signaling satiety or hunger, often seems to be impaired in bulimics evidencing clinical symptoms such as dyspepsia (indigestion) and early satiety (Kaplan & Woodside, 1987).

Geracioti and Liddle (1988) concluded that bulimics do not have normal satiety and that they also have impaired secretion of cholecystokinin in response to a meal. A key suggestion made by these authors is that both of these

abnormalities may be improved by tricyclic antidepressants (a class of antidepressant drugs), although the mechanism by which they may augment cholecystokinin secretion is unknown.

Endorphins

The role of endogenous opiates (endorphins) may also be implicated. Studies of the fluctuations of blood levels of endorphins in obese individuals and as a function of conditioning and exercise may contribute to the knowledge base regarding the etiology of bulimia. The hypothesized involvement of endorphins in the causes of bulimia is based on their function. Endorphins are proteins that may act as neurotransmitters and are structurally similar to opiates; they have been linked to pain control, addictive behaviors, and to the control of eating (Mitchell, Laine, Morley & Levine, 1986). Their relationship to eating behavior is supported by findings that elevated endorphin levels were found in genetically obese rats (Margules, Moisset, Lewis, Shibuya, & Pert, 1978). In satiated rats, endorphins injected into the ventromedial hypothalamus were found to stimulate food intake (Grandison & Guidotti, 1977). A narcotic agonist, Naloxone, which suppresses the effects of endorphins, has been shown to stop the overeating behavior of genetically obese rats (Holtzman, 1974) and to cause weight loss and appetite loss when given to humans (Hollister, Johnson, Bookhabzer & Gillespie, 1981).

Mitchell et al. (1986) performed a human double-blind

study using trials of Naloxone in bulimics to examine elevated endorphin levels that exist in some obese people. Investigators sought to determine if eating behavior would be suppressed using narcotic antagonists. Presumably these antagonists, such as Naloxone, antagonize the endorphins (endogenous opioids), as they have been shown to antagonize exogenous narcotics such as morphine (Kyriakides, Silverston, Jeffcoate & Laurence, 1980; Morley & Levine, 1980). Mitchell et al. (1986) found that Naloxone significantly suppressed food intake during binge episodes in hospitalized bulimics. Jonas and Gold (1986) also supported these findings, but their experiment was an open label trial, i.e. the experimenters and the subjects were aware of the drug that was being used in the study. More double-blind, placebo-controlled trials of Naloxone would be needed to confirm positive effects on bulimics.

Cholesterol

Mira, Stewart, Vizzard and Abraham (1987) found that women with an eating disorder (anorexia nervosa, bulimia, or unclassified) had significantly higher cholesterol levels than their control group and significantly lower levels of potassium in their blood plasma. Decreased blood plasma potassium has been found more commonly in those women who induced vomiting or abused laxatives, common occurrences among bulimics (Mira et al., 1987). Large amounts of potassium are lost through the gastrointestinal tract in

people who vomit or purge as a means of controlling weight. Bulimics were shown to evidence elevated plasma cholesterol but less consistently than in the patients with anorexia nervosa. Mira et al. (1987) were unsure as to why cholesterol was elevated in subjects with eating disorders but hypothesized that it may be due to impaired clearance of Low Density Lipid (LDL) cholesterol, placing the eating disordered patient at an increased risk of cardiovascular disease (Mira et al., 1987). It has also been hypothesized that elevated cholesterol levels in bulimics may be a genetic marker but this has yet to be substantiated (Weninger & Getzfeld, 1992, in press).

Cortisols

Musisi and Garfinkel (1985) reported that cortisol secretion may be disturbed in bulimics. Abnormal results were noted in the dexamethasone suppression test (DST) in bulimics of normal weight. The DST's major purpose was to test an individual for endogenous depression and results may be due to depressive symptoms themselves, to abnormal eating patterns, or to a biologically underweight state even though normal weight is maintained. As a point of comparison, abnormal DST results may be related to weight loss and have been seen in nondepressed patients who had lost weight for other reasons (Fichter, Pirke & Holsboer, 1986).

As the literature demonstrates, no definite conclusions regarding the physiology of bulimia can be stated; however,

a number of hypotheses are suggested. If a hypothalamic dysfunction is involved in bulimia, the specifics of that dysfunction and the nature of its involvement are not yet clear. Serotonin and norepinephrine are shown in relation with the feeding-satiety process and may be dysfunctional in people who manifest bulimia and other eating disorders. It can also be hypothesized that serotonin and norepinephrine are related to the function of the hypothalamus and may reflect abnormalities. There is literature to support this view yet the origin of dysfunctional effects and the nature of interaction still remains unsubstantiated.

It is also possible that endorphins, related to obesity, may be implicated in bulimia. More importantly, the literature hypothesizes that the satiety function is impaired in bulimics and that serotonin and norepinephrine levels are also impaired. Included in this hypothesis is the notion that cholecystokinin levels are also impaired (Geracioti & Liddle, 1988). However, these physiological manifestations may be the results of disorders in eating, not the etiology. In effect, these studies demonstrate that a relationship appears to exist between a person's physiology and bulimia, but the actual cause of bulimia has yet to be identified.

Assessment Instruments

Bulimics are notoriously difficult to assess. Bulimics tend to be secretive about their behavior and are usually

diagnosed when their behavior has reached a crisis level (Gandour, 1984). Some prior studies (e.g. Drewnowski et al., 1988; Halmi et al., 1981; Mintz & Betz, 1988) have relied upon clinical interviews in order to gather data and to draw conclusions. This method may lead to experimenter and response biases. Some paper-and-pencil measures that have been used in previous research on bulimia are the Eating Disorder Inventory (Garner et al., 1983), and the Beck Depression Inventory (Beck et al., 1961), and less frequently the Bem Inventory (Bem, 1978).

Eating Disorder Inventory

The Eating Disorder Inventory (Garner et al., 1983) consists of sixty-four, forced-choice items that measure various behavioral and psychological traits that are relevant to anorexia nervosa and bulimia nervosa. The Eating Disorder Inventory (EDI) was not designed to be used as a diagnostic tool but rather as an additional piece of assessment information in making clinical judgements about clients who may have eating disorders. The authors recommend that the EDI be used as a screening device, outcome measure, or as an aid in research. It is appropriate for people who are 12 years of age and older and can be used in individual and group settings. An examiner should always be present and the examinee should be able to ask questions about items on the EDI. The item responses are based on a six-point Likert scale, with the choices

ranging from "Always" to "Never".

The items are classified according to one of eight constructs that are measured by the EDI: Drive for Thinness (DT), Bulimia (B), Body Dissatisfaction (BD), Ineffectiveness (I), Perfectionism (P), Interpersonal Distrust (ID), Interoceptive Awareness (IA), and Maturity Fears (MF). The most extreme anorectic response receives a raw score of "3". The response that is immediately adjacent to the most extreme response receives a "2", and the next response receives a "1". The three choices that are opposite to the most anoretic response receive scores of "0". Subscale scores are the summation of all of the item raw scores for that particular subscale. The test is untimed but usually completed in about twenty minutes.

Two groups of subjects were used in the validation of the EDI. The criterion group, called "AN" (anorectic) by Garner et al. (1983), consisted of three subsamples of women, mostly anorectics, who were seen in consultation at the Clarke Institute of Psychiatry. These women averaged 20% below both the expected height and weight, according to norms from Health and Welfare Canada. Of these women (N= 129), 56 were classified as anorectic "restricters" (they greatly restricted their food and liquid intake) and 73 were classified as "bulimic". There were significant differences ($p < .0001$) between the two groups in the mean percentage of average weight. The comparison group (FC) consisted of

three independent samples of female undergraduates (N= 770) who were enrolled in first- and second- year psychology classes. Items were retained if they demonstrated significant differences between the AN and FC groups, and if they correlated highly with their own subscales.

Garner et al. (1983) reported the psychometric information based on their final sample of 271 college women and 155 anorectics (53 "restricters" and 102 "bulimics"). Cronbach's alpha, used to determine internal consistency, was greater than .80 for each of the eight subscales. The average item total correlation was .63. Criterion related validity studies were conducted by comparing the self-report EDI client profiles with the clinical judgements of clinicians who were familiar with the clients' psychological makeups. The raters were asked to assess the relevancy of each of the eight constructs to the client compared to other anorectics whom they had treated. All interrater correlations were significant ($p < .001$); the correlations ranged from .43 (Maturity Fears subscale) to .68 (Ineffectiveness subscale). Criterion related validity was also demonstrated in that the "bulimic" and "restricter" anorectic groups scored in the theoretically expected manner on specific subscales. The EDI also differentiated between "restricters" and "bulimics" in a discriminant functional analysis which correctly classified 85% of the criterion group (AN) subjects into "bulimic" and "restricter" subtypes

based on their Bulimia subscale score. In a sample of 53 "restricters" and 102 "bulimics," the groups had significantly different scores on the Drive for Thinness, Ineffectiveness and Interoceptive Awareness subscales.

The EDI's reliability data were based upon the final sample of 271 college women who took the EDI. Reliability coefficients for the criterion group (AN) ranged from .83 on the Interoceptive Awareness subscale to .93 for the Ineffectiveness subscale. Reliability coefficients for the comparison group (FC) ranged from .72 for the Maturity Fears subscale to .92 for the Body Dissatisfaction subscale.

Swassing (1989) described the Eating Disorder Inventory (EDI) as an excellent instrument to use in assessing eating disorders. He explained that it adds to the clinical knowledge regarding the psychological and behavioral dimensions of anorexia nervosa and bulimia, and that it is useful in differentiating between these two disorders. He also noted that is can be used to differentiate between college women who are preoccupied with their weight from those who are anorectic, thus qualifying for a DSM-III-R (American Psychiatric Association, 1987) diagnosis of an eating disorder.

Klemchuk, Hutchinson, and Frank (1990) administered the EDI on two college campuses to three female undergraduate samples who did not have clinically diagnosed eating disorders. Very high rates of body dissatisfaction were

obtained from self-reports. Factor analyses of the EDI produced an Eating Disorders factor that was a combination of three of the EDI's subscales: Drive for Thinness, Bulimia, and Interoceptive Awareness. The other five factors were identical to the other five EDI subscales (Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, and Maturity Fears). Extreme EDI factor scores identified two risk groups: a binge/purge group with poor psychological adjustment and a body-dissatisfied group. Thus, the EDI has utility in campus intervention programs, particularly in screening those who are dissatisfied with their bodies and those who are at risk for bulimia (Klemchuk et al., 1990).

