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The affective variant hypothesis: How is bulimia nervosa related to depression?

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The Affective Variant Hypothesis: How is Bulimia Nervosa Related to Depression?

A Dissertation

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Abstract

Bulimia nervosa patients frequently score from mild to severe on measures of depressive symptoms. This association between bulimia nervosa and depression led some researchers to hypothesize that bulimia nervosa is a variant of the affective disorder of depression. The main purpose of this study was to provide further evaluation of the affective variant hypothesis of bulimia nervosa. It was postulated that bulimia nervosa patients with and without depression would differ from each other and from depressives without eating disorders on specific measures of cognitive, behavioral, and somatic symptoms associated with depression. Secondly, it was hypothesized that the nondepressed and depressed bulimic patients would differ from depressives but not from each other on measures of anxiety and obsessiveness which would lend support to the anxiety model of bulimia nervosa. The results did not support either the affective variant hypothesis or the anxiety model of bulimia nervosa. The depressed bulimia nervosa patients evidenced higher levels of somatic symptoms, cognitive
dysfunction, state anxiety, and obsessiveness than nondepressed bulimics. These two groups were similar in severity only in trait anxiety and anhedonia. The bulimic and nonbulimic depressed patients did not statistically differ on any measure when the bulimic was also clinically depressed. The dually diagnosed patients were more disturbed than either the nondepressed bulimics or the nonbulimic depressed patients. The nonbulimic depressed patients' scores were more severe than the normals' on four of the six significant measures (somatic symptoms, cognitive dysfunction, state anxiety, trait anxiety), the depressed bulimics' more severe on all six, and the nondepressed bulimics' more severe on only three measures (cognitive dysfunction, anhedonia, trait anxiety). While no measure was associated with bulimia nervosa, several measures were highly associated with depression.
The Affective Variant Hypothesis: How is Bulimia Nervosa Related to Depression?

Introduction

This dissertation presents an overview of bulimia nervosa and depression as well as a review of the research literature addressing the affective variant hypothesis of bulimia nervosa. The affective variant hypothesis explains bulimia nervosa as a variant of affective disorder and not distinct from the disorder of depression. An association between the two disorders has repeatedly been demonstrated, but the nature of the relationship has been unclear. This investigation was undertaken to identify variables that would be useful in defining the relationship between bulimia nervosa and depression.

Bulimia and Bulimia Nervosa

Bulimia nervosa first emerged as a subclinical variant of anorexia nervosa and since then has carried various labels as well as different diagnostic criteria. The disorder has been known as dysorexia (Guiora, 1967), bulimarexia (Boskind-Lodahl & White, 1978), subclinical anorexia (Crisp, Kalucy, Lacey, &

Since 1979, four sets of diagnostic criteria have been reported most frequently in the research literature. Russell's (1979, 1983) criteria have been utilized in Britain while the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM-III, 1980; DSM-IIIR, 1987) criteria have been preferred in the United States. These sets of diagnostic criteria are listed in Tables 1 through 3.

Three major symptoms have been found to comprise the bulimic syndrome (Fairburn & Garner, 1986). The primary feature of this eating disorder, regardless of the label, has been episodic, uncontrolled overeating. A second clinical feature is engaging in one or more extreme behaviors designed to control body weight. These weight control behaviors include self-induced vomiting, laxative or diuretic use, fasting or severe dieting, and excessive exercise. A third symptom,
Table 1.
Russell’s Diagnostic Criteria for Bulimia Nervosa

Bulimia nervosa—original criteria (Russell, 1979)
1. The patients suffer from powerful, intractable urges to overeat;
2. They seek to avoid the "fattening" effects of food by inducing vomiting or abusing purgatives;
3. They have a morbid fear of becoming fat.

Bulimia nervosa—revised criteria (Russell, 1983)
1. Preoccupations with food, irresistible cravings for food and repeated episodes of overeating;
2. Devices aimed at counteracting the "fattening" effects of food;
3. A psychopathology resembling that of classical anorexia nervosa;
4. A previous overt or cryptic episode of anorexia nervosa.
Table 2. DSM-III Diagnostic Criteria for Bulimia

A. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than two hours);
B. At least three of the following:
   1. consumption of high-caloric, easily ingested food during a binge
   2. unconspicuous eating during a binge
   3. termination of such eating episodes by abdominal pain, sleep, social interruption, or self-induced vomiting
   4. repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics and/or diuretics
C. Awareness that the eating pattern is abnormal and fear of not being able to stop eating voluntarily;
D. Depressed mood and self-deprecating thoughts following eating binges;
E. The bulimics episodes are not due to anorexia nervosa or any known physical disorder.

American Psychiatric Association (1980)

Table 3. DSM-IIIR Diagnostic Criteria for Bulimia Nervosa

A. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time);
B. A feeling of lack of control over eating behavior during the eating binges;
C. The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain;
D. A minimum average of two binge eating episodes a week for at least three months;
E. Persistent overconcern with body shape and weight.

preoccupation with body size, has been found to be associated with bulimia nervosa (Fairburn & Garner, 1986). These three symptoms are included as diagnostic criteria for bulimia nervosa in the recent revision of DSM-III (DSM-III-R, American Psychiatric Association, 1987).

DSM-III (American Psychiatric Association, 1980) required both depressed mood and self-deprecating thoughts following binge eating. The revision of DSM-III (American Psychiatric Association, 1987) eliminated this criterion, however. The elimination of this symptom might be due to findings that the mood immediately following binge eating is varied (cf. Mizes, 1985), and when present, the depressive mood is often more pervasive and not limited to the period following binging (Davis, Freeman, & Solyom, 1985; Fairburn & Cooper, 1982; Herzog, 1982, 1984; Johnson & Larson, 1982; Lee, Rush, & Mitchell, 1985; Mitchell, Hatsukami, Eckert, & Pyle, 1985; Williamson, Prather, Upton, Davis, Ruggiero, & VanBuren, 1987). Fairburn and Garner (1986) suggested that the psychopathology associated with bulimia nervosa is less specific than
the three main symptoms of binge eating, purging, and preoccupation with body weight and shape. However, they report that anxiety and depression are the most commonly associated symptoms. Others have suggested that depression is not an associated feature of bulimia nervosa but is the central problem (Hudson, Pope, Jonas, Laffer, Hudson, & Melby, 1983). This issue will be discussed in more detail in a subsequent section.

Bulimia is a relatively common disorder, with prevalence rates reported in the literature ranging from 1 to 16 percent (American Psychiatric Association, 1980; Cooper & Fairburn, 1983; Johnson, Lewis, Love, Stuckey, & Lewis, 1983; Mizes, 1985; Pyle, Mitchell, Eckert, Halvorson, Neuman, & Goff, 1983). The large variability in estimates of prevalence is probably due to differences in the diagnostic criteria utilized and whether or not self-induced vomiting was required for a positive diagnosis of bulimia. Although DSM-IIIR criteria are more restrictive than DSM-III, vomiting is still not essential for the diagnosis of bulimia nervosa. A patient may be diagnosed with bulimia nervosa if the
patient engages in either fasting, vigorous exercise, uses laxatives or diuretics, or self-induces vomiting to prevent weight gain between binges. Results from prevalence studies requiring the presence of self-induced vomiting suggested an incidence rate of approximately 4 percent (Johnson, Lewis, Love, Stuckey, & Lewis, 1983), while higher rates have been reported in studies not requiring self-induced vomiting.

Onset usually occurs between the ages of 15 to 18 years (Fairburn & Cooper, 1982; Pyle, Mitchell, & Eckert, 1981), however age of onset may range from 13 to 30 years (Russell, 1979). Ninety-five percent of those diagnosed with bulimia have been females.

**Major Depression and Dysthymia**

The diagnosis of major depression is indicated when a designated cluster of symptoms has been present for a period of at least two weeks (American Psychiatric Association, 1987). Refer to Table 4 for complete DSM-III-R diagnostic criteria. The symptom cluster includes a relatively pervasive and prominent dysphoric mood or decreased interest in usual
Table 4. DSM-IIIR Diagnostic Criteria for Major Depression

A. At least five of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood, or (2) loss of interest or pleasure:

1. depressed mood most of the day, nearly every day;
2. markedly diminished interest or pleasure in all, or almost all, activities most of the days, nearly every day;
3. significant weight loss or gain when not dieting or decrease or increase in appetite nearly every day;
4. insomnia or hypersonmia nearly every day;
5. psychomotor agitation or retardation nearly every day;
6. fatigue or loss of energy nearly every day;
7. feelings of worthlessness or excessive or inappropriate guilt;
8. diminished ability to think or concentrate, or indecisiveness nearly every day;
9. recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or specific plan for committing suicide

activities every day during the worst point of the depression. In addition, at least four of the following eight symptoms must also be present to satisfy diagnostic criteria. These symptoms include (a) appetite disturbance, (b) sleep disturbance, (c) psychomotor retardation or agitation, (d) a decrease in sexual interest and/or interest in usual activities, (e) loss of energy or fatigue, (f) feelings of worthlessness and/or excessive guilt, (g) indecisiveness and concentration difficulties, and (h) persistent suicidal ideation or attempts (DSM-IIIR, American Psychiatric Association, 1987).

Prevalence rates indicate that approximately 18 to 23 percent of females have had a major depressive episode at some time, and this rate is twice as high as that for males (Boyd & Weissman, 1981). Onset can occur at any age, and occurrence is relatively evenly distributed across adult age groups (American Psychiatric Association, 1980).

Dysthymia is similar to major depression with symptoms differing only in duration and severity (American Psychiatric Association, 1987). The DSM-IIIR diagnostic criteria for dysthymia are listed in
Table 5. Dysthymia is essentially a chronic disturbance of mood, and impairment in social and occupational functioning is usually mild to moderate. As in major depression, dysthymia is a common disorder and is identified more frequently in adult females. Onset generally occurs before or during early adult life.

Affective variant hypothesis

As discussed earlier, bulimia nervosa is frequently accompanied by depressive symptoms which are pervasive and sometimes severe (cf. Mizes, 1985; Williamson et al., 1987). Thus, it has been hypothesized that bulimia nervosa is another behavioral manifestation of depression and is not an independent syndrome. In the affective variant hypothesis, bulimia nervosa and depression are viewed as equivalent disorders with the same organic etiology (cf. Hudson, Pope, Jonas, Laffer, Hudson, & Melby, 1983).

Biological or organic formulations of depression are based on three main classes of observations. First, depressive disorders are accompanied by changes
Table 5. DSM-III-R Diagnostic Criteria for Dysthymia

A. Depressed mood most of the day, more days than not for at least two years.
B. Presence, while depressed, of at least two of the following:
   1. poor appetite or overeating;
   2. insomnia or hypersomnia;
   3. low energy or fatigue;
   4. low self-esteem;
   5. poor concentration or difficulty making decisions;
   6. feelings of hopelessness;
C. During a two-year period of the disturbance, never without the daily depressed mood for more than two months at a time.

In biological functions such as appetite and sleep, psychomotor activity, and sexual interest; second, depressed patients frequently report a positive history of affective disorder in first degree relatives which may support the theory of genetic transmission; third, biological treatments such as antidepressant medication frequently result in amelioration of depressive signs and symptoms (Carson & Carson, 1984; Davis & Sharma, 1986) although the mechanism of antidepressant action is unclear (Gerner & Bunney, 1986).

