Age of Onset and Duration Variables: Relationship to Primary and Secondary Psychopathology of Bulimia Nervosa.

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Age of onset and duration variables: Relationship to primary and secondary psychopathology of bulimia nervosa

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The Louisiana State University and Agricultural and Mechanical Col., 1990
AGE OF ONSET AND DURATION VARIABLES:
RELATIONSHIP TO PRIMARY AND SECONDARY
PSYCHOPATHOLOGY OF BULIMIA NERVOSA

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
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in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in
The Department of Psychology

by
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For me and Darrell, and for my Daddy
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ABSTRACT

Over the past decade there has been growing concern about the syndrome of bulimia nervosa. Current theoretical models of the etiology and symptomatology of bulimia predict that the psychopathology of the eating disorder worsens over time. The specific purposes of this study was to examine the effect of onset age and duration of the disorder on the primary and secondary psychopathology of bulimia nervosa at the time of presentation for treatment. The results suggest that binging and purging worsen with increasing duration and older age of onset, accompanied by increased weight and greater distortion of body size estimation. Furthermore, subjects appeared to become increasingly inactive and socially isolated with longer duration and older age of onset. These conclusions were discussed in terms of current theoretical models of bulimia nervosa and the need for integration of developmental variables into future research regarding the etiology of eating disorders.
CURRENT CONCEPTUALIZATIONS

Etiology

In an attempt to define the origins of bulimia nervosa, diverse etiological theories of this eating disorder have been proposed. These theories postulate causative factors ranging from sociocultural influences and dysfunctional interpersonal relationships to affective and biological abnormalities. Each of these will be described briefly, followed by a discussion of multi-determined models of bulimia nervosa which combine these proposed etiological factors.

Sociocultural influences have often been considered a determining factor in the etiology of eating disorders, including bulimia nervosa (Boskind-White & White, 1983; Bruch, 1973; Kaffman & Sadeh, 1989). The environmental emphasis on thinness observed in societies and social classes with abundant food supply is believed to contribute to pervasive concern with body size. This preoccupation is proposed to increase the vulnerability of some young women to engage in restrictive dieting. Recent research supports hypotheses that restrained eating is a precursor to more extreme forms of weight control (Duchmann, Williamson, & Stricker, 1989; Shisslak, Crago, Neal, & Swain, 1987). The development of bulimia nervosa as a consequence of restrained eating will be discussed in a following section. Another aspect of the social environment implicated in the
development of bulimia nervosa is interpersonal functioning, particularly in family relationships (Johnson & Pure, 1986; Strober & Humphrey, 1987). Dysfunctional eating patterns develop as solutions or adaptations to conflicts with control, autonomy, and dependency issues in these relationships.

In addition to sociological explanations, biological factors accounting for eating disturbances have also been examined. Reviews of this literature (Kaplan & Woodside, 1987; Witcher, 1986) detail the range of central neurophysiological systems and peripheral hormonal mechanisms which include possible sources of appetite and satiety dysfunction.

One biological factor which has received considerable attention as a determinant of bulimia nervosa is history of major affective disorder in the eating-disordered individuals or their family members (Hudson, Pope, Jonas, & Yurgelun - Todd, 1983; Pope & Hudson, 1985). Due to the prevalence of depressive symptoms in bulimia nervosa subjects, as well as their frequent improvement in binging and purging in response to antidepressant medication (Stewart, Walsh, Wright, Roose, & Glassman, 1984), the eating disorder symptoms have been considered a variant of the same biological abnormalities responsible for endogenous depressive disorders. Although evidence has not been conclusive on this position, the association of
depression and bulimia nervosa is well documented in the literature. Depression, as a secondary symptom to the eating disorder, will be reviewed in a following section.

A different line of research (Leitenberg, Rosen, Gross, Nudelman, & Vara, 1988; Rosen & Leitenberg, 1985) also implicates affective disturbance in the development of bulimia nervosa. Parallels are drawn between the central role of anxiety in obsessive-compulsive disorder and in bulimia nervosa. Fear of weight gain motivates purging and thus perpetuates binging. Although success has been demonstrated for anxiety deconditioning treatment pursued by this line of research, it has also become increasingly clear that anxiety is not singularly responsible for bulimia nervosa. Rather, it is more likely that these various causative agents interact with one another to produce the syndrome of bulimia nervosa.

Out of this proliferation of proposed etiological factors came several integrative models of bulimia nervosa’s development. One of these was from Mizes (1985) who presented a complex model of psychological factors in which familial and societal factors are seen as the sources of irrational beliefs that produce neurotic and dysfunctional personality styles. Deficits in self-management in turn give rise to chronic states of anxiety and depression which serve as binge precipitants. As will be seen in a following discussion of the primary symptoms
of bulimia nervosa, the binge-purge cycle is self-perpetuating, once established.

The multi-determined nature of eating disorders was also recognized by Garner, Garfinkel, and Irvine (1986) in their integrated, sequenced approach to treatment. They emphasize the importance of addressing social, familial, biological, and psychological factors in treatment of bulimia nervosa. It is recommended that appropriate sequencing of treatment take into consideration such factors as age of the patient and duration of the disorder. These two variables have received relatively little attention in the bulimia nervosa literature, and will be proposed in this research as variables of interest.

Most recently a comprehensive, yet parsimonious, model of etiological as well as maintaining factors operating in bulimia nervosa has been presented (Williamson, Davis, Duchmann, McKenzie, & Watkins, 1990). The etiological portion of this model portrays the sociocultural emphasis upon thinness as a general, or "umbrella", variable which encourages preoccupation with body size that leads to dieting behavior. More specific predisposing factors increase an individual's susceptibility to developing an abnormal eating pattern. Predisposing factors include personality dysfunction, familial problems, and affective disturbance along with nutritional and genetic influences. The social climate in combination with these factors leads
to the development of core bulimia nervosa symptoms which interact in a bi-directional manner with secondary psychopathology. This model has been the focus of ongoing research, particularly with regard to the primary (Duchmann, Williamson, & Stricker, 1989; Williamson, Goreczny, Davis, Ruggiero, & McKenzie, 1988; Williamson, Kelley, Davis, Ruggiero, & Vietia, 1985) and secondary (Prather & Williamson, 1988; Williamson, Prather, Goreczny, Davis, & McKenzie, 1989; Williamson, Prather, Upton, Davis, Ruggiero, & Van Buren, 1987) symptomatology and provides a clear and coherent picture of the fundamental variables implicated in the etiology and maintenance of bulimia nervosa. These aspects of the disorder will be examined further in the next section.

In summary, bulimia nervosa has come to be conceptualized as a disorder with multiple etiologies. It has been proposed that this view of etiology allows for less fragmented assessment approaches (Foreyt & McGavin, 1988) and more comprehensive and individualized treatment efforts (Garner, Garfinkel, & Irvine, 1986; Williamson, Davis, Duchmann, McKenzie, & Watkins, 1990). The host of etiological variables seen in bulimia nervosa lead to a common symptom picture to be discussed next.

**Primary Symptomatology**

To distinguish among the multitude of symptoms
manifested in syndrome of bulimia nervosa, the term primary psychopathology was coined (Williamson, Kelley, Davis, Ruggiero, & Vietia, 1985) to refer to the principal components of the eating disorder (e.g., binging, purging, etc.). Other associated features of the syndrome (e.g., depression, obsessive-compulsiveness, etc.) are considered secondary psychopathology (Williamson, Davis, Duchman, McKenzie, & Watkins, 1990).

The etiological conceptualization offered by this Williamson et al. model is illustrated in Figure 1. The consequence of prolonged, severe dieting (e.g., fasting) is a state of starvation or semistarvation. In other words, essential nutrients and energy are depleted by limited nutritional intake. The well documented starvation related endocrine, hematological, and gastrointestinal complications of bulimia nervosa are offered as support for this idea (Foreyt & McGavin, 1988; Kaplan & Woodside, 1987; Mizes, 1985). Thus, restrained eating is likely to produce physiological deprivation and accompanying sensations of hunger.

In this state, a natural behavioral response is to break dietary restraint by eating (see Figure 1). Garfinkel, Mouldofsky, and Garner (1980) reported bulimics' onset of binging followed approximately one and a half years of dieting. It was first suggested by Slade (1982) that this biologically deprived state created by starvation
CONCEPTUAL MODEL OF BULIMIA NERVOSA

FIGURE 1
in anorexia nervosa serves as the impetus for subsequent binge eating. Further evidence of this sequence is garnered from a review of the literature (Shisslak, Crago, Neal, & Swain, 1987). At least five studies are cited which report an extended period of severe dieting prior to onset of binging and purging in their subjects.

A contributory factor in the onset of binging behavior is cognitive distortions that are frequently present in bulimic's thinking. With regard to food, bulimics typically formulate rigid rules about what are safe or acceptable foods to eat. Breaking the rules by ingesting even a small amount of the food is likely to result in uncontrolled consumption. Breaking of dietary restraint can be likened to Jellinek's (1969) view that abstinence violation by alcoholics leads to loss-of-control drinking. Such counter-regulated eating behavior has been observed experimentally in bulimics and dieters (Duchmann, Williamson, & Stricker, 1989; Polivy & Herman, 1985).

Once binging has occurred, a negative affective state is created in the bulimic. This state consists largely of anxiety about weight gain. The emotional response a bulimic experiences to overeating, or even to normal eating, is highly related to fears of fatness and excessive concern about body size. This relationship is depicted in Figure 1.
Psychophysiological evidence of anxiety in response to eating in bulimia nervosa has been presented by Williamson, Goreczny, Davis, Ruggiero, and McKenzie (1988). Bulimics who ate test meals and were not allowed to purge responded with modest levels of physiological arousal, suggestive of anxiety. Also, Leitenberg, Gross, Peterson, and Rosen (1984) noted increases in self-rated anxiety and the urge to vomit during and following eating. There is also behavioral evidence of anxiety in response to eating. Subjects ate less when they knew vomiting would be prevented (Rosen, Leitenberg, Gross, & Wilmut, 1985) suggesting that they were avoiding eating in order to reduce anxiety. Thus, overeating appears to produce anxiety in bulimia nervosa.