Paludni (1987), however, found some serious problems with the EDI. She noted that the EDI is sensitive to positive response bias by the client, further compounded in that denial is an inherent part of eating disorders (Gandour, 1984). Paludni (1987) also concluded that the EDI may lack external validity since the EDI was developed based on its ability to differentiate between a criterion group (AN) and nonclinical samples (FC) (See also Swassing, 1989). Another problem is that some clients may misunderstand or misinterpret some of the items, thus answering them inaccurately. Paludni (1987) stated that certain items that the EDI scores as "deviant" (those items that contribute to a subscale's score) are concerned with body image, areas

with which adolescents are typically concerned. Thus it is hazardous to correlate high scores on these items with anorexia nervosa. She concluded by stating that the EDI does not address the importance of gender role development and cultural aspects of a female's ideal body image.

Cooper, Cooper and Fairburn (1985) stated that Garner et al. (1983) did not indicate which of the eight subscales specifically dealt with eating disorders; some of the scales may assess aspects of psychological problems that are common in clients who have many psychological troubles, not necessarily eating disorders. Cooper et al. (1985) found that only three of the subscales correlated highly with eating disorders: Drive for Thinness, Body Dissatisfaction, and Bulimia. This adds support to the finding that the Drive for Thinness and Bulimia subscales of the EDI may be measuring the presence and severity of an eating disorder (Klemchuk et al., 1990). The five other scales had a much lower correlation with anorectics. Thus, these other five scales may indeed be assessing general psychopathology rather than only eating disorders.

Bem Inventory

The Bem Inventory is more commonly known as the Bem Sex Role Inventory (Bem, 1978). Steiger et al. (1989) concluded that sex-role disturbances might be active in the etiology of bulimia. However, the current literature is scarce and inconclusive.

The Bem Sex Role Inventory (BSRI) separately assesses psychological femininity and masculinity. It also has a category called "androgynous", referring to people who possess both feminine and masculine traits. The BSRI does not assume that masculinity and femininity form a single dimension but that they are conceptually and empirically distinct.

The BSRI asks respondents to rate themselves on 20 stereotypically feminine traits, 20 stereotypically masculine traits, and 20 filler items that are considered neutral. The rating system is a seven-point Likert scale, ranging from a low of "1" ("Never or almost never true") to a high of "7" ("Always or almost always true"). The test is untimed; most respondents are able to complete the test in about 15 minutes. The BSRI is designed for people who are of high school age and older.

A Femininity score, a Masculinity score, and a Femininity minus Masculinity score, also called an Androgyny score are produced from the BSRI. The Masculine and Feminine scales are totaled, and the median score for each scale is then calculated. A scoring system advocated by Bem was developed by Spence, Helmreich and Stapp (1975) and which revises the original scoring system. The system uses a median split on both the Masculine and Feminine scales that produces a fourfold classification table: Masculine, Feminine, Androgynous (above the median on both scales), and

Undifferentiated (below the median on both scales). The BSRI has two subscales, plus 20 items that are classified as neutral adjectives and do not figure either in the scoring or in the final analysis of the respondent.

The BSRI has good internal consistency and reliability. Using the scoring system developed by Spence et al. (1975), Bem (1981) reported the following coefficient alphas for the 1978 Stanford undergraduate sample of 340 females and 476 males: for women, .75 for the Femininity scale and .87 for the Masculinity scale; for men, .78 for the Femininity scale and .87 for the Masculinity scale. The BSRI Manual (1981) does not give evidence of the discriminant validity of the instrument.

The Bem Inventory (BSRI) Manual (1981) lists some of the most important factor analytic studies that have been conducted on the device, and most of these studies concluded that the BSRI's scales were not factorially pure. Bem (1981) responded to this point by stating that society's stereotypes are not necessarily consistent. This rationale was used in the development of the BSRI.

According to Lippa (1985), factor analyses have yielded the following results: two masculinity factors which could be labeled "Dominance/Aggression" and "Self-Reliance/Personal Control", one femininity factor which could be labeled "Interpersonal Sensitivity and Warmth", and one factor which is often correlated with biological sex,

encompassing three characteristics which are BSRI items: "Masculine", "Feminine", and "Athletic". "Athletic" is an item that is part of the BSRI's Masculinity scale and is an item that Bem (1981) believed was positively correlated with male sex role beliefs.

Schmitt and Millard (1988) performed factor analyses on all items of the BSRI for traditional sex-typed (masculine and feminine) and androgynous subjects separately. Their results showed that the two types of subjects (androgynous and sex-typed) responded differently to the BSRI's items. Bipolar masculinity/femininity factors were found for the sex-typed group and unipolar factors were found for the androgynous group, suggesting that the BSRI had discriminant validity in distinguishing the two groups.

Taylor and Hall (1982) noted that many validation studies of the BSRI suggested that the BSRI Femininity and Masculinity scales were correlated with gender-related behaviors. For example, studies have shown correlations between the BSRI Masculinity and Femininity scales and conformity, nurturance and, interpersonal sensitivity.

Larsen and Seidman (1986) assessed the BSRI as an appropriate device for investigating gender schema theory (i.e. that males have masculine sex-typed behaviors while females have feminine sex-typed behaviors). Their results showed that there were consistent and theoretically predictable differences in the factor analyses of masculine

and feminine groups. The gender schematic (sex-typed) group obtained bipolar factors, with the masculine items loading onto one factor while the feminine items loaded onto the other factor. The sex of each subject loaded highly for almost every factor for each group. The non-sex typed (androgynous) group obtained few dichotomous factors, and the sex of the subjects in this group loaded only on the weaker factors. Their results supported the construct validity of the BSRI.

Payne (1985), on the other hand, said that the BSRI is not clear in what it purports to measure, whereas Bem (1981) stated that the scales are indices of global femininity and masculinity. Payne (1985) disagreed, stating that the evidence to support Bem's (1981) contentions is lacking. Indeed, the BSRI seems to show that self-description of the subject agrees with his/her overt conduct. There is no real proof that the BSRI's scales are getting at the deeper processes of male and female gender identity issues (Payne, 1985).

One problem is that the content of the scales does not cover a full range of the way that men and women differ stereotypically in the United States (Payne, 1985). An even more pressing concern is that the BSRI's scales are factorially complex. This is particularly true of the Femininity scale, which is a combination of socially desirable expressive traits and of socially undesirable (at

least according to Payne) traits, such as "shy", "gullible", etc.

Beck Depression Inventory

The Beck Depression Inventory (Beck et al., 1961) is purported to measure an individual's degree of depression. The Beck Depression Inventory (BDI) was specifically designed to measure the behavioral manifestations of depression. There appear to be common physiological and affective characteristics (such as disturbed sleep) between bulimia and major depression that warrant further investigation.

The BDI is generally used for treatment planning and evaluation in mental health settings. It consists of twenty-one multiple-choice items that assess an individual's complaints, symptoms, and attitudes related to his/her current emotional state. The subjects are asked to respond to items based upon how they have been feeling during the past week, including the day of the BDI's administration. The BDI is designed for adults, with questions presented on an eighth grade reading level, and it takes approximately 15 to 20 minutes to administer. It can be self-administered or the examiner can read the items to the subject and record the subject's responses. An examiner is required to be present during the administration.

Beck et al. (1961) constructed the BDI based on observations and records of the characteristic attitudes and

symptoms of depressed patients who were in psychoanalysis with the author. He selected a group of attitudes and symptoms that appeared to be specific for these depressed patients and which were consistent with the descriptions of depression contained in the literature at that time. On the basis of this procedure, the BDI was constructed.

The items were chosen based on their relationship to the overt behavioral manifestations of depression and do not reflect any theory regarding the etiology or the underlying psychological processes of depression (Beck et al., 1961). Examples of some of the symptom category attitudes are: "Crying Spells", "Sleep Disturbance", and "Loss of Libido." In effect, each question is a separate subscale.

The statements in each item are ranked to reflect the severity of the symptom that is being assessed. Numerical values, ranging from a low of zero to a maximum of three, are assigned to each statement to indicate the degree of severity of the symptom. All scores from the items are summed to yield a total global score.

The original norm group consisted of psychiatric outpatients at the University of Pennsylvania Hospital and of psychiatric inpatients from a metropolitan hospital, Philadelphia General. Diagnostic interviews were conducted immediately before or after the BDI was administered by experienced psychiatrists who also rated the patients on various symptoms including memory, anxiety, reality testing,

vegetative signs, and so on. The norm groups were predominantly white, 15-44 years of age, and primarily from lower socioeconomic strata. Two data sets were collected: The first was called the "original group" (N= 226) and the second was called the "replication group" (N= 183).

BDI global scores were compared with the psychiatrists' diagnostic ratings for each of the two norm groups. For the original and replication groups, analyses of variance (anova) produced statistically significant ($p < .001$) differences among the means of all four "Depth of Depression" categories. That is, with each increase in the magnitude of depression, there was a progressively higher mean global score on the BDI (Beck et al., 1961). The Pearson biserial correlation coefficients were .65 for the original group and .67 for the replication group. The highly significant relationship between the global scores on the BDI and the clinical ratings of the psychiatrists attested to the BDI's validity (Beck et al., 1961).

A two-step process was used in establishing the BDI's reliability. First, protocols of 200 consecutive cases were analyzed and scores for each of the 21 BDI questions were compared with the global BDI score for each subject. An anova revealed that all categories showed a highly significant relationship to the global BDI score ($p < .001$); the exception was the "Weight-Loss" category, Question 19 ($p < .01$).

Next, an evaluation of internal consistency was done through the determination of split-half reliability. The data were based on 97 cases in the original patient sample. The Spearman-Brown R between the odd and even categories was .93. Thus, the high correlation coefficient on the split-half item analysis and the highly significant relationship between the individual item scores and the global BDI scores indicated that the BDI was highly reliable (Beck et al., 1961). The BDI was later re-administered to neurotic outpatients by Beck and Steer (1984), and a new Spearman-Brown R of .86 was obtained, comparable to that of the original BDI.

Taylor and Klein (1989) added support to the validity of the BDI by using the instrument, among other measures, to assess the recovery of depressed patients. Global BDI scores decreased from an average of 19.82 in the pre-treatment phase to an average of 6.88 in the post-treatment phase. Even though treatment protocols, changes in behaviors, and severity of symptoms were not described, Taylor and Klein (1989) concluded that the BDI was a moderately good predictor of recovery from depression, and that it might be useful in outcome studies.