The affective variant hypothesis of bulimia nervosa was derived from these same three areas of research. In addition, several studies have suggested a high prevalence of depressive symptoms to be associated with bulimia nervosa. Research addressing the family history of depression in those diagnosed as bulimic, results of biological tests for depression in bulimia, and outcome using antidepressant medications to treat bulimia have led to the theory that bulimia nervosa is a variant of depressive disorder. These three areas of research as well as studies looking at the association of depression and bulimic symptoms
will be reviewed.

**Depressive symptoms in bulimia nervosa.** An association between depression and bulimia nervosa has frequently been reported (Pyle et al., 1981). Some studies have shown that bulimic patients do evidence mood disturbance (cf. Davis, Freeman, & Solyom, 1985; Fairburn, 1983; Herzog, 1984; Russell, 1979). Although survey studies are methodologically weak, results from these studies suggest that from 17 to 75 percent of subjects who endorsed bulimic behaviors also endorsed symptoms indicative of either current or past depression (Abraham & Beaumont, 1982; Fairburn & Cooper, 1982; Johnson, Stuckey, Lewis, & Schwartz, 1983; Hatsukami, Eckert, Mitchell, & Pyle, 1984; Herzog, 1982; Johnson, Stuckey, Mitchell, Hatsukami, Eckert, & Pyle, 1985). This wide range in number of subjects endorsing both bulimia and depression exists in part to methods of assessment as well as differences in the criteria used to identify both bulimia and depression. These studies have used unstructured interviews and questionnaires designed for the specific project and the psychometric
properties were not reported. Studies in which standardized diagnostic criteria and structured interviews were employed found the lower incidence of depression associated with bulimia (Herzog, 1984; Walsh, Roose, Glassman, Gladis, & Sadik, 1985). Another problem with survey studies is that because investigators were not blind to diagnosis the data were subject to experimenter bias. Hinz and Williamson (1987) pointed out that the incidence rate of current affective disorder in bulimic subjects was lower (24 to 33 percent) when structured interviews and standardized diagnostic criteria were used, and the high incidence rates occurred only when lifetime (past and present depressive episodes) data were included.

A number of studies have used standardized objective measures and included a non-eating disordered control group (Hatsukami, Owen, Pyle, & Mitchell, 1982; Prather & Williamson, 1988; Weiss & Ebert, 1983; Williamson, Kelley, Davis, Ruggiero, & Blouin, 1985; Williamson, Prather, Upton, Ruggiero, Davis & VanBuren, 1987). Without exception depression scores were found to be significantly higher in
bulimic groups than the control groups. Williamson et al. (1985) found that 35 to 40 percent of their bulimic subjects obtained clinically significant scores on objective measures of depression (T-score above 70 on the MMPI Depression subscale and/or a Beck Depression Inventory score above 20). Prather and Williamson (1988) replicated this incidence of clinically elevated scores using the MMPI and BDI. In addition, one study documented that more frequent self-induced vomiting was associated with higher scores on measures of depression (Williamson, Prather, Upton, Davis, Ruggiero, & VanBuren, 1987).

An association between bulimia nervosa and depression has been consistently documented in approximately 30 percent of patients with bulimia nervosa. However, causality should not be inferred from this association. Depression has been found to be associated with several disorders. One can entertain other hypotheses which have not been empirically eliminated to explain the association between depression and bulimia nervosa. For example, depression could occur due to the lack of control the
bulimic individual begins to feel, or depressive symptoms could be a reflection of inadequate nutrition.

Family history of depression. The second area of research used to support the affective variant hypothesis is familial association of depression. The heritability of depression is measured by investigating the incidence of affective disorders occurring in bulimics' relatives. Some researchers have reported a high occurrence of affective disorders among the relatives of bulimia nervosa patients (Hudson, Laffer, & Pope, 1982; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Pyle, Mitchell, & Eckert, 1981). The first investigators of the family history of depression in bulimia nervosa reported that approximately 60 percent of bulimics had a positive family history of affective disorder (Hudson, Laffer, & Pope, 1982; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Lee, Rush, & Mitchell, 1985). There were several methodological problems with these studies which included small sample size, investigators not blind to diagnosis, and use of patient report of family history of affective disorder when relatives
were not available.

Controlled studies using investigators blind to diagnosis have found a much lower occurrence of affective disorder in relatives of individuals diagnosed as bulimic. In fact, Stern, Dixon, Nemzer, Lake, Sansone, Smetzer, Lantz, and Schrier (1984) using a control group, blind diagnosis, and interviews of subjects' mothers found no difference between the frequency of affective disorder in relatives of bulimics and relatives of a control group. There were 368 relatives of bulimics and 384 relatives of control subjects included in the Stern et al. (1984) study. Rates of familial depression have been the highest when using retrospective, self-report methods. To date no studies have been reported investigating the familial association of depression in monozygotic and dizygotic twins who are also bulimic. This information is important as it has been shown that monozygotic twins have a higher concordance rate of affective disorder than do dizygotic twins (Bertelson, Harvald, & Hauge, 1977).

In addition to incidence rates of affective
disorder in relatives of bulimia nervosa patients, it
would also be relevant to investigate the incidence
rates of bulimia nervosa in relatives of affective
patients. If bulimia nervosa and affective disorder
had the same etiology the relatives of affective
disorder patients would be expected to develop bulimia
nervosa more frequently than would occur in the
general population. In a study described by Strober
and Katz (1987) first-degree relatives of patients
diagnosed with affective disorders had a low incidence
rate of eating disorders. The rates of eating
disorders in relatives of depressed, manic, and
schizophrenic patients ranged from 0.6 to 1.3 percent
which was not significantly different than lifetime
expectancy in the general population (Strober & Katz,
1987).

Dexamethasone suppression test of depression. A
third group a studies addressing the affective variant
hypothesis were designed to investigate the results of
a biological test of depression. The dexamethasone
suppression test (DST) has been purported to identify
depressive patients due to their abnormal or
nonsuppression of cortisol following injection of
The dexamethasone suppresses cortisol in nondepressed patients. Results of DST studies have shown similarities in nonsuppression of cortisol between bulimia nervosa and affective disorder patients (Gwirtsman, Roy-Byrne, Yager, & Gerner, 1983; Hudson, Laffer, & Pope, 1982; Hudson et al., 1983; Lindy & Walsh, Roose, Gladis, & Glassman, 1985). Four studies found that a high percentage of bulimic patients evidenced abnormal DST responses, that is, failed to suppress cortisol. Percentages varied from 35 percent (Lindy et al., 1985) to 67 percent (Gwirtsman et al., 1983). The abnormal DST responses in bulimic patients could be a result of other factors such as weight or nutrition abnormalities.

The ability of the DST to identify depressives has been adequately challenged (Birch, 1985; Walsh, Roose, Glassman, Gladis, & Sadik, 1985), and results of the DST are even more questionable for eating disordered individuals. It is known that weight change and nutrition affect the outcome of the DST (Carroll, Feinberg, & Greden, 1981; Fichter, Pirhe, & Holsboer, 1986; Smith, Bledsoe, & Chetri, 1975; Stahl & Kravitz,
Bulimic patients often have frequent weight fluctuations and inconsistent nutritional intake. Gwirtsman et al. (1983) did find that low body weight was related to abnormal DST responses. Lindy et al. (1985) failed to confirm this relationship between body weight and DST response. Other studies (Hudson et al., 1982; Hudson et al., 1983) did not control for weight status.

The DST has also been faulted for not being specific. Other disordered populations such as alcoholics, obsessive-compulsive patients, and patients diagnosed with borderline personality disorder have been found to evidence an abnormal DST response (Hinz & Williamson, 1987).

Antidepressant medications. The final area of research included in the affective variant hypothesis comes from the treatment of bulimic patients with antidepressant medications. It has been reported that bulimia nervosa patients reduced bulimic behaviors when treated with antidepressant medications (Agras & McCann, 1987; Pope & Hudson, 1986). Early uncontrolled reports (Jonas, Pope, & Hudson, 1983; Mendels, 1983; Moore, 1977; Pope & Hudson, 1982; Rich,
1978; Roy-Byrne, Gwirtsman, & Edelstein, 1983; Shader & Greenblat, 1982; Walsh, Stewart, Wright, Harrison, Roose, & Glassman, 1982) were suggestive that some bulimic patients may improve when treated with antidepressants.

The first controlled study to be reported (Sabine, Yonance, Farrington, Barratt, & Wakeling, 1983) treated 50 bulimic patients with the antidepressant mianserin. They found no differences in bulimic symptoms between placebo and drug conditions. They also reported that no significant improvement occurred in binge eating or self-induced vomiting during pharmacological treatment. In contrast, Pope, Hudson, Jonas, & Yurgelun-Todd (1983) reported a significant difference between placebo and imipramine groups in decreased binge eating behavior. At the end of treatment 35 percent of their patients treated with imipramine were reported to be in remission of bulimic symptoms (Pope et al., 1983). Pope and Hudson (1986) suggested that the differing results between Sabine et al. (1983) and Pope et al. (1983) may have been due to insufficient dosage in Sabine et al. (1983) and/or
more severely ill patients in Pope et al. (1983). Walsh, Stewart, Roose, Gladis, and Glassman (1984) reported a significant difference between placebo and phenelzine treated bulimic patients in binge eating frequency but no differences for measures of depression. Very few of the original 35 patients were included in the final data analysis (15 patients) so results must be interpreted cautiously. Placebo responders were eliminated during a wash-out phase thus biasing the results in favor of phenelzine.

In studies assessing blood plasma levels to insure a therapeutic medication dose was reached treatment outcome results have been inconsistent. Three studies to date have been reported (Agras, Dorian, Kirkley, Arnow, & Bachman, 1987; Hughes, Wells, Cunningham, & Ilstrup, 1986; Mitchell & Groat, 1984). One study (Hughes et al., 1986) reported significant differences between placebo and desipramine treated bulimic patients in improved binge eating frequency and one depression measure. Agras et al. (1987) found imipramine to be superior over placebo in reducing bulimic symptoms, however depression was not significantly improved. Mitchell and Groat (1984)
comparing amitriptyline to placebo found no differences between placebo and drug groups on any bulimic symptoms. They did report significant improvement in the drug group on one measure of depression (Hamilton Depression Scale) but not on another (Zung Depression Scale).

Due to the inconsistent treatment outcome in these studies treating bulimic patients with antidepressant medications one cannot conclude that bulimia nervosa is a variant of affective disorder with the same organic etiology. The fact that some antidepressants were found to be effective in reducing binge eating frequency in some patients does not mean that bulimia is an affective disorder. One must consider the fallacy of inferring causality from treatment results. The mechanism of antidepressant treatment effects is unclear. These medications have been used in treating disorders that have not been considered affective variants (Hinz & Williamson, 1987). As Walsh et al., (1985) stated, imipramine has been used to treat cardiac arrhythmias even though cardiac disorders are not a variant of affective disorder. It is
inappropriate to assume the circular logic that two or more disorders are the same because the same intervention is effective across the disorders. More importantly, when comparing the outcome of treatment studies, treatment of bulimia with antidepressant medications has been no more effective than cognitive-behavioral treatments. Typically, a 30 to 40 percent abstinent rate is reported in studies incorporating either treatment modality (Agras, 1987; Kirkley, Schneider, Agras, & Bachman, 1985).

Summary of research evidence. In summary, it has been documented that bulimia nervosa has been frequently accompanied by depression. Depression is known to be associated with many chronic psychological and physical disorders, e.g., agoraphobia (Munjack & Moss, 1981), alcoholism (Cadoret & Winokur, 1974), chronic pain (Keefe, 1982). The depressive symptoms associated with bulimia nervosa may also be due to the chronic, cyclic pattern of this eating disorder. As noted by Klerman (1980) pathological depression may exist as a separate syndrome or in association with another disorder.