The model then predicts decreased anxiety following purging. Self-monitored anxiety was found to decrease after purging (Johnson & Larson, 1982). Purging has frequently been proposed to serve an anxiety reduction role (Rosen & Leitenberg, 1985; Williamson, Prather, Goreczny, Davis, & McKenzie, 1989). In this role, anxiety reduction increases the likelihood of future purging through negative reinforcement. However, purging also perpetuates the binge-purge cycle in another important way. It creates continued elimination of energy that exacerbates the original physiological deprivation brought on by limited intake. Jarrell (1985) reported expected increases in
bulimics' blood insulin and glucose levels after eating, followed by the dramatic decreases in glucose and insulin levels after purging. Subsequent eating led to recovered levels of glucose and insulin. Additionally, in order to compensate for their initial caloric deficit bulimics who purged ate larger quantities of food than did normal controls.

Furthermore, the deleterious effects of purging are not limited to nutrient depletion. A more long-term consequence of caloric deprivation is starvation-induced lowered resting metabolic rate (Apfelbaum, 1978; Keys, Brozek, Hanschel, Mickelson, & Taylor, 1950), as also shown in Figure 1. In bulimia nervosa, subjects who show greater eating disorder pathology as measured by the BULIT (Smith & Thelen, 1984) and purge with greater frequency have been shown to have lower resting metabolic rates than have less severe bulimics or normal control subjects (Bennet, Williamson, & Powers, 1989). Lowered metabolic rate refers to a decrease in the rate at which the body burns energy, which functions to increase the probability of weight gain. This metabolic effect in turn, impacts directly on the bulimics fear of weight gain which motivates further purging.

Thus once established, the cycle of binging and purging is usually self-perpetuating. The initial dietary restraint sets the occasion for binge eating which creates
a state of anxiety about weight gain. Subsequent purging alleviates anxiety, thereby negatively reinforcing the purgative habit for quieting fears of weight gain. Behavioral learning theory would predict that more chronic or frequent occurrence of this cycle would lead to worsening of the eating disorder over time.

While these are the components necessary for a diagnosis of bulimia nervosa, it may be that intensity or saliency of the features vary as a function of individual bulimics age at onset, or the duration of their disorder at the time of presentation for treatment. As noted previously these are two variables that have not been the focus of investigation. Current research suggests that primary psychopathology is influenced by, and influences, the severity of secondary psychopathology (Williamson et al., 1987). These symptoms will be examined next.

Secondary Symptomatology

In addition to the primary features that are diagnostic of bulimia nervosa, other psychopathology has also been found to be consistently associated with bulimia. As mentioned previously, these features are conceptualized as secondary to the core cycle of binging and purging (Williamson, Davis, Duchmann, McKenzie, & Watkins, 1990; Williamson, Kelley, Davis, Ruggiero, & Blouin, 1985). They lie outside the core syndrome and interact with it by means of bidirectional influences (i.e., the secondary problems
affect primary symptoms and vice versa). In terms of development, the model predicts that if primary psychopathology worsens then secondary psychopathology also worsens. While the primary symptoms are common to all bulimics, the presence of secondary symptoms vary among individual bulimics. Examples of secondary psychopathology include depression, anxiety and obsessive-compulsive habits, interpersonal and family problems, and personality disorders. Additionally, a unique type of pathology observed in bulimia and other eating disorders is a disturbance of body image. This construct is assumed to be a primary underlying disturbance which is highly related to fear of weight gain and overconcern with body size. This disturbance is most evident in the primary symptomatology at the point where anxiety is experienced prior to purgative behavior. These aspects of the bulimic syndrome will be addressed in the following discussion.

**Depression.** Prior to the time that bulimia nervosa was formally recognized as a distinct eating disorder its occurrence in anorexic patients had been noted. Several researchers (Beumont, George, & Smart, 1976; Casper, Eckert, Halmi, Goldberg, & Davis, 1980; Strober, 1981) observed that anorexics who incorporated binging and purging into their disordered eating habits had a greater disposition to depression than did anorexics who strictly fasted.
The bulimia nervosa literature frequently contains psychometric studies linking bulimia nervosa to depression. Several early studies using the MMPI (Hatsukami, Owen, Pyle, & Mitchell, 1982; Pyle, Mitchell, & Eckert, 1981) and Hopkins Symptom Checklist (Johnson, Stuckey, Lewis, & Schwartz, 1982) obtained data indicative of clinical depression. In a comparison of the nature of depressive symptoms reported by bulimics and major depressive disorder patients (Cooper & Fairburn, 1986), it was found that response to the Montgomery and Asberg Depression Rating Scale differed significantly for the two groups. In contrast to the depressed group bulimics had less "apparent sadness," but experienced greater "obsessional ideas and ruminations."

Several investigations conducted by Williamson and colleagues (Prather & Williamson, 1988; Williamson, Kelley, Davis, Ruggiero, & Blouin, 1985; Williamson, Prather, Upton, Davis, Ruggiero, & Van Buren, 1987) provide further evidence of the relationship between bulimia and depression. A greater proportion of bulimics than of other eating disordered groups or controls obtained clinical elevations on MMPI scale 2 (depression) and the Beck Depression Inventory. When compared to normal obese and control groups, the only MMPI scale whose mean was clinically elevated for bulimics was the depression scale. In a later study, frequency of purging, a measure of eating
disorder severity, was found to positively correlate with Beck Depression Inventory scores (Williamson et al., 1987). It has been suggested, but not empirically demonstrated, that bulimia worsens over time. Given the above correlation, it might also be expected that depression and other secondary psychopathology follow a similar course of worsening. To date empirical research has not addressed this question.

Depression, as the most commonly identified secondary psychological feature of bulimia nervosa (Prather & Williamson, 1988), has led some researchers to view the disordered eating as a manifestation of a more fundamental, underlying affective disorder (Hudson, Pope, & Jonas, 1985; Pope & Hudson, 1985). This affective variant hypothesis has been the focus of reviews and debate in recent years (Hinz & Williamson, 1987; Mizes, 1985; Swift, Andrews, & Barklage, 1986; Wilson & Lindholm, 1987). This position has found empirical support (Stunkard, 1985), though most reviews agree it is premature to conclude that binging and purging are a form of affective disorder. To do so minimizes the unique (primary) symptoms of bulimia nervosa. As mentioned previously, one unexplored aspect of the relationship is depression’s course with regard to the primary symptom’s course and chronicity. The relationship of primary symptom’s course to other types of secondary
psychopathology, to be examined next, has received equally little attention.

**Obsessive-Compulsiveness.** In addition to depression, other neurotic psychopathology has been found to be associated with bulimia nervosa. From clinical observations, Loro (1984) noted the ritualism with which some bulimics plan and carry out binging and purging. Because of the role of anxiety in binging and purging, bulimia nervosa has sometimes been likened to an obsessive-compulsive disorder (Rosen & Leitenberg, 1985; Williamson, Prather, Goreczny, Davis, & McKenzie, 1989). Exposure to the feared stimulus (i.e., food and calories) by breaking dietary restraint and engaging in subsequent binging leads to anxiety and obsessional thinking about weight gain. Purging then provides an "escape" from the feared stimulus and "protection" against weight gain. These behaviors are analogous to compulsive hand washing which provides escape from germs and protection from disease, thus temporarily alleviating anxiety. These aspects of the bulimia syndrome illustrate how the propensity for anxiety and obsessive-compulsive habits play a role as secondary psychopathology components.

Close examination of Cooper and Fairburn's (1986) comparison of bulimia nervosa and depressed patients further clarifies the relationship of depression and anxiety symptoms to bulimic symptoms. Although the two
groups appeared identical in overall severity of depression and other psychiatric symptoms, they differed significantly on items comprising total severity. Bulimics scored higher on items reflective of "inner tension" and "pessimistic thoughts" and more frequently displayed syndromes of "obsessional neurosis", "situational anxiety", "hypomania", and "special feature of depression." The symptom which contributed most to discontinuity was the greater frequency of "obsessional ideas and ruminations" in the bulimic group. Thus, bulimics reported an experience of depression which differed from that of depressed patients by inclusion of a salient anxiety component. Currently, how anxiety fluctuates with changes in eating disorder symptoms (over time) is unknown.

**Interpersonal problems.** Disordered interpersonal functioning has also been found to be associated with bulimia nervosa. Consistent with findings regarding the relationship between severity of eating disorder symptoms and depression, social maladjustment scores have also been positively correlated with binging and purging frequencies. In bulimic graduate students (Herzog, Norman, Rigotti, & Pepose, 1986) and medical students (Herzog, Pepose, Norman, & Rigotti, 1985), significantly poorer adjustment scores were obtained in comparison to a non-bulimic control group in social, vocational, and family role performance. The bulimic students displayed maladjustment similar to a
sample of clinic bulimics. These findings are consistent with earlier studies (Abraham & Beumont, 1982; Clement & Hawkins, 1980; Katzman & Wolchik, 1982; Loro, 1980) that produced convergent evidence of deficits in interpersonal coping skills from maladjustment in several related areas of social role functioning (i.e., assertiveness).

Attempts to measure specific social skills, such as assertiveness deficits, have not produced clear findings. Direct measurements of vocational performance have not appeared in the literature, and studies of family interactional patterns have historically focused on anorexia nervosa rather bulimia nervosa as it is now defined.