Dorus, Kennedy, Gibbons, and Ravi (1987) examined depressive symptoms of 50 alcoholic patients using the BDI. The alcoholic subjects who had major depression had higher BDI scores than did those subjects who did not have major

depression. They concluded that their analyses and results confirmed the validity of the BDI.

Self-Reports

As stated earlier, bulimics have been notoriously difficult to assess. They often will not volunteer information unless their behavior has reached a crisis level (Gandour, 1984). Bailey (1978) notes that it is easier for experimenters to secure confidential information through a nonthreatening questionnaire than through a structured interview. The subject is more likely to provide accurate, personal information through this method than through a personal interview. The subject can also take his/her time in answering each question and can take his/her time in answering the more difficult items. Bailey (1978) also notes several other advantages of self-report measures:

1. Standardized wording of the instruments will ease the comparison of the respondents' answers because each respondent is exposed to exactly the same wording of each item. However, this can be offset by the differential understanding of items due to such factors as differences in educational levels, geographic backgrounds, and so on.

2. There is no opportunity for the respondent to be biased by the experimenter. It is very easy for the experimenter to bias the subject's answers in a variety of ways, such as: prompting through voice inflection, the interviewer misunderstanding the respondent, or the

interviewer assuming that the subject will answer in a certain way.

There are also disadvantages in using self-report measures (Bailey, 1978):

1. If an item is left unanswered, the lack of an interviewer will not allow the interviewer to explore the reasons why the item was not answered. The lack of an interviewer also allows the respondents' nonverbal behaviors to go unrecorded.

2. A serious problem is that questionnaires, in most cases, cannot control for response bias in both a positive and negative fashion. This occurs when a subject wishes to paint either a better or worse picture of the situation than is actually the case. It is difficult to control for this; thus, a certain amount of error will occur in the measuring of subjects on most questionnaires. It is also difficult to gather spontaneous first opinions, as the subject can erase a hasty answer because it is not in the subject's best interests, etc.

3. Response bias is also a problem when one is measuring addictive disorders or eating disorders, since denial is a major part of the spectrum (e.g. Gandour, 1984). For example, if a subject is binging every day, that subject may only admit to binging once a week instead in order to prove to the experimenter that s/he is not bulimic.

4. The sample itself may be biased. The respondents

need to be able to read, write, comprehend and see. Many questionnaires and devices lose their standardization if they have to be read and explained to the respondent. On very emotional or controversial issues, those subjects who are strongly in favor and those who are strongly opposed may respond, while those who are neutral may not respond. Finally, some subjects may refuse to answer some or all of the questions, harming the final results.

Thus, self-reports, though having disadvantages associated with them, cannot be surpassed as a nonthreatening means of obtaining personal information from many subjects who might otherwise be reluctant to divulge this information to experimenters. Since the present study examined a very sensitive subject, bulimia (including the component of denial), there seemed to be no better way to gather clinical information than through the combination of clinical interviews and self-report questionnaires, along with sound psychometric instruments and with physiological measures such as blood plasma examinations.

Bulimia and Affective Disorders

As previously stated, there appear to be common physiological and affective characteristics (such as disturbed sleep, low self-esteem, feelings of sadness, and general lethargy) in the symptomatology of bulimia and affective disorders; consequently these affective symptoms may be related to bulimia (Bazire, 1988; Hudson & Pope,

1990; Siegel, 1989; Swift, Andrews & Barklage, 1986).

Familial Incidences

Studies have demonstrated that some bulimics have family histories of major affective disorders (e.g. Geracioti & Liddle, 1988; Jampala, 1985). Mitchell (1990) noted that most of these studies demonstrated that an individual who has bulimia is more likely to have relatives with affective disorders than someone who does not have bulimia. He suggested that these findings were important because it suggested a common etiology for both affective disorders and bulimia (Mitchell, 1990).

Jampala (1985) studied genetic patterns and found that a higher incidence of bulimics' relatives had histories of primary affective disorder. Geracioti and Liddle (1988) examined family histories and found that 8 of 14 bulimic patients had first degree relatives who had a major affective disorder. Major depression has also been identified as a specific diagnosis among immediate family members (Geracioti & Liddle, 1988; Logue, Crowe & Bean, 1989; Siegel, 1989). Keck et al. (1990) compared 119 bulimic women with nonpsychiatric controls and found that the bulimic women differed significantly from the controls in their prevalence of personal and familial major affective disorders. Keck et al. (1990) concluded that their data support the hypothesis that a relationship exists between bulimia and major affective disorders.

Such a relationship was investigated by Schulman, Kinder, Powers, Prange, and Gleghorn (1986). The Bulimia Cognitive Distortions Scale (a multifaceted scale developed by the authors for their study) and the Beck Depression Inventory (BDI) was administered to 55 bulimic and 55 non-bulimic women who ranged in age from 17-45 years. Results indicated that the severity of bulimia, as measured by the global score of the Bulimia Cognitive Distortions Scale was significantly correlated to the global score on the BDI for bulimic women ($r = .60$, $p < .0001$). The results should be used with caution since they were based on a new instrument that had not been subject to validity or reliability checks.

Goebel, Spalthoff, Schulze and Florin (1989) compared 44 bulimic women with 38 control women and discovered that elevated global BDI scores were highly predictive of the severity of bulimia as measured by the Eating Disorders Questionnaire, an inventory that measures a subject's history of eating behavior. They also discovered that bulimic women were moderately depressed according to their BDI global scores. They concluded that depressive symptoms were predominant in bulimic women.

Schlesier-Carter, Hamilton, O'Neil, Lydiard and Malcolm (1989) found that bulimics showed more maladaptive thinking associated with depression and were significantly more depressed than normal control subjects. The differences on the cognitive and behavioral measures remained when the

level of depression was statistically controlled. The authors concluded that even though depression can be frequently diagnosed in bulimics, specific maladaptive cognitions and behaviors may reflect bulimia itself and not an affective disorder. These results may refute Mitchell's (1990) hypothesis that comorbidity may exist between bulimia and affective disorders, specifically major depression.

Hudson, Pope, Yurgelun-Todd, Jonas and Frankenburg (1987), in structured diagnostic interviews, found that bulimics had high lifetime rates of major affective disorders and anxiety disorders. Pope, Hudson and Yurgelun-Todd (1989) compared the scores of 45 bulimics, 21 people in treatment for major depression and 27 normal controls on the Hamilton Rating Scale for Depression and found that the bulimic and depressed subjects were virtually indistinguishable on all items. Both of these groups were easily distinguished from the controls, thus adding support to the hypothesis that major depression and bulimia may be associated.

Hsu (1990) noted that about one-third of those patients who had an eating disorder (anorexia nervosa or bulimia) also had major depression. However, he noted that current evidence did not support the view that eating disorders are necessarily related to affective disorders or are variants of them. The present study was designed to further investigate the relationship between major depression (an

affective disorder) and bulimia.

Hypothesizing that bulimia may share a pathophysiological abnormality with major depression and with panic disorder, Hudson and Pope (1990) reviewed literature that examined all treatment studies using four classifications of antidepressants: tricyclics, monoamine oxidase inhibitors, serotonin uptake inhibitors, and atypical antidepressants such as trazedone. Hudson and Pope (1990) evaluated disorders that consistently responded to antidepressants and that were not classified as affective disorders. They discovered three placebo-controlled studies for each drug that demonstrated the efficacy of using desipramine, imipramine and phenelzine in the treatment of bulimia, with little or no counterevidence. They also discovered ten placebo-controlled studies that demonstrated the effectiveness of treating panic disorder with imipramine. Hudson and Pope (1990) concluded that the treatment-response model they developed identified bulimia and panic disorder as possible components of the overall affective disorder spectrum, which typically is seen as only consisting of major depression.

Antidepressant Medications

The efficacy of pharmacological treatment for chronic and severe depression has been substantiated over time; it has been a relatively brief period that these same pharmacological agents have been used in treatment for

bulimia. Patterns of positive response may add support to a common physiological base for some depression and eating disorders.

As with most of the drugs used to treat psychiatric problems, the mechanisms of action of tricyclic antidepressants are not completely understood (Baldessarini, 1985). The literature poses several hypotheses about how these drugs may work. Baldessarini (1985) states that, in addition to the acute sedative effects of the tricyclics, they also serve to enhance the action of norepinephrine and may block the effects of tyramine, a neurotransmitter. These observations have led to support for the amine hypothesis of affective disorders, suggesting that major depression is associated with a relative lack of activity of certain amine neurotransmitters in the brain, most probably norepinephrine. Other amines such as acetylcholine and serotonin may also be involved in major depression. As noted earlier, both norepinephrine and serotonin have been implicated in physiological studies of bulimics and therefore have been hypothesized as being dysfunctional in physical substrates of bulimia (Kaplan & Woodside, 1987; Mitchell & Eckert, 1987).

Baldessarini (1985) said that a large part of the problem in understanding how antidepressants work is the lack of knowledge of the causes of the major affective (mood) disorders. Although much research has been done, a

cause-effect relationship between physiological changes and depression has not been sufficiently shown. For example, many metabolic and physiological changes that occur in major affective disorders tend to return to normal once the affective disorder is in remission. These biological changes in major depression may be secondary to, or concomitant with, depression; they are not necessarily a cause of depression or an indication of a biological vulnerability to it. A review of psychopharmacological treatment shows that no single biochemical nor neurophysiological hypothesis can account for the actions of all antidepressants (Baldessarini, 1985).

Bulimia and Antidepressant Medication Treatment

Geracioti and Liddle (1988) raise key issues regarding the treatment of bulimia nervosa with tricyclic antidepressants. Research has shown some interesting results concerning the efficacy of treating bulimia with antidepressants. Imipramine, a tricyclic antidepressant, has been shown to increase the available amount of the monoamine neurotransmitters norepinephrine or serotonin (or both) in the central nervous system. The reuptake of norepinephrine and serotonin into the postsynaptic neurons is blocked by the tricyclics, thus allowing serotonin and norepinephrine to accumulate and prolonging their action (Barnhart, 1986; Gilman, Rall, Nies & Taylor, 1990; Springhouse Corporation, 1989). The tricyclics need an

average of two to four weeks for effective levels, most often the latter to attain maximum effectiveness. Primary indications have been for various conditions, such as major depression, enuresis (especially using imipramine), panic disorder, and bulimia. These drugs may be effective in the treatment of organic affective disorders, obsessive-compulsive disorder, and posttraumatic stress disorder (Hyman & Arana, 1987).