Studies investigating family history of
depression, the dexamethasone suppression test for depression, and treatment of bulimia nervosa with antidepressant medications have not provided conclusive evidence that bulimia nervosa is a variant of affective disorder. It has been established that there is an association between depression and bulimia nervosa in some patients, however, these studies are incapable of determining the etiology of bulimia nervosa. A review of the research literature related to the affective variant hypothesis conducted by Swift, Andrews, and Barklage (1986) summarized that although there may be some type of relationship between eating disorders and affective disorder, the nature of the relationship is unclear. In a more recent review Hinz and Williamson (1987) concluded that although some research has supported the affective variant hypothesis of bulimia the propositions which follow from this hypothesis have not been conclusively supported. They suggested further research directly comparing bulimics and depressives using measures related to depression as well as measures related to bulimia (Hinz &
Comparison of bulimia nervosa and depression.

Prior to this study only one study has directly compared the symptoms of patients diagnosed with bulimia nervosa against patients diagnosed with major depression (Cooper & Fairburn, 1986). Both bulimia nervosa and major depression groups were assessed using the Present State Examination (Wing, Cooper, & Sartorius, 1974) and the Montgomery and Asberg Depression Rating Scale (Montgomery & Asberg, 1979). Bulimia nervosa patients were diagnosed using criteria from Russell (1979). A diagnosis of depression was based on the Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978). The affective variant hypothesis was evaluated by comparing the presence of depressive symptoms in the two clinical groups. Bulimia nervosa patients were found to evidence more anxiety, obsessional ideas, and pessimistic thought. These same subjects were found to endorse less simple depression, apparent sadness, sleep disturbance, and suicidal thought than the major depression patients. The authors concluded that there were different symptom patterns evident for the two patient groups.
and that depressive symptoms are likely to be secondary to bulimia nervosa (Cooper & Fairburn, 1986). They did not compare bulimia nervosa with a dual diagnosis of major depression to bulimia nervosa without major depression. If the affective variant hypothesis is valid bulimia nervosa patients with and without clinical depression should not differ on a variety of measures of psychopathology. Whereas, if depression is secondary to bulimia nervosa, then only those bulimics who are also depressed should evidence psychopathology similar to a depressed sample.

Affective variant hypothesis: Further evaluation.

Bulimia nervosa subjects and major depression subjects consistently achieve similar scores on general measures of depressive symptomatology, but research does not support the hypothesis that these two disorders are necessarily derived from the same etiology (Hinz & Williamson, 1987; Swift, Andrews, & Barklage, 1986). Cooper and Fairburn (1986) further questioned the hypothesis by finding that bulimia nervosa and depressive patients have different symptom patterns.
The important question then becomes, are depressed bulimia nervosa patients different from nondepressed bulimia nervosa patients? What variables besides specific eating behaviors might define the relationship between bulimia nervosa and depression? Researchers frequently evaluate depression on three dimensions which include cognitive, behavioral, and somatic symptoms. Perhaps bulimic and depressed individuals differ on one or more of these specific dimensions of depression even though they do not differ on general measures of depression.

**Cognitive Dimension.** Cognitive theories of depression (Beck 1967, 1976) postulate that depressives experience negative cognitions that distort their view of the self, world, and future. Depressive symptoms are seen as a direct result of this negative cognitive set. In cognitive theory of depression it has been proposed that individuals learn, through early experiences, cognitive styles (or schemata) that function to screen, categorize, and evaluate stimuli (Beck, 1974). These schemata result in self-concepts and personal expectations that are global, rigid, and negative (Hollon & Beck, 1979).
Several systematic cognitive distortions may result from these schemata (Beck, 1967). The most common of these errors in logic are explained in Table 6. The relationship of the biased cognitive style to depression has been widely researched and supported. Hammen, Krantz, and Cochran (1981) found that perceptions of globality (belief that the event would affect other aspects of the person's life) and low controllability were predictive of depression onset. This finding has been replicated in other investigations (cf. Golin, Sweeney, & Shaeffer, 1981). Because the presence of a negative cognitive style has been adequately linked to the onset of depression, it is plausible that measures of dysfunctional cognitions would not discriminate between bulimia nervosa and depression subjects if the affective variant hypothesis was correct. The questions of interest were, (a) do bulimics and depressives have similar cognitive dysfunction and (b) do depressed bulimics differ from nondepressed bulimics on measures of cognitive dysfunction?

Behavioral Dimension. In behavioral theories of
Table 6
Common Cognitive Errors

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Arbitrary inference: drawing a conclusion in the absence of evidence or when the evidence is contrary to the conclusion.</td>
</tr>
<tr>
<td>2</td>
<td>Selective abstraction: focusing on a negative detail in a situation and conceptualizing the entire experience on the basis of this negative fragment.</td>
</tr>
<tr>
<td>3</td>
<td>Overgeneralization: drawing a general rule or conclusion on the basis of one isolated incident and applying the concept indiscriminately to both related and unrelated situations.</td>
</tr>
<tr>
<td>4</td>
<td>Magnification and minimization: overestimating the significance or magnitude of undesirable events and underestimating the significance or magnitude of desirable events.</td>
</tr>
<tr>
<td>5</td>
<td>Personalization: relating external events to oneself without evidence.</td>
</tr>
<tr>
<td>6</td>
<td>All-or-none thinking: thinking in absolute black or white, all-or-none terms.</td>
</tr>
</tbody>
</table>

Beck (1967).
depression it has been postulated that depression is a consequence of reduced response-contingent positive reinforcement (Lewinsohn, 1974). Reinforcement, in this context, refers to the quality of one's interactions with one's environment. Productive, social behavior of depressed persons does not result in sufficient positive reinforcement to maintain the behavior. This low rate of pleasurable activity provides little or no rewarding interactions with the environment and is assumed to lead to dysphoric feelings that lead to further reduction in responses that might be positively reinforcing. A positive correlation between positive mood and frequency of participation in pleasant activities has been documented by Lewinsohn and Graf (1973) and Lewinsohn and Libet (1972). The average rate of participation in pleasurable activities as well as the subjective enjoyability of potentially reinforcing events were both lower in depressed subjects relative to psychiatric and normal control subjects (Lewinsohn & MacPhillamy, 1974; MacPhillamy & Lewinsohn, 1974). Again, the question to be answered was, would the interaction between depression and bulimia nervosa
result in more severe scores indicating less participation in and enjoyability of activities?

**Somatic Dimension.** Endogenous depression or depression with an organic etiology is well known to be accompanied by physical or somatic signs which have been described as vegetative. These somatic signs include appetite disturbance, sleep disturbance, psychomotor disturbance, and fatigue or loss of energy. The presence of these somatic features in depressed patients has been used to support the biological theory of affective disorders. Cooper and Fairburn (1986) reported a significant difference between bulimics and depressives in sleep disturbance. Other somatic signs were either not significant or not reported.

Bulimics have been found to endorse test items reflecting somatic distress arising from concerns about bodily dysfunction (Williamson et al., 1985). Bulimics achieved higher scores than normals did on the Somatization subscale of the Symptom Checklist 90-Revised. The Somatization subscale measures complaints focused on cardiovascular,
gastrointestinal, respiratory systems, and other signs with strong autonomic mediation (Derogatis, 1977). The signs and symptoms included on this subscale are reported to be somatic equivalents of anxiety (Derogatis, 1977). Available reports concerning somatic symptoms suggest that bulimic individuals may experience anxiety symptoms, but the presence of vegetative signs associated with depression in bulimic individuals is unclear. Knowing whether vegetative signs differ between bulimics with and without a diagnosis of depression would be useful in evaluating the affective variant hypothesis of bulimia nervosa, especially since this hypothesis was based on a biological theory.

**Obsessions-compulsions.** As noted by Lehmann (1985), an association between depression and compulsive and obsessional features has long been recognized. This relationship, however, has been reported to be a complex one since not all depressed patients develop obsessive-compulsive problems nor do all obsessive-compulsive patients develop depressive complaints (Sturgis, 1984). Co-occurrence rates have been reported to range from 20 to 45 percent
Cooper and Fairburn (1986) found that only 2.5 percent of the depressed subjects evidenced obsessional ideas and ruminations. It appears that obsessive-compulsive patients may also evidence symptoms of depression but few depressive show obsessive-compulsive symptoms.

The anxiety model of bulimia, incorporated obsessive-compulsive components as a central feature of bulimia nervosa (Rosen & Leitenberg, 1982). Anxiety and fear of weight gain are reported to follow binge eating. As can be seen in Figure 1, purgative behavior reduces this anxiety and fear thus negatively reinforcing purging (Williamson, Prather, Goreczny, Davis, & McKenzie, 1989). The DSM-IIIR (American Psychiatric Association, 1987) diagnostic criteria for obsessive-compulsive disorder follow a similar pattern. Ruminative obsessions result in anxiety and distress; engaging in compulsive behavior is said to aid the patient in avoiding or escaping anxiety and obsessive thoughts (DSM-IIIR, 1987).

Several studies have consistently demonstrated...
Depression & Low Self-Esteem

Distorted Body Image

Interpersonal & Family Problems

Biological Deprivation & Hunger

Binge Eating

Anxiety & Worry concerning Weight Gain

Purge

Anxiety Reduction

Elimination of Energy and Nutrients

Obsessive Compulsive Habits

Substance Abuse

Stress

Anxiety Reduction

Elimination of Energy and Nutrients

Figure 1. Anadet Model of Bulimia, Williamson, Prather, Garfinkel, Davis, & Mckenzie (1989).
that bulimic individuals who purge evidence obsessive-compulsive characteristics (Cooper &
Fairburn, 1986; Hatsukami, Owen, Pyle, & Mitchell,
1982; Prather & Williamson, 1988; Scott & Baroffio,
1986; Williamson, Kelley, Davis, Ruggiero, & Blouin,
1985). Cooper and Fairburn (1986) found that 80
percent of their bulimic sample endorsed items on the
Present State Examination indicative of obsessional
ideas and ruminations. Obsessional ideas and
ruminations contributed most to the discrimination of
bulimia nervosa subjects from depressives (Cooper &
Fairburn, 1986). Williamson et al. (1985) reported
that bulimics differed significantly from a normal
control group on the MMPI clinical scale number seven
(Psychasthenia) which is associated with obsessive
found that 62 percent of bulimia nervosa subjects
obtained clinically elevated scores (T-score equal to
or greater than 70) on MMPI scale seven or
Psychasthenia.

In summary, although obsessive-compulsive patients
may also evidence symptoms of depression few
depressives show obsessive-compulsive symptoms. In
comparison, several studies have documented that bulimia nervosa subjects experience obsessive-compulsive characteristics. From these data, it could be expected that bulimic patients but not depressives would frequently endorse obsessive-compulsive symptoms.

**Anxiety.** Anxiety symptoms may accompany depressive disorders although anxiety disorders are less commonly accompanied by depression (Lehmann, 1983). A 58 percent incidence rate of anxiety symptoms in a group of depressed patients was reported by Leckman, Weissman, Merikangas, Pauls, and Prusoff (1983). They also concluded that depressed patients feeling anxiety symptoms had early onset and severe depression (Leckman et al., 1983). Cooper and Fairburn (1986) found that within their depressed group 60 percent endorsed general anxiety while 37.5 percent endorsed situational anxiety.