Recently, however, studies have begun to examine bulimic's family interaction patterns, including marital relationships. Family-of-origin studies have found that there is greater conflict avoidance, rigidity, and repression in families with an eating disordered member than in normal control families (Kog & Vandereycken, 1989; Stern, Dixon, Jones, Lake, Nemzer, & Sansone, 1989). Unfortunately both anorexic and bulimic subjects were studied together, so that patterns of normal weight bulimics alone cannot be clearly discerned. Strober and Humphrey's (1987) review of familial contributions to anorexia and bulimia nervosa points up differences between bulimic and non-bulimic subtypes of eating disorders.
Tendencies for greater hostility, lack of parental nurturance, and alcoholism generally occur more often in bulimic families. Recent findings by Head and Williamson (in press) however, indicate that characteristics of family dysfunction are more highly associated with secondary rather than primary symptomatology observed in bulimia nervosa.

In a study of the spouses of anorexics and bulimics, Van den Broucke and Vandereycken (1989) found that these spouses display and more psychiatric symptoms than did spouses of normal control subjects and reported a significant degree of maladjustment in their marriage. Marital relationships were also the focus of a study by Van Buren and Williamson (1988) in which bulimic subjects and their spouses were compared to distressed and normal couples. Bulimics were similar to maritally distressed females in degree of marital dissatisfaction, irrational relationship beliefs, decreased problem solving, and withdrawal from conflict.

While these investigations of specific areas of interpersonal dysfunction are relatively new, previous global measurement studies were indicative of problems in overall personality functioning. Studies of bulimic responses to the MMPI and similar self-report inventories provide a summary of dysfunctional personality patterns. In a review of symptomatology of bulimia nervosa, Mizes
(1985) noted a collection of evidence from MMPI studies conducted to that time consistent with significant personality disturbance. For example, codetypes suggestive of social withdrawal, argumentativeness, hostility, passive-aggressiveness, and manipulativeness were frequently found. Subsequent controlled studies have yielded consistent findings with regard to MMPI profiles (Prather & Williamson, 1988; Scott & Baroffio, 1986; Williamson, Kelley, Davis, Ruggiero, & Blouin, 1985). In contrast to normals, bulimia nervosa subjects produced codetypes indicative of manipulative, immature individuals who may withdraw or struggle for control in relationships.

Additionally, signs of personal maladjustment can be seen in SCL-90 subscale elevations found in some of these same investigations. Of particular relevance here are elevations that appeared on Interpersonal Sensitivity, Paranoia, and Psychoticism subscales. This pattern suggests that bulimics are fragile, cautious, mistrustful, and disengaged in relationships. There is also evidence that more severe bulimics (i.e., more frequent purgers) show a greater degree of maladjustment when compared to normals than do less severe bulimics. This finding may be related to duration, or chronicity, of the disorder. In terms of behavioral learning theory, a behavior which is reinforced and repeated becomes strengthened and more likely to occur with increasing frequency over time.
As noted for other aspects of secondary psychopathology, it is not known how chronicity of the eating disorder or age at onset are related to the severity of interpersonal dysfunction symptoms. In keeping with the guiding model of this paper this area of secondary symptomatology, in addition to depression, anxiety, and obsessive-compulsiveness, can be seen as interacting with the primary symptoms of the bulimic syndrome in a bidirectional manner. The primary symptoms exacerbate secondary ones, and secondary symptoms potentiate primary ones. The secondary symptoms of bulimia nervosa are general, or universal, types of psychopathology also seen in individuals without eating disorders. A unique type of secondary pathology however, can be seen in body image disturbances. In the Williamson et. al (1990) model, body image disturbances lie outside of the core, or primary, symptom picture of bulimia nervosa and impact on it unidirectionally. As an area of secondary psychopathology that is exclusive to eating disorders, body image issues will be reviewed next.

Body image. One of the earliest definitions of body image came from Schilder (1935) who said it is the "picture of our own body which we form in our mind, that is, the way in which our body appears to ourselves." Negative evaluation of one's body by anorexic patients was a phenomenon first clinically observed by Bruch (1962).
Since gaining increased attention as a component of eating disorders, body image has come to be conceptualized as more complex than simple perception of one's body features. An affective or attitudinal facet of body image has also been identified in anorexia nervosa (Garner & Garfinkel, 1981) and bulimia nervosa (Ruff & Barrios, 1986). These two aspects of body image have been termed, respectively, body size distortion and body dissatisfaction. Whereas size distortion concerns inaccuracies in estimating physical dimensions, body dissatisfaction involves feelings about the body.

Corresponding to the rise in eating disorders with associated body image issues is the development of techniques to measure body image disturbance. Of the two facets of body image discussed, size estimation has received the most attention in assessment. Three basic procedures have been employed in size estimation. Distorting image techniques (Freeman, Thomas, Solyom, & Miles, 1983; Glucksman & Hirsch, 1969; Traub & Orbach, 1964) and silhouette selection (Counts & Adams, 1985; Williamson, Kelley, Davis, Ruggiero, & Blouin, 1985) are used as measures of whole body distortion, while body part size estimation (Ruff & Barrios, 1986; Slade & Russell, 1973) requires the individual to judge the width of several body areas. Review of these various techniques indicates that silhouette selection is the preferred method of
assessment for bulimics given its greater availability, ease of administration, and cost effectiveness.

The Body Image Assessment procedure (Williamson et al., 1989) has been shown to be a reliable and valid silhouette selection technique for bulimia nervosa. Both current and ideal size estimates are obtained, from which body image distortion and preference for thinness are derived. In the conceptual framework of Williamson et al. (1989), the elevation of current body-size scores from normals represents body image distortion, and the depression of ideal body size scores from normals represents strong preference for thinness. The discrepancy between these two scores can be thought of as a measure of body size dissatisfaction. A recent construct validity study (Williamson, Watkins, & Schlundt, 1990) has supported this conceptualization of body image.

From the anorexia nervosa literature, findings emerge of a relationship between body image disturbance and other variables. Greater body image distortion has been related to the presence of vomiting and higher chance of relapse (Button, Fransella, & Slade, 1977), poorer treatment outcome (Garfinkel, Moldofsky, & Garner, 1977) and higher degree of neuroticism (Garner, Garfinkel, Stancer, & Moldofsky, 1976). So it may be that disordered body image is a fluctuating symptom that worsens during acute stages of anorexia and bulimia.
This pattern is consistent with findings in the bulimia nervosa literature that body image disturbances are associated with indicants of greater pathology in other areas. More disordered body image has been associated with greater chance of relapse (Freeman, Beach, Davis, & Solyom, 1985) and less progress in therapy (Norris, 1984). Additionally, greater size estimation has been associated with a previous history of anorexia nervosa (Freeman, Thomas, Solyom, & Koopman, 1985). However, other studies have found less overestimation in samples of bulimics with a history of anorexia nervosa (Birtchell, Lacey, & Harte, 1985; Thompson, Berland, Linton, & Weinsier, 1986).

There is agreement that much inconsistency exists in body image literature (Cash & Brown, 1987; McCrea, Summerfield, & Rosen, 1982). One explanation is that variability results from differing techniques used for measuring body image disturbances. Another possibility is that size estimation is confounded by the subject's actual body size. Ben-Tovim, Whitehead, and Crisp (1979) reported an inverse relationship between overestimation and actual body width in anorexics, while Birtchell, Lacey, and Harte (1985) found a positive relationship between overestimation and weight index in bulimics. A recent study by Williamson, Davis, Bennet, Goreczny, and Gleaves (1989) found weight to be positively correlated with estimation of current body size which is conceptually related to body
image distortion, and to ideal body size which is conceptually related to drive for thinness. Findings such as these highlight the importance of considering body size in the interpretation of size estimation scores, and led Williamson et al. (1989) to develop standardized scores for their Body Image Assessment procedure.

Recent findings that bulimics detect fear related words (i.e., FAT) more often than neutral words on a dichotic listening task (Schotte, McNally, & Turner, 1990) offer a potential explanation for the worsening of body image disturbance over time. These findings show that bulimics exhibit "enhanced perceptual sensitivity and physiological reactivity" (pg. 113) to stimuli relevant to their body image concerns. This information processing bias for cues that enhance their fear may lead them to increasingly attend to such cues in the environment. Thus, it might be speculated that environmental cues which elicit overconcern with body size become more prevalent over time, just as "safe" and "germ-free" stimuli become less frequent with worsening of agoraphobia and obsessive-compulsive disorder. Williamson et al.'s (1989) standardized Body Image Assessment procedure was used to measure body image disturbance in this investigation. Scores from this measure were related to the duration of binging and purging and to their age at onset of bulimic symptoms. As noted
for other secondary psychopathology variables, this relationship has not been previously investigated.

Summary. Secondary psychopathology symptoms, such as depression, obsessive-compulsiveness, interpersonal problems, and body image disturbance, have been found to accompany the primary symptoms of bulimia nervosa. The relationship between primary and secondary symptoms posited by the guiding model (Williamson et al., 1985) is that they affect one another bidirectionally. Worsening of both primary and secondary symptoms is a pattern suggested by the collection of data, but not directly investigated thus far.

A decade ago it was speculated that secondary psychopathology such as personality disorders, psychiatric illness, social maladjustment, and body image distortion are factors which predispose to eating disorder chronicity (Strober, Goldenberg, Green, & Saxon, 1979). However, there has not been systematic research in eating disorders aimed at identifying which factors are influential in the course of symptom development. As a guide to such research, the next section will explore variables which have been found to be influential in the development of psychopathology other than eating disorders.
DEVELOPMENTAL RESEARCH

Affective disorders, anxiety disorders, and alcoholism are three psychiatric disorders which have been extensively studied from a developmental perspective. Review of this research may serve as a guide for identifying variables associated with symptom development of bulimia nervosa over the course of time. Also the methodology used for studying such variables will be reviewed in order to evaluate their utility.