Pope and Hudson (1982) performed one of the first studies on the treatment of bulimia with the antidepressant imipramine. Although their sample size was small (N=8), they found that 75% of those subjects who were treated with imipramine showed a significant decrease in their binging behavior, lasting from two to seven months. However, Pope and Hudson (1982) did not have a control group in their study and could not rule out that the results were caused by experimental bias, imipramine therapy, or some extraneous variable. Hudson, Pope, and Jonas (1983) found supporting data in a subsequent study where bulimic symptoms were significantly reduced in treatment with imipramine.

Mitchell, Pyle, Eckert, Hatsukami, Pomeroy and Zimmerman (1990) reported that the improvement experienced by bulimic outpatients was superior for those who were in intensive group psychotherapy when compared to those who were taking imipramine alone. The addition of imipramine to the intensive group psychotherapy component did not

significantly affect the improvement of bulimic symptoms when compared to the addition of a placebo, but it did result in greater improvement in the symptoms of depression and anxiety. All three active treatment phases (imipramine alone, imipramine with psychotherapy and placebo with psychotherapy) resulted in significant reductions in target eating behaviors (binging and purging) and a significant improvement in mood (Mitchell et al., 1990).

Desipramine is another tricyclic antidepressant whose mechanisms of action, primary indications, and time of effective therapeutic onset are similar to imipramine; however the adverse anticholinergic side effects (dry mouth, constipation, urinary hesitancy, etc.) and sedation are less severe than imipramine's (Gilman et al., 1990; Hyman & Arana, 1987). In a six week trial of desipramine with bulimics, Hughes et al. (1986) measured serum desipramine levels during the fifth week of the study in order to see how many of their subjects had achieved blood plasma levels within the therapeutic range for desipramine. Their results demonstrated that bulimics who were treated with desipramine showed an 87% binge decrease rate. The results are interpreted with caution, however, due to two factors: a decrease in sample size caused by attrition and the use of self-report measures (created by the authors and called a "global clinical ratings scale" and a "bulimia symptom rating scale") for reporting data which were of questionable

validity.

Barlow, Blouin, Blouin and Perez (1988) assessed 47 normal weight bulimics who were treated with desipramine during a 15 week, double-blind crossover experiment. Twenty-four subjects completed the 15 week trial, with desipramine being significantly more effective than a placebo in reducing the frequency of weekly binging and regurgitation. No significant effect of desipramine was found on the overall Eating Disorder Inventory (EDI) score, nor were desipramine's antibulimic effects associated with an alleviation of depressive symptoms.

Subsequently, Blouin, Blouin, Perez and Barlow (1989) performed a randomized double-blind crossover trial of desipramine on 24 normal weight bulimics, again over a period of 15 weeks. Desipramine was significantly more effective than a placebo in reducing the frequency of weekly binging and vomiting. Seven subjects responded to desipramine by reducing their binging, seven were classified as borderline responders, and the other ten were non-responders. Interestingly, responders were found to have had lower EDI scores on the Bulimia subscale even though no significant effect of desipramine was obtained on the overall EDI score, and they also had a higher frequency of purge episodes than the non-responders.

Monoamine oxidase inhibitors (MAOI's), such as phenelzine, promote the action of monoamine

neurotransmitters by inhibiting the enzyme monoamine oxidase in the central nervous system, peripheral nervous system and nonnervous tissues such as the liver and gut. The time required for effective levels to build for MAOI's is from one to four weeks, approximately equivalent to the onset time for the tricyclics. The primary indication for these drugs is for the treatment of major affective disorders and for panic disorders (Barnhart, 1986; Gilman et al., 1990; Hyman & Arana, 1987; Springhouse Corporation, 1989). These drugs have been used with some success in the treatment of bulimia. However, since complex, severe and/or unpredictable interactions between MAOI's and other drugs and food-derived amines may occur, using them is difficult and may be potentially hazardous (Gilman et al., 1990).

Pope, Hudson and Jonas (1983) reported that MAOI's may be superior to other antidepressants in the treatment of bulimia. Of 65 patients who were treated in their clinic for bulimia, 17 out of 26 (65%) bulimic patients showed a marked response or remission with MAOI's, even though many had responded poorly to tricyclics or to trazedone. Walsh et al. (1988) noted that phenelzine was successful in 64% of their bulimic clients in reducing binge frequency. However, MAOI's have some serious side effects and require the user to be on a tyramine-free diet. This dietary restriction may be prohibitively difficult to maintain with bulimics and may carry serious health consequences when breached (Walsh et

al., 1988).

Chemically different from the tricyclics or MAOI's, the antidepressant trazedone strongly inhibits serotonin reuptake in the brain. The time of therapeutic onset is similar to the tricyclics and to MAOI's, and its primary indication is for the treatment of major affective disorders. One advantage of trazedone is that its anticholinergic side effects are minimal; however, it retains sedative effects and may still produce drowsiness and dizziness (Barnhart, 1986; Gilman et al., 1990; Hyman & Arana, 1987; Springhouse Corporation, 1989).

Solyom, Solyom and Ledwidge (1989) used trazedone with 13 bulimics in an open label, flexible dose study. Three subjects dropped out before the fourth week; however, in those subjects who remained, the number of binging and vomiting episodes was significantly decreased. The number of episodes was reduced to zero in four subjects. The total EDI score decreased but was not statistically significant; however, the Bulimia subscale score of the EDI did decrease significantly. The mean weight of the subjects was virtually unchanged from pre- to post-treatment: 58.5 kg to 57.3 kg. Solyom et al. (1989) concluded that this lack of weight gain is an advantage of using trazedone over the other antidepressants since the fear of becoming fat is part of the bulimic's pathology.

Fenfluramine is an anorectic drug that stimulates the

ventromedial nucleus of the hypothalamus, may affect the sympathetic and parasympathetic nervous systems and may have serotonin depleting properties. In addition, it may alter the hunger and/or satiety mechanisms. It is primarily indicated for use by obese individuals as an appetite suppressant and as a weight reducer (Gilman et al., 1990; Springhouse Corporation, 1989). Blouin, Blouin, Perez, Bushnik, Zuro and Mulder (1988) ran a 15 week, double-blind, placebo controlled crossover trial of 22 normal weight bulimic subjects. 12 subjects were randomly assigned to a group receiving fenfluramine and the other 10 subjects received desipramine. Blouin et al.'s (1988) results demonstrated that both drugs had beneficial effects on the frequency of binging and regurgitation, although more subjects responded positively to fenfluramine than to desipramine. Both drugs were also effective in reducing the psychological symptoms of bulimia such as the urge to binge.

Russell, Checkley, Feldman and Eisler (1988) performed a 12 week double-blind, placebo controlled trial of fenfluramine with 42 bulimics. Forty percent (17 out of 42) of the subjects dropped out halfway through the 12 weeks. Among the noncompleters, those on fenfluramine had reportedly experienced relief of their bulimic symptoms; the authors hypothesized that the persistence of depressive symptoms and of the features of bulimia may have contributed to the high dropout rate. Those taking fenfluramine did not

evidence weight loss or serious side effects, but fenfluramine by itself did not significantly benefit some of the subjects who had severe bulimia. Due to this high dropout rate, the authors advised caution when interpreting their results, and they called for more research into treating bulimia with fenfluramine.

Based upon the literature, using antidepressant medications in the treatment of bulimia would seem to be a logical course of treatment, even if we are not sure how or why these medications work or if bulimia and major affective disorders are somehow related. However, there were limitations in the earlier research.

For example, Brotman, Herzog and Hamburg (1988) discovered that three out of seven of their clients who received at least one trial of antidepressants showed a significant decrease in their binging behavior. However, even though Brotman et al. (1988) found positive treatment effects, they also had a small sample, thus hindering their study's external validity. Other studies as well (e.g. Hughes et al., 1986; Russell et al., 1988) also suffered from sample sizes which grew even smaller due to attrition.

Gender Issues Related to Eating Disorders

Hsu (1990) noted that objective studies which examine personality characteristics of eating disordered patients are few. Most of these studies used the Minnesota Multiphasic Personality Inventory (MMPI), which, according

to Hsu (1990), is more a measure of present symptomatology than a personality indicator. Striegel-Moore et al. (1986) and Steiger et al. (1989) found that the literature examining the relationship between bulimia and female sex roles yielded inconsistent results. Some studies showed no relationship between bulimia and feminine sex roles while others showed bulimics exhibiting sex role disturbances.

Steiger et al. (1989) found that all of the 31 women with eating disorders (Anorectics, "Bingers" who were bulimiarectics, or Bulimics) in their study showed hyperfeminine sex role identifications. "Hyperfeminine," as used by Steiger et al. (1989), referred to those women who endorsed fewer masculine traits on the BSRI than normal control women. "Hyperfeminine" can also describe women who are unassertive, dependent, eager to please and concerned with social approval (Striegel-Moore et al., 1986). Rost, Neuhaus and Florin (1982) found that the sex role beliefs of bulimiarectics were significantly less liberated (i.e. they were more unassertive, more dependent, etc.) than those of a control group.

Hsu (1990) also noted that many factors can influence the development of sexual attitudes, behaviors and sex roles. For example, Hsu (1990) suggested that a fear of loss of femininity may accompany success in a woman, and that a woman's fear of success was more prevalent among those who were nontraditional (i.e. masculine) in their sex

roles. Bulimic women often came from successful, high achieving, financially stable families (Gandour, 1984; Hsu, 1990). Striegel-Moore et al. (1986) found that sex-role messages delivered to young girls could often be quite confusing and might lead to an eating disorder. For example, girls were told to be successful and work hard at school, yet remain feminine, popular, and pretty. Adolescent girls who strove to be superwomen (i.e. having the perfect family and career, and possessing great beauty) had elevated eating pathology scores on the Eating Attitudes Test, a measure of disordered eating. Striegel-Moore et al. (1986) discovered that a girl's ability to separate societal ideals about her appearance and sex roles from her own expectations for her appearance and sex roles was associated with decreased empirical evidence of eating disturbance as measured by the Eating Attitudes Test. These authors also found that their female bulimics, who were in psychotherapy, expressed confusion about their sex roles. Striegel-Moore et al. (1986) further stated that future research should examine the sex roles of bulimic women who are not in psychotherapy in order to determine the presence of sex role confusion. The present study was designed to address that question; empirical data (sex roles) of bulimic women who were not involved in psychotherapy was collected.