High levels of anxiety have been reported in bulimic samples (Cooper & Fairburn, 1986; Fairburn & Cooper, 1982; Pyle, Mitchell, & Eckert, 1981; Williamson et al., 1985). Some researchers have
proposed that the cycle of binging and self-induced vomiting may be directly linked to anxiety (Leitenberg, Gross, Peterson, Janis, & Rosen, 1984; Rosen & Leitenberg, 1982). Others have associated anxiety with the anticipated lack of control experienced by bulimics (Palmer, 1979; Rau & Green, 1975). Cooper and Fairburn (1986) reported that 48.6 percent of bulimia nervosa subjects endorsed generalized anxiety items and 71.4 percent endorsed situational anxiety items on the Present State Examination. Williamson et al. (1985) reported significantly more severe scores for bulimic subjects than normal controls on the anxiety scale of the Symptom Checklist 90-Revised. Using behavioral observation procedures Rosen and Leitenberg (1982) and Leitenberg et al. (1984) found that bulimics reported increased subjective anxiety following eating a large test meal which they were kept from purging. Several studies utilizing psychophysiological recording have reported increased heart rate in bulimics after eating (Janata, Klonoff, & Ginsberg, 1985; Leitenberg et al., 1984; Jarrell, Johnson, & Williamson, 1986; Williamson, Goreczny, Davis, Ruggiero, & McKenzie, 1986).
1988). Two studies also reported a reduction heart rate immediately following purging a test meal (Janata et al., 1985; Jarrell et al., 1986). In a controlled study by Williamson et al. (1988) bulimics responded to eating with an immediate increase in heart rate, whereas the heart rate of normals decreased after eating. Thus, studies using subjective, behavioral, and psychophysiological measures consistently support the role of anxiety in bulimia nervosa. This anxiety is clearly associated with eating but may also be more generalized.

Current research has suggested that bulimic subjects experience more situational anxiety, while depressed subjects report more generalized anxiety. The unanswered question was, how do bulimics with depression differ on situational and general anxiety symptoms from bulimics without depression and depressive patients without bulimia nervosa?

In summary, no past study of the affective variant hypothesis has controlled for depression in bulimia nervosa. It is unknown how bulimics with depression differ from bulimics without depression. In addition,
it is not known how these two eating disordered groups differ in cognitive style, behavioral style, or somatic signs from depressives or normal subjects. Comparison of research on bulimia nervosa and research on depression suggests that bulimia nervosa subjects may exhibit more obsessive and situational anxiety characteristics than do both depressives and normals and more general anxiety than normals.

**Summary**

Bulimia nervosa is frequently accompanied by depressive symptoms which are often pervasive and severe. This association has led some researchers to hypothesize that bulimia nervosa is a variant of affective disorders. The only study comparing bulimics to depressives found that bulimics reported a different pattern of symptoms than the depressive patients (Cooper & Fairburn, 1986). The bulimic sample was found to be more anxious and obsessive and less depressed than were the depressives.

The purpose of this study was to further examine the affective variant hypothesis expanding upon the findings of Cooper and Fairburn (1986) using specific measures of depression, anxiety, and obsessions—
compulsions. The main goal was to compare bulimia
nervosa patients with and without depression,
depression patients without eating disorders, and
normal control subjects on cognitive, behavioral, and
somatic variables. These patients were compared on
cognitive dysfunction, behavioral activity and
anhedonia, somatic signs of depression, general and
situational anxiety, and obsessive-compulsive symptoms
in an attempt to identify variables other than eating
and purging behaviors that could define the
relationship between bulimia nervosa and depression.

Two main questions were addressed. First, would
there be an interaction effect of bulimia nervosa and
depression? Would bulimics with depression be more
disturbed than nondepressed bulimia nervosa patients
as reflected by scores on specific measures of
different dimensions of depression? If the affective
variant hypothesis was valid then these patients
should not be disparate. It was hypothesized that
bulimia nervosa patients without depression would be
less disturbed than bulimia nervosa patients with
depression who would be more disturbed than depressed
patients on cognitive, somatic, and behavioral aspects of depression.

Second, would there be a main effect of bulimia nervosa, main effect of depression, and/or an interaction effect of bulimia nervosa and depression on the measures of anxiety and obsessiveness? The second hypothesis proposed in this study was that there would be a main effect of bulimia nervosa but no main effect of depression or interaction effect of bulimia nervosa and depression. Nondepressed and depressed bulimics were predicted to endorse more anxiety and obsessiveness than depressives but not differ from each other. If the anxiety model of bulimia nervosa, which is analogous to an anxiety model of obsessive-compulsive disorder, was cogent bulimia nervosa patients with and without depression would not differ from each other on measures of anxiety and obsessive-compulsiveness.
Method

Research Participants

Clinical subjects were recruited from patients presenting for evaluation and treatment during the 1987-88 year at three fee-for-services outpatient clinics and two inpatient hospitals. These treatment centers served both student and nonstudent patients. Normal subjects were recruited through publicity from undergraduate psychology classes and from the community.

Included in the clinical sample were seventeen patients diagnosed with bulimia nervosa without depression, 18 patients diagnosed with bulimia nervosa and depression, and seventeen patients diagnosed with depression without eating disorders using DSM-III-R criteria for bulimia nervosa, major depression, and dysthymia. The bulimia nervosa with depression group was comprised of 15 patients with major depression and three patients with dysthymia. The depression without eating disorders group included 11 patients with major depression and six patients with dysthymia. The normal control group consisted of 20 subjects who reported no depression or eating disorder problems.

The 72 subjects were all Caucasian. Using
Kruskal-Wallis nonparametric test of homogeneity of demographic variables, it was determined that the four groups were from the same population. Chi square analysis confirmed the groups were comparable in distribution of gender and marital status (see Table 7). The distribution of educational status was also similar across groups, \( \chi^2 (.05, 3) = 0.59, p > .05 \). Analysis of variance (ANOVA) verified the similarity of groups on the variables of age, height, and weight. Comparing the clinical groups there were no differences in either distribution of outpatient and inpatient or medication status across the groups. Comparing the depression groups there were no significant differences in distribution of major depression or dysthymia. To ensure that the clinical groups did not differ in severity of either depression or bulimia nervosa they were compared on the respective scores on the BDI and BULIT. The depressed bulimics and the noneating disordered depressed subjects did not differ on the BDI. The nondepressed bulimics were more depressed than the normals, however. The nondepressed and depressed bulimics did not differ on the BULIT. Complete demographic statistics can be reviewed in Table 7.
Table 7. Group Demographics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Group Means</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BN(Y)</td>
<td>BN(Y)</td>
<td>BN(N)</td>
</tr>
<tr>
<td>Age</td>
<td>21.7</td>
<td>26.4</td>
<td>29.2</td>
</tr>
<tr>
<td>Height</td>
<td>65.3</td>
<td>64.8</td>
<td>66.3</td>
</tr>
<tr>
<td>Weight</td>
<td>134.4</td>
<td>141.4</td>
<td>143.1</td>
</tr>
<tr>
<td>BDI</td>
<td>10.0</td>
<td>31.9</td>
<td>27.8</td>
</tr>
<tr>
<td>BULIT</td>
<td>116.6</td>
<td>125.7</td>
<td>56.9</td>
</tr>
</tbody>
</table>

Group Distributions

<table>
<thead>
<tr>
<th>Gender</th>
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<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
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<td>n</td>
</tr>
<tr>
<td>Male</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
<td>17</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Marital</th>
<th>Married</th>
<th>Single</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Married</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>Single</td>
<td>14</td>
<td>11</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outpatient</th>
<th>Yes</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Yes</td>
<td>16</td>
<td>1</td>
</tr>
<tr>
<td>NO</td>
<td>1</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication</th>
<th>Yes</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Yes</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>NO</td>
<td>15</td>
<td>10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Depression</th>
<th>Major Dep.</th>
<th>Dysthymia</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>n</td>
<td>n</td>
</tr>
<tr>
<td>Major Dep.</td>
<td>NA</td>
<td>15</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>NA</td>
<td>3</td>
</tr>
</tbody>
</table>

BN(Y)=Bulimia Nervosa, BN(N)=No Bulimia Nervosa, D(Y)=Depression, D(N)=No Depression.
Groups with different superscripts are significant at p < .05.
Assessment Instruments

Demographic data was collected from each subject using a questionnaire developed from the first two pages of the Diagnostic Survey of Eating Disorders (Johnson, 1985). Race, age, gender, height, weight, marital and educational status information was reported on the Demographic Questionnaire (see Appendix A).

The Diagnostic Interview for Bulimia Nervosa (DIBN), developed from Johnson's (1985) Diagnostic Survey of Eating Disorders, was used as a structured interview to elicit information following the DSM-IIIR criteria for a diagnosis of bulimia nervosa. Areas of information covered during this interview were weight history, weight and body shape concerns, dieting behavior, binge eating behavior, feelings of control while binging, purging behaviors, and exercise habits (see Appendix B).

The Bulimia Test (BULIT) designed by Smith and Thelen (1984) is a 32-item questionnaire which assesses abnormal eating attitudes and behaviors associated with bulimia (see Appendix C). Temporal stability was reported as $r = .54$, $p < .0001$. Smith and Thelen (1984) recommended using the research cut-off
score of 102 to reduce false positives, but that a score above 88 warrants clinical attention. Scores obtained on the BULIT were used for inclusion/exclusion criteria in this study. Normal control subjects had to score below 88 to be included while clinical subjects meeting the DSM-IIIR diagnostic criteria for bulimia nervosa had to also receive a score of 102 or above to be included.

The Schedule for Affective Disorders and Schizophrenia (SADS) developed by Endicott and Spitzer (1978) provide distinctive guidelines for interviewing patients to establish the presence of depressive symptoms and signs. Also available is a subtype of the SADS (see Appendix D) using questions that elicit information concerning signs and symptoms of major depression and dysthymia according to DSM-IIIR criteria (Leber, Beckham, & Danker-Brown, 1985). The SADS depression Scale has been demonstrated to have high inter-rater reliability ($r = .95$ or higher) and high internal consistency ($r = .95$ or higher). Concurrent validity for a diagnosis of depression has also been demonstrated (Endicott & Spitzer, 1978). The SADS, DSM-III Subtype, was used to determine the diagnosis of major depression or dysthymia.
Scores obtained on the **Beck Depression Inventory** (BDI) were used as an inclusion/exclusion criterion for subjects diagnosed as depressed. Depressed subjects had to obtain a score of 20 or above to remain in the study. Normal control subjects had to score below 11 on the BDI. The BDI is a standardized 21-item self-report questionnaire or interview measure which taps cognitive symptoms, somatic signs, and behavioral signs associated with clinical depression (see Appendix E). A score of 20 or above has been recommended for research inclusion (Beck, 1978; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). A score above 10 has been considered clinically significant (Beck, 1978). Test-retest reliability has been reported to range from r = .69 to .90 (Gallagher, Nies, & Thompson, 1982; Strober, Green, & Carlson, 1981). Numerous studies of concurrent validity have demonstrated the BDI to have moderate to good validity (Shaw, Vallis, & McCabe, 1985).

The **Center for Epidemiological Studies Depression Scale** (CES-D) developed by Radloff (1977) is a self-report measure designed to provide an index of the number and frequency of depressive signs and symptoms (Orme, Reis, & Herz, 1986). The CES-D can be reviewed
in Appendix F. The CES-D has been found to have good internal consistency across diverse population subgroups as well as good test-retest stability (Aneshensel, Clark, & Frerichs, 1983; Fava, 1983; Radloff, 1977; Roberts, 1980; Ross & Mirowsky, 1984). The CES-D differentiates between those in treatment and those not in treatment for depression (Orme et al., 1986). Factor analysis of the CES-D resulted in four subscales related to depression, (a) depressed affect, (b) positive affect, (c) somatic signs, and (d) interpersonal difficulty (Radloff, 1977). The Somatic subscale (CESD-SC) was used to measure whether differences existed in somatic signs of depression among the four groups.