Affective Disorders

In a study of affective disorders, Hirschfield and Cross (1982) found that age of onset most significantly predicted severity of depression. Comparison of bipolar and unipolar depressed patients at time of presentation for treatment showed differences in average age of onset for the two disorders. Bipolar disorder had an earlier average age of onset, in the late twenties. Unipolar depression, in contrast averaged onset in mid to late thirties. Thus, onset age was related to the nature of the affective disorder observed. In this instance the more severe of the affective disorder subtypes, bipolar disorder, was associated with a younger age of onset. No mention was made of the duration of symptoms from onset to presentation for treatment, which might be expected to be shorter for the earlier onset subtype if its symptoms were experienced
as more severe and disruptive. These findings regarding severity and age of onset are in apparent opposition to those described for anxiety disturbances.

**Anxiety Disorders**

In a comparison of different types of phobias, Ost (1987) found that age of onset was related to type of phobia. There was a trend for younger to older age of onset to be associated with (in order) animal, dental, claustrophobic, social, and agoraphobic fears. Thus, simple or less severe phobias had an earlier age of onset than complex phobias that were more severe in terms of symptoms and disability. In addition to age of onset, duration of symptoms was also related to the type of phobia presented. Shorter to longer duration of symptoms was associated with (in order) agoraphobic, claustrophobic, social, animal, and dental fears. Thus, there was an inverse relationship between age of onset and duration. Phobias with a younger age of onset were associated with longer duration at time of presentation for treatment, and older age at onset phobias was associated with shorter durations before presenting for treatment.

This pattern of development and presentation makes conceptual sense considering the level of cognition involved and degree of lifestyle disruption present with each type of phobia. The progression from simple to
complex phobias requires increasingly sophisticated cognitive processes, as would occur with increasing age. Likewise, the more complex the phobia the greater the adjustments in daily functioning required to accommodate them, making prolonged tolerance of symptoms unlikely. To generalize to bulimia nervosa, one would predict that increasingly severe symptoms would appear at older ages and would be of shorter duration at the time of presentation for treatment.

These studies in the area of affective and anxiety disorders point to the importance of age of onset and chronicity variables in disorders that appear as secondary to bulimia nervosa. Studies of alcohol abuse have been fruitful in further identifying how these two variables interact.

Alcohol Studies

This line of research has approached investigation of age and chronicity variables by categorizing subjects on the basis of these variables and examining differences that emerge in personality and other functioning. Several such studies have consistently found differences between young and old alcoholics. Abelsohn and Van der Spuy (1978) and Foulds and Hassall (1969) examined psychopathological correlates of age of onset. Early onset of drinking was associated with more neuroticism, anxiety, immaturity,
personality disorder, and social instability. Rosenberg (1969) identified younger, rather than older, alcoholics as being more disturbed. However, Rosenberg's investigation confounded age with length of drinking history.

Later studies improved this basic experimental design. These studies examined subjects as a function of both age and duration of disorder. Kliz and Parsons (1977) used current age and found more cognitive deficits in older alcoholics with no duration effects. Lee and DiClemente (1985) assessed a range of psychological characteristics as a function of age of onset and duration of problem drinking. Early onset alcoholism was found to have more severe primary and secondary symptoms than later onset. Furthermore, age of onset correlated more strongly with secondary psychopathology than did duration of drinking.

Although, duration was not found to be a significant predictor variable in most of the alcoholism research, the Lee and DiClemente study serves as an example of an appropriate and effective design to study these variables. In this investigation age of onset and duration of disorder were arranged in a two-by-two matrix to allow for detection of main and interaction effects. In the next section, it will be proposed that bulimia nervosa research could benefit from examination of the development of eating disorder pathology with this type of experimental design.
Eating Disorders

Much of the information available about the developmental aspects of anorexia and bulimia nervosa is derived from demographic data. Age of onset is a commonly reported characteristic in surveys of the symptoms of anorexia and bulimia nervosa. In anorexia nervosa, age of onset usually ranges from age fourteen to eighteen (Crisp, Hsu, Harding, & Hartshorn, 1980; Garfinkel, Kaplan, Garner, & Darby, 1983; Halmi, Casper, Eckert, Goldberg, & Davis, 1979; Martin, 1983). Early studies related older age of onset (past sixteen years) to poor prognosis and greater psychiatric maladjustment (Halmi, Brodland, & Loney, 1973; Morgan & Russell, 1975). Bulimia nervosa's average age of onset has been reported as about eighteen or nineteen years (Agras & Kirkley, 1986; Fairburn & Cooper, 1982) for community and clinical samples, respectively. When assessed separately, binging reportedly begins at about age seventeen on average, with onset of vomiting following a year later at about eighteen (Mitchell, Hatsukami, Eckert, & Pyle, 1985). Based on these figures Foreyt and McGavin (1988) concluded that anorexia usually occurs in early adolescence while bulimia usually occurs in late adolescence.

Although less frequently reported in the eating disorders literature, duration of symptoms from onset to presentation for treatment has appeared to be an important
variable in other areas of psychopathology. In anorexia nervosa reports of average duration range from eight months (Martin, 1983) to three years (Crisp et al., 1980). Given this variability in duration it might be speculated that other factors, such as age of onset, may influence symptom severity which in turn determines presentation for treatment. This interaction between severity and duration was most clearly illustrated in the affective disorders literature. A recent treatment outcome study for bulimia nervosa (Hsu & Holder, 1986) identified duration as a significant prognostic indicator. This relationship was in the direction of longer duration predictive of poorer outcome at follow-up. A recent treatment outcome study of inpatient and outpatient bulimia nervosa subjects (Williamson, Prather, Bennet, Davis, Watkins, & Grenier, 1989) found increased secondary psychopathology to be associated with poor outcome. Since primary and secondary psychopathology influence one another, as shown in Figure 1, it might be hypothesized that increases in secondary psychopathology associated with longer duration are paralleled by similar increases in primary psychopathology with longer duration. These findings suggest that more severe, entrenched symptoms occur with longer duration, but direct examination of this relationship has not been carried out for bulimia nervosa.
Although age of onset and duration of disorder emerge as important variables in the development of other psychopathology, including some of the secondary psychopathology of bulimia nervosa, little attention has been given to chronicity and age of onset in the bulimia nervosa literature. These variables may partially determine the severity of both bulimic behavior and secondary problems, which may have implications for treatment. Hence, there is a need for empirical investigation of the natural development of bulimia nervosa over time.
STATEMENT OF THE PROBLEM

The current state of bulimia nervosa literature is itself at a developmental stage. With the sequence of symptoms now clearly defined, there is a need for empirical data to describe the development of these symptoms, both primary and secondary, over time. The available evidence suggests that there is a worsening of both primary and secondary aspects of the disorder over time, but there has been no direct empirical demonstration of this developmental process. Research in other areas of psychopathology suggests that two variables most strongly related to symptom progression are age of onset and duration. Furthermore, clarification of the relationship of these variables to the primary and secondary symptoms of the bulimic syndrome may prove important to treatment planning given suggestions in the literature that they are related to poor prognosis. Yet neither age of onset nor duration, as related to bulimic symptoms, have been systematically observed or reported in bulimia nervosa research. It was the goal of this study to investigate whether the primary and secondary symptoms of bulimia nervosa worsen as a function of age of onset and/or duration of the disorder.

One way to approach such a study is using the methodology employed by Lee and DiClemente (1985) to group
subjects into short or long duration and young or old age of onset categories. However, a problem arises with defining age of onset and subsequently determining duration of illness in bulimia nervosa. The difficulty lies in choosing which of the primary symptoms (i.e., dieting, binging, or purging) to use as the starting point of the disorder. Since dieting is a behavior in which many individuals engage, it does not seem sufficiently definitive of bulimia nervosa to warrant its use as the variable from which age of onset and duration are derived. Binging, on the other hand, has been recognized as the cardinal feature of bulimia nervosa by its inclusion in the original DSM-III diagnostic criteria (American Psychiatric Association, 1980). Although purging has recently been added to the diagnostic criteria, pilot data collected for this study showed that binging and purging have a significant moderate correlation ($r = .64, p<.001, N = 23$). Thus, use of only one of these variables can be justified empirically. The use of binging, rather than purging, as the point of onset of the eating disorder is warranted because of its position in the guiding model as the precursor to purging. Therefore, binging was used to determine age of onset and duration for the purposes of this study.

Another concern is whether to include both age of onset and duration as variables of interest due to the
possibility that they measure the same thing. Again, pilot data collected for this study indicated that inclusion of both variables is appropriate. Preliminary analysis showed they are related by a rather low negative correlation ($r = -.31, p < .0001, N = 155$). Thus, they measure different aspects of the development of bulimia nervosa and therefore can be assumed to have potentially different relationships to the primary and secondary symptomatology. In addition to two-way blocking of these variables to observe their effect on the dependent variables, age of onset and duration can also be used as predictor variables regressed against each dependent variable. Dependent variables in this case were scores from two standardized self-report measures of eating pathology (i.e., EAT and BULIT), standardized scores from the Body Image Assessment (BIA) procedure, Beck Depression Inventory (BDI) scores, and MMPI subscale scores. Based on available data the following hypotheses are offered:

1. That the earlier the age of onset the more severe are the primary and secondary psychopathologies,

2. That the longer the duration of symptoms the more severe the primary and secondary psychopathologies,

3. Conversely, that less severe symptomatology emerges as a function of a) later age of onset and b) shorter duration, and
4. That when an interaction effect exists the most severe symptoms are a function of young age of onset \( \times \) long duration.
METHOD

Subjects

Data was drawn from the records of approximately 500 patients assessed for an eating disorder at a university based community psychology clinic, or at a local hospital. Data from 153 subjects who were staffed by a licensed psychologist and who met DSM-IIIR (American Psychiatric Association, 1987) criteria for bulimia nervosa were selected for the study. All subjects signed consent forms (Appendix A) agreeing that their assessment data could be used for research purposes. All subjects were females (three males were excluded due to lack of appropriate body image data). The sample was composed of 149 Caucasian subjects, two Hispanic subjects, one Black subject, and one Oriental subject. One hundred fifteen (75%) were single, 26 (17%) were married, and twelve (8%) were divorced. Average education was fourteen years.