Another gender issue, body image dissatisfaction, is rather prevalent in the United States, especially among

women. In a national survey, 31% of the women and 24% of the men reported negative attitudes about their physical appearances (Butters & Cash, 1987). Negative body image was found to be related to the presence of poor self-esteem and increased one's vulnerability to depression. People who have eating disorders often have dysfunctional perceptions of their physical appearance (Butters & Cash, 1987).

The results of a study by Mintz and Betz (1988) supported the findings of Butters and Cash (1987). They found an inverse relationship between the severity of a woman's eating disorder (bulimia being the most severe; anorectics were excluded from their study) and self esteem and body image satisfaction.

Steiger et al. (1989) compared 31 women with eating disorders (anorexia nervosa, bulimiarexia or bulimia) with 11 normal control women and found that only the anorectics exhibited body-image distortions.

Bulimia also seems to be more prevalent among women. Connors and Johnson (1987) reviewed many studies on bulimia and found that the prevalence of bulimia in females ranged from 5% of college females to 20% of female college seniors. Bulimia in males had been reported as ranging from zero percent of college males to 5% of college males. Of the 355 bulimics Halmi et al. (1981) sampled, 87% were female and 13% were male. Stangler and Printz (1980) sampled 500 students who were seen at a university psychiatric clinic.

3.8 percent of them were diagnosed as being bulimic; of the bulimics, 89.5% were female and 10.5% were male.

Steiger (1989) examined the literature on anorexia nervosa and bulimia in males in order to discover if differences existed between males and females with these disorders. He concluded that fundamental male-female differences did not exist as far as the etiology or symptomatology was concerned and hypothesized that the lower incidence of eating disorders among men suggested that the biological and social determinants of eating disorders were more active in females.

Pope, Hudson and Jonas (1986) compared 15 bulimic males to female bulimics and found no demography, family history, associated psychopathology or treatment response differences. They also found little evidence to support previous hypotheses that increased homosexuality, atypical sexuality or sexual conflict existed in these men.

Conclusion

Many questions remain for future research of the mechanisms related to the etiology and treatment of bulimia. The literature points to potential physiological bases and similarities to the physiological aspects of major affective disorders. These hypotheses are supported by the relative efficacy of treatment models for both conditions which include antidepressant agents, especially the tricyclics (e.g. Hughes et al., 1986). However, much of the literature

is flawed. For example, some researchers have relied only on self-report data to determine compliance with medication.

Improved methodology would allow use of validated paper-and-pencil measures with the examination of the bulimic's blood plasma levels of the drug, like the Hughes et al. (1986) study. A study using these techniques plus self-reports, clinical and behavioral observations, and chemical and urine analyses may help detect the efficacy of antidepressant, psychotherapeutic, or behavior management strategies on bulimic behavior. The value in measuring blood levels, which yields vital information including such data as compliance as well as levels of cholesterol, potassium, etc., cannot be underestimated.

A potential problem exists with examining the relationship of elevated cholesterol and depleted potassium levels in bulimics. In frequent and/or chronic purging episodes, whether due to laxative abuse, self-induced vomiting, or other problems, fat soluble elements, like cholesterol, may be retained while water soluble elements, such as potassium, will be eliminated. Thus, elevated cholesterol levels and/or depleted potassium levels are positively correlated with the presence of bulimia and provide a positive indication of a differential diagnosis, but these indicators may be less related to its etiology than to the secondary effects of the purging behaviors.

Although psychopharmacological research provides some

evidence for positive and efficacious treatment avenues, other problems may arise. Many antidepressants have side effects, some of which may be unpleasant (Baldessarini, 1985; Hyman & Arana, 1987). One way to counteract this is to use a drug that has low sedating effects and low anticholinergic effects. Hyman and Arana (1987) recommend desipramine, among other drugs, in order to achieve these results. MAOI's also appear to be effective but the fact that they would require the bulimic to remain on a tyramine-free diet makes their usage unwise. The concept of treating bulimia nervosa with tricyclic antidepressants remains controversial for many reasons, not the least of which is that the front-line usage of these drugs is for depression, not for treating bulimia (Gilman et al., 1990; Springhouse Corporation, 1989). Regardless, investigations into drug therapy as an effective treatment modality for bulimia may add to the support that drug therapy may be used as a viable treatment option and that bulimia may somehow be linked with affective disorder.

Rationale for the Study

Although research has focused upon the efficacy of using antidepressant medications for the treatment of bulimia, many of these studies had flaws (e.g. Pope & Hudson, 1982). Evidence has also been presented to support the hypothesis that bulimia is more prevalent in women than in men (e.g. Striegel-Moore et al., 1986); however,

empirical research (using validated instruments such as the BSRI) into the sex role beliefs of these bulimic women has been scarce (Hsu, 1990).

One Canadian study compared the sex role beliefs of eating disordered women (using the BSRI) with a normal female control group (Steiger et al., 1989). However, their conclusions lack external validity since their experimental group had bulimics, anorectics, and bulimiarectics. Their sample was also not balanced, as they had 20 more subjects in the experimental group than in the control group. Rost et al. (1982) had evenly balanced experimental and control groups when they compared sex roles. Their study, conducted in West Germany, was limited to bulimiarectics, whose pathology and behaviors may differ from normal-weight bulimics (American Psychiatric Association, 1987). The data from Rost et al. (1982) and Steiger et al. (1989) might not be applicable to normal-weight bulimics in general, and to women in the United States in particular.

Since bulimia, like major depression, is disproportionally represented in women, Striegel-Moore et al. (1986) stressed the importance of producing more research examining the connection between the two disorders. Mitchell (1990), unlike Hsu (1990), stated that if there is a relationship between major depression and bulimia, confirming the existence of that relationship would allow researchers to gain knowledge about major depression and

about bulimia. That is, does bulimia lead to the development of major depression or is major depression a primary causal factor of bulimia? Mitchell (1990) cited at least three studies (e.g. Swift et al., 1986) that pointed to a comorbidity between major depression and bulimia and concluded by stating that bulimia may not be a unique homogeneous diagnostic entity.

Data revealing sex role beliefs of bulimics are needed to explore the possible connection between sex role beliefs and the severity of bulimic symptoms. Unlike some previous studies which have used anorectics, bulimiarectics and bulimics in various combinations, the present study only included normal-weight bulimic women, thus eliminating confounding variables and producing more externally valid results. Empirical studies of normal-weight bulimics using psychometrically sound instruments are quite scarce, thus making generalizations difficult (Swift et al., 1986).

Statement of the Problem

A review of previous literature reveals a trend towards using antidepressant medication in the treatment of bulimia (Hughes et al., 1986; Pope & Hudson, 1982; Walsh et al., 1988). In this study, desipramine was chosen to treat bulimics, assessing treatment compliance and effectiveness by examining blood plasma levels. The design of this study allowed collection of a unique data base. Only three other studies have examined desipramine blood plasma levels of

bulimics (Barlow et al., 1988; Blouin et al., 1989; Hughes et al., 1986). In addition, the present study included bulimics from the southeast, unlike Barlow et al. (1988) and Blouin et al. (1989) which used Canadians, and Hughes et al. (1986) which sampled from the midwest. This study also used DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria, unlike Hughes et al. (1986) which used DSM-III (American Psychiatric Association, 1980) diagnostic criteria.

The relationship between perceived gender roles and bulimia was also examined. The literature on the sex role beliefs of bulimics is scarce, and only a few studies were found in which the Bem Inventory (Bem, 1978) was used to assess bulimics' sex role beliefs (e.g. Steiger et al., 1989). Steiger et al. (1989) concluded that sex-role disturbances were present in the bulimics they studied. More importantly, they concluded that sex-role disturbances might be active in the etiology of bulimia. Striegel-Moore et al. (1986) found that the literature examining the relationship between bulimia and female sex roles was inconsistent. Gandour (1984) addressed the relationship between bulimia and female sex roles but offered no empirical support for her conclusions. Because the data base is limited and inconclusive, it was important to further investigate the relationship between gender issues and bulimia. The present study used the BSRI to measure sex

role beliefs of bulimics.

Finally, the relationship between bulimia and major depression was examined. Some authors (e.g. Bazire, 1988; Hudson & Pope, 1990; Siegel, 1989) have hypothesized that bulimia and affective (mood) disorders were somehow related. The severity of bulimia also seemed to be related to the global score on the Beck Depression Inventory (Goebel et al., 1989; Schulman et al., 1986). This study further adds to the research base which defines the relationship between bulimia and major depression.

As previously stated, data was mostly obtained through self-reports. These data included, for example, purge and binge attempts, medication compliance and side effects. The self-report data, combined with physiological data, was used to answer the research questions posed in this study.

Research Questions

1. Is there a relationship between bulimia and major depression as measured by the Beck Depression Inventory?

2. Is there a relationship between bulimia and perceived sex roles as measured by the Bem Inventory?

3. Is there a relationship between the severity of bulimic symptomatology and desipramine treatment?

4. Is there a relationship between bulimia and dysfunctional body image perceptions?

Research Hypotheses

Evidence was gathered to investigate the following a priori hypotheses:

1. A significant positive correlation coefficient will be obtained between the severity of bulimic symptoms, as measured by the Bulimia subscale (B) of the Eating Disorder Inventory (EDI), and depression as measured by the Beck Depression Inventory (BDI), for bulimic women in a pretest administration (Goebel et al., 1989).

2. A significant positive correlation coefficient will be obtained between the severity of bulimic symptoms, as measured by the B subscale of the EDI and perceived female sex roles, as measured by the Feminine subscale of the Bem Inventory (BSRI), for bulimic women in a pretest administration (Steiger et al., 1989).

3a. Mean gain scores on the B subscale of the EDI will be significantly lower in the experimental group of bulimic women as compared with the mean gain scores on the B subscale of the EDI of the control group of bulimic women in a posttest administration.

3b. Increased blood levels of desipramine in bulimic women in the experimental group will be significantly negatively correlated with a decrease in scores on the B subscale of the EDI in a posttest administration.

4. A significant positive correlation coefficient will be obtained between the severity of bulimic symptoms, as

measured by the B subscale of the EDI and dysfunctional body image perceptions, as measured by the Body Dissatisfaction (BD) subscale of the EDI for bulimic women in a pretest administration.