The Dysfunctional Attitude Scale (DAS) was developed by Weissman and Beck (1978) to assess cognitive dysfunction centering around general irrational themes in thinking and negative thoughts (see Appendix G). The DAS has been shown to have good internal consistency (Weissman, 1979) and stability over time (Hamilton & Abramson, 1983; O'Hara, Rehm, & Campbell, 1982; Riskind, Beck, & Smucker, 1983). Construct validity was demonstrated by use of the DAS with both clinical and nonclinical populations (Eaves
& Rush, 1984; Hamilton & Abramson, 1983). Concurrent validity has been demonstrated in several studies (Dobson & Breiter, 1983; Riskind et al., 1983; Weissman & Beck, 1978). Differences in cognitive dysfunction among the groups were assessed using DAS.

MacPhillamy and Lewinsohn (1972) designed the Pleasant Events Schedule (PES) to assess self-reported frequency and enjoyment of engaging in common positive, reinforcing activities (see Appendix H). It was their contention that depressed individuals engage in fewer activities and experience less pleasure from activities. The psychometric properties of the PES reported by MacPhillamy and Lewinsohn (1972, 1974, 1982) were quite good. Test-retest reliability ranged from .69 to .88 for one-month test-retest. Validity studies reported satisfactory concurrent and predictive validity, and several studies also demonstrated construct validity showing significant correlations between mood and engagement in pleasant events (Hammen & Krantz, 1985). The PES was used to compare groups on both frequency (PES-F) and pleasantness (PES-P) of activities.

The State-Trait Anxiety Inventory (STAI) consists of two twenty-item self-report scales (see Appendix I)
which assess general anxiety proneness (trait anxiety) and current tension and apprehension or state anxiety (Spielberger, Gorsuch, & Lushene, 1970). Adequate validity has been demonstrated as has satisfactory reliability (Spielberger, 1983). The STAI scores were included to compare situational (STAI-S) and general (STAI-T) anxiety symptoms across groups.

Hodgson and Rachman (1977) developed the Maudsley Obsessive-Compulsive Inventory (MOCI) to assess both general and specific obsessive-compulsive symptoms (see Appendix J). The MOCI yields an overall obsessive-compulsive score plus five subscores of checking, cleaning, slowness, doubting-conscientiousness, and ruminations. The MOCI total score has been found to have adequate reliability and validity, and two of the subscales (checking and cleaning) have been validated (Rachman & Hodgson, 1980). The MOCI total score was used to compare groups on obsessive-compulsiveness.

Procedure

All subjects were given an informed consent form to read and sign before participating in this study (Appendix K). All patients and control subjects were informed that they could decline participation at any
point without penalty.

Subjects were interviewed for diagnostic purposes using the structured interview for diagnosing bulimia nervosa (DIBN) and diagnosing depression (SADS). Height and weight was measured for all subjects and the Demographic Questionnaire, BDI, and BULIT were administered. A diagnosis of bulimia nervosa with or without depression and depression without eating disorders was made during staffing supervised by licensed psychologists with extensive experience in both eating disorders and depressive disorders. Subjects meeting all inclusion criteria then completed a battery of instruments which included the DAS, PES-F, PES-P, CESD-SC, STAI-S, STAI-T, and MOCI.

Normal control subjects were interviewed and administered the BDI and BULIT. To qualify the control subjects had to score below 88 on the BULIT and below 10 on the BDI. Those subjects not endorsing either bulimia nervosa or depressive signs and symptoms during the interview or on the BDI and BULIT were then measured for height and weight and administered the Demographic Questionnaire, DAS, PES-F, PES-P, CESD-SC, STAI-S, STAI-T, and MOCI.
Results

Group Comparisons

A 2 X 2 between-subjects multivariate analysis of variance (MANOVA) was calculated on the seven dependent variables: PES-F, PES-P, STAI-S, STAI-T, MOCI, CESD-SC, and DAS. Independent variables were bulimia nervosa (yes and no) and depression (yes and no). Results of the multivariate test for homogeneity of variance-covariance revealed statistically similar variance-covariance matrices, Box's M = 12.83, p > .05. Total number of subjects included was 72. The MANOVA was followed by 2 X 2 factorial analyses of variance (ANOVA), and significant ANOVAs were subjected to the Scheffe' post hoc comparison test. The strength of each effect was tested using canonical correlational analyses (r ). To further describe these patients, c

ANOVA of group effects was also conducted. Summaries of the results are presented in Table 8 and Table 9.

Using Wilks' criterion, the 2 X 2 MANOVA resulted in significant main effects for both bulimia \( F(7,62) = 8.25, \ p < .0001 \) and depression \( F(7,62) = 22.80, \ p < .0001 \). There was a significant main effect of bulimia nervosa on all measures except the PES-F and CESD-SC. The results reflected a relatively strong
An association between bulimia nervosa and the combined dependent variables, $r = .69$.

The main effect for depression, $F(7,62) = 22.80, p < .0001$, resulted in a significant effect on five of the seven measures. The association was quite substantial between depression and the dependent variables, $r = .85$. There was no main effect of depression on the PES-F or PES-P. The summary of main effects is presented in Table 8.

There was a significant interaction between bulimia nervosa and depression [$F(7,62) = 3.07, p < .01$] on two dependent variables (see Table 8). The STAI-T and PES-P were found to be high for all three clinical groups. These two measures were low only in the nondepressed subjects that were also not bulimic. The addition of depression to bulimia had a significant effect on scores obtained by the clinical patients on the STAI-T and PES-P. The association between the interaction of the independent variables and the dependent variables was moderate, $r = .51$.

Analyses of variance (ANOVAs) investigating the combined effects of bulimia nervosa and depression yielded significant group effects for six of the seven variables. Only PES-F was not significant. A
Table 8. TESTS OF BULIMIA, DEPRESSION, AND THEIR INTERACTION

<table>
<thead>
<tr>
<th>IV</th>
<th>DV</th>
<th>Univariate F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulimia</td>
<td>CESD-SC</td>
<td>0.50</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>STAI-T</td>
<td>34.08</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>4.60</td>
<td>.0360</td>
</tr>
<tr>
<td></td>
<td>DAS</td>
<td>22.55</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>MOCI</td>
<td>5.78</td>
<td>.0190</td>
</tr>
<tr>
<td></td>
<td>PES-P</td>
<td>6.16</td>
<td>.0260</td>
</tr>
<tr>
<td></td>
<td>PES-F</td>
<td>0.39</td>
<td>NS</td>
</tr>
<tr>
<td>Depression</td>
<td>CESD-SC</td>
<td>116.56</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>STAI-T</td>
<td>49.58</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>74.92</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>DAS</td>
<td>36.83</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>MOCI</td>
<td>19.80</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>PES-P</td>
<td>3.52</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-F</td>
<td>3.57</td>
<td>NS</td>
</tr>
<tr>
<td>Bulimia</td>
<td>CESD-SC</td>
<td>0.18</td>
<td>NS</td>
</tr>
<tr>
<td>by Depression</td>
<td>STAI-T</td>
<td>15.75</td>
<td>.0001</td>
</tr>
<tr>
<td></td>
<td>STAI-S</td>
<td>2.33</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>DAS</td>
<td>2.12</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>MOCI</td>
<td>0.10</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-P</td>
<td>4.15</td>
<td>.0460</td>
</tr>
<tr>
<td></td>
<td>PES-F</td>
<td>2.18</td>
<td>NS</td>
</tr>
</tbody>
</table>

1 Degrees of freedom for all ANOVAs was (1,68).
complete summary of these analyses is presented in Table 9. The Scheffe post hoc test revealed that four measures (CESD-SC, STAI-S, DAS, and MOCI) distinguished between the two bulimia nervosa groups (depressed and nondepressed). The depressed bulimics obtained more severe scores than did the nondepressed bulimics suggesting that the former is more obsessive, more situationally anxious, experiencing more somatic signs of depression and cognitive dysfunction. Two measures differentiated the nondepressed bulimics and the depressed group. The depressed patients (without bulimia nervosa) received higher scores on the CESD-SC and STAI-S. The nondepressed bulimic patients endorsed more symptoms than the normals on three measures including the STAI-T, DAS, and PES-P. The depressed bulimia nervosa and depressed groups did not differ statistically on any dependent variable. The depressed bulimics scored higher than the normals on all six of the significant measures, while the depressed group scored higher than the normals on four of the six measures (CESD-SC, STAI-S, STAI-T, and DAS). These results suggest that the depressed bulimia nervosa patients were the most disturbed group.
Table 9. Summary of Combined Effects of Bulimia Nervosa and Depression

<table>
<thead>
<tr>
<th>Measure</th>
<th>BN</th>
<th>BN+D</th>
<th>D</th>
<th>NC</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D,SC</td>
<td>2.9</td>
<td>9.1</td>
<td>8.9</td>
<td>2.2</td>
<td>39.6</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>(2.4)</td>
<td>(2.7)</td>
<td>(3.3)</td>
<td>(1.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAI-T</td>
<td>65.8</td>
<td>72.2</td>
<td>68.3</td>
<td>45.1</td>
<td>35.7</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>(5.4)</td>
<td>(6.9)</td>
<td>(8.3)</td>
<td>(12.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAI-S</td>
<td>53.4</td>
<td>69.8</td>
<td>68.3</td>
<td>44.9</td>
<td>28.3</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>(8.2)</td>
<td>(7.5)</td>
<td>(9.4)</td>
<td>(12.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DAS</td>
<td>140.4</td>
<td>170.6</td>
<td>149.1</td>
<td>99.8</td>
<td>21.9</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>(24.3)</td>
<td>(25.8)</td>
<td>(27.5)</td>
<td>(31.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PES-P</td>
<td>330.4</td>
<td>333.6</td>
<td>338.1</td>
<td>414.2</td>
<td>4.6</td>
<td>&lt;.005</td>
</tr>
<tr>
<td></td>
<td>(74.0)</td>
<td>(99.1)</td>
<td>(99.3)</td>
<td>(50.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PES-F</td>
<td>233.4</td>
<td>227.7</td>
<td>209.8</td>
<td>256.8</td>
<td>2.0</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>(56.3)</td>
<td>(73.0)</td>
<td>(65.6)</td>
<td>(37.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MOCI</td>
<td>8.7</td>
<td>14.3</td>
<td>11.1</td>
<td>6.3</td>
<td>9.0</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td></td>
<td>(4.1)</td>
<td>(6.6)</td>
<td>(5.3)</td>
<td>(3.5)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 MANOVA F(21,179)=9.26, p<.0001.
2 Standard Deviations are presented in parentheses. Means with different superscripts differ significantly (p < .05).
3 BN=Bulimia Nervosa. BN+D=Bulimia Nervosa with Depression. D=Depression, NC=Normal Control.
4 Degrees of freedom for all ANOVAs was (3,68).
Correlational and Multiple Regression Analyses.

The results of the 2 X 2 Factorial MANOVA yielded strong main effects for both bulimia nervosa and depression, however, the interaction effect suggested that the combination of the independent variables had only an additive rather than a multiplicative effect on the dependent variables across the clinical subjects. Correlational analyses were computed among the seven variables and the BDI and BULIT to determine patterns of association among the seven measures of secondary psychopathology and measures of primary psychopathology of depression or bulimia nervosa (see Table 10). Stepwise multiple regression analyses were used to determine the variables most highly associated with either depression or bulimia nervosa (see Table 11 and Table 12). Both correlational and stepwise multiple regression analyses were computed on bulimia nervosa subjects alone, depressed subjects alone, and total sample. These analyses were done to assess the association between level of severity of bulimia nervosa and depression and the measures of secondary psychopathology.