Measures

The primary variables of interest, age of onset and duration, were obtained from subject’s self-report on the Diagnostic Survey for Eating Disorders (DSED) (Appendix B). This self-report inventory was designed by Johnson (1985). It provides extensive information on demographic factors, eating and weight history, and related psycho-social functioning. Current age at the time of assessment and age
of onset of binging were defined as responses to items 23-24 on card 1 and 52-53 on card 4, respectively. These variables were then used to determine duration of symptoms. Average age in this sample was 23 years and the range was thirteen to 46 years. Average age of onset was eighteen years and average duration was five years. Dependent variables were subject’s scores on self-report measures of primary and secondary psychopathology as follows.

Eating Attitudes Test (EAT). (Appendix C). This questionnaire is a 40-item self-report inventory which assesses eating habits and cognitions associated with anorexia nervosa (Garner & Garfinkel, 1979). It was selected to provide a measure of anorectic symptoms (e.g., overconcern with dieting and body size) which are also commonly found in bulimia nervosa (Williamson, Prather, et al., 1987). Average EAT score for the sample was 46, which is about the average found in other studies of bulimia nervosa (Williamson et al., 1990).

Bulimia Test (BULIT). (Appendix D). This questionnaire assesses the primary symptoms of bulimia (Smith & Thelen, 1984) including binging and purging, restrictive eating patterns, and concern with dieting and body size. It has been used in previous studies (e.g., Bennet, Williamson, & Powers, 1989) as a measure of severity of bulimic symptoms. Average BULIT score for the
sample was 120, which is comparable to that found in other studies of bulimia nervosa (Williamson et al., 1990).

**Body Image Assessment (BIA).** (Appendix E). This procedure utilizes nine cards containing female silhouettes which range from thin to obese. Subject's selection of silhouettes for perceived current body size and ideal body size are standardized according to their actual height and weight. These scores then serve as measures of body image distortion and drive for thinness. This procedure has been shown to be reliable and valid for bulimia nervosa (Williamson, Davis, Bennet, Goreczny, & Gleaves, 1989). The average current body size t-score (BIAC) was 60. The average ideal body size t-score (BIAI) was 44.

**Beck Depression Inventory (BDI).** (Appendix F). This 21-item measure assesses severity of depressive symptoms (Beck, 1978). Previous research concerning bulimia nervosa has consistently found depression to be highly associated with bulimia nervosa (Hinz & Williamson, 1987). Average BDI score of this sample was 21, which is comparable to that found in other studies of depression in bulimia nervosa (Williamson et al., 1990).

**Minnesota Multiphasic Personality Inventory (MMPI).** This instrument has been widely used to assess general psychopathology (Dahlstrom, Welsh, & Dahlstrom, 1978). It measures various aspects of psychopathology via ten clinical subscales: Hypochondriasis (Hs), Depression (D),
Hysteria (Hy), Psychopathic deviance (Pd), Masculine-Feminine (Mf), Paranoia (Pa), Psychasthenia (Pt), Schizophrenia (Sc), Mania (Ma), and Social introversion (Si). The validity scale F has been described as an index of the extent and severity of psychopathology (Greene, 1980). The MMPI was used in this investigation to evaluate the relationship of general psychopathology to the age of onset and duration of eating disordered symptoms. Average t-scores in this sample were: 63 (F), 63 (Hs), 72 (D), 66 (Hy), 73 (Pd), 45 (Mf), 67 (Pa), 72 (Pt), 72 (Sc), 61 (Ma), and 61 (Si). This mean MMPI profile is quite similar to that found in other studies of bulimia nervosa which have used the MMPI (Williamson et al., 1990).

Procedures

Subject’s files were examined for assessment data that included the previously described scores. Data were included for analysis from subjects who met DSM-IIIR criteria for bulimia nervosa. One analysis utilized canonical correlation to evaluate the relationship of clusters of dependent variables to the two independent variables. Another set of analyses entered age of onset and duration as continuous variables into regression equations as predictors of each of the criterion, or dependent, variables. Linear and quadratic solutions were used to discern both linear and curvilinear trends.
Further analyses were conducted in which age of onset and duration were transformed into discrete variables by means of a median split. The median for age of onset was seventeen years, and the median for duration was three years. This transformation yielded a two-by-two design of young/old age of onset and short/long duration. Dependent variables were analyzed using MANOVA to describe how age of onset and duration of bulimia nervosa influenced primary and secondary psychopathology. Finally, simple correlation matrices were computed to evaluate the relationship of primary to secondary psychopathology.
RESULTS

The data were examined using four types of statistical analyses: canonical correlation, stepwise multiple regression, multivariate analysis of variance, and simple correlation. The findings from each of these analyses will be described in turn.

Canonical Correlations

Three canonical correlations were performed to evaluate the association between a linear composite of the predictor variables age of onset and duration, and three linear composites of criterion variables. The first set of criterion variables was composed of those measures reflective of primary psychopathology, specifically the Eating Attitudes Test, Bulimia Test, current body size estimates, and ideal body size estimates. The second set of criterion variables was composed of measures reflective of secondary psychopathology such as the Beck Depression Inventory (BDI), one MMPI validity scale (F), and ten MMPI clinical scales (Hs, D, Hy, Pd, Mf, Pa, Pt, Sc, Ma, Si). The third set of criterion variables was a combination of both primary and secondary measures of psychopathology.

Each of the three correlations yielded two canonical functions. In each case, the first canonical function was statistically significant and the second was not. Tables 1-3 summarize the canonical correlations for the three sets.
of criterion variables, including the second (nonsignificant) function which is presented for purposes of comparison.

**Primary psychopathology variables.** The first canonical function yielded a canonical correlation coefficient of .27 ($p < .05$). The pattern of significant canonical loadings indicated that younger age of onset and longer duration were associated with lower EAT scores and higher BULIT and BIAC scores. The canonical root for this function estimated shared variance of 7% between the predictor and criterion composites.

**Secondary psychopathology variables.** The first canonical function for this correlation yielded a coefficient of .52 ($p < .0001$). Canonical loadings may be interpreted as showing that older age of onset and longer duration were associated with lower scores on Hs, D, and Pt, and with higher scores on Hy and Si. The canonical root for this function estimated shared variance of 27% between the two composites.

**Combined psychopathology variables.** The first function for this coefficient yielded a coefficient of .53 ($p < .0001$). The canonical loadings indicated that older age of onset and longer duration were associated with lower scores on Hs and D, and higher scores on Hy and Si. The canonical root estimated shared variance of 28% between the predictor and criterion composites.
### TABLE 1

**CANONICAL CORRELATIONS: PRIMARY PSYCHOPATHOLOGY**

<table>
<thead>
<tr>
<th>Canonical Function</th>
<th>Canonical Correlation</th>
<th>Canonical Root</th>
<th>F</th>
<th>df</th>
<th>Prob.</th>
</tr>
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<tr>
<td>1.</td>
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<td>.07</td>
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<td>2.</td>
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**STANDARDIZED CANONICAL COEFFICIENTS**  
**FOR AGE OF ONSET AND DURATION**

<table>
<thead>
<tr>
<th>Predictor Variables</th>
<th>Canonical Function 1</th>
<th>Canonical Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of Onset</td>
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<td>.74</td>
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<td>Duration</td>
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<td>.94</td>
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**STANDARDIZED CANONICAL COEFFICIENTS**  
**FOR PRIMARY PSYCHOPATHOLOGY VARIABLES**

<table>
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<th>Criterion Variables</th>
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<th>Canonical Function 2</th>
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<tr>
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<td>BULIT</td>
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<td>-.17</td>
</tr>
<tr>
<td>BIAC</td>
<td>.65*</td>
<td>.46</td>
</tr>
<tr>
<td>BIAI</td>
<td>.02</td>
<td>.66</td>
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* > .50 interpreted as a significant contribution
TABLE 2
CANONICAL CORRELATIONS: SECONDARY PSYCHOPATHOLOGY

<table>
<thead>
<tr>
<th>Canonical Function</th>
<th>Canonical Correlation</th>
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<th>df</th>
<th>Prob.</th>
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STANDARDIZED CANONICAL COEFFICIENTS
FOR AGE OF ONSET AND DURATION

<table>
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<th>Predictor Variables</th>
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<th>Canonical Function 2</th>
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</thead>
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<td>Age of Onset</td>
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<td>.69</td>
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<td>Duration</td>
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STANDARDIZED CANONICAL COEFFICIENTS
FOR SECONDARY PSYCHOPATHOLOGY VARIABLES

<table>
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<th>Criterion Variables</th>
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<td>BDI</td>
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<td>F</td>
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</tr>
<tr>
<td>Hs</td>
<td>-.56*</td>
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<tr>
<td>D</td>
<td>-.55*</td>
<td>.42</td>
</tr>
<tr>
<td>Hy</td>
<td>.61*</td>
<td>.00</td>
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<tr>
<td>Pd</td>
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<tr>
<td>Mf</td>
<td>.11</td>
<td>.40</td>
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<tr>
<td>Pa</td>
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<tr>
<td>Pt</td>
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<td>-.51</td>
</tr>
<tr>
<td>Sc</td>
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<td>Ma</td>
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<tr>
<td>Si</td>
<td>1.13*</td>
<td>.34</td>
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* > .50 interpreted as a significant contribution
TABLE 3