CHAPTER TWO

METHOD

Subjects

To be eligible for this study, subjects were required to meet the DSM-III-R (American Psychiatric Association, 1987) criteria for bulimia. This study eliminated developmental variables and controlled for gender variables so that the segment of the population most often affected by bulimia-- young women-- could be investigated (American Psychiatric Association, 1987). Therefore the following criteria also had to be met for participation in the study: the participant was required to be between 18-40 years old; the participant had to be female whose weight fell between 80%-120% of ideal body weight; the participant could not have any acute or chronic medical problems and the participant could not be on any psychotropic medications within the previous month.

Participants who fulfilled DSM-III-R (American Psychiatric Association, 1987) criteria for anorexia nervosa, schizophrenia, active chemical dependence, or bipolar affective disorder were excluded from the study. Data from participants who declined to take the medication, or who refused to take any of the tests, were excluded. However, they remained in the study until their eight week period was concluded. This was to insure that they did not talk to those participants who were still involved in the

study, possibly confounding the results.

Participant Characteristics

All 27 potential participants were volunteers gathered from eastern Tennessee. The participants were self-referrals to a Health Center or to the experimenter(s). Three participants were excluded during the initial interview phase because their primary diagnosis was not bulimia (One only binged twice a year, one was bulimiarectic and the other was alcohol dependent). The initial sample size thus consisted of 24 women who were randomly assigned to either the control or the experimental group. During the study, two of the control participants and two of the experimental participants contacted the experimenter(s) by the seventh week of the study and stated that they were no longer interested in participating in the study (One wished to engage in psychotherapy, two did not wish to continue on the medication, and the final participant did not offer a reason); one control participant never returned for the final stage of the study and the debriefing session; and one experimental participant had to be excluded during the medication phase of the study because she contracted a bisymmetric skin rash over her chest, a potential side effect of using desipramine.

The final sample size consisted of 18 women, divided evenly between the control and experimental group. At the time of the initial screening interview, the ages of the

experimental subjects ranged from 18 to 24 years (M = 22.6 years, sd = 4.7), their average weight was 122.0 pounds (sd = 16.02), they binged an average of 2.91 times per day (sd = .70) and they purged an average of 2.64 times per day (sd = 1.43). The control subjects ranged in age from 18 to 24 (M = 21.1 years, sd = 2.13), their average weight was 130.3 pounds (sd = 10.06), they binged an average of 2.73 times per day (sd = .83) and they purged an average of 2.32 times per day (sd = 1.20). Table 1 presents some additional participant demographics. Thirteen of the participants used alcohol; eight were sexually active; six had a family member (limited to first degree biological relatives) whom they believed was alcohol dependent; 10 had a family member who suffered from a major affective disorder; three had a family member whom they believed had some type of mental illness other than a major affective disorder (such as schizophrenia) and three said that a family member had attempted or successfully completed suicide.

Measures

The present study used three paper and pencil measures: The Eating Disorder Inventory (Garner et al., 1983), the Bem Inventory (Bem, 1978) and the Beck Depression Inventory (Beck et al., 1961). The descriptions and reviews of these instruments, as well as reliability and validity information, were presented in Chapter One. In addition, the physiological measures used were a Multichannel Blood

Table 1

Demographic Characteristics

Variables	Experimental Group	Control Group	t Statistic	p Value
N	9	9		
Age				
M	22.6	21.1	.99	.334
SD	4.7	2.1		
Initial Weight				
M	122.0	130.3	-1.45	.116
SD	16.02	10.06		
Binge(s) Per Day				
M	2.91	2.73	.56	.584
SD	.70	.83		
Purge(s) Per Day				
M	2.64	2.32	.25	.802
SD	1.63	1.20		
Uses Alcohol	7	6		
Sexually Active	5	3		
Alcoholic(s) in Family	3	3		
Major Affective Disorder(s) in Family	6	4		

Table 1 Continued

Variables	Experimental Group	Control Group	t Statistic	p Value
Mental Illness in Family	1	2		
Suicide(s) in Family	2	1		

Chemistry Screening (SMA-25), a Complete Blood Count (CBC), and a Urine Analysis (UA).

Procedure

All participants were seen initially for an intake interview by the consulting psychiatrist and/or by the author. The experimenter(s) assessed the participant and interviewed the participant to obtain a detailed history of eating disorders and of mood or affective disturbances. The consulting psychiatrist and the experimenter then made a diagnosis, which had to be agreed upon before any participant was allowed to enter the study. All those participants meeting participation criteria were then offered the opportunity to take part in the study. All participants who agreed to take part received the pretest BDI, EDI and BSRI. These were given in a counterbalanced order to prevent order effects. Participants were assigned randomly to either the experimental group or to the placebo control group by using a random number generator. The experimenter(s) then explained the nature of the study in detail. During the active phase of the study participants were unaware of their group status.

Participants in the placebo control group did not receive any form of active treatment until the eight week experimental phase was concluded, at which time active treatment (drug therapy) was presented as an option. Control group participants were then asked to complete the

EDI, BSRI and the BDI again in a counterbalanced order.

Participants were prohibited from engaging in psychotherapy during the study; this eliminated a confounding variable. If any participant wished to engage in psychotherapy, or if the experimenter(s) deemed psychotherapy necessary for any participant, she was to be excluded from the study and was to be referred to a counseling center or elsewhere. The participant also had the option to continue to see the consulting psychiatrist. This did not occur in the present study.

The study lasted eight weeks. Those participants who were in the experimental group initially received a single blind placebo washout that lasted two weeks, following Walsh et al. (1988). If the participants continued to binge and purge at least two times per week, they then entered the active phase of the study and received desipramine. If they discontinued their binging and purging or showed a significant decrease in these behaviors, they were to be excluded from the study, as they would be demonstrating a placebo response. None of the subjects in the present study demonstrated a placebo response.

Baldessarini (1985) notes that placebos should be identical in appearance to the active drug. The placebo phase was included for comparison to control for beneficial effects (or placebo effects) not related to the actions of a drug. Approximately 60% of people will have some type of

reaction to a placebo; about 30% will have positive results and about 30% will get negative reactions from placebos (Gilman et al., 1990).

The experimental participants were started on a dosage of 10 milligrams (mg) of desipramine per day at the conclusion of the second week of the study, the placebo washout phase. This dosage was gradually increased until a maintenance level was attained, which for most people occurs by the end of the fourth week of administration. The end point of the administration of desipramine is to a specific blood level. For desipramine, that blood level is in the range of 100-150 nanograms (ng), which is considered to be an adequate therapeutic range. The consulting psychiatrist was solely responsible for the prescribing of the medicines and was responsible for the monitoring of the participants while they were on the medicines (In this case, "Medicines" also refers to the placebo that was administered during the first two weeks of the study).

The experimental participants were seen biweekly by the experimenter(s) for 15 minute appointments. No psychotherapy was provided. The visits focused on the participant's report of medication compliance, side effects, and weekly reports of binge/purge episodes. The participants were asked to keep a daily diary in which they recorded the number of binges and the number of successful purges. A purge attempt included any attempts at self-

induced vomiting or regurgitation, or the use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain. This information was collected by the experimenter(s) during each visit.

The participants in the experimental group were required to have a baseline Multichannel Blood Chemistry Screening (SMA-25), a Complete Blood Count (CBC), and a Urine Analysis (UA). This occurred at the second week of the study. These screenings were not expected to show any presence of desipramine because all participants who were in the experimental phase were taking the placebo for the first two weeks and, following the exclusionary criteria for the active part of the study, were expected to be drug-free. This baseline screening let the researchers find out what other drugs, if any, were present. Also, the experimental participants were required to submit to a blood level measure, SMA-25, CBC and UA at the fourth, sixth, and eighth week of the study. Blood levels may be an accurate way to assess how effective a drug is with a particular participant, but they are most effective at measuring treatment compliance. However, since blood levels do not provide a complete picture, SMA-25, CBC and UA tests were also used to provide the experimenters with additional information. If the participants did not reach an adequate blood level (or did not show significant improvement) at the conclusion of the eight week trial, they were fully

debriefed and were allowed to continue with the drug therapy. Their data at the end of their eight week trial were included in the final analyses.

At the conclusion of the study, the BDI, EDI and BSRI were re-administered to all participants in the experimental and control groups, and all participants were fully debriefed. The option to continue drug therapy was offered to all experimental participants at that time, and the option to engage in drug therapy was offered to all control participants. Five of the nine experimental participants did not reach "clinically effective" desipramine blood plasma levels at the end of the eight week trial. Of these five, two opted to continue with drug therapy, two wished to try a combination of drug treatment and psychotherapy and one did not wish to continue with any form of treatment. Of the final nine control group participants, four wished to engage in drug therapy but did not wish to participate in the study, two had no desire to seek any form of treatment, two wished to participate in psychotherapy which eliminated their eligibility for the study and one graduated and left the area.

Statistical Analyses

At the conclusion of the study, all data were compiled and all paper and pencil measures were scored. The following statistical analyses were then performed:

Hypotheses 1 and 2 were analyzed by using Pearson

Product Moment Correlation Coefficients. Hypothesis 3a was analyzed by using a t test. Hypothesis 3b was analyzed by using a Pearson Product Moment Correlation Coefficient. Hypothesis 4 was analyzed by using a Pearson Product Moment Correlation Coefficient.

CHAPTER THREE

RESULTS

Included in this chapter are results relevant for investigating the relationship between bulimia and depression, the relationship between bulimia and perceived sex role beliefs, the effectiveness of desipramine treatment in reducing bulimic symptoms and the relationship between bulimia and dysfunctional body image perceptions. Pre-and posttest Eating Disorder Inventory (EDI), Beck Depression Inventory (BDI) and Bem Sex Role Inventory (BSRI) results were analyzed, along with final blood plasma desipramine levels of the experimental group.

Participant Characteristics

The characteristics of all 18 participants were described in Chapter Two and presented in Table 1. To ensure that the experimental group did not differ significantly from the control group and that the randomization process was successful, t tests were performed on the ages, initial weights, binge(s) per day and purge(s) per day of the participants. The results demonstrated that both groups did not differ significantly on any of these variables (See Table 1). Therefore, it can be assumed that both groups were homogeneous on these variables, and that the randomization process was successful.