Bulimia nervosa. Correlational analysis including only bulimic subjects (both depressed and
Table 10. Correlational Analyses

<table>
<thead>
<tr>
<th>Psychopathology</th>
<th>Groups</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>Secondary</td>
<td>Bulimic (n=35)</td>
<td>Depressed (n=35)</td>
</tr>
<tr>
<td>BULIT</td>
<td>STEAI-T</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>DAS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>BDI</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>STEAI-S</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>CESD-SC</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>MOCI</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-P</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-F</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>BDI</td>
<td>CESD-SC</td>
<td>.75*</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>STEAI-S</td>
<td>.77*</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>DAS</td>
<td>.58*</td>
<td>.41*</td>
</tr>
<tr>
<td></td>
<td>STEAI-T</td>
<td>.51*</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>MOCI</td>
<td>.46*</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>BULIT</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-P</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>PES-F</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = No significant correlation.

* Correlations listed differ significantly at p < .01.
nondepressed) resulted in no significant associations between the BULIT and the seven dependent variables. There were several significant positive correlations between the BDI and secondary measures. The STAI-S, CESD-SC, DAS, STAI-T, and MOCI scores were significantly correlated with the BDI scores within the bulimic group (see Table 10).

Stepwise multiple regression analysis was unsuccessful in producing any secondary measure that was significant in prediction of the primary psychopathology (BULIT) in the bulimic groups (see Table 11). Stepwise multiple regression analysis predicting BDI score for only the bulimic subjects found only two variables contributed significantly to prediction of the BDI score (see Table 12). The STAI-S and CESD-SC were significantly correlated with the BDI scores and accounted for a large proportion of the variance ($R^2 = .69$).

**Depression.** Correlational analysis including only the depressed subjects resulted in no significant correlations between the primary psychopathology of bulimia nervosa (BULIT) and the secondary variables. One significant correlation between the primary psychopathology of depression (BDI) and the DAS was
Table 11. Stepwise Multiple Regression Analyses for BULIT

<table>
<thead>
<tr>
<th>Variables</th>
<th>Groups</th>
<th>in Equation</th>
<th>Beta</th>
<th>$R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bulimic (n=35)</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Depressed (n=35)</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>All (n=72)</td>
<td>STAI-T</td>
<td>0.53</td>
<td>.28</td>
<td>&lt;.0001</td>
</tr>
</tbody>
</table>

Table 12. Stepwise Multiple Regression Analyses for BDI

<table>
<thead>
<tr>
<th>Variables</th>
<th>Groups</th>
<th>in Equation</th>
<th>Beta</th>
<th>$R^2$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bulimic (n=35)</td>
<td>STAI-S</td>
<td>0.49</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>CESD-SC</td>
<td>0.43</td>
<td></td>
<td>.69</td>
</tr>
<tr>
<td></td>
<td>Depressed (n=35)</td>
<td>DAS</td>
<td>0.34</td>
<td>.12</td>
<td>&lt;.05</td>
</tr>
<tr>
<td></td>
<td>All (n=72)</td>
<td>CESD-SC</td>
<td>0.48</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>STAI-S</td>
<td>0.44</td>
<td></td>
<td>.70</td>
</tr>
</tbody>
</table>
Stepwise multiple regression analysis correlating BDI score for the depressed groups found one variable to be significantly correlated with level of depression. The DAS was found to be correlated with the BDI ($R = .12$). Stepwise multiple regression analysis found no associations between the BULIT and secondary variables within the depressed subjects.

**Total sample.** There were several significant correlations between the BULIT and BDI and the secondary variables when all subjects were included. The BULIT was found to correlate with the STAI-T, DAS, BDI, and STAI-S. Stepwise multiple regression analysis, however, showed the STAI-T to be the only measure of secondary psychopathology to be associated with BULIT scores ($R = .28$). The BDI was found to correlate with all of the other measures except PES-F. Stepwise multiple regression analysis found the CESD-SC and STAI-S to account for the most variance in BDI scores ($R = .57$).

Finally, there was no significant correlation between the BULIT and the BDI within either the bulimia nervosa or the depression subjects. When the normal control subjects were included a significant
correlation between these two measures of primary psychopathology was found.
Discussion

It has been hypothesized that bulimia nervosa is a form of affective disorder and that the two disorders share a common etiology (cf. Hudson et al. 1983). Although the research has not supported the hypothesis of common etiology, the fact remains that bulimia nervosa patients frequently report depressive signs and symptoms (cf. Mizes, 1985). Cooper and Fairburn (1986), however, found depressive symptoms in bulimia nervosa patients to be nonspecific and different than the symptom pattern of depressed patients (Cooper & Fairburn, 1986). In addition to finding differing patterns of depression between the bulimia nervosa and the depressed group, Cooper and Fairburn (1986) also found that bulimia nervosa patients evidenced higher levels of situational anxiety and obsessiveness, while depressives were more generally anxious. Their findings suggested a difference between bulimia nervosa and depression. However, it was not known how many of the bulimia nervosa patients also met criteria for depression. The question unanswered was how nondepressed and depressed bulimia nervosa patients compared to depressed patients on measures of depression, anxiety, and obsessiveness.
This study was designed to address that question by testing the affects of bulimia nervosa and depression on various dimensions of depression, anxiety, and obsessive-compulsive characteristics. Dimensions of depression included in this investigation were somatic complaints, cognitive style, and behavioral style. Specific measures of state and trait anxiety and obsessive-compulsiveness were included.

The first hypothesis addressed the affective variant hypothesis. In this study it was proposed that bulimics without depression would be healthier than both depressed bulimics and non-eating-disordered depressives on various dimensions of depression. The second hypothesis supporting the anxiety model of bulimia nervosa stated that there would be a main effect of bulimia nervosa but no interaction effect of bulimia nervosa and depression on the anxiety measures. Nondepressed and depressed bulimics were predicted to not differ from each other but to achieve more severe scores than depressives on measures of anxiety.

Results suggested both bulimia nervosa and depression have strong main effects, but the
interaction of the two does not appear to have more than an additive affect upon the secondary psychopathology measured in this study. Depression was more clearly associated with these measures than was bulimia nervosa. Level of depression was highly associated with dysfunctional cognitions, somatic complaints, state anxiety, trait anxiety, and obsessiveness. Level of bulimia nervosa was not associated with any secondary measure.

Hypothesis One. The results of this investigation argue against the affective variant hypothesis. The proposition that depressed and nondepressed bulimia nervosa and depressed patients would be similar was not supported. The depressed bulimia patients were more disturbed than the nondepressed bulimics. In addition, there was no main effect of bulimia nervosa on somatic signs and frequency of activity, and there was no main effect of depression on either frequency of activity or pleasure rating of activities. Even though nondepressed bulimics did not evidence the somatic signs of depression they were not completely free from symptoms associated with depression (anhedonia and cognitive dysfunction). It should be kept in mind that the nondepressed bulimic subjects
did score higher than the normals on the BDI. It may be that even a very mild level of depression may affect the scores on these two variables, although there was no influence on somatic signs.

Cooper and Fairburn (1986) reported more pessimistic thinking in bulimia nervosa patients than depressed patients. In this study, although cognitive dysfunction occurred in both depressed and nondepressed bulimics, the dysfunction was associated only with depression. The dysfunction of the depressed bulimic was significantly more severe than nondepressed bulimics. Cooper and Fairburn (1986) found that their depressed group endorsed more somatic signs of depression than did the bulimics. The current study confirmed and expanded upon that finding; somatic complaints were associated only with depression and not bulimia nervosa.

Results related to hypothesis one have relevant assessment implications. Although incidence reports of affective disorder in bulimia nervosa have averaged around 50 percent (Lee, Rush, & Mitchell, 1985), Katz (1987) has questioned whether depression may be missed in the remaining bulimia nervosa patients. The findings in this study argue against that explanation.
Bulimia nervosa patients not diagnosed with affective disorder were different from both the depressed bulimia nervosa and the depressed patients. The bulimia nervosa group was generally less disturbed than either the depressed bulimia nervosa group or the depressed group.

In contrast to Katz's (1987) questioning whether a diagnosis of depression is being overlooked, Altshuler and Weiner (1985) questioned whether those bulimia nervosa patients diagnosed with affective disorder actually had a depressive disorder. These authors stated that core sign and symptom of somatic difficulties and anhedonia are typically not present in "depressed" bulimia nervosa patients. Because the depressed bulimics in this study achieved a score similar to depressives on the somatic scale of the CES-D suggests the presence of these somatic difficulties. Anhedonia measured by the PES-P was similar across both bulimic groups and the depressed group indicating that not only is anhedonia a problem for depressed bulimics but also for nondepressed bulimics. More research investigating the relationship between bulimia and anhedonia is needed.

Hypothesis Two. The second proposition of this
investigation stated there would be a main effect of bulimia nervosa on the anxiety measures. That is, the nondepressed bulimics and depressed bulimics would not differ from each other but differ from depressives on anxiety and obsessive-compulsive variables. Following from the anxiety model of bulimia nervosa, bulimic patients were believed to be more anxious and obsessive than depressives. This hypothesis was not supported. There was a main effect of bulimia nervosa on these measures, however there was also a main effect of depression. The interaction effect suggested that the addition of depression to bulimia did not statistically affect the scores of bulimics on any of the anxiety measures beyond that achieved by depression alone. Trait anxiety was similar across the clinical patients and was low only in the normal subjects. Nondepressed bulimics shared only trait anxiety with depressed bulimics, and the depressed bulimics did not differ from the depressives on any of the measures. The results of this study differ from Cooper and Fairburn's (1986) findings. Those authors found bulimics to be more obsessive and situationally anxious than depressives. The current study found that only depression correlated with state anxiety and
obsessiveness.

Researchers have consistently confirmed the presence of anxiety and obsessiveness resulting from food and weight related situations in bulimic patients (Barrios & Pennebaker, 1982; Cooper & Fairburn, 1986; Janata et al., 1985; Leitenberg et al., 1984; Jarrell et al., 1986; Rosen & Leitenberg, 1982; Williamson et al., 1988). Investigations of nonfood and nonweight anxiety and obsessiveness have been less convincing. Johnson, Stuckey, Lewis, and Schwartz (1983) found that bulimics did not differ from normals on anxiety and obsessive-compulsive subscales of the Hopkins' Symptom Checklist. Others have found that bulimics scored higher than normals on the Psychasthenia scale (Scale 7) of the MMPI (Prather & Williamson, 1988; Scott & Barrofio, 1986; Williamson et al., 1985). Cooper and Fairburn (1986) measured obsessiveness and anxiety as either situational or general. They found situational anxiety and obsessiveness but not general anxiety in bulimics. None of these studies controlled for depression status. The current study found trait anxiety in bulimics regardless of depression while state anxiety and obsessiveness were problematic only in depressed bulimics.
Although there has been consistent empirical support for the anxiety model in relationship to eating disorder signs and symptoms, the results from this study suggest that the model may not generalize to nonfood areas. Noneating related anxiety and obsessiveness appear to be related to depression.

A recent study (Burrows, Ribordy, & Johnson, 1989) found that bulimic individuals were accurate when defining their emotional states. Thus, we have confidence that our patients were able to discriminate and apply the dimensions measured in this investigation. Burrows et al. (1989) also found that bulimic patients were deficient in coping with their emotions and this deficiency was related to depression. The results of the present study combined with Burrows et al. (1989) and other research suggest that depression may seriously affect the secondary psychopathology as well as reduce coping abilities of bulimia nervosa patients.