CANONICAL CORRELATIONS: COMBINED PSYCHOPATHOLOGY

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<td>.09</td>
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STANDARDIZED CANONICAL COEFFICIENTS
FOR AGE OF ONSET AND DURATION

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<tr>
<th>Predictor Variables</th>
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<th>Canonical Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of Onset</td>
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<tr>
<td>Duration</td>
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<td>.58</td>
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STANDARDIZED CANONICAL COEFFICIENTS
FOR COMBINED PSYCHOPATHOLOGY VARIABLES

<table>
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<th>Criterion Variables</th>
<th>Canonical Function 1</th>
<th>Canonical Function 2</th>
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<tbody>
<tr>
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<td>BULIT</td>
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<td>BIAC</td>
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<td>Hs</td>
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<td>-.31</td>
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<td>Hy</td>
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<tr>
<td>Ma</td>
<td>.22</td>
<td>-.16</td>
</tr>
<tr>
<td>Si</td>
<td>1.10*</td>
<td>-.10</td>
</tr>
</tbody>
</table>

* > .50 interpreted as a significant contribution
Summary. These canonical correlations provided a multivariate analysis of the relationships between the predictor variables age of onset and duration and three sets of criterion variables related to the primary and secondary psychopathology of bulimia nervosa. The results of these analyses suggested that age of onset and duration accounted for more variance in secondary psychopathology (> 25%) than in primary psychopathology (< 10%).

Multiple Regression

Stepwise multiple regression equations were performed for each of the dependent variables, using linear, quadratic, and interaction terms of predictor variables (i.e., age of onset and duration). The results for each dependent variable will be discussed, focusing on those regression models which accounted for ten percent or more of the variance.

Primary psychopathology variables. The regression models predicting scores on the EAT, BULIT, BIAC, and BIAI accounted for only 3-5% of the variance of these dependent variables. It was therefore concluded that the independent variables age of onset and duration did not accurately predict scores on these dependent measures.

Secondary psychopathology variables. The regression models predicting scores on the BDI, MMPI validity scale F, and the first eight MMPI clinical scales (Hs, D, Hy, Pd, Mf, Pa, Pt, Sc) accounted for 0-3% of the variance in these
dependent variables. Therefore, it was concluded that age of onset and duration did not predict these aspects of secondary psychopathology.

However, the models generated for MMPI clinical scales Ma and Si accounted for 11% and 14% of the variance of these scores, respectively. Tables 4 and 5 summarize the findings for these two variables. As can be seen from Table 4, the best fitting regression model for Ma yielded an $R^2$ of .11. As shown, the first predictor entered into the model was duration with a regression coefficient of -.81. The second step entered the age of onset term with a coefficient of -.57. Finally age of onset X duration interaction term was entered with a coefficient of -.08. This model indicates that Ma scores decreased as age of onset and duration increased.

Table 5 shows that the best fitting regression model for Si yielded an $R^2$ of .14. Duration was the first predictor entered with a regression coefficient of .83. The second step entered the age of onset term with a regression coefficient of .96. The last term entered was age of onset squared with a coefficient of -.03. This model indicates that Si scores increased as age of onset and duration increased.

**Summary.** Consistent with results of the canonical correlation analysis, multiple regression analyses indicated that age of onset and duration were more powerful predictors of the secondary than of the primary psychopathology of
TABLE 4

STEPWISE MULTIPLE REGRESSION FOR Ma

$R^2 = .11$

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>Mean Square</th>
<th>F</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression</td>
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<td>2439.79</td>
<td>813.26</td>
<td>6.15</td>
<td>.0006</td>
</tr>
<tr>
<td>Error</td>
<td>149</td>
<td>19718.11</td>
<td>132.34</td>
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<td></td>
</tr>
<tr>
<td>Total</td>
<td>152</td>
<td>22157.90</td>
<td></td>
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<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter estimate</th>
<th>Standard error of squares</th>
<th>Type II Sum of squares</th>
<th>F</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>60.33</td>
<td>.97</td>
<td>513973.74</td>
<td>3883.85</td>
<td>.0001</td>
</tr>
<tr>
<td>Age of Onset</td>
<td>-.57</td>
<td>.19</td>
<td>1184.89</td>
<td>8.95</td>
<td>.003</td>
</tr>
<tr>
<td>Duration</td>
<td>-.81</td>
<td>.21</td>
<td>1921.59</td>
<td>14.52</td>
<td>.0002</td>
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<tr>
<td>Age X Dur</td>
<td>-.08</td>
<td>.03</td>
<td>958.89</td>
<td>7.25</td>
<td>.008</td>
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<table>
<thead>
<tr>
<th>Step</th>
<th>Variable entered</th>
<th>Partial $R^2$</th>
<th>Model $R^2$</th>
<th>F</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Duration</td>
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<td>.03</td>
<td>3.99</td>
<td>.05</td>
</tr>
<tr>
<td>2</td>
<td>Age of Onset</td>
<td>.04</td>
<td>.07</td>
<td>6.60</td>
<td>.01</td>
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<tr>
<td>3</td>
<td>Age X Dur</td>
<td>.04</td>
<td>.11</td>
<td>7.25</td>
<td>.01</td>
</tr>
</tbody>
</table>
TABLE 5

STEPWISE MULTIPLE REGRESSION FOR Si

$R^2 = .14$

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>Sum of Squares</th>
<th>Mean Square</th>
<th>F</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression</td>
<td>3</td>
<td>3567.14</td>
<td>1189.05</td>
<td>8.01</td>
<td>.0001</td>
</tr>
<tr>
<td>Error</td>
<td>149</td>
<td>22128.39</td>
<td>148.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>152</td>
<td>25695.53</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Parameter estimate</th>
<th>Standard error</th>
<th>Type II Sum of squares</th>
<th>F</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>62.10</td>
<td>1.08</td>
<td>489699.13</td>
<td>3297.36</td>
<td>.0001</td>
</tr>
<tr>
<td>Age of Onset</td>
<td>.96</td>
<td>.25</td>
<td>2160.56</td>
<td>14.55</td>
<td>.0002</td>
</tr>
<tr>
<td>Duration</td>
<td>.83</td>
<td>.19</td>
<td>2736.96</td>
<td>18.43</td>
<td>.0001</td>
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<tr>
<td>Age Onset Sq.</td>
<td>.03</td>
<td>.02</td>
<td>611.06</td>
<td>4.11</td>
<td>.04</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step</th>
<th>Variable entered</th>
<th>Partial $R^2$</th>
<th>Model $R^2$</th>
<th>$F$</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Duration</td>
<td>.05</td>
<td>.05</td>
<td>8.62</td>
<td>.0004</td>
</tr>
<tr>
<td>2</td>
<td>Age of Onset</td>
<td>.06</td>
<td>.12</td>
<td>10.34</td>
<td>.002</td>
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<tr>
<td>3</td>
<td>Age Onset Sq.</td>
<td>.02</td>
<td>.14</td>
<td>4.12</td>
<td>.04</td>
</tr>
</tbody>
</table>
bulimia nervosa. In particular, MMPI variables Ma and Si were most highly associated with the predictor variables.

**Multivariate Analysis of Variance (MANOVA)**

Three MANOVA's were conducted to examine the effect of age of onset and duration on the primary psychopathology dependent variables, secondary psychopathology dependent variables, and combined primary and secondary psychopathology dependent variables. The independent variables were transformed from continuous to discrete variables via a median split. The median for age of onset was 17, with 49.7% of the cases falling at or below this age. Thus, the two groups for this analysis were defined as younger age of onset equal to or less than seventeen years (N=38), and older age of onset equal to or greater than eighteen years (N=33). The median for duration was three years with 54.2% of the cases falling at or below this age. Shorter duration was thus established to be three years or less (N=38) and longer duration equal to or greater than four years (N=44). The means and standard deviations for each of these four cells were: in the young age of onset/short duration cell mean age of onset was 15.5 years (SD=1.5) and mean duration was 2.2 years (SD=.72); in the young age of onset/long duration cell mean age of onset was 13.6 years (SD=3.1) and mean duration was 9.5 years (SD=7.2); in the old age of onset/short duration cell mean age of onset was 22.4 years (SD=5.8) and mean duration was
1.8 years (SD=.75); in the old age of onset/long duration cell mean age of onset was 19.8 years (SD=2.8) and mean duration was 8.3 years (SD=5.2).

**Primary psychopathology variables.** The first MANOVA, which included dependent variables related to the primary psychopathology of bulimia nervosa (i.e., EAT, BULIT, BIAC, BIAI), was not statistically significant for age of onset, duration, or the interaction of age of onset and duration.

**Secondary psychopathology variables.** The second MANOVA, which included measures of secondary psychopathology (i.e., BDI, F, Hs, D, Hy, Pd, Mf, Pa, Pt, Sc, Ma, Si), was significant for age of onset ($F = 2.46$, $p = .006$), but not for duration or the interaction term. Subsequent examination of the univariate analysis of variance showed that age of onset was significant for dependent variables Ma ($F = 6.64$, $p = .01$) and Si ($F = 13.51$, $p = .0003$). Comparing the means for younger and older age of onset presented in table 6 shows that mean Ma scores were higher in the group with younger age of onset than in the group with older age of onset, and mean Si scores were higher in the group with older age of onset.