Hypotheses Results

Bulimia and Depression

In order to examine the relationship between bulimia and depression, a Pearson Product Moment Correlation was conducted on pretest scores of the Bulimia subscale of the EDI (M = 12.89, sd = 4.42) and pretest global scores of the BDI (M = 13.56, sd = 7.51) for all 18 participants. A moderately strong, positive correlation exists between pretest global scores on the BDI and pretest scores on the Bulimia subscale of the EDI (r = .60, p = .009). The correlation coefficient was squared to yield a coefficient of determination of .36. Thus, global BDI scores predicted 36% of the variance of pretest Bulimia subscale scores on the EDI.

Bulimia and Perceived Sex Roles Beliefs

The hypothesized relationship between the severity of bulimic symptoms and perceived female sex roles was investigated by conducting a Pearson Product Moment Correlation on pretest scores of the Bulimia subscale of the EDI (M = 12.89, sd = 4.42) and pretest scores on the Feminine subscale of the BSRI (M = 94.56, sd = 20.01) for all 18 participants. A mildly strong negative correlation exists between these two variables (r = -.30, p = .224).

Bulimia and the Effectiveness of Desipramine Treatment

The analysis investigating the effectiveness of desipramine treatment in reducing bulimic symptoms as

measured by the Bulimia subscale of the EDI was performed in two stages. To ensure that both the experimental and the control groups were homogeneous, t tests were performed on the demographic data. Since no statistically significant differences existed between the experimental and control groups on any of the demographic variables, the groups were assumed to be homogeneous (See Table 1).

A t test was then performed, comparing the mean gain scores of the Bulimia subscale of the EDI for both the experimental and control groups. For the nine experimental group participants, their Bulimia subscale scores decreased an average of 5.89 points (*sd* = 4.31); for the nine control group participants, their Bulimia subscale scores increased an average of 1.33 points (*sd* = 3.08). These score differences were obtained by subtracting each participant's posttest Bulimia subscale score from her pretest Bulimia subscale score. These values were then used in the final statistical analysis. The results demonstrate that a statistically significant difference exists between the mean gain scores of the experimental group and the mean gain scores of the control group on the Bulimia subscale of the EDI (t (16) = 4.09, p = .001).

Bulimia and Desipramine Blood Plasma Levels

Hypothesis 3b was investigated by examining the relationship between blood levels of desipramine in the experimental group (N = 9) and posttest results of the

experimental group on the Bulimia subscale of the EDI (M = 6.67, sd = 5.05) by conducting a Pearson Product Moment Correlation. The results showed that a low negative correlation exists between the last blood level measured (M = 95.44, sd = 62.66, Range = 30 - 189 nanograms) and the Bulimia posttest score (r = -.17, p = .6877).

Bulimia and Dysfunctional Body Image Perceptions

The final hypothesis was investigated by using a Pearson Product Moment Correlation to examine the relationship between dysfunctional body image perceptions, as measured by pretest scores on the Body Dissatisfaction subscale of the EDI (M = 17, sd = 8.06), and the severity of bulimic symptoms, as measured by pretest scores on the Bulimia subscale of the EDI (M = 12.89, sd = 4.42) for all 18 participants. The results indicate that there is virtually no relationship at all between these two variables as measured (r = -.05, p = .855).

CHAPTER FOUR

DISCUSSION

The design of this study allowed for the investigation of the effectiveness of desipramine (a tricyclic antidepressant) in treating the symptoms of bulimia nervosa in young women. In order to eliminate developmental variables and control for gender variables only young women were used in this study. Pretest scores for all subjects on the Eating Disorder Inventory (EDI), the Beck Depression Inventory (BDI) and the Bem Sex Role Inventory (BSRI) were analyzed in examining the relationships between bulimia and depression, bulimia and perceived sex role beliefs, and bulimia and dysfunctional body image perceptions. Pre- and posttest results of the experimental and control groups on the Bulimia subscale of the EDI were compared to analyze desipramine effectiveness. Posttest Bulimia subscale scores of the experimental group were compared with final desipramine blood level measures to examine the relationship between those two variables.

Bulimia and Depression

Previous research has demonstrated that there appear to be common physiological and affective characteristics (such as disturbed sleep and low self-esteem) between bulimia and affective disorders which would suggest that they might be related (Bazire, 1988; Hudson & Pope, 1990; Siegel, 1989; Swift et al., 1986). For example, Schulman et al. (1986)

found that the severity of bulimia, as measured by the Bulimia Cognitive Distortions Scale was positively correlated to global BDI scores. The results of the present study corroborated earlier investigations. Depressive symptoms were common in these bulimic women (The mean pretest global BDI score for all 18 participants was 13.56, indicating mild depression (Beck et al., 1961)). Additionally, the severity of bulimia had a moderately strong, direct correlation with the severity of depression, supporting previous research and providing evidence for the possible comorbidity of bulimia and depression (Mitchell, 1990).

Goebel et al. (1989) found that elevated global BDI scores were highly predictive of the severity of bulimia as measured by the Eating Disorders Questionnaire, an inventory that measures a subject's history of eating behavior. I obtained similar results as noted above; global BDI scores predicted 36% of the variance of pretest Bulimia subscale scores on the EDI. Furthermore, some studies have demonstrated that bulimics will often have family histories of major affective disorders (Geracioti & Liddle, 1988; Jampala, 1985). In this study, ten of eighteen participants (56%) had a first degree biological relative who had a major affective disorder, specifically depression.

These results suggest that when clinicians are either assessing or treating bulimic clients, they should also

investigate, and treat if present, any major affective disorder. The clinician will have to decide which disorder warrants primary attention. Once the presence of other mental disorders (in addition to bulimia) is detected, clinicians can begin to design more effective treatment modalities for bulimia.

The causal factor(s) of bulimia are still unknown. Regardless of what these factors may be, it remains unclear whether bulimia contributes to the manifestation of depression or whether depression contributes to the manifestation of bulimia (Mitchell, 1990).

Bulimia and Perceived Sex Roles Beliefs

There exist few objective studies which examine the personality characteristics of eating disordered patients (Hsu, 1990). Past research into the relationship between bulimia and perceived sex role beliefs has been inconclusive. Striegel-Moore et al. (1986) and Steiger et al. (1989) found that some studies showed no relationship between bulimia and perceived sex role beliefs while others showed bulimics exhibiting sex role disturbances.

The relationship between perceived sex role beliefs and bulimia was examined in the present study. One of the DSM-III-R (American Psychiatric Association, 1987) diagnostic criteria for bulimia is that a person must have a persistent overconcern with body weight and shape. Based on this it was hypothesized that bulimic women who were overconcerned

with their bodies would possess hyperfeminine sex role beliefs (Steiger et al., 1989). However, results of the present study found a non-significant negative correlation between perceived female sex role beliefs and the severity of bulimic symptoms. The low correlation (r = - .30) only indicated a possible inverse trend between these two variables.

The present study's result thus provided no conclusive evidence to support the hypothesis that a positive relationship exists between perceived feminine sex role beliefs and the severity of bulimic symptoms (Steiger et al, 1989). One explanation for the different results between the present study and Steiger et al. (1989) is that Steiger et al. (1989) used Canadians in their study while the present study used women from the southeastern United States. In addition, Steiger et al. (1989) included anorectics and bulimiarectics as well as bulimic women in their sample while the present study's sample was limited to bulimic women. Thus, the present study's result follows the trend in the literature which has been inconclusive in discovering the relationship between sex role beliefs and bulimia (Striegel-Moore et al., 1986). A more geographically and socioeconomically representative sample, or a sample gathered from a different section of the United States, might produce different results.

Bulimia and the Effectiveness of Desipramine Treatment

Previous research has provided some evidence for the efficacy of antidepressants in the treatment and subsequent reduction of bulimic symptoms (e.g. Hughes et al., 1986; Solyom et al., 1989). However, much of the previous research was flawed, for reasons ranging from the use of open label medication trials (Solyom et al., 1989) to the use of self-report measures that had not been previously validated (Hughes et al., 1986).

This study improved upon past research in a number of ways, from using the EDI (a valid instrument) to measure the severity of bulimic symptoms to using a single blind design. Pre- and posttest measures of the severity of bulimic symptoms were analyzed across experimental and control groups to determine desipramine treatment effectiveness. The findings indicated a significant difference between the severity of bulimic symptoms for the experimental and the control group, suggesting that involvement in the active phase of the study (desipramine treatment) was related to the bulimic symptom decrease in the experimental group and supporting previous research findings. It can be inferred that the desipramine treatment alone may have been responsible for the posttest score decrease in the experimental group because the strength of the experimental design allowed for the elimination of many multiple confounding variables (e.g. the participants neither

received nor participated in any form of psychotherapy during the study).

Bulimia and Desipramine Blood Plasma Levels

It is unclear whether blood plasma levels measure medication effectiveness, but they are an effective way of measuring medication compliance (Hughes et al., 1986; Russell et al., 1988). Before conducting their study, Hughes et al. (1986) speculated that inadequate or widely varying drug levels might occur in bulimia as they do in depression. Their data supported this hypothesis, with patient desipramine levels ranging from 44 to 501 nanograms at a target dose of 200mg of desipramine yet their sample had a 91% decrease in binge frequency. They concluded that an effective therapeutic blood level range for desipramine still needed to be established.

The present study examined whether a relationship existed between blood levels of desipramine and a decrease in the severity of bulimic symptoms in the experimental group. The correlation between final blood levels and a posttest measure of the severity of bulimic symptoms displayed a low inverse relationship. Generally, as the amount of desipramine increased in a participant's blood her bulimic symptoms decreased. Seventy-eight percent of the participants evidenced a decrease in the severity of bulimic symptoms, yet only four of nine participants had blood levels in the suggested therapeutic range for desipramine

(100-150 nanograms). In addition, the present study supports the speculation made by Hughes et al. (1986) that inadequate or widely varying drug levels might occur in bulimia. Subjects in the present study had final blood levels ranging from 30 to 189 nanograms at a dose of 200mg of desipramine.

It can be inferred that the severity of bulimic symptoms in the experimental group decreased because of the desipramine treatment. The inverse relationship between the final desipramine blood level and the severity of bulimic symptoms was not strong in the context of the present study; however, it can be inferred that whatever amount of desipramine was present in the experimental subjects' blood might have been effective in reducing the severity of bulimic symptoms.

These results mandate further investigation into establishing a therapeutic range for desipramine in the treatment of bulimia and continued investigation into what factors contribute to a decrease in bulimic symptoms. Additionally, further research is required to investigate whether participation in a medication study alone without psychotherapy contributes to the decrease in bulimic symptoms. Regardless, the importance of using blood levels as a measure of treatment effectiveness remains unclear; however they do provide evidence of medication compliance (Russell et al., 1988).