It appears that bulimia nervosa has less association with secondary psychopathology than does depression. The two bulimic groups shared similarities in symptoms of general anxiety and anhedonia. Anhedonia and general anxiety were the
only measures that were independent of clinical depression status in bulimia nervosa patients suggesting that the dual diagnosis of depression with bulimia nervosa had no affect on these two symptoms for the nondepressed bulimia nervosa patients. Although nondepressed bulimics evidenced moderate cognitive dysfunction, the depressed bulimics' dysfunction was significantly more severe.

Differences in signs and symptoms across these groups argue against the hypothesis of a unitary affective or anxiety disorder underlying bulimia nervosa. Instead, these data suggest that nondepressed bulimia nervosa patients experience some of the same problems reported by depressed or anxiety patients. (i.e., trait anxiety, anhedonia, and cognitive dysfunction). They did not, however, report other problems common to depression or anxiety, (i.e., somatic complaints, state anxiety, obsessiveness).

Treatment implications. The combined results from both hypothesis one and hypothesis two may help explain some of the current inconsistencies in the bulimia nervosa treatment outcome research. The literature is inconclusive concerning the effectiveness of frequently used treatment components.
The debate includes three basic treatment modalities which include (1) antidepressant medication (cf. Hudson, Pope, & Jonas, 1986), (2) exposure with response prevention (cf. Wilson, Rossiter, Kleifeld, & Lindholm, 1986), and (3) cognitive-behavioral (cf. Agras, Schneider, Arnow, Raeburn, & Telch, 1989). Although studies vary in their outcomes, reports of patients remaining abstinent from purging activity at six-month follow-up range from 20 to 60 percent. The most frequent abstinent rate reported has been around 40 percent (Freeman, Barry, Dunkeld-Turnbull, & Henderson, 1988; Kirkley et al., 1985; Pope et al., 1983; White & Boskind-White, 1981). When comparing treatment outcome of E/RP, cognitive-behavioral, and antidepressants it is uncertain which program has been most effective.

What is not clear from any of the treatment outcome studies are the patient characteristics associated with improvement following interventions using different treatment components. From the results of this study it could be postulated that the depression status of the patient could have affected the outcome of treatment studies.

The results of the current study suggest that
there may be a subgroup of bulimia nervosa patients that are clinically depressed and might benefit from antidepressant medications plus E/RP and cognitive-behavioral treatment that is both specific to eating/weight related problem areas and noneating/nonweight problem areas. In this study, bulimia nervosa patients with depression were clearly different from patients that were not depressed. Failure to address these differences in treatment could help explain the high relapse rate (approximately 60 to 70 percent) in bulimia nervosa patients.

Research has suggested that depressed patients continue to improve following therapy, while bulimia nervosa patients tend to relapse. Keller, Herzog, Lavori, Bridges, Ott, and Klerman (1986) reported a 35 percent recovery rate at six months for bulimic patients compared to 64 percent at six months for depressed patients. The probability of full recovery from unipolar depression has been reported to range from 42 percent at three months to 79 percent at two years (Keller, 1985). Of particular interest is that out of the 60 percent of bulimic patients also classified with depression only one patient had
recovered from both disorders at six months follow-up (Keller et al., 1986). This low recovery rate may be explained by the results of this study. The dual diagnosed patients in this study were found to be more disturbed than nondepressed bulimics. It may not only be useful to design more comprehensive treatment of the depressed bulimia nervosa patient, but also to design interventions of longer duration to overcome both bulimia nervosa and depression.

Future Directions

Since this was the first study to compare nondepressed and depressed bulimia nervosa patients, these findings need to be replicated and extended.

Differences in the symptom profiles of bulimia nervosa patients in this study and the symptom profiles presented by Cooper and Fairburn (1986) could be explained by hypotheses independent of the presence or absence of depression in bulimia nervosa. First, different methods were used; the former authors used a structured interview format to establish the presence/absence and severity of symptoms. Secondly, since different measures were used, it is possible that variations of similar dimensions rather than the same dimensions (e.g., pessimistic thought versus
dysfunctional cognitions) were actually measured. Lastly, duration of the disorder was not controlled in either study. Perhaps depressed bulimia nervosa patients develop depression due to the chronic, cyclic nature of the eating disorder. Directionality is also an issue. These patients could also develop depression due to the situational anxiety and obsessiveness they experience or develop situational anxiety and obsessiveness due to the depression they experience.

These data suggest that research continue to address the development of depression in bulimia nervosa as well as treatment for bulimia nervosa patients with and without clinical depression. Unanswered questions are whether (a) treating situational anxiety, obsessions, and cognitive dysfunction associated with and independent of eating situations in the depressed bulimia nervosa patients and (b) treating both eating and noneating related general anxiety in all bulimia nervosa patients will increase the improvement rate.

Another area for further investigation is cognitive dysfunction. There has been an abundance of clinical observation and anecdotal information
suggesting that bulimics suffer from maladaptive cognitions. The presence of dysfunctional cognitions related to eating disorder symptoms in bulimia nervosa patients has been supported (Scanlon, Ollendick, & Bayer, 1986), however, more general cognitive dysfunction in bulimia has yet to be empirically validated. In a recent review of cognitions and bulimia ReynaMcGlone and Ollendick (1989) suggested several questions that need to be addressed: (1) do bulimics evidence cognitive errors, (2) if yes, to what extent, (3) are they exclusive to weight related concerns (4) if not, what are the other areas, and (5) are they distinguishable from depressives and normals?

The results from this study help answer some of those questions. First, bulimics obtained scores on the DAS indicative of clinical levels of cognitive dysfunction. Second, dysfunction appears to be mild to moderate unless the bulimic is also clinically depressed. The third and fourth questions asked by ReynaMcGlone and Ollendick (1989) can only be partially addressed by these results. Although the cognitive dysfunctions do not appear to be only related to weight concerns, it is unclear whether the depressed bulimics were experiencing different
dysfunctions, more dysfunctions at similar severity levels, or similar dysfunctions at higher severity levels. Lastly, scores on the DAS suggest that bulimics are distinguishable from normal subjects but not from depressed patients. It is evident from these results that further research into the cognitive dysfunction of the bulimia nervosa population is needed to clarify the relationship between bulimia, depression, and cognitive dysfunction.

**Summary**

The data from this study support previous research arguing against the affective variant hypothesis. Nondepressed and depressed bulimia nervosa patients achieved different symptom profiles. The results of this study also expanded upon the anxiety model. Nondepressed bulimics and depressed bulimics also reported different anxiety symptom profiles. In addition to different profiles on both depression and anxiety measures, bulimics who were clinically depressed were more disturbed than bulimics who were not diagnosed with depressive disorder. Depression clearly was the important variable affecting the dependent measures. There were several correlations between depression and secondary psychopathology but
no correlation between bulimia nervosa and secondary psychopathology.

These results call for further research investigating the development of depression in bulimia nervosa. Another important area needing further research is the cognitive dysfunction associated with bulimia nervosa. It is also suggested that research investigating treatment outcome report the relationship between outcome and the depression status of the bulimia nervosa patient. It may be that choices of treatment for bulimia nervosa patients are dependent upon whether they are also clinically depressed. Standard treatments for eating disordered patients may be acceptable for the nondepressed bulimic, however, the depressed bulimic may require the addition of other treatment components and longer treatment duration.
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APPENDICES

A through L

Assessment Measures
Appendix A

Demographic Questionnaire
## Identifying and Demographic Information

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<thead>
<tr>
<th>Sex</th>
<th>Male □</th>
<th>Female □</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race (Check one)</td>
<td>White □</td>
<td>Black □</td>
</tr>
<tr>
<td>Marital Status (Check one)</td>
<td>Present in first marriage □</td>
<td>Divorced and presently remarried □</td>
</tr>
<tr>
<td>What is your present primary role? (Check one)</td>
<td>Wage earner □</td>
<td>Husband-wife □</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Highest Occupational level attained</th>
<th>Self</th>
<th>Father</th>
<th>Mother</th>
<th>Spouse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Higher executive, proprietor of large concern.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Major professional</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Business manager of large concern, proprietor of medium sized business, lesser professional</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Administrative personnel, owner of small independent business, minor professional, owner of large farm</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Clerical or sales workers, technician, owner of little business, owner of medium sized farm</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Skilled manual employee, owner of small business</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Office operator, semiskilled employee, tenant farmer who owns little equipment</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Unskilled employee, sharecropper</td>
<td>□</td>
<td>□</td>
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<tr>
<td>Does not apply (Never worked in paid employment)</td>
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<tr>
<td>Does not apply (No spouse)</td>
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<td>□</td>
</tr>
<tr>
<td>Information not available</td>
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Current living arrangement (Check one)

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<th>With Parents or relatives</th>
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<tr>
<td>Alone</td>
<td>□</td>
</tr>
<tr>
<td>Shared apartment with Extend</td>
<td>□</td>
</tr>
<tr>
<td>Conjoint (kinship relationship with one other person, including spouse, boyfriend, etc.)</td>
<td>□</td>
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</table>

Highest level of education

<table>
<thead>
<tr>
<th>Self</th>
<th>Father</th>
<th>Mother</th>
<th>Spouse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completed postgraduate training</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Some postgraduate training</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Completed college, received four year academic degree</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Attended college, but didn't receive four year academic degree</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Completed high school, may have attended or completed trade school or other non-academic training requiring high school completion</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Attended high school</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Completed grammar school (8th grade)</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Attended grammar school</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>No schooling</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Does not apply (No spouse)</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>Information not available (Specify why)</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>
Appendix B

Diagnostic Interview for Bulimia Nervosa
DIAGNOSTIC INTERVIEW FOR BULIMIA NERVOSA

1. Have you ever been on a diet?
   Yes____  No____

2. At what age did you begin to restrict your food intake due to concern over your body size?
   _______ years old

3. Over the last year how often have you begun a diet?
   _______ number of times

4. Have you ever had an episode of eating a large amount of food in a short period of time (an eating binge)?
   Yes____  No____

5. How old were you when you binged for the first time?
   _______ years old

6. How characteristic are the following of your binge eating?
   Never—Rarely—Sometimes—Often—Always

   Consuming a large amount of food N R S O A
   Eating very rapidly N R S O A
   Feeling out of control N R S O A
   Feel down or annoyed afterward N R S O A
   Get uncontrollable urges to eat until physically ill N R S O A
   Binge eat in private N R S O A

7. Within the last three months, what has been your average number of binge episodes per week?
   _______ Number of binges/week

8. Have you ever vomited or spit out food after eating in order to avoid weight gain?
   Yes____  No____

9. How old were you when you induced vomiting for the first time?
   _______ years old
10. Have you ever used laxatives or diuretics to control your weight or "get rid of food"?
   Yes___ No___

11. How old were you when you first took laxatives/diuretics for weight control?
   _______Years old

12. During the entire last month, what is the average frequency that you have engaged in the following behaviors to control weight?
   Ginge eating:
   Never--1/Month--1/Week--1/Day--1/Day
   Self-induced vomiting:
   Never--1/Month--1/Week--1/Day--1/Day
   Laxative use:
   Never--1/Month--1/Week--1/Day--1/Day
   Use of diet pills:
   Never--1/Month--1/Week--1/Day--1/Day
   Use of enemas:
   Never--1/Month--1/Week--1/Day--1/Day
   Exercise:
   Never--1/Month--1/Week--1/Day--1/Day
   Fasting:
   Never--1/Month--1/Week--1/Day--1/Day

13. How many minutes a day do you currently exercise (including going on walks, riding bicycle, etc.)?
   _______Minutes
14. Weight History

Current weight ____________________ lbs.
Current height ____________________ inches
Desired weight ____________________ lbs.