**Summary.** The results of the MANOVA were consistent with both canonical correlation and multiple regression findings. Age of onset was more associated with changes in secondary psychopathology than in primary psychopathology. The MANOVA also confirmed findings of the multiple regression that MMPI
TABLE 6

COMPARISON OF AGE OF ONSET GROUP MEANS FOR DEPENDENT MEASURES

<table>
<thead>
<tr>
<th>Primary Psychopathology</th>
<th>Younger Age of Onset</th>
<th>Older Age of Onset</th>
<th>F (1, 149)</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>EAT</td>
<td>44.6</td>
<td>47.6</td>
<td>1.44</td>
<td>NS</td>
</tr>
<tr>
<td>BULIT</td>
<td>120.5</td>
<td>119.2</td>
<td>.19</td>
<td>NS</td>
</tr>
<tr>
<td>BIAC</td>
<td>60.1</td>
<td>59.6</td>
<td>.04</td>
<td>NS</td>
</tr>
<tr>
<td>BIAI</td>
<td>44.7</td>
<td>42.7</td>
<td>.84</td>
<td>NS</td>
</tr>
</tbody>
</table>

| Secondary Psychopathology | | | |
|---------------------------| | | |
| BDI                       | 21.1                 | 20.3               | .26        | NS      |
| F                         | 63.5                 | 63.4               | .00        | NS      |
| Hs                        | 64.2                 | 62.4               | .69        | NS      |
| D                         | 71.6                 | 72.8               | .29        | NS      |
| Hy                        | 66.9                 | 65.4               | .67        | NS      |
| Pd                        | 74.4                 | 72.5               | .88        | NS      |
| Mf                        | 44.9                 | 44.3               | .18        | NS      |
| Pa                        | 66.5                 | 67.6               | .29        | NS      |
| Pt                        | 72.5                 | 72.4               | .00        | NS      |
| Sc                        | 72.1                 | 71.4               | .08        | NS      |
| Ma                        | 63.3a                | 58.4b              | 6.64       | .01     |
| Si                        | 57.8a                | 65.2b              | 13.51      | .0003   |

* Means with different superscripts (a, b) differ significantly (p < .05)

BIA and MMPI scores are t-scores.
scales Ma and Si were the secondary measures which varied as a function of age of onset.

**Simple Correlation**

Correlation matrices were computed to evaluate the association between measures of primary and secondary psychopathology for the short and long duration groups. The correlations of short and long duration groups were then compared to determine if groups differed. Correlations between measures of primary and secondary psychopathology were not significantly different for the short and long duration groups. These results suggest that the degree of association between primary and secondary psychopathology did not change significantly over the course of the disorder.
DISCUSSION

Based on the results of canonical correlation, a general finding of this study was that components of both primary and secondary psychopathology varied as a function of age of onset and duration. Worsening of some variables, reflected by higher scores on the BULIT, body image assessment, and social introversion scale, supported predictions that both primary and secondary psychopathology would worsen. The guiding model of this research (Williamson et al., 1990) predicted worsening of both sets of symptoms based on their bidirectional relationship. Thus, the secondary psychopathology influences primary symptoms and vice versa. It was predicted that bulimic symptoms would worsen over time because of strengthening of these habits via positive and negative reinforcement.

Other changes (i.e., decreases) in measures of primary and secondary psychopathology were also observed as a function of age of onset and duration. The relationship of both primary and secondary psychopathology to age of onset and duration will each be discussed in turn.

Primary Psychopathology

Both increases and decreases occurred in the various components of primary symptomatology as a function of duration and age of onset. Young age of onset in conjunction with long duration was associated with changes
in three measures of primary symptomatology. One might suspect that an individual who develops a disorder at a younger age (i.e., early adolescence) may be more likely to experience long duration of the disorder than an individual who develops it later in life. This relationship was found in this sample. Mean age of onset was 16.5 in the long duration group and 19.2 in the short duration group. Previously discussed research on anxiety disorders (Ost, 1987) found the same association of young age of onset and long duration for phobic symptoms.

The pattern of young age of onset and long duration in this study was associated with lower scores on the EAT, and increasing BULIT and current body size estimation scores. The EAT assesses eating related habits and cognitions of anorexia nervosa (Garner & Garfinkel, 1979), and as such can serve as a measure of the severity of anorexic symptoms (e.g., overconcern with dieting and body size) also commonly found in bulimia nervosa. The BULIT assesses the primary symptoms of bulimia nervosa (Smith & Thelen, 1984), and has been used previously as a measure of the severity of bulimic symptoms (Bennet, Williamson, & Powers, 1989) which includes binging and purging.

The canonical correlation analysis, thus, suggested that anorexic symptoms (e.g., restrained eating pattern) became less problematic with greater chronicity. This change may be a function of lessening peer pressure to
adhere to sociocultural ideals for thinness during adolescence. At the same time, this analysis found that bulimic symptoms (e.g., binging and purging) became more problematic as bulimia nervosa continued and was accompanied by higher estimates of body size. As noted previously, increased binging and purging is predicted by the theoretical model based on the negative reinforcement of purging. The increase in standardized scores on current Body Image Assessment is suggestive of worsening of body image distortion as bulimia became a more chronic condition.

The nature of these findings can be further clarified by examining the relationship between weight and duration. In this study, the correlation between duration of bulimia nervosa and weight (.33, p < .0001) indicated that the weight of this sample increased over time. This increase was reflected in the difference between mean weights for the short duration group (128 lbs.) and long duration group (142 lbs.). Additionally, mean weight increased from short duration (125 lbs.) to long duration (144 lbs.) within the younger age of onset group and from short duration (131 lbs.) to long duration (140 lbs.) within the older age of onset group. A recent study (Williamson et al., 1989) found weight to be positively correlated with estimation of current body size, which is conceptually related to body image distortion. That report was consistent with an
earlier one (Birtchell, Lacey, & Harte, 1985) which also found a positive relationship between weight index and overestimation of body size in a bulimic sample. The occurrence of actual weight gain over the duration of the disorder in this study helps support the interpretation of decreased dieting coupled with increased binging, and previous research helps to explain the associated increases in current body size estimation.

Following a discussion of secondary psychopathology, an explanation will be offered regarding how changes in primary and secondary psychopathology are related.

**Secondary Psychopathology**

In this study, mean MMPI scale scores showed clinical elevations on Depression, Psychopathic deviance, Psychasthenia, and Schizophrenia scales. This profile is consistent with previous research using the MMPI with bulimic samples (Williamson et al., 1990). The majority of literature identifies a typical bulimic profile as being characterized by elevations or near elevations on these same scales (Mizes, 1985; Prather & Williamson, 1988, Williamson, Prather, Upton, Davis, Ruggiero, & Van Buren, 1987). Additionally, elevated mean BDI scores are consistent with previous findings (Prather & Williamson, 1988; Williamson et al., 1987) of clinically significant levels of depression in bulimics and reports that
depression is the most common secondary psychopathology observed in bulimia nervosa (Hudson, Laffer, & Pope, 1982; Katzman & Wolchik, 1984).

As was the case for primary psychopathology, the combination of age of onset and duration was associated with a specific pattern of secondary symptoms. Increasing age of onset in conjunction with longer duration was associated with pattern of decreases on three MMPI scales (Hypochondriasis, Depression, Psychasthenia) and increases on two scales (Hysteria, Social introversion). Graham (1977) offers several descriptions of increased Hysteria scale scores. Individuals with these scores may display lack of anxiety, tension, depression, and insight as well as superficiality and immaturity in interpersonal relationships.

Increasing Social introversion scale scores are indicative of avoidance, withdrawal, and discomfort in interpersonal relationships which is likely to be accompanied by greater sensitivity to what others think (Trimboli & Kilgore, 1983). Greene (1980) points out that lack of contact with significant others may further exacerbate existing problems by limiting available support. These findings support predictions of the guiding conceptual model for worsening of interpersonal problems and sensitivity.
Decreasing Hypochondriasis scale scores may reflect increasing denial of physical complaints (Greene, 1980). Decreasing Depression scale scores suggest that individuals report fewer symptoms of depression. Additionally, these individuals may adopt a less worrisome, anxiety-laden approach to situations following a previous period of painful anxiety; they may even be seen as unmotivated because they respond with less anxiety than most other people (Duckworth & Anderson, 1986). Thus, individuals who become bulimic at an older age and have a longer duration of bulimic problems were characterized by less anxiety, depression, and somatic complaints along with increasing interpersonal problems and avoidance.

When viewed individually as a function of age on onset and duration, the pattern of MMPI scales which vary significantly was slightly different. Social inroversion scores continued to increase as duration and age of onset increased. However, the only other MMPI subscale which showed systematic changes as a function of age of onset or duration was the Mania scale. Rather than increasing over the course of duration and age of onset, regression equations predicted decreasing Mania scale scores over both duration and age of onset.

The pattern of age of onset and duration for Mania scores suggests that the older an individual is when she becomes bulimic and the longer she is bulimic, the more
likely it is that Mania scores will decrease. Low scores on the Mania scale for older people is consistent with other reports which consider low scores an indication of decreased energy levels (Colligan, Osborne, Swenson, & Offord, 1984). In general, this scale can be considered a reflection of psychic and physical energy levels (Duckworth & Anderson, 1986). Significantly lower Mania scale scores for a sample of low frequency purgers as compared to a normal sample was previously reported in the bulimia nervosa literature (Williamson et al., 1987).

Increased Social introversion scores and decreased Mania scores are also meaningful in combination. Good and Brantner (1974) have called this pattern the "nonsocializer" profile. Examination of the table of means shows that Schizophrenia scale scores were elevated above a t-score of 70 in this study. Duckworth and Anderson (1986) reported that elevations on Schizophrenia and Social introversion scales tend to accentuate one another and reflect increasing isolation. This interpretation is consistent with description of the "schizoid V" characterized by high Schizophrenia, low Mania, and high Social introversion scores (Tromboli & Kilgore, 1983). They consider such a profile reflective of a slowly developing, chronic pattern of maladjustment.