Bulimia and Dysfunctional Body Image Perceptions

Body image dissatisfaction and distortion may be present in people who manifest eating disorders (Butters & Cash, 1987), may be inversely related to the severity of bulimia (Mintz & Betz, 1988), or may not be present in bulimic women at all (Steiger et al., 1989). The authors of the EDI (Garner et al., 1983) found that body dissatisfaction was related to other body image disturbances which have been considered a basic deficit in anorexia nervosa but not in bulimia nervosa. The EDI's Body Dissatisfaction subscale was designed with these relationships in mind. Although it provides useful diagnostic information for all eating disorders, the highest scores on the Body Dissatisfaction subscale are expected to occur with anorectic subjects (Garner et al., 1983).

The present study further investigated the relationship between the severity of bulimic symptoms and dysfunctional body image perceptions. Correlational analyses indicated that virtually no relationship existed between measures of the severity of bulimic symptoms and measures of dysfunctional body image perceptions, supporting the findings of Garner et al. (1983). These results also supported the findings of Steiger et al. (1989), who compared 31 women with eating disorders (anorexia nervosa, bulimiarexia or bulimia) with 11 normal control women and found that only the anorectics exhibited body-image

distortions.

Dysfunctional body image perceptions, while common in the United States (Butters & Cash, 1987), is not a diagnostic criterion for bulimia nervosa but is a required diagnostic criterion for anorexia nervosa in the DSM-III-R (American Psychiatric Association, 1987). Therefore, if clients are exhibiting extreme dysfunctional body image perceptions, clinicians should not use this as a diagnostic criterion in assessing for the presence or absence of bulimia. Further investigation is required into this relationship.

Factors Contributing to the Severity of Bulimia

The results of the present study indicated that desipramine treatment was an effective way to reduce the severity of bulimic symptoms in women. In order to investigate the combined predictive effect of the posttest treatment variables on the reduction of the severity of bulimic symptoms a multiple linear regression was performed for experimental group participants, using posttest Bulimia subscale scores of the EDI as the criterion variable and posttest global BDI scores, Masculine and Feminine subscale scores of the BSRI, and final desipramine blood levels as predictor variables. As shown in Appendix A, none of the predictor variables in isolation or in combination appears to be a significant predictor of the severity of bulimic symptoms in a posttest phase. Due to a variety of factors,

these results were not unexpected. For example, because desipramine is an antidepressant and could be presumed to have alleviated any depressive symptoms in the experimental subjects, a significant relationship between the severity of bulimic symptoms and depression would not be expected.

Limitations of the Study

Limitations of the present study should be noted. Bulimia is a disorder that is characterized by denial and secrecy (Gandour, 1984); because of this, it was difficult to gather a large sample size. In addition, this study was not geographically representative of the United States population. Therefore, the results may only be externally valid to the southeastern United States. The sample also consisted solely of Caucasian women, thus limiting the generalizability of the results to other racial and ethnic groups.

An additional concern is that some of the participants seemed to have a decrease in their bulimic symptoms even though they did not reach "clinically effective" blood levels of desipramine of note. "Effective levels" have yet to be determined (Hughes et al., 1986).

The single-blind design was also a limitation. To prevent possible experimenter bias and increase external validity, future research examining the effectiveness of antidepressants on reducing bulimic symptoms should use double-blind placebo controlled designs.

A final limitation concerns the socioeconomic backgrounds of the participants. Most were college-aged and came from lower to upper middle class backgrounds. In most cases, the participants' mother or father (or both) was employed as a professional (i.e. as an executive, a teacher, etc.). More generalizable results might have been obtained if the sample were more representative of the socioeconomic strata in the United States.

Recommendations for Further Research

The results of the present study suggest that bulimia and depression are directly related. Existing research has hypothesized that bulimia shares many characteristics, both physiological and otherwise, with major affective disorders (e.g. Hudson & Pope, 1990; Mitchell, 1990). Further research is needed to determine whether major affective disorders are secondary characteristics of bulimia, or whether the presence of depressive symptoms in bulimics is symptomatic of another, unique disorder that may not yet be identified. Further research is also needed to determine if people who manifest major affective disorders as a primary DSM-III-R diagnosis (American Psychiatric Association, 1987) also have bulimic symptoms. Once the relationship between bulimia and major affective disorders is clearer, more effective treatment modalities for bulimia could be designed and the potential cause(s) might be discovered.

Present findings also support further examination of

perceived sex role beliefs of bulimics and what role, if any, these beliefs have in the manifestation and maintenance of bulimic symptoms. Past research has been inconclusive in discovering the perceived sex role beliefs of bulimics (e.g. Steiger et al., 1989). The results of this study found no support for the hypothesis that a relationship existed between perceived feminine sex role beliefs and the severity of bulimic symptoms. Stronger, more conclusive evidence with a more geographically representative sample is needed before externally valid conclusions can be reached.

Additional research is needed into the effectiveness of desipramine and other antidepressants in the treatment of bulimia. Larger, more representative samples should be used with double-blind placebo controlled designs to guard against experimenter bias. Other antidepressants (in addition to desipramine) should also be used to investigate their effectiveness. Ideally psychotherapy would not be allowed in future studies. Blood levels should continue to be used, as no better method presently exists to check on participants' compliance (Russell et al., 1988). Blood tests will also provide researchers with abundant additional diagnostic information that may be useful in detecting the cause(s) of bulimia, or discovering if genetic markers exist.

Finally, additional research into body image perceptions of bulimics is warranted. Results of the

present study found virtually no relationship between dysfunctional body image perceptions and the severity of bulimic symptoms. Past research was also inconclusive on this issue (Butters & Cash, 1987; Mintz & Betz, 1988; Steiger et al., 1989). Further research will help resolve these issues and will provide useful diagnostic information into the self-perceptions of bulimics.

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APPENDICES

Appendix A

Multiple Linear Regression to Predict Posttest Bulimia Subscale Scores on the Eating Disorder Inventory (N = 9)

Variable	R-Sq.	B Weight	F value	p value
Final Desipramine Blood Level	.0288	.044		
Posttest BSRI Masculine Score	.019	.2989	.116	.745
Posttest BSRI Feminine Score	.013	.3482	.079	.7844
Posttest Global BDI Score	.0647	.704	.415	.5432

For overall model: R-Sq = .3875; Adj R-Sq = .4291

F (4,4) = .475, p = .7580

Note: BSRI stands for Bem Sex Role Inventory, BDI stands for Beck Depression Inventory

Appendix B

List of Copyrighted Materials Used

Complete descriptions of the paper-and-pencil questionnaires used in this study can be found in Chapter One. For further information please write to the addresses listed below.

1. Eating Disorder Inventory

   Psychological Assessment Resources, Inc.

   P.O. Box 98

   Odessa, FL 33556

2. Beck Depression Inventory

   Center for Cognitive Therapy

   Room 602

   133 South 36th Street

   Philadelphia, PA 19104

3. Bem Inventory

   Consulting Psychologists Press, Inc.

   577 College Avenue

   Palo Alto, CA 94306

Appendix C

Additional Pretest and Posttest Comparison Data for all Subjects (N = 18)

Variable	*t* value@	*p* value
BSRI Masculine Scale	.7033	.4920
BSRI Feminine Scale	.0238	.9813
Global BDI Score	2.331	.0332*
BD Subscale	.9200	.3713

@ df = 16

* $p < .05$

Note: BSRI stands for Bem Sex Role Inventory, BDI stands for Beck Depression Inventory, BD stands for Body Dissatisfaction subscale of the Eating Disorder Inventory.

Appendix D

Informed Consent Form

I, _____, agree to participate in the research on bulimia nervosa that is being conducted at the University of Tennessee. The director of this project is Andrew Getzfeld. All procedures will be fully supervised by a licensed psychiatrist. I am aware that the medicine used in this study may cause me to become slightly drowsy, may cause me to experience constipation, and may cause me to have a dry mouth. I am aware that the medicine should not be combined with alcohol or other sedating chemicals or drugs. I am aware that caution must be used while driving and operating heavy machinery while I am taking the medicine. I also agree to submitting to blood tests at the second, fourth, sixth and eighth week of the study. The amount of time needed for the lab work will be approximately sixty minutes total. I also agree to fill out several questionnaires; the total time needed for this will be approximately ninety minutes. I will have the option of continuing drug treatment with the psychiatrist, should I desire, once the study is concluded.

I realize that, by participating in this study, my binging and purging may decrease and I may feel better about myself. I realize that participation may enable me to control my bulimic behaviors such as vomiting, binging and purging. I also realize that participation may help me to overcome any depressive symptoms that I may have.

I realize that all participation is voluntary and that refusal to participate will involve no penalty. If I refuse to participate, or decide to discontinue participation at any time, I understand that I may continue to receive therapy. All information will remain fully confidential; if the results of the study are reported in scientific journals, only aggregate group data will be used. I am aware that I am receiving a copy of this release form.

By signing this form, I agree to participate in this project.

_____ _____

Printed Name Date

_____ _____

Signature Age

For Information or Questions Contact:
Andrew Getzfeld
20 East Ninth St.
NYC, NY 10003
(212)-674-6955

VITA

Andrew Roy Getzfeld was born in New York City, New York on December 1, 1959. He attended high school at Stuyvesant High School in Manhattan and graduated in June 1977. He entered Vassar College and in May 1981 received the degree of Bachelor of Arts in Psychology. He then entered the University of Wisconsin, Madison in August 1981 and in May 1983 received a Masters of Science Social Work degree. In June 1984 he became a Certified Social Worker in New York State; soon after that he joined the Orange County Department of Mental Health in Newburgh, N.Y. as a Staff Therapist. After working there for sixteen months he joined the Bergen Regional Counseling Center in Hackensack, N.J. in the fall of 1985, where he worked as a Staff Therapist/Alcoholism Counselor. In September 1986 he became a Certified Alcohol Counselor in New Jersey. In September 1987 he entered the University of Tennessee, Knoxville. His graduate assistantships there included being a Teaching Assistant and an Instructor of both undergraduate and masters level courses. He also worked for two years as a research assistant for Dr. Michael T. Nettles, the Vice-President for Assessment. In December 1992 he received a Doctor of Philosophy degree in Education with a specialization in School Psychology.