Adult Years

Highest adult weight since age 10 ______ lbs. at age______
Lowest adult weight since age 18 ______ lbs. at age______
How long did you remain at your lowest adult weight?
_______ days _______ months _______ years

Adolescent years

Highest weight between ages 12-18 ______ lbs. at age______
Lowest weight between ages 12-18 ______ lbs. at age______

15. Childhood

How did you perceive your weight as a child between ages 12-18 years old?
Very thin--Somewhat thin--Normal--Somewhat overwt.--Very overwt.

16. At your current weight do you feel that you are:

Very thin--Somewhat thin--Normal--Somewhat overwt.--Very overwt.

17. How much does a two pound weight gain affect your feelings about yourself?
Extreme--Very much--Moderately--Slightly--Not at all

How much does a two pound weight loss affect your feelings about yourself?
Extreme--Very much--Moderately--Slightly--Not at all
10. How much does a five pound weight gain affect your feelings about yourself?
   Extremely—Very much—Moderately—Slightly—Not at all
   How much does a five pound weight loss affect your feelings about yourself?
   Extremely—Very much—Moderately—Slightly—Not at all
17. Has there ever been a time when your feelings about yourself or your social life have changed substantially as a result of losing weight?
   Yes_______ No_______
   If the answer is yes - Please tell me about it.

20. How often do you weigh yourself?
   More than daily____ Daily____ More than weekly____
   Weekly____ Monthly____ Less than monthly____
21. How dissatisfied are you with the way your body is proportioned?
   Extremely dissatisfied—Very dissatisfied—Moderately dissatisfied—Slightly dissatisfied—Not at all dissatisfied
22. How often do you think about your body shape?
   Always—Often—Sometimes—Rarely—Never
23. How do you feel about the different areas of your body?

Face:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Arms:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Shoulders:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Breast:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Stomach:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Buttocks:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

Thighs:
- Strongly positive—Moderately positive—Neutral
- Moderately negative—Strongly negative

24. How often do you measure your body size?

More than daily____ Daily_____ More than weekly____
Weekly_____ Monthly_____ Less than monthly____
Appendix C

The Bulimia Test (BULIT)
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These consist of pages:

115–121
123–130
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152–153
Appendix D

Schedule for Affective Disorders
Appendix E

The Beck Depression Inventory (BDI)
Appendix F

Center for Epidemiological Studies

Depression Scale
Appendix G

Dysfunctional Attitude Scale
Appendix H

Pleasant Events Schedule
Appendix I

Maudsley Obsessive-Compulsive Inventory
Appendix J

State-Trait Anxiety Inventory
SELF-EVALUATION QUESTIONNAIRE

Developed by Charles D. Spielberger
in collaboration with

STAI Form Y-1

Name _____________________________ Date _________ Sex: M ____ F ____
Age ________

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1. I feel calm .................................................................
2. I feel secure ............................................................... ...
3. I am tense ................................................................. ...
4. I feel strained ............................................................. ...
5. I feel at ease ............................................................... ...
6. I feel upset ................................................................. ...
7. I am presently worrying over possible misfortunes .......
8. I feel satisfied ............................................................ ...
9. I feel frightened ......................................................... ...
10. I feel comfortable ..................................................... ...
11. I feel self-confident .................................................. ...
12. I feel nervous ........................................................... ...
13. I am jittery ............................................................... ...
14. I feel indecisive ......................................................... ...
15. I am relaxed ............................................................ ...
16. I feel content ........................................................... ...
17. I am worried ............................................................ ...
18. I feel confused ..........................................................
19. I feel steady .............................................................. ...
20. I feel pleasant ........................................................... ...

Consulting Psychologists Press
577 College Avenue, Palo Alto, California 94306
SELF-EVALUATION QUESTIONNAIRE
STAI Form Y-Z

Name ________________________________ Date ________________________________

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

21. I feel pleasant ................................................................. O O O O
22. I feel nervous and restless ................................................ O O O O
23. I feel satisfied with myself ................................................ O O O O
24. I wish I could be as happy as others seem to be ..................... O O O O
25. I feel like a failure ............................................................... O O O O
26. I feel rested ................................................................. O O O O
27. I am "calm, cool, and collected" ............................................. O O O O
28. I feel that difficulties are piling up so that I cannot overcome them O O O O
29. I worry too much over something that really doesn't matter ....... O O O O
30. I am happy ................................................................. O O O O
31. I have disturbing thoughts .................................................... O O O O
32. I lack self-confidence .......................................................... O O O O
33. I feel secure ........................................................................ O O O O
34. I make decisions easily ......................................................... O O O O
35. I feel inadequate .................................................................. O O O O
36. I am content ......................................................................... O O O O
37. Some unimportant thought runs through my mind and bothers me O O O O
38. I take disappointments so keenly that I can't put them out of my mind ................................................................. O O O O
39. I am a steady person ........................................................... O O O O
40. I get in a state of tension or turmoil as I think over my recent concerns and interests ................................................................. O O O O
Appendix K

Informed Consent Form
CONSENT FORM FOR EATING DISORDERS RESEARCH

I, __________________________________, voluntarily consent to participate in the Eating Disorders Research program directed by Donald A. Williamson, Ph.D. This research involves both normal and eating disordered individuals. Therefore, provision of my consent does not imply that I have problems related to eating. By my signature, I agree to participate in the research activities indicated below and to allow data pertaining to me to be reported in scholarly publications, scholarly meetings, or in educational programs related to the Eating Disorders Research project. The activities which I agree to participate are those checked below:

1. Clinical interviews
2. Height and weight measurement
3. Psychological testing

All of my questions have been answered and I understand that I may withdraw for the research project without penalty.

_________________________________________ / ____________________________
Signature                                               Date

_________________________________________ / ____________________________
Signature of Witness                                    Date
Appendix L

DSM-III-R Diagnostic Checklists:
Bulimia Nervosa
Major Depression
Dysthymia
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These consist of pages:

160-162
VITA

Name: RITA C. PRATHER

ACADEMIC HISTORY

1989  Ph.D.  Louisiana State University
      Baton Rouge, Louisiana
      Major: Clinical Psychology
      Specialty Area: Behavioral Medicine
      Minor: Behavioral Neurology

      Dissertation title:
      The Affective Variant Hypothesis: How is
      Bulimia Nervosa Related to Depression?

1987-88  Internship:
        University of Mississippi Medical Center
        Department of Psychiatry & Human Behavior
        Jackson, Mississippi

1985  M.A.  Louisiana State University
      Baton Rouge, Louisiana
      Major: Clinical Psychology

      Thesis title:
      Differential Diagnosis of Eating
      Disorders: Purgers, Bingers, Obese,
      and Normals.

1983  B.A.  University of Central Florida
      Orlando, Florida
      Major: Psychology
      Honors: Summa Cum Laude

HONORS

1986  Outstanding Young Women of America
1983  Summa Cum Laude, University of Central Florida
1983  Who's Who Among Students in American Universities
1982  PHI KAPPA PHI National Honor Society
1981  PSI CHI National Honor Society
1981  DELTA TAU KAPPA International Honor Society
1981  OMICRON DELTA KAPPA National Honor Society
TEACHING ACTIVITIES

1988-1989  Assistant Professor. Appointed to Graduate faculty September, 1988
Texas A & M University
Department of Psychology

University of Mississippi Medical Center
Department of Psychiatry & Human Behavior

Department of Psychology
Louisiana State University

1983-1984  Teaching Assistant.
Department of Psychology
Louisiana State University

RESEARCH ACTIVITIES

1987-1988  Research Associate
Eating Disorders Unit
University of Mississippi Medical Center

Research Associate
UMC Headache Clinic
University of Mississippi Medical Center

Research Associate
Behavioral Cardiology
Veterans Administration Medical Center
Jackson, MS

1983-1987 Research Associate
Eating Disorders Clinic
Psychological Services Center
Louisiana State University

CLINICAL EXPERIENCE

University of Mississippi Medical Center
Department of Psychiatry & Human Behavior
Jackson, MS
1983-1987 **Clinical Psychology Trainee.**
Eating Disorders Clinic
Psychological Services Center
Louisiana State University, Baton Rouge, LA
Supervisor: D. A. Williamson, Ph.D.

1985-1986 **Psychological Consultant.**
Center on Problem Eating
Baton Rouge General Medical Center
Baton Rouge, LA

1985-1987 **Clinical Psychology Trainee.**
Neuropsychological Clinic
Psychological Services Center
Louisiana State University
Supervisor: Wm. D. Gouvier, Ph.D.

1985-1986 **Clinical Psychology Trainee.**
Adult Services Clinic
Psychological Services Center
Louisiana State University
Supervisor: Wm. D. Gouvier, Ph.D.

1984-1985 **Clinical Psychology Trainee.**
Child Services Clinic
Psychological Services Center
Louisiana State University
Supervisor: June Tuma, Ph.D.

**ADMINISTRATIVE/SERVICE ACTIVITIES**

1988-1989 Texas A & M University
Clinical Search Committee
Library Committee
TAMU MENTORS Program
In Search of a Speaker Program

1984-1985 **Graduate Assistant.**
Psychological Services Center
Louisiana State University
Supervisor: Donald A. Williamson, Ph.D.

1984-1985 **Graduate Student Representative**
Clinical Training Committee
Department of Psychology
Louisiana State University

1981-1983 **Peer Advisor**
Department of Psychology
University of Central Florida
Orlando, FL
1981-1982  Charter President
PSI CHI National Honor Society
University of Central Florida

OTHER PROFESSIONAL ACTIVITIES

1988  Eating disorders and psychopathology. Presented at University of Mississippi Medical Center Psychiatry Grand Rounds February 19.

1988  Body Image: Impossible dreams and unrealistic means. Presented at the University of Mississippi Medical Center Community Education meeting March 17.

1988  Overview of Eating Disorders. Presented at the Mississippi State University annual Women's Week March 29.

PROFESSIONAL MEMBERSHIPS

1981-Present  American Psychological Association
1986-Present  Division 12, American Psychological Association
1988-Present  Division 38, American Psychological Association
1982-Present  Association for Advancement of Behavior Therapy
1984-Present  Southeastern Psychological Association
1985-Present  The Society of Behavioral Medicine

GRANTS

Coauthor with D. B. Penzien, Ph.D.
Behavioral versus pharmacological treatment of recurrent migraine headache.
Funded 1/88 by Clinical Research Center, University of Mississippi Medical Center. $2500.00

Coauthor with D. B. Penzien, Ph.D.
Menstrual versus non-menstrual migraineurs: Fluctuations of reproductive hormones and physical symptoms across the menstrual cycle.
Funded 1/88 by Clinical Research Center, University of Mississippi Medical Center. $3000.00


PRESENTATIONS AND SYMPOSIA


Prather, R. C. (December, 1982). \textit{The importance of thorough assessment as demonstrated in the elimination of insomnia: A case study.} Paper presented at the annual meeting of the Florida Association of Behavioral Analysis, Tampa, FL.

DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Rita C. Prather

Major Field: Psychology

Title of Dissertation: The Affective Variant Hypothesis: How Is Bulimia Nervosa Related to Depression?

Approved:

[Signatures]

Major Professor and Chairman
Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination: Sept. 26, 1989