When viewed as a function of discrete categories of age of onset and duration, Mania and Social introversion
scales were the only variables which varied as a function of age of onset. Duration did not affect any of the dependent variables. This finding is consistent with previously discussed alcohol research (Lee & DiClemente, 1985) which reported that age of onset of drinking was correlated more strongly with secondary psychopathology than was duration of drinking. In this study the findings suggest that later age of onset bulimics (eighteen years or older), whose mean duration was 4.7 years, reported greater social introversion and lower energy and activity levels. One possible interpretation is that the presence of greater social introversion in this group may have facilitated the development of bulimic behaviors as a solitary way of dealing with psychological conflict, and the secrecy of the disorder encourages further social withdrawal.

This finding also suggests that earlier age of onset bulimics (seventeen years or younger), whose mean duration was 5.9 years, reported less social introversion and higher energy level. This pattern is consistent with adolescents’ greater involvement in peer-related activities.

It has been previously noted that decreased Mania scores are often characteristic of older people (Colligan et al., 1984). The lower activity level which these scores suggest may serve to exacerbate the weight gain that accompanies increased binging behavior with longer duration. As was previously noted, mean weight increased
from 128 lbs. in the short duration group to 142 lbs. in the long duration group. Thus, the longer an individual is bulimic the more weight they are likely to gain. Weight gain would further contribute to body image problems and fear of weight gain which drive binging and purging.

It is worth noting that elevations of the Social introversion scale score is an uncommon finding in a bulimic sample. As observed in the discussion of typical bulimic MMPI profiles, previous research has not reported elevations of Social introversion scores. This difference in results may have occurred because of the inclusion of duration and age of onset as factors with which scores on this scale covary. One way to view these results is to consider the generalized distress profile reported by the majority of the literature as a constant across bulimics. This constant level of psychopathology may then be subject to variations in interpretation depending on further assessment of the bulimic's activity patterns and social involvement.

Integration

With regard to the model of bulimia utilized in this study, the changes in primary psychopathology can be related conceptually, if not statistically, to changes in secondary psychopathology. For example, given that dieting (a socially acceptable behavior) becomes less prominent
over time and that binging and purging (socially unacceptable behaviors) become more prominent, the increase in social introversion can be understood as increased secretiveness which functions to guard against the discovery of bulimia nervosa thus maintaining sufficient opportunities to engage in binging and purging. The increased weight and body image distortion which appears to accompany greater chronicity is also likely to be related to greater interpersonal sensitivity and social withdrawal due to fears of being seen as fat by others. Additionally, the continued elimination of energy and nutrients that would accompany increased binging and purging according to the theoretical model may account for decreases in mania, which can be considered an index of psychic and physical energy (Duckworth & Anderson, 1986). Although engaging in more bulimic behavior over the course of the disorder, bulimics may also report somewhat less distress with increased age of onset. When accompanied by lowered activity levels and greater social avoidance in later stages of bulimia, reporting of less distress may be reflective of denial of symptoms and an indication of progressive worsening, rather than improvement, of secondary psychopathology.

Conclusions

This study undertook the investigation of primary and
secondary aspects of bulimic psychopathology as a function of age of onset and duration. One of the limitations of this study concerns the use of cross-sectional data to draw conclusions about the development of bulimia nervosa. Although this approach may be an acceptable first step in developmental research, future studies might attempt to find support for these results through a longitudinal approach that follows high risk individuals (e.g., young female dieters) to observe changes in eating habits and psychological adjustment.

A second limitation of this study is the use of global scores to describe changes in psychopathology. More specific measures of dieting (e.g., number of calories consumed), bulimic behavior (e.g., frequency of binging or purging), activity level, and interpersonal and social functioning could offer more detailed information regarding dysfunction in the areas identified as significant in this study. Thus, this study can be considered a preliminary description of factors operating in the development of bulimia nervosa, which ideally should be followed by additional studies to garner support and further clarification of these results.
REFERENCES


anorexia, and bulimia (pp. 405-499). New York: Basic Books.


in the objective assessment of body distortion in seven eating disorder groups. *International Journal of Eating Disorders, 5*, 113-120.


*Behavior Therapy, 19*, 1-9.


APPENDIX A

CONSENT FORM
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. I understand that my identity will remain anonymous and that
my name will not be used in any publications or presentations which are derived
from this research. The activities in which I agree to participate are those
checked below:

   1. Clinical interviews
   2. Height and Weight measurement
   3. Psychological testing
   4. Assessment of therapy outcome
   5. Assessment of depression
   6. Body Image assessment
   7. Group Therapy
   8. Individual Therapy
   9. Self monitoring of eating
   10. Survey of oral habits

All of my questions have been answered and I understand that I may withdraw
from the research project without penalty at any time.

Signature Date

Signature of Witness Date
APPENDIX B

DIAGNOSTIC SURVEY FOR EATING DISORDERS
PLEASE NOTE

Copyrighted materials in this document have not been filmed at the request of the author. They are available for consultation, however, in the author's university library.

86-106
108-110
112-118

University Microfilms International
APPENDIX C

EATING ATTITUDES TEST
APPENDIX D

BULIMIA TEST
APPENDIX E

BODY IMAGE ASSESSMENT
Administration

The experimenter places the cards randomly on a table in front of the subject and provides the instructions:

"Select the card that most accurately depicts your current body size, as you perceive it to be. Please be honest. You must choose only one card and you may not rearrange the cards to directly compare them."

After the subject selects a card, the experimenter records the card number 1 to 9 (written on the back of the card) as the score for current body size. The cards are then reshuffled and again randomly placed in front of the subject with the instructions:

"Select the card that most accurately depicts the body size that you would most prefer. Again, be honest and do not rearrange the cards."

This card number is recorded as the score for ideal body size. These raw scores are converted to t-scores using the normative data (table following).
### Conversion Table for Body Image Assessment (t scores)

<table>
<thead>
<tr>
<th>Body Size</th>
<th>N</th>
<th>Height (In)</th>
<th>Height Mean</th>
<th>Weight (lbs)</th>
<th>Weight Mean</th>
<th>Raw Score: CBS</th>
<th>Raw Score: IBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low Weight</td>
<td>118</td>
<td>60-68</td>
<td>63</td>
<td>91-115</td>
<td>107</td>
<td>26 37 48 59 70 81 92 103 114</td>
<td></td>
</tr>
<tr>
<td>Normal Weight</td>
<td>204</td>
<td>60-70</td>
<td>65</td>
<td>116-134</td>
<td>124</td>
<td>28 38 47 57 67 76 86 96 106</td>
<td></td>
</tr>
<tr>
<td>High Weight</td>
<td>103</td>
<td>63-71</td>
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<td>115-166</td>
<td>144</td>
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APPENDIX F

BECK INVENTORY
PLEASE NOTE

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University Microfilms International
VITA
Darlene Broussard Witcher

Date of Birth: June 12, 1957   Marital Status: Married
Place of Birth: New Orleans, LA   Current Residence: Houston

Education

1979   B.A., Psychology   University of New Orleans
       New Orleans, LA

1984   M.A., Psychology   Louisiana State University
       Baton Rouge, LA

Thesis: Cognitive and Social Desirability
Factors in Marital Adjustment for a Black and
White Sample.

1990   Ph.D., Clinical Psychology   Louisiana State University
       Baton Rouge, LA

Dissertation: Age of Onset and Duration
Variables: Relationship to Primary and
Secondary Psychopathology of Bulimia Nervosa.

Training

1986-1987   Veterans Administration Medical Center,
             Houston: Clinical psychology internship -
             rotations in family therapy, substance abuse,
             neuropsychology, and impatient psychiatry.
Experience

1982-1985
Psychological Services Center, Department of Psychology, Louisiana State University:
Graduate practica in adult, child, and eating disorders psychology.

10/88-
1988-present
The Methodist Hospital
Baylor College of Medicine
Houston, TX
Psychological consultant to Physical Medicine Rehabilitation, Behavioral Medicine, and Institute for Preventive Medicine.

9/87-
10/88
Mainland Center Hospital
Bay Area Institute for Living
Texas City, TX
Psychotherapist for acute psychiatry unit.

1/86-
8/86
CPC Meadow Wood Hospital
Baton Rouge, LA
Psychological consultant to eating disorders program.

3/85-
Feliciana Forensic Facility
Jackson, LA
Psychological Associate position in maximum security facility for felony offenders with psychiatric diagnoses.

9/83- 2/85
East Louisiana State Hospital
Oakcrest House, Intermediate Care Facility for Mentally Retarded
Jackson, LA
Psychological Testing Assistant position in residential setting for profound to mild mentally retarded adults.

2/80- 8/82
Ochsner Foundation Hospital
Adolescent Psychiatry
New Orleans, LA
Mental Health Associate position in long-term inpatient unit.

Assistantship
1982 - 1983 National Institute of Mental Health
Teaching/Research Assistant
Research

Publications


Readability assessment of questionnaires frequently used in sex and marital therapy. *Journal of Sex and Marital Therapy, 13*, 137-141.

Presentations

Cognitive and social desirability factors in marital adjustment for black and white samples. Paper presented at the meeting of the Southeastern Psychological Association, Atlanta.

Readability assessment of questionnaires frequently used in sex and marital therapy. Paper presented at the meeting of the Southeastern Psychological Association, Orlando.
DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Darlene Broussard Witcher

Major Field: Psychology

Title of Dissertation: Age of Onset and Duration Variables: Relationship to Primary and Secondary Psychopathology of Bulimia Nervosa

Approved:

[Signatures]

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

[Signatures]

Date of Examination:

July 12, 1990