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Factors Associated with Bulimia Nervosa
And Their Relationships with Bulimic Symptoms:
Examining the Role of the Family Environment Factor

by

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

Master of Arts

Department of Psychology

Carleton University

Ottawa, Ontario

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Abstract

This study integrated a list of risk factors for bulimia nervosa into major risk factor categories and then examined the relationships between the risk factor groups and bulimic symptomatology.

Ninety-two women with bulimia nervosa participated in this study by providing information about their exposure to a variety risk factors over the course of their lifetimes. A confirmatory factor analysis suggested that three risk factor classes better described the data than two factor classes. The three factor classes identified were the General Psychiatric factor, the Dieting and Obesity factor, and the Family Environment factor. A series of regression analyses revealed that only the General Psychiatric factor and the Dieting and Obesity factor significantly predicted bulimic symptomatology. The Family Environment factor was found to be a suppressor variable, which suggested it was indirectly affecting bulimic symptomatology through the other two factors. A path analysis that examined the direct and indirect effects of the Family Environment factor on bulimic symptomatology, confirmed this finding.

Acknowledgments

To my husband Michael: Your love and support made this dream possible.

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Factors Associated with Bulimia Nervosa
and Their Relationships with Bulimic Symptoms:
Examining the Role of the Family Environment Factor

Bulimia nervosa (BN) research has received very little attention until recently. The formal diagnosis 'Bulimia Nervosa' did not appear in the Diagnostic and Statistical Manual (DSM) until the revised third edition in 1987 (APA, 1987). Because a standardized diagnosis for BN has only existed since this time, the current research surrounding this particular disorder is still at an early stage of development (Fairburn, Welch, Doll, Davies & O'Connor, 1997).

The knowledge of risk factors and the way they interact to give rise to BN, are aspects of BN research that currently face certain limitations. Much of the existing research has focused on a variety of disparate risk factors that have the potential to give rise to BN, or at very least have reciprocal relationships with bulimic symptomatology. This research has identified a large list of putative risk factors including; socio-cultural pressure (Schwartz, Thompson & Johnson, 1982; Stice & Shaw, 1994; Striegel-Moore, Silberstein & Rodin, 1986), negative parental attitudes (Johnson & Connors, 1987; Johnson & Flach, 1985; Pike & Rodin, 1991), internalization of a thin ideal (Striegel-Moore, Schreiber, Pike, Wilfley & Rodin, 1995), adiposity (Fairburn & Cooper, 1982; Thelen & Corimer, 1995), history of abuse (Welch & Fairburn, 1996) as well as co-morbid psychopathological traits such as affective instability (Leon, Fulkerson, Perry &

Cudeck, 1993; Stice & Agras, 1998) and negative self-esteem and self-worth (Johnson & Connors, 1987; Wagner, Halmi & Maguire, 1987). Many of these studies have approached the issue of etiology with a very specific focus. As a result, the list of possible risk factors has continued to grow whereas very few attempts have been made to conceptualize or integrate these factors.

One of the major criticisms of the risk factor literature to date is that it lacks a vision of ‘the big picture’ (Crowther, Wolf & Sherwood, 1992). So far, the research has failed to explain how all of the putative risk factors associated with BN interact and contribute to the development of the disorder. Garner and Garfinkle (1987) commented, “It appears that any one-factor theory of this complex disorder is far too limited..... We hope that the next decade of eating disorders research will lead to greater refinement of etiological parameters and more specific knowledge of how they interact to produce bulimia nervosa” (p. 6). The purpose of the present study was to provide some insight into the ‘big picture’ by classifying the putative risk factors into conceptualized groups, and then to examine the way these risk factor groups interacted and contributed to bulimic pathology.

History of Bulimia Nervosa

The name ‘Bulimia’ was derived from two ancient Greek words, *limos* meaning “hunger” and *boul* meaning “a great amount of” (Ziolko & Schrader, 1985). Thus binge eating behaviour, a characteristic symptom of BN, was the first to be recognized as a problematic eating behaviour (Lidell & Scott, 1972). Descriptions of this disorder resurfaced again in the 18th century when James (1743) described *caninus appetitus*, a

disorder in which excessive overeating was followed by vomiting. Despite the early descriptions of bingeing and purging behaviours, a formal recognition of this disorder was overlooked until recently (Stunkard, 1993). In contrast, anorexia nervosa (AN), which has historical roots grounded in medieval times (Bell, 1985), became a recognized medical diagnosis twenty years before the formal recognition of BN. The first modern medical description of BN was given by Russell (1979) who described this disorder as an “an ominous variant of anorexia nervosa” (p. 429).

As a result, AN was the primary focus of the eating disorders literature until recently (Holland, Hall, Murray, Russell & Crisp, 1984; Morgan & Russell, 1975; Rastam & Gillberg, 1992; Rivinus, Biederman, Herzog, Kemper, Harper, Harmatz & Houseworth, 1984; Swift & Stern, 1982). Only in the last two decades have researchers in the eating disorders literature focused on BN (Fairburn & Beglin, 1990). The efforts to develop BN research have been considerable but further studies are needed to refine the knowledge of possible risk factors for this disorder.

Defining Characteristics of Bulimia Nervosa

The DSM-IV describes three core characteristics of BN (APA, 1994). The first characteristic is the presence of recurring binge eating. These binge eating episodes are marked by two salient features:

- 1) Eating an amount of food that is significantly larger than ‘normal’ within a discrete period of time.
- 2) Feeling a sense of loss of control during an eating episode such that the person cannot control what or how much they eat.

The second characteristic is the presence of recurring 'inappropriate' compensatory behaviour including self-induced vomiting, misuse of laxatives (purging type), fasting or excessive exercise (non-purging type). In order to meet the diagnostic criteria these two primary characteristics must occur twice a week, for a period of at least three months (APA, 1994). The third diagnostic characteristic is a pervasive negative self-evaluation that is unduly influenced by body shape and weight.

Prevalence

According to the practice guidelines of the American Psychological Association (1993) BN is diagnosed in 1% to 10% of middle and upper class adolescent and college-age women. Epidemiological studies within the last fifteen years have found prevalence rates of similar proportions, ranging from 1% to 12% (Fairburn & Beglin, 1990; King, 1986; Pope, Hudson & Yurgelun-Todd, 1984; Rand & Kulda, 1992; Williams, Schaefer & Shisslak, 1986). These studies have used a variety of sampling methodologies and consequently the results have not been directly comparable. Despite the methodological differences however, several common themes have emerged. First, BN affects a predominantly female population with ages ranging from young adolescence to young adulthood (Fairburn & Wilson, 1993). Second, two to three times more women are affected by BN than by AN (Crowther, Wolf & Sherwood, 1992; Rastam & Gillberg, 1992).

Over the last forty years there has been an upward surge of reported cases of BN (Fairburn & Beglin, 1990; Gordon, 1990). Although the prevalence rates of AN have remained relatively stable since the 1960s, the reported prevalence of BN has more than

doubled in North America (Fairburn & Wilson, 1993; Garner & Fairburn, 1988). Gordon (1990) suggested that BN is a disorder that is approaching “social epidemic” proportions. The development of clearer definitions of BN and a more wide spread awareness of this disorder over the last several decades might explain much of the drastic increase in the prevalence rates for BN (Fairburn & Wilson, 1993). However, since the diagnostic criteria was standardized for BN, epidemiological research consistently finds that the prevalence rates for BN heavily outweigh the rates for AN. Fairburn and Beglin (1990) suggested that BN has replaced AN as the primary eating disorder in North America. This research emphasizes the importance of further research that focuses on the nature, course and etiology of BN (Fairburn & Beglin, 1990).

Chapter II

Factors Associated with Bulimia Nervosa

The Use of the Term ‘Risk Factor’

Before the eating disorders literature is reviewed it is important to establish the intended meaning of the term ‘risk factor’ for this study. The term ‘risk factor’ implies that there is a temporal relationship between the individual’s exposure to these factors and the subsequent onset of BN. Understanding the term risk factor in this sense implies a potential causal relation between the risk factors and BN. However, presuming a causal connection for this study would be premature.

The term ‘risk factor’ is used in this study to describe the life events, personality traits, and a host of other variables that tend to be associated with BN. The term was chosen because these variables are referred to as ‘risk factors’ in the eating disorders literature. The majority of the research does suggest that these risk factors are involved in the development and maintenance of BN, however the research rarely uses ‘risk factor’ to imply a causal relationship. This is because most of the ‘risk factor’ studies have used retrospective methodologies, relying on self-report techniques to determine the variables involved with BN. Until new prospective research can demonstrate the temporal relations between these variables and BN, cause cannot be inferred. However retrospective studies can suggest the direction for future prospective research.

For this study the term ‘risk factor’ was not used to infer cause. Instead throughout this paper, ‘risk factor’ should be taken to mean any personality trait, attitude, event or relationship that has been shown by the literature to be associated with BN.

Literature Review

The literature contains a wide variety of studies of possible risk factors for eating disorders. Although a small number of these studies pertain to BN (Leon, Keel, Klump & Fulkerson, 1997; Lowe, Gleaves & Murphy-Eberenz, 1998; Stice & Agras, 1998), most of the risk factor literature has focused on AN (Halmi, Eckert, Marchi, Sampugnaro, Apple & Cohen, 1991; Rastam & Gillberg, 1992; Strober, Morrell, Burroughs, Salkin & Jacobs, 1985; Swift & Stern, 1982) or eating disorders in general (Schmidt, Tiller & Treasure, 1993; Steiger, Goldstein, Mongrain & Van der Feen, 1990). As stated earlier, one of the primary problems with this research has been that each study has had a circumscribed focus, examining only a few possible risk factors at a time. This oversight in the research has resulted in a current knowledge of many putative risk factors for eating disorders, with a poor understanding of the relative contributions and interactions of different classes of risk factors.

A few studies have conceptualized broad classes of risk factors from the existing risk factor research (Fairburn & Cooper, 1984; Fairburn et al., 1997; Gleaves, Williamson & Barker, 1993; Tobin, Johnson, Steinberg, Staats & Baker Dennis, 1991). From these studies, three major risk factor groupings have been proposed for BN. These three major factors are the General Psychiatric factor, the Dieting and Obesity factor, and the Family

Environment factor. The studies that have been responsible for the development these concepts will now be reviewed.

Early clinical descriptions of BN included the eating patterns and compensatory behaviours of this population, as well as the typical family history, personality traits, childhood experiences, and developmental progress for this eating disorder (Rau & Green, 1975; Russell, 1979). Pyle, Mitchell and Eckert (1981) described a group of women with BN who were typically of normal weight, had a high degree of co-morbid psychiatric problems, and some history of AN. The personality characteristics, low self-esteem and a low sense of self-control were consistent throughout this group. Although these descriptions were critical in terms of profiling this particular population, this study was faulted for being purely descriptive with no standardized system for assessing these clinical features (Fairburn & Cooper, 1984).

Fairburn and Cooper (1984) developed standardized questionnaires to measure these clinical features. Participants (N=620) meeting the DSM-III criteria for BN (APA, 1987), responded to questions about their demographic characteristics, eating habits, co-morbid psychiatric illness, family history and non-specific psychopathology. The results of this study revealed that the participants were predominantly young adult females, of normal weight, with a morbid fear of weight gain. Only 3% of the participants had previously suffered from AN. This finding differed from earlier studies by Pyle, Mitchell and Eckert (1981) and Russell (1979), and provided support for the notion that BN was not just a chronic complication of AN.

Fairburn and Cooper (1984) also examined the participants' exposure to a variety of risk factors and conceptualized the variables into groups. The conceptualizations were based on the results of two self-report questionnaires, a mental health questionnaire and a personal obesity history questionnaire. On the mental health questionnaire, 68% of the participants with BN scored above the 'normal' threshold, indicating the presence of co-morbid psychiatric disorders among many of these participants. The mental health scale revealed that most of the participants were suffering from depression and/or anxiety. Based on this finding, Fairburn and Cooper (1984) suggested that personal vulnerabilities to psychiatric disorders in general might be a predisposing group of risk factors for the development and maintenance of BN. Results from the personal obesity questionnaire revealed that 75% of the participants had been over weight at some point in their lives. This rate was significantly higher in participants with BN compared to the control participants matched for age and height. Furthermore, 82% of the participants with BN reported that they "persistently felt fat" despite the fact that 60% of them were within a normal weight range at the time of the study (Fairburn & Cooper, 1984). Based on these results, Fairburn and Cooper (1984) suggested that personal vulnerabilities to overweight and dieting might be another predisposing group of risk factors for the development and maintenance of BN.

These findings were important because they provided some insight into the possible groupings of risk factors for the development and maintenance of BN. The two groups of risk factors, the General Psychiatric factor and Dieting and Obesity factor, were the first two basic classes of risk factors proposed.

Following this study (Fairburn & Cooper, 1984), two subsequent studies were performed to conceptualize the possible risk factors for BN using a multi-factorial approach. Tobin et al. (1991) performed an exploratory factor analysis on a limited set of potential risk factors for BN. Participants with BN (N=245 women) completed a self-report battery of psychological tests relating to mental health and eating attitudes and behaviours. The results of the factor analysis indicated a general factor that grouped all of the possible risk factors for BN into one large category. Tobin et al. (1991) also found three second-order factors and suggested that “bulimia nervosa could be described more complexly” (p. 14). Tobin et al. (1991) labeled the three second-order factors *Affective and Personality Disorder*, *Bulimic Behaviours*, and *Restricting Behaviours*. Gleaves et al. (1992) performed a confirmatory factor analysis to test the three-factor multidimensional model using *Affective and Personality Disorder*, *Bulimic Behaviours*, and *Restricting Behaviours* (Gleaves et al., 1993). The results confirmed Tobin et al.’s (1991) findings by demonstrating that this three-factor model provided a good fit to the data. This early work in multi-factorial assessment of BN provided a statistical framework for further research on this eating disorder.

Tobin et al. (1991) suggested that for the first factor *Affective and Personality Disorder*, participants with BN also tended to suffer from a variety of co-morbid psychiatric disorders. This factor was made up of variables such as co-morbid depression and anxiety. These variables matched those risk factors in Fairburn and Cooper’s (1984) General Psychiatric factor. The second and third factor within Tobin’s (1991) study, *Bulimic* and *Restricting Behaviours*, suggested that participants with BN exhibited dieting

behaviours, weight control difficulties and over concern with body shape. These risk factors were similar to the variables in the second factor identified by Fairburn and Cooper (1984), the Dieting and Obesity factor. Although the results were not directly comparable because of differences in study methodologies, the similarity in factors between these two studies provided strong support for the relevance of these two factors in BN pathology.

These early factor analytic studies were limited because only a few potential risk factors for BN were assessed. None of the above mentioned studies examined the role of the family and the environment in BN. Fairburn et al. (1997) included a comprehensive list of risk factors in which family relationships and life events were assessed as possible risk factors along with personality characteristics, co-morbid psychiatric illness, personal weight histories, etc. Fairburn et al. (1997) interviewed 102 participants with BN and compared them with 204 non-clinical control participants and 102 psychiatric control participants. One of the noteworthy methodological features of this study was the use of comparison groups. The bulimia group was compared to a group of non-clinical controls and to a group of psychiatric controls. This methodological approach allowed Fairburn et al. (1997) to define the combination of risk factors that were associated with BN specifically.

Prior to analyses, Fairburn et al. (1997) grouped all of the possible risk factors in the study into three major factor classes. The first two factor classes, the General Psychiatric factor and the Dieting and Obesity factor, were derived from Fairburn and

Cooper's (1984) study. The third factor class focused on the family relationships and environment of the individual.

Instead of testing an overall statistical model for these factors, Fairburn et al. (1997) performed chi-square comparisons on each of the three factors in order to differentiate the three groups of participants. Overall, univariate comparisons showed that the participants with BN had significantly greater exposure to each of the three major factor classes than the participants in the non-clinical control group.

Univariate comparisons between the bulimia group and the psychiatric group revealed that these two groups were quite similar. Both groups reported high levels of co-morbid psychiatric illness, as well as personality vulnerabilities such as affective instability and low self-esteem. The univariate results did show a significant difference between the bulimia group and the psychiatric control group on the Dieting and Obesity factor. The bulimia group had much higher scores than the psychiatric group on items such as critical comments about shape or weight by peers and family members, as well as obesity history. Therefore, the General Psychiatric factor separated the non-clinical control group from the two clinical groups, whereas the Dieting and Obesity factor separated the bulimia group from the psychiatric control group. These two factors helped define risk factors specifically for BN. Fairburn et al. (1997) suggested that the combination of these two factors, namely the General Psychiatric factor and the Dieting and Obesity factor, would be a good framework for conceptualizing the potential risk factors associated with the development and maintenance of BN.

One of unexpected findings within this study was the emergence of a possible third factor in this model. The univariate results for the Family Environment factor revealed that the bulimia group was not significantly different from the psychiatric control group on the overall family factor. But a breakdown analysis of the specific items on the Family Environment factor revealed that the bulimia group was significantly different from the psychiatric group on some items. Specifically, the bulimia group experienced higher parental expectations, and more negative parent-child relationships than the psychiatric control group. Therefore, Fairburn et al. (1997) suggested that the Family Environment factor might be an important risk factor associated with the development and maintenance of BN.

Fairburn et al.'s (1997) results were important because they emphasized the need to include the family environment variables in the understanding of the development and maintenance of BN. However it was unclear whether these family environmental variables would be best understood as a separate factor class or as a part of the General Psychiatric factor. The analysis showed that the participants with BN were not significantly different from the participants in the psychiatric control group on the overall Family Environment factor. However a more detailed examination suggested that these two groups did differ on some of the items on the Family Environment factor. Fairburn et al. (1997) suggested that the Family Environmental factor would be best conceptualized as a separate factor but further research would be required to confirm this postulation.

To date there has been a limited amount of research that has attempted to integrate the list of putative risk factors into broad factor classes. It is clear that more research is

needed that focuses on these broad classes of putative risk factors, their interactions and relationships with bulimic symptomatology. Crowther, Wolf and Sherwood (1992) confirmed the need for this type of research, “Furthermore, rather than investigate these factors in isolation, researchers must develop models that foster understanding and yield testable hypotheses concerning the dynamic interaction among various individual and familial risk factors” (p. 18).

Two major questions grew out of this research. First, how should these disparate risk factors be integrated to create a parsimonious model for the factors associated with the development and maintenance of BN? The present study compared two models that classified the list of putative risk factors in different ways. The first model divided the risk factors into three separate factor classes, the General Psychiatric factor, the Dieting and Obesity factor, and the Family Environment factor. The second model divided the risk factors into only two separate factor classes, the General Psychiatric factor and the Dieting and Obesity factor. In this two-factor model the family environment risk factors were grouped together with risk factors on the General Psychiatric factor. Comparing these two models examined the need for the Family Environment factor as a separate factor class for BN.

The second question was, ‘Given that these risk factors have been shown to be associated with BN, how are they related to the actual symptoms of BN?’ The study by Fairburn et al. (1997) used risk factor groupings to differentiate individuals with BN from individuals with other psychiatric disorders and individuals with no diagnosed disorders. However, so far there have been no studies that have explained what these risk factors

have to do with the actual symptoms of BN. The present study attempted to answer this question by examining the relationships between the risk factor classes and three hallmark characteristics that have often been used to define BN.

Risk Factor Variables

Although it is necessary to explore the theory that will serve to develop the model and hypotheses for this study, the research for the specific risk factor variables will first be examined. For the sake of clarity, the literature for each disparate risk factor will be reviewed in order, starting with the variables that made up the General Psychiatric factor, followed by the Dieting and Obesity factor, and finally the Family Environment factor. A review of the theory underlying the factor classes will be presented in Chapter 3.

General Psychiatric Factor

Certain risk factors associated with the development of BN are more appropriately understood as risk factors that increase the likelihood of the development of a psychiatric disorder in general. For example, a history of affective instability is seen as a good predictor not only for the development of an eating disorder, but also of the development of a variety of anxiety and mood disorders (Lilenfeld, Kaye, Greeno, Merikangan, Plotnicov, Pollice, Radhika, Strober, Bulik & Nagy, 1998). Thus, these putative risk factors tend to predict the potential onset of some type of psychological problem, but cannot be used as the sole indicators for the development of an eating disorder such as BN.

Co-morbid Psychiatric Disorders

Bulimia nervosa is often accompanied by other co-morbid psychological illness such as depression and anxiety disorder (Lilenfeld et al., 1998). Although much of the research to date has focused on eating disorders in general, there is evidence to suggest

that these psychological problems also accompany BN (Bulik, 1987; Leon et al., 1997; Wonderlich, Swift, Slotnick & Goodman, 1990).

A variety of studies have examined the degree of co-morbid psychiatric illness in patients diagnosed with BN. Halmi et al. (1991) interviewed participants with two subtypes of AN, restricting and bulimic type (N=62). The results showed that 84% of all of the participants had a lifetime history of some kind of affective disorder. The most prevalent co-morbid diagnosis was major depression, which tended to be most prevalent in the anorexia-bulimic group. Lilenfeld et al. (1998) compared participants with restricting AN (N=26) and with BN (N=47) to a group of non-clinical controls (N=44). The results showed that co-morbid depression and anxiety were significantly higher in participants with AN and BN as compared to non-clinical controls. Mangweth, Pope, Hudson and Biebel (1996) compared women with BN from Austria and the United States to two groups of non-clinical controls from corresponding countries (N of 33 in each group). Results showed that the participants with BN from both countries experienced significantly more depression and anxiety disorders than the non-clinical control participants. Thus anxiety related disorders and depression are common co-morbid psychiatric illnesses in individuals diagnosed with BN.

Co-morbid Substance Abuse

Several studies have examined the degree of co-morbid substance abuse in individuals with BN. Bulik (1987) compared patterns of drug and alcohol abuse in 35 participants with BN to a group of non-clinical control participants (N=35). In the bulimia group, 49% of the participants reported alcohol abuse and 34% of the participants

reported some form of drug abuse. These rates were significantly higher than the non-clinical control group whose rates of alcohol and drug abuse were 9% and 3% respectively. Lilenfeld et al. (1998) examined the rates of drug and alcohol abuse in participants with BN, restricting AN and a non-clinical group. The results showed that although the drug and alcohol rates were similar for participants in both eating disordered groups, their rates were significantly higher than the drug and alcohol abuse rates in the non-clinical control group. Mangweth et al. (1996) compared rates of drug and alcohol abuse in participants with BN and participants in a non-clinical control group from Austria and the USA and found 46% of American participants with BN reported a substance abuse or dependence problem. This rate was significantly higher than all other groups. These studies have suggested that drug and alcohol abuse are not only common co-morbid problems in the general psychiatric population but also in individuals diagnosed with BN.

Self-Harm Behaviours

There is some evidence to suggest that self-harm behaviours are prevalent not only in the general psychiatric population but also in the BN population (Lacey, 1993; Welch & Fairburn, 1996). Root and Fallon (1989) suggested that self-mutilation behaviours could be understood as inappropriate mechanisms for pain management that helped to reduce tension and provided relief from emotional turmoil. Favaro and Santonastaso (1998) suggested that self-harm behaviours may not only 'reduce tension' but might also be a way to "experience one's body, to seek a sense of reality and identity" (p. 163). Because purging behaviours can be considered self-injurious acts, self-harm

behaviours have been understood as possible risk factors for not only psychiatric disorders in general but also for BN (Favaro & Santonastaso, 1998).

Welch and Fairburn (1996) compared 102 participants with BN, 102 participants with general psychiatric disorders and a 204 non-clinical participants . Deliberate self-harm by cutting or repeated burning was found to be significantly more prevalent in participants with BN (26%) as compared to participants in the non-clinical control group (2%) and participants in general psychiatric control group (11%). Favaro and Santonastaso (1998) examined self-cutting and burning behaviours, hair pulling and nail biting in 125 women with BN. The results showed that 72% of the women reported nail biting and hair pulling and 22% of the women reported skin cutting or burning. These studies have shown that while self-harm behaviours tend to occur among individuals with many different psychiatric conditions, they also occur among individuals with BN.

Suicidal Ideation

Suicidal ideation and suicide attempts are known to occur in the general psychiatric population and in individuals with BN. Suicide attempts have typically not been classified with other forms of self-harm behaviour. Self-harm behaviours are often used to temporarily relieve emotional distress, while suicide attempts are usually intended as cries for help or to end the suffering (Favaro & Santonastaso, 1998).

Russell (1979) found that out of 32 women with BN, 11 of them had made at least one suicide attempt in their lives. Another study revealed that 9 out of 32 patients with BN had made a suicide attempt (Abraham & Beaumont, 1982). In a study by Weiss and Ebert (1983), 40% of participants with BN reported at least one suicide attempt in their

lives. This rate was significantly higher than the suicide rates of participants in the non-clinical normal control group, where 0% of the participants had made an attempt. Overall the results have suggested that suicide and suicidal ideation are not only associated with psychiatric disorders in general but also with BN.

Personality Vulnerabilities

A variety of personality vulnerabilities have been shown to be associated with BN as well as with other general psychiatric disorders. Four personality traits have been singled out as possible risk factors for BN. They include affective instability, negative self-esteem, poor impulse regulation and poor interoceptive awareness (an inability to recognize and respond to emotional and physical states (Garner, 1991)).

Leon et al. (1993) performed a four-year prospective study with 2000 high school girls and boys. The participants reported on their exposure to a variety of risk factors for each year of the study. For both genders, negative self-esteem was the strongest predictor of disordered eating. Weiss and Ebert (1983) studied impulse regulation in 15 participants with BN and 15 participants in a non-clinical control group. The results showed that significantly more participants with BN (67%) admitted to shop lifting than the non-clinical participants (13%). Pyle, Mitchell, and Eckert (1981) showed that 53% of the participants with BN in their study admitted to stealing prior to the onset of the eating disorder. Wonderlich and Swift (1990a) examined the correlations between personality vulnerabilities and bulimic symptoms in participants with BN. The results showed that affective instability and impulsivity were strongly correlated with bulimic symptomatology. This finding was confirmed by a follow up study (Ruderman &

Besbeas, 1992). Stice and Agras (1998), studied 215 adolescents for nine months.

Among the personality variables measured, affective instability and poor interoceptive awareness were found to be significant predictors of the onset of eating disorders. Favaro and Santonastaso (1998) found that virtually all of the participants with BN in their study suffered from some degree of poor interoceptive awareness and poor impulse regulation.

Overall, four personality traits have been shown to be associated with the BN.

They include affective instability, negative self-esteem, poor interoceptive awareness, and poor impulse regulation.

Dieting and Obesity Factor

The variables that make up this factor class are those that have a strong influence on dieting behaviour. Personal feelings about shape and body weight, as well as harsh criticism by peers and family members have been shown to be strong influences on extreme dieting behaviours. Factors that have the potential to give rise to dieting behaviours are important in BN because extreme dieting has been cited as a key factor for the development of the bulimic binge-purge cycle (Polivy & Herman, 1985).

Mother's Preoccupation with Weight and Dieting

Family system theories maintain that interaction between family members can influence the development of dieting behaviours through behavioral modeling processes (Costanza & Woody, 1985). Therefore, dieting behaviours may be greatly influenced by the dieting patterns and weight concerns within the family. The dieting behaviour of the mother has been the focus of this research because both the mother and daughter are more likely to be influenced by social pressure to be thin, than male members of the family

(Pike & Rodin, 1991). Daughters that develop eating disorders often have mothers that have difficulties themselves managing these societal expectations for thinness (Pike & Rodin, 1991; Pyle, Mitchell & Eckert, 1981).

Pike and Rodin (1991) performed a study in which 350 high school girls completed a battery of tests that examined their attitudes about body shape and eating behaviours as well as the strength of family relationships, particularly the mother-daughter dyad. One of the key research interests was to assess the mother's preoccupation with her own weight and the extent to which she dieted. The results revealed that significantly more mothers of daughters with eating disorders had dieted and dieted for longer periods of time than mothers of daughters in the non-eating disordered group (Pike & Rodin, 1991). Mangweth et al. (1996) studied the weight and dieting histories of first-degree relatives of women with BN in Austria and the United States. The results showed that in both countries, relatives of women with BN dieted significantly more than the relatives of non-clinical control participants in both countries.

Overall, these studies have shown that the dieting behaviour in family members is a potential risk factor for BN. More specifically it has been suggested in the literature that the mother's preoccupation with dieting tends to be an important influence on the daughter's dieting behaviour and subsequent development of BN.

Mother's Criticism of Daughters' Weight

Studies have shown that dieting behaviours are also influenced by the mother's criticism about shape and weight (Pike & Rodin, 1991). The mother plays an important role because of her own potential sensitivity to social influences about shape and weight.

The mother that tends to be very sensitive about her own shape and weight is also likely to influence her daughter to maintain low weights through dieting behaviour (Pike & Rodin, 1991).

Pike and Rodin (1991) showed that mothers of daughters with eating disorders had significantly more negative evaluations of their daughters' appearances than a control group of mothers of daughters without eating disorders. Moreno and Thelen (1990) found that mothers of daughters with BN encouraged significantly more dieting and exercise than the mothers of daughters in a non-clinical control group. Of particular interest, this study also examined the role of the father's criticism on daughters' dieting behaviour. The results showed that the fathers were much less likely than the mothers to criticize their daughters about weight. There was no difference in the amount of criticism by the father between the groups with or without eating disorders. Moreno and Thelen (1990) concluded that the mothers played a more pivotal role than fathers in their daughters' eating problems. They argued that criticism has the potential to lead to severe body dissatisfaction and dieting behaviours which are key triggers for the development of binge-purge cycle in BN (Heatherton, Herman & Polivy, 1991; Heatherton & Polivy, 1992; Polivy & Herman, 1985).

Teasing by Peers

The impact of teasing and criticism on severe dieting behaviours have been studied as potential risk factors for BN (Thelen, Lawrence & Powell, 1992). It has been argued that teasing by peers may be an important factor during adolescence because of the strong influence of peer pressure during this age (Levine & Smolak, 1992).

Several studies have shown that peer evaluations are perceived to be more negative in the girls with eating disorders than in girls without eating disorders (Brown Cash & Lewis, 1989; Fabian & Thompson, 1989; Irwin, 1981). Irwin (1981) reported a case-history of a nine-year-old girl with AN who reported that the primary precipitating factor for her eating disorder was the extent to which she was teased about her weight by the boys in her class. Since this study, 'teasing by peers' has been empirically examined as a potential risk factor for eating disorders. Fabian and Thompson (1989) found that negative attention from peers was significantly related to negative body image in premenarcheal girls. Brown et al. (1989) found a link between childhood teasing about appearance and negative body image in a nation wide survey in the United States. Overall it has been suggested in the literature that teasing by peers is a potential risk factor for the development of eating disorders in general. Therefore it is likely that teasing by peers will be a potential risk factor for BN as well.

Personal Obesity

Obesity appears to play an important role in both the risk of dieting and in the development of BN. Because individuals with BN tend to be within a normal body-mass-index (BMI) weight range, it has been argued that these individuals are actually using compensatory purging behaviours to manage a naturally high set-point weight (Fairburn & Cooper, 1982).

Fairburn and Cooper (1982) studied the weight histories of participants with BN and participants in a non-clinical control group. The results showed that significantly more of the participants with BN had been overweight at some point since the onset of

menarche, as compared to the non-clinical control group. Johnson, Stuckey, Lewis and Schwartz (1982) studied the retrospective weight histories of participants with BN, and found that 40% of them reported having been overweight in the past. Patton (1988) compared 21 high school girls with AN and 22 girls with BN to an equal number of non-clinical participants. The results showed that a history of obesity along with a history of depression could predict the severity of eating disorder symptoms. Overall these studies have shown that a history of obesity or overweight is a potential predisposing risk factor in BN.

Family Environmental Factor

The risk factor research for BN has also examined the role of social risk factors in the development of BN. Much of the emphasis has been put on the role of the family and the family environment.

Parent-Child Interactions

The role of family factors in the development of eating disorders in general were not formally investigated until the mid 1980s. Speculation around the development of AN and the early mother-daughter relationship as well as broader systems perspectives were discussed (Jacob, 1975; Minuchin, Rosman & Baker, 1978; Palazzoli, 1978) but very little data existed to support this early work.

Strober et al. (1985) were the first to examine the nature of the family environment in AN using standardized scales. The findings revealed that individuals with AN tended to come from families with difficulties in conflict management and affective expression. A more detailed comparison revealed that participants with

bulimic-type AN reported higher levels of conflict interaction and negative self-expression within the family than participants with restricting-type AN. Furthermore, parents of participants with bulimic-type AN were reported to have significantly more marital discord and less closeness with the children as compared to the parents of the participants with restricting-type AN. These findings suggested that while environmental family difficulties were common to eating disorders in general, the type of family environmental problems might contribute to the specific type of eating disorder that developed.

Johnson and Flach (1985) examined the family environment of 105 participants with BN. The results revealed that the participants with BN perceived their families as being less supportive, helpful and generally less involved as compared to participants in a group of non-clinical controls. Participants with BN reported that their families were highly critical and had higher expectations as compared to the participants in the non-clinical control group.

Overall, it has been suggested in the literature that the family environment of individuals with BN tends to be one in which the parent-child interaction is poor and riddled with conflict. Therefore a poor parent-child relationship has been suggested to be a potential risk factor for BN.

Supportive Relationships

A perceived lack of social support from both family members and close friends is often seen in individuals with BN (Fairburn et al., 1997). This lack of supportive

relationships has been seen as a potential risk factor for eating disorders and for BN specifically (Johnson & Connors, 1987).

Norman and Herzog (1984) studied participants diagnosed with BN, alcoholism, depression or schizophrenia. The participants responded to questions on a social adjustment scale. The scale was designed to capture life maladjustments such as poor social relationships, stress and coping. The t-tests revealed that the participants with BN were significantly better adjusted than participants with severe clinical depression. However the participants with BN were significantly less well-adjusted than the participants with schizophrenia or alcoholism. Johnson et al. (1982) found that 68% of a community sample of 316 individuals with BN reported that their eating disorder interfered with interpersonal relationships. Johnson and Larson (1982) studied social isolation in participants with BN compared to participants in a non-clinical control group. Participants reported whether or not they were alone, when randomly signaled by a beeper. The participants with BN were found in isolation significantly more times (49%) than the non-clinical control participants (32%). Overall the results have suggested that a lack of supportive relationships and isolation are potential risk factors for BN.

Life Events

The family research on eating disorders has also stressed the importance of the association between disruptive life events and the development and maintenance of the eating disorder (Schmidt et al., 1993; van der Kolk, 1996). Although the families of individuals with BN have often been typically described as 'chaotic' and poorly

supportive (Shisslak, McKeon, & Crago, 1990), there is some evidence to suggest that abrupt life changes are also common to individuals suffering from BN.

Schmidt, Tiller, and Treasure (1993) examined the family environments as well as the disruptive family events of participants with BN, AN and non-clinical controls. The results revealed that participants with BN experienced significantly more changes and disruptive events in their lives as compared to participants with AN or participants from the non-clinical control group. Levine and Smolak (1992) devised a model of eating disorders that examined life stressors in the form of poor relationships and disruptive life events. The results of the regression analysis revealed that disruptive life events such as death of a parent and family and personal illness were found to be significant predictors of eating disturbances. Overall, disruptive life events and life stressors have been shown to be potential risk factors for BN.

Abuse

The issue of sexual and physical abuse in the development of eating disorders began receiving considerable attention following the interest in the role of the family environment in the development of eating disorders. Both aspects of abuse have been shown to be risk factors for general psychiatric conditions but they have also been studied as potential risk factors for the development of BN (Welch & Fairburn, 1994).

The typical families of individuals diagnosed with BN have been described as having difficulty in conflict resolution and a tendency toward high levels of anger and violence (Strober, 1981). This description differs considerably from the typical family environment of an individual diagnosed with AN, where the tendency is toward conflict

avoidance (Strober, 1981). It is for this reason perhaps, that the incidence of physical abuse for individuals with BN has often been found to be significantly higher than for individuals with AN (Strober et al., 1985). Johnson and Connors (1987) described a case history of a 32-year-old woman with BN and co-morbid borderline personality disorder. An examination of her childhood revealed that she had suffered from excessive physical abuse by her father. This apparently led to the development of the self-destructive need to binge and purge and was cited to be a key risk factor for the development of her eating disorder (Johnson & Connors, 1987). Schmidt et al. (1993) examined the rates of physical abuse for participants with BN and participants with AN, finding that both violence against a family member and violence against the participant were significantly more prevalent in the participants with BN. They therefore suggested that physical abuse was a potential risk factor for the development of BN.

The role of sexual abuse in the development of an eating disorder has also been examined. The plausibility for this link was supported because of the similar personality characteristics between victims of sexual abuse and individuals with eating disorders, including low self-esteem, and negative body image (Oppenheimer, Howells, Palmer & Chaloner, 1985). Welch and Fairburn (1994) conducted a community-based study in which 50 participants with BN, 50 participants in a psychiatric control group and 100 participants in a non-clinical control group responded to questions about sexual and physical abuse. The results showed that the rates of sexual abuse were significantly higher for both the BN group and general psychiatric group as compared to the non-clinical group. There was no difference between rates of sexual abuse in the participants

with BN and the participants in the psychiatric control group. Schmidt et al. (1993) found that sexual abuse in childhood involving a family member was significantly higher in participants with BN as compared to participants with AN.

The overall results have indicated that individuals with BN report significantly higher rates of sexual abuse than do individuals with no diagnosed clinical disturbance (Oppenheimer et al., 1985; Welch & Fairburn, 1994). Sexual abuse has been shown to be a potential risk factor for BN as well as the general psychiatric population.

Conclusion

An examination of the current literature detailing putative risk factors for BN clarifies the need for a model that simplifies the understanding of BN development and maintenance. Although many risk factors have been examined, there is a paucity of literature that describes a comprehensive view of these potential risk factors, how they interact and how they contribute to bulimic symptomatology. The purpose of this study was to conceptualize the disparate risk factors into heuristic factor classes and then to examine the way these factor classes interact and relate to the symptoms of BN.

Chapter III

Theory Development and Hypotheses

Socio-cultural Perspective

Just a century ago, the preferred female form was quite opposite to the one that is idealized today. Dimpled flesh, and rounded hips and buttocks were characteristics in a woman that were considered desirable. "Fat was seen as a 'silken layer' that graced the frames of elegant ladies" (Seid, 1994, p. 5). More importantly, plumpness was seen as a sign of good mental and physical health. Today the perception of fat is quite the opposite. Not only are rounded curves and cellulite seen as unattractive and unappealing, fat is seen as unfit and unhealthy. The underlying assumption is that overweight individuals simply lack the will power and self-control to reduce their food intake. "Indeed, in modern America, being fat is as shameful as being dirty"(Seid, 1994, p. 7).

The fat-phobic North American culture affects women more profoundly than it does men (Perlick & Silverstein, 1994). One of the reasons that women struggle with weight control more than men is because of the extreme difference between the socially desired and realistically attainable female body shapes and weights (Seid, 1994). The emerging weight and shape standards idealize narrow hips, thin thighs and body fat contents that are genetically impossible for most women (Levine & Smolak, 1992). Because of this unattainable goal, women are in a constant 'pursuit of thinness' (Streigel-Moore, Silberstein & Rodin, 1986).

Despite the growing feminist movement that emphasizes the importance of internal strength and beauty, woman are still being judged and judging themselves based

on their external appearance (Seid, 1994). Some theorists have argued that the social expectation for thinness is a counter attack on the growing feminist movement (Wolf, 1994; Wooley, 1994). Noami Wolf suggested,

“The great weight shift must be understood as one of the major historical developments of the century, a direct solution to the dangers posed by the women’s movement and by economic and reproductive freedom. Women’s advances had begun to give them high self-esteem, a sense of effectiveness, activity, courage and a clarity of mind. Prolonged and periodic caloric restriction is a means to take the teeth right out of this revolution” (p.97).

As a result of this constant and unattainable ‘pursuit of thinness’, women are in a constant struggle to maintain low weights through dieting and exercise (Garner, Garfinkle, Schwartz & Thompson, 1980; Garner, Rockert, Olmstead, Johnson, & Coscina, 1985). When weight ideals still cannot be achieved the resulting personal evaluation is of poor self-control, low self-esteem and low self-worth (Heatherton & Polivy, 1991; Polivy, Garner & Garfinkle, 1986).

This socio-cultural perspective provides some insight into the increase in prevalence rates for eating disorders among young adult women over the last several decades (Garner, Garfinkle & Olmstead, 1983; Garner, Garfinkle, Schwartz & Thompson, 1980; Mazur, 1986). The unrealistic social expectation for thinness has the potential to contribute to the development of eating disorders for women living within North American society. However it is important to note that not all women living in this socio-cultural environment develop an eating disorder, even though these weight and

shape ideals exert a relatively constant influence on all North American women (Rosch & Sheperd, 1992). Therefore, Rosen (1992) proposed that this socio-cultural perspective can not be considered a specific risk factor for BN. Instead it is best understood as a context or a foundation for other specific risk factors to interact and contribute to the development and maintenance of BN (Crowther & Mizes, 1992). Garner et al. (1983) suggested that while 'psychologically robust' individuals respond favorably to the increased social pressures to be thin, those individuals faced with additional and or alternative negative pressures might find the new role expectations overwhelming and destabilizing. It is these alternative negative pressures that specifically contribute to the development of an eating disorder such as BN (Garner et al. 1983). Therefore, while societal influences lend themselves to the development of eating disorders for young women, they are best seen as a context or a framework that provide support for other specific risk factors in the development and maintenance of BN (Rosch & Sheperd, 1992; Rosen, 1992).

The risk factor studies to date have examined the associations between the putative risk factors and BN. However, the empirical research has not examined the theoretical foundations for these relationships. For the purposes of model development within this thesis however, it is important to examine the theories that describe and explain BN development. It should be noted, that the purpose of this section is not to prove or disprove any aspect of the proposed theories, but to examine them in an effort to generate the risk factor models and a set of hypotheses for this study.

The Psycho-Biological Impasse

In order to examine the risk factors involved in the development and maintenance of BN, one must first examine the psychological factors that contribute to this disorder. This is important because psychological factors play a prominent role in various eating disorders (Polivy & Herman, 1993).

Psychological factors such as depression and low self-esteem have the potential to leave an individual feeling emotionally vulnerable and fragile (Johnson & Wonderlich, 1992). An emotionally vulnerable individual may be more influenced by the socio-cultural expectation for extreme thinness than someone that does not suffer from this type of emotional vulnerability. Therefore, although most women do not achieve the ideal female body shape or weight, women who are faced with additional psychological vulnerabilities may have more difficulty coping with this and consequently experience extreme body dissatisfaction and negative self-evaluation (Herman & Polivy, 1988). These negative emotions have been shown to lead to episodes of binge eating and severe dieting behaviours that are major triggers for the bulimic binge-purge cycle (Leon et al., 1993).

The stress created from feelings of inadequacy and low self-esteem has been seen as a primary trigger for binge eating behaviour (Attie & Brooks-Gunn, 1987; Hirsh & Rapkin, 1987; Pyle, Mitchell & Eckert, 1981). Heatherton and Beaumeister (1991) argued that distress brought about by feelings of low self-worth leads to unavoidable and painful feelings of self-awareness. The individual seeks to escape the suffering by narrowing her cognitive focus to concrete stimuli such as food. A focus on food shifts

this focus away from emotions and cognitions that are painful and unappealing.

However, once higher level cognitions are suppressed, the signals are removed that indicate satiety and normally stop eating behaviours. As a result, eating continues beyond a 'normal' level (Heatherton & Beaumeister, 1991). Within this context, binge eating has been seen as an adaptive response to excessive distress brought about by psychological factors such as low self-esteem (Heatherton & Polivy, 1992; Polivy & Herman, 1993).

The "psycho-biological impasse" is a term that has been used to describe the competing biological and psychological management systems in individuals with BN (Johnson & Connors, 1987). Binge eating has not only been seen as an adaptive response to psychological distress, but also as a biological compensatory response. Typically, individuals with BN who experience extreme body image dissatisfaction accompanied by low self-esteem are driven, at least temporarily, to engage in severe dieting and/or excessive exercise (Heatherton, Herman & Polivy, 1991). By achieving a weight loss at or below the body's set point, the self-esteem deficits are compensated for, however a variety of physiological side effects occur. The body's effort to prevent starvation or at very least to maintain a predetermined set-point weight, results in a relentless drive to consume calories and reestablish its base-line fat content. At this point the individual is at a 'psycho-biological impasse' (Johnson & Connors, 1987).

The relationship between dieting and binge eating was examined by Polivy and Herman (1985). Using a prospective methodological approach they established that chronologically, dieting typically preceded binge eating behaviours. They therefore argued that dieting is better seen as the cause of binge eating, instead of the reverse

relation. Johnson and Connors (1987) argued that the individual who establishes self-esteem through weight loss, is threatened by the body's need to reestablish its set-point weight through binge eating. As a result, the two systems begin compensating for one another through the development of a binge-purge cycle, leaving the individual feeling inadequate and out of control (Johnson & Connors, 1987).

This psycho-biological struggle is even more dramatic when a woman's set-point weight is somewhat above average. Dieting and Obesity risk factors are especially important for these individuals. Individuals who have a naturally high set-point weight will struggle with dieting and exercise just to maintain a 'normal' fat level. Because individuals with BN tend to be of normal weight despite their purging behaviours, it has been suggested that these individuals are struggling with a higher natural set-point weight (Fairburn & Cooper, 1982). When a 'normal' weight is achieved, the body attempts to regain the higher set-point weight through binge eating behaviour. The binge eating creates distress for the individual because it threatens the weight loss and the subsequent feelings of heightened self-esteem. In order to reduce the distress, the individual begins to engage in compensatory behaviours such as purging, once again establishing the binge-purge cycle (Johnson & Connors, 1987).

The psycho-biological impasse theory is one that accommodates many of the putative risk factors for BN. It lends support to the two well established factors, the General Psychiatric factor and the Dieting and Obesity factor. The psychological factors included in the psycho-biological impasse theory, such as low self-esteem and self-worth, high anxiety and depression, suggest a grouping of risk factors that might put an

individual at risk for a psychiatric disorder in general. Those risk factors will be examined within the General Psychiatric factor in the current study. When an individual is exposed to a variety of these risk factors, within the socio-cultural context that promotes unrealistically thin ideals, a second group of factors that predict the risk of dieting emerge. The Dieting and Obesity factor includes difficulties with obesity, teasing by peers, as well as observed dieting behaviours in other family members. When an individual is exposed to both of these general risk factors in some combination, the risk of the development of BN is greatly increased (Johnson & Connors, 1987; Patton, 1988).

Although these theories account for many of the putative risk factors that have been examined in BN research, the risk factors that describe the family environment have been left unexplained. The following section will review the theories that emphasize the importance of Family Environment risk factors in BN development and maintenance.

Developmental theory

The empirical research that has examined the family environment of individuals with BN has described the typical 'bulimic family' as one that is chaotic, disorganized, unemotional and non-supportive (Johnson & Flach, 1985). However two criticisms of these family descriptions are of note. First, most risk factor studies that have examined family relationships have been retrospective and have been based solely on the patient's perception of those relationships (Wonderlich & Swift, 1990b). Therefore the typical family description as chaotic and disorganized is largely retrospective and subjective. Second, this description of the typical family of an individual with BN does not take into account the reciprocal nature of family relationships. Although the relative contribution

to the parent-child relationship is more strongly influenced by the parent, it is important to recognize the interaction between the parent and child and the way that they influence one another. Scarr (1987) pointed out that differences in parent-child relationships are largely due to the match between the personality of the parent and the temperament of the child. If these personalities clash the resulting parent-child relationship may be a negative one in which the parent and child contribute to the relationship in a reciprocal fashion.

Attachment theory (Bowlby, 1977) is useful for examining the role of the family in the development of eating disorders because it emphasizes the reciprocal nature of parent-child relationships. Attachment theory was originally developed in order to understand normal childhood development. Bowlby (1977) postulated that a secure attachment between a parent and child provides the foundation for the child's future development. He suggested that a secure attachment develops from the parents provision of love and security. Given a good match with the child's temperament, the child then responds to this loving behaviour by becoming confident, communicative and generally content. The child's response then promotes more loving behaviour from the parent (Bowlby, 1977).

Attachment theory has also been used to understand psychopathological childhood development (O'Kearney, 1995). Ainsworth, Blehar, Waters and Wall (1978) used Bowlby's original attachment theory to identify three distinct attachment styles; secure, avoidant, and anxious-ambivalent. Ainsworth and her colleagues suggested that all three types of infants are attached to their primary care-givers, but the quality of the attachment relationship is the critical difference in the development of psychological problems. The

child that is securely attached to the parent will develop a host of personality traits such as self-confidence and a trusting nature, that will allow the child to forge strong relationships with family members and others throughout his or her life. A child that has an insecure attachment to the parent may not develop these critical personality traits and consequently will have difficulties developing trusting and loving relationships throughout life.

Ainsworth et al. (1978) described the typical interaction process between the parent and child for the two insecure attachment styles. The avoidant infant is characterized by a social withdrawal, a lack of exploring activity and a general lack of response to the mother. The mother in this attachment style usually responds slowly and erratically to the child's cries. This interaction results in an insecure attachment style in which avoidant behaviours are maintained by both the mother and the child in a reciprocating fashion. The anxious-ambivalent infant is characterized by general anxiety, excessive crying and a constant attempt to draw the mother's attention. The mother in this attachment style is typically emotionally intrusive and can force 'affection' on her child at times when it interferes with her child's activities and development. This interaction results in an insecure attachment style in which anxious and ambivalent behaviours are maintained by both the mother and the child in a reciprocating fashion.

Blatt and Shichman (1983) used attachment theory to explore the development of psychological well-being in adulthood. They postulated that healthy psychological development involves two fundamental processes. The first is an ability to develop stable, mutually satisfying and reciprocal interpersonal relationships. The second is to

achieve a differentiated, stable, autonomous sense of self. These two processes describe the interplay between the development of a self-concept as it relates to both independence and autonomy as well as healthy interdependence in personal relationships (Blatt & Blass, 1990). They argued that the attachment styles of early childhood provide the framework for the development of these two fundamental processes for psychological well-being (Blatt & Shichman, 1983).

Blatt and Shichman (1983) argued if these two fundamental processes are not developed in childhood as a result of an insecure attachment style, the child will have difficulty developing strong, meaningful and trusting relationships in adulthood, and will fail to develop self-esteem and autonomy. Deficits in both of these fundamental processes are seen in many psychological disorders such as borderline personality disorder, depression and anxiety disorder, as well as BN (Blatt & Shichman, 1983).

Chassler (1998) examined the role of the attachment style and the development of BN more closely. Individuals suffering from low self-esteem and poor self-image as a result of an insecure attachment style in childhood, are at risk of developing a psychiatric disorder in general. This disturbance in psychological well-being in a society that promotes extreme thinness in women, will have the potential to give rise to an eating disorder such as BN.

Integrating Theories

Many theories exist that describe the way a specific group of risk factors influence the development of BN. However, there is a paucity of literature that integrates these theories to include all of the putative risk factors, their interactions and their relationships

with BN symptomatology. The psycho-biological impasse theory emphasizes the importance of psychological factors that might predispose an individual to a psychiatric disorder in general. The psycho-biological impasse theory also emphasizes the importance of biological predisposition to obesity and other risk factors than can influence dieting. The attachment theory proposes that family relationships and social environments are also important for the development of BN. By integrating these two eating disorder theories a more comprehensive conceptualization becomes available for understanding the development and maintenance of BN. These theories support the notion that three major risk factor classes are involved in BN development, the General Psychiatric factor, the Dieting and Obesity factor and the Family Environment factor.

It is interesting that although each of these individual risk factors alone do not seem to give rise to BN, a combination of some or all of these biological, family, and dieting risk factors have the potential to be involved in the development and maintenance of BN. Hsu, Chesler and Santhouse (1990) suggested that “dieting provides the entree into an eating disorder but since the majority of those who embark on a diet do not develop an eating disorder.... genetic, psychological, biological, personality and family factors may increase the vulnerability of a dieting individual to an eating disorder” (p. 276).

Hypotheses and Model Development

The theories for the development and maintenance of BN have provided an heuristic conceptualization of the risk factors that have been studied. They outlined the importance of the three major classes of risk factors; the General Psychiatric factor, the

Dieting and Obesity factor and the Family Environment factor. However this theoretical conceptualization has not been empirically tested. Furthermore there has been very little research that has examined the way that these risk factors might interact and relate to bulimic symptomatology.

Hypothesis A- Is the Family Environment a Separate Class of Risk Factors

Much of the previous literature has supported the conceptualization of the first two major factors classes in the development and maintenance of BN (Fairburn & Cooper, 1984; Tobin et al., 1991). However, the concept of the Family Environment factor still remains relatively unexplored even though the eating disorder theories have suggested the three major factor classes in the development and maintenance of BN. Furthermore because the developmental theory emphasized the importance of the family in BN, it was assumed that the Family Environment factor was best understood as a separate factor.

In order to examine the conceptualization of the Family Environment factor as a separate risk factor group, a confirmatory factor analysis was performed to compare the two and three-factor models (See Figure 1 and 2). It was hypothesized that a) the variables associated with the development and maintenance of BN would be multi-dimensional, and b) the three-factor model involving a separate factor for family environmental variables would provide a better fit than the two-factor model.

Figure 1: Three-Factor Model to Conceptualize Risk Factors Associated with Bulimia Nervosa

Note: E1 through E20 represent the errors associated with the corresponding risk factor variables

Figure 2: Two-Factor Model to Conceptualize Risk Factors Associated with Bulimia Nervosa

Note: E1 through E20 represent the errors associated with the corresponding risk factor variables

Hypothesis B- How Do the Risk Factor Classes Relate to Bulimic

Symptomatology?

It was unclear how each of these untested risk factor classes contributed to the development and maintenance of BN. The eating disorder theories suggested that each of these risk factor classes would be important predictors for BN. The Psycho-Biological Impasse theory suggested that both General Psychiatric factor and the Dieting and Obesity factor would be important predictors for BN. The developmental theories suggested that the Family Environment factor would also be an important predictor of BN. It was therefore hypothesized that all three risk factor classes would significantly predict bulimic symptomatology.

Chapter IV

Method

Data

All data were taken from an archival database, from the Regional Center for the Treatment of Eating Disorders at the Ottawa Hospital (General Campus).

Participants

Four-hundred and eighty women, referred to the Regional Center for the Treatment of Eating Disorders at the Ottawa Hospital (General Campus) by community psychiatrists and family doctors, were candidates for this study. Among these 480 potential participants, 92 women, who met the diagnostic criteria for BN (purging type) according to the DSM-IV (APA, 1994), were participants in this study. The minimum age for these participants was 18, and there was no upper age limit.

The participants were between 18 and 51 years of age. The mean age was 29.77 years ($SD=8.89$) with a median of 29 years. The average body mass index was 26.76 ($SD=7.0$), which was considered to be just slightly higher than the 'normal' healthy range. This was consistent with BMI scores from other studies for BN (Fairburn & Cooper, 1982; Fairburn et al., 1997). Participants reported to have suffered from some type of eating disorder for 10.87 years ($SD=7.75$).

Procedure

An initial consultation was performed for each patient by a psychiatrist or a clinical psychologist within the treatment program. This consultation phase consisted of a semi-structured interview in which previous medical history, family history, eating

behaviours and psychiatric functioning were assessed, in order to determine if an active eating disorder was present. Each patient was given a multi-axial diagnosis according to the DSM-IV (APA, 1994). Those patients who were not diagnosed with an eating disorder were typically referred elsewhere for individual psychiatric counseling. Participants who were diagnosed with an eating disorder according to the DSM-IV, were given one of the following diagnosis; AN (restricting type), AN (binge/purge type), BN (purging type), BN (non-purging type), binge eating disorder, or eating disorder not otherwise specified. Following the initial assessment, a variety of treatment options were discussed including an educational program, a day treatment program, individual counseling, out-patient group therapy, and other community resources.

Following the initial consultation phase all participants were asked to complete a battery of psychological tests including the Eating Disorders Inventory-2 (EDI-2; Garner, 1991), the Personality Assessment Inventory (PAI; Morey, 1991), and the Ottawa Diagnostic Survey of Eating Disorders (DSED; adapted from Johnson & Connors, 1987).

As a check on inter-rater reliability between clinicians, a random selection of 10% of all the consultations were reevaluated by an independent clinician blind to the original diagnosis. Inter-rater reliability was found to be 86%, which indicated a high level of agreement (Demidenko, 2000).

The present study considered only participants diagnosed with BN (purging type). The participants diagnosed with BN (non-purging type) were not included for several reasons. First the sample size for these participants was considerably less than the BN (purging type) group, (N of 15). Second, research has suggested that the etiological

issues surrounding these two diagnoses are quite different (Bruch, 1961; Cooper & Fairburn, 1983). As this study focused on the theoretical models for purge-type BN, only the data for those participants assessed with (purge-type) BN were examined.

Standardized Assessment Tests

Personality Assessment Inventory (PAI)

The Personality Assessment Inventory (Morey, 1991) is a relatively new standardized test that was designed to provide a comparative personality profile for non-clinical and clinical populations. It was designed to assess a broad range of attitudinal, experiential and behavioral characteristics that have been shown to be relevant in contemporary diagnostic practice. It has recently become a well accepted tool and is considered comparable to, and even an improvement upon the Minnesota Multiphasic Personality Inventory (Morey, 1991).

The PAI is comprised of eleven clinical scales that measure a variety of personality characteristics and general psychiatric conditions. These clinical scales include Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression, Mania, Paranoia, Schizophrenia, Borderline Features, Antisocial Features, Alcohol Problems, and Drug Problems. Each clinical scale is derived from 3 to 4 subscales. There are also five treatment scales designed to assess severity and response potential to treatment. These treatment scales include Aggression, Suicidal Ideation, Stress, Nonsupport and Treatment rejection. There are two interpersonal scales designed to assess social interaction. These interpersonal scales include dominance and warmth. Finally there are four validity scales designed to assess response bias due to changes in mood, and or

desire to represent oneself in a particular light. These validity scales include Inconsistency, Infrequency, Negative Impression Management, and Positive Impression Management.

The PAI was written at a grade four reading level and was designed for the clinical assessment of individuals ranging from age 18 through to adulthood. Response norms were based on a standardization sample of 1000 adults in the United States who matched consensus projections based on race, gender and age. These standardized averages provide the 'normal' response set, upon which comparative scores are assessed.

This particular standardized test has been shown to have good internal and external reliability based on alpha coefficients and alternative major instrument comparisons such as the Beck Depression Inventory, Maudsley Obsessive-Compulsive Inventory and the Hamilton Rating Scales for Depression (Morey, 1991). This test is also particularly useful because of four point response range which provides the necessary variability for many statistical analyses (Morey, 1991).

For the present study the PAI was used to measure many of the general psychiatric risk factors such as indices of depression and anxiety, indices of substance abuse problems, as well as some the relevant personality risk factors including affective instability and tendencies toward self-harm and suicidal ideation.

Eating Disorders Inventory-2

The Eating Disorders Inventory -2 (Garner, 1991) is a well accepted standardized test which was designed to assess eating disorder symptomatology and pathology. More

specifically it was designed to measure attitudes and behaviours revolving around eating, weight and shape that are typically associated with AN and BN.

The EDI-2 is comprised of three original subscales that specifically focus on weight and shape concerns. These include Drive for Thinness, Bulimia, and Body Dissatisfaction. There are also five subscales that are designed to assess the psychological traits that are directly related to eating disordered attitudes and behaviours. These include Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness, and Maturity Fears.

The normalized scores for the EDI-2 were derived from a sample of a variety of psychiatric groups, including those individuals with BN and AN, as well as a group of non-disordered female college students. The scores for each of these groups allow for clinical comparisons with eating disordered populations, general psychiatric populations and a normal college population.

Several studies have shown that the EDI-2 is strong in internal and external validity (Garner, 1991) as well as reliability (Garner & Olmstead, 1984). This test is also particularly useful because of its four point response range that provides the necessary variability for many statistical analyses.

For the present study this standardized test was used as a measure of some of the risk factors of personality vulnerabilities such as interoceptive awareness, self-esteem, and impulse regulation.

Diagnostic Survey for Eating Disorders (DSED)

The Diagnostic Survey for Eating Disorders (DSED; adapted from Johnson & Connors, 1987) is a self-report questionnaire that was designed to capture participants' demographic information as well as a wide range of information regarding behavioral and family history. The specific DSED used by the Ottawa Hospital Regional Center for the Treatment of Eating Disorders was adapted from an earlier version by Johnson and Connors (1987). It measures specific risk factors such as teasing by peers, family history of various psychiatric disorders, history of sexual and physical abuse, and social-environmental difficulties associated with the onset of the eating disorder.

The remaining risk factors under examination for the present study were assessed using this questionnaire. This particular variation of the DSED was designed to explore general life risk factors. It is not a standardized test but was created in order to attain a qualitative overview. Several modest modifications were made to the original DSED (Johnson & Connors, 1987), however we do not feel that these modifications have in any way altered the integrity of this survey.

Independent/ Exogenous Variables

The observed variables for this study were derived from the PAI (Morey, 1991), The EDI-2 (Garner, 1991), and the revised version of the DSED (Johnson & Connors, 1987). This section will review the variables that were used in this study, where they were derived from and their intended interpretation. Table 1 contains information about the number of items on each variable and the associated alpha reliabilities taken from the PAI (Morey, 1991) and EDI-2 (Garner, 1991) manuals.

Table 1

Reliability of the Exogenous and Endogenous Variables

	Number of Items	Alpha Reliability
Index for Related Disorders		
Anxiety Subscale	24	0.90
Depression Subscale	24	0.87
Index of Substance Abuse		
Alcohol Abuse Subscale	12	0.84
Drug Abuse Subscale	12	0.74
Affective Instability	6	0.71
Self-Esteem	10	0.90
Interoceptive Awareness	10	0.83
Impulse Regulation	11	0.77
Index of Self-Harm Behaviours	6	0.62
Suicidal Ideation	12	0.85
Non-supportive Environment	8	0.72
Stress	8	0.74
High Expectations	2	N/A
Parent-Child Relationships	2	N/A
Disruptive Life Events	9	N/A
Abuse	2	N/A
Negative Relationships	6	0.63
Mother's Preoccupation with Dieting	1	N/A
Mother's Criticism of Daughters' Weight	1	N/A
Childhood Weight	1	N/A
Teenage Weight	1	N/A
Teasing By Peers	1	N/A
Drive For Thinness	7	0.83
Body Dissatisfaction	9	0.92
Bingeing/Purging	7	0.86

General Psychiatric Risk Factors

Index for Related Disorders: This variable was derived from two subscales on the PAI. The scores for this variable were calculated by averaging depression and anxiety index scores for each participant. It was intended to be an index of the disorders that are related to BN. This index was designed to measure the clinical features common to the experience of anxiety and depression. The item content ranged across various features of anxiety and depression including subjective feelings of apprehension and stress, ruminative worry, pessimism and negative expectations, unhappiness and apathy (Morey, 1991). A high score on this variable indicated that the participant was extremely anxious, experienced excessive rumination, and had feelings of hopelessness and sadness.

Index of Substance Abuse: This variable was derived from two subscales on the PAI. The scores for this variable were calculated by averaging alcohol abuse and drug abuse index scores for each individual. This index was designed to measure clinical features associated with drug and alcohol abuse such as loss of control, drug and alcohol related cravings as well as the frequency and type of use of these substances (Morey, 1991). A high score on this variable indicated that the participant experienced great difficulty in controlling urges to use drugs and alcohol in excess.

Affective Instability: The scores for this variable were taken directly from the PAI. This scale was designed to assess the elements of affective instability as it related to a severe personality disorder. The scale measured the individual's tendency to respond emotionally, experience mood instability, and have difficulty with anger management

and control (Morey, 1991). A high score on this variable indicated that the participant suffered from extreme mood instability and expressed great hostility and poorly controlled anger.

Self Esteem: This variable was taken directly from the EDI-2 where it was called 'ineffectiveness'. This scale was designed to assess feelings of inadequacy, insecurity, worthlessness, and lack of control over one's life (Garner, 1991). In his description of this particular EDI-2 scale Garner (1991) explained "It is conceptually very closely related to poor self-esteem....., but goes beyond this construct to include feelings of emptiness and aloneness" (p. 5). For the purposes of this study this variable was called 'self-esteem' as it was a more intuitively understandable variable label than 'ineffectiveness'. A high score on this variable indicated that the participant had very low self-worth and self-confidence, along with feelings of isolation and despair.

Interceptive Awareness: The scores for this variable were taken directly from the EDI-2. This scale was designed to measure "confusion and apprehension in recognizing and accurately responding to emotional states" (Garner, 1991, p. 6). This variable also examined the participant's mistrust of visceral sensations within the body that indicate hunger and satiety. A high score on this variable indicated that the participant had difficulty in understanding their emotional responses and identifying bodily sensations of hunger and satiety.

Impulse Regulation: The scores for this variable were taken directly from the EDI-2. This scale was designed to assess "tendencies toward impulsivity, recklessness, hostility, destructiveness in interpersonal relationships and self-destructiveness" (Garner,

1991, p. 6). This variable was intended to measure an inability for self-control in areas of life other than eating. A high score on this variable indicated that the participant had difficulty controlling their urges to shop lift both food and non-food related items and to engage in other highly impulsive activities.

Index of Self Harm Behaviours: The scores for this variable were taken directly from the PAI. This scale was designed to measure self-destructive and self-harm behaviours such as dangerous sex, reckless driving, and self-mutilation. A participant scoring high on this scale would be more likely to have cut or burned themselves as well as to have engaged in behaviours such as reckless driving and dangerous sexual activity.

Suicidal Ideation: The scores for this variable were taken directly from the PAI. This scale was designed to assess the extent to which the participant had “thoughts and ideation related to death and suicide” (Morey, 1991, p.19). The scale item content ranged from general thoughts of death to specific plans for a suicidal act. A high score on this variable indicated that the participant had a very hopeless view of their world and had a strong conviction that suicide was a solution.

Family-Environmental Risk Factors

Non-supportive environment: The scores for this variable were taken directly from the PAI. Non-supportive environment was designed to measure the participants “perceived lack of social support, tapping both the availability and quality of the respondent’s social relationships.” (Morey, 1991, p. 20). The item content within this scale focused on close relationships with an emphasis on family members. A high score

on this variable indicated that the participant perceived a poor support network, and family members and close friends that were uncaring and rejecting.

Stress: The scores for this variable were taken directly from the PAI. The stress scale was designed to measure the extent to which the participant had suffered from a variety of difficult events and consequently felt stressed. The item content for this scale ranged from problems in family relationships, problems at work, major life changes, financial instability and their impact on the individual. A high score indicated that the participant's life was tumultuous and unpredictable, with difficult family relationships and difficulties at work.

High Expectations: The scores for this variable were derived from two specific questions on the EDI-2. The two questions directly asked about the level of expectation that the parent had on the child, and the consistency with which those expectations were high. The scores were averaged for each participant. A high score on this variable indicated that the participant perceived her parents' expectations to be very high and without exception. Typically an individual who scored high on this variable would feel extreme pressure to perform well in every aspect of their lives.

Parent-Child Relationships: The scores for this variable were derived from the DSED. The parent-child relationship variable was actually the combined average of the participant's perceived relationship with her mother and her father. For those cases when only one parent had been involved with raising the child, the score only reflected the relationship with that parent. A high score on this variable indicated that the parent-child

relationship was very poor and that there was a great deal of perceived hostility and poor communication.

Disruptive Life Events: The scores for this variable were derived from the DSED. The scores for this variable were calculated by averaging the number of disturbing life events the participant reported. These life events included the death of a family member, family problems, romantic problems, leaving home, difficult sexual experience, illness of a family member, personal illness, and failures at work or school. A high score on this variable indicated that the participant had experienced all or almost all of the disruptive life events listed.

Abuse: The scores for this variable were derived from the DSED. The abuse variable was calculated by asking whether the participant had ever been sexually and or physically abused by anyone in their life time. The variable was then calculated by averaging each participant's score. A high score on this variable indicated the participant had suffered from both physical and sexual abuse at some point in their lives.

Negative Relationships: The scores for this variable were taken directly from the PAI. This variable was designed to measure the overall history of the participant's relationships with friends, spouses and family members. The scale range assessed relationship characteristics such as instability, intensity, closeness, security and ambivalence. A high score on this variable indicated that the participant had a long history of unstable relationships in which they felt betrayed or rejected.

Dieting and Obesity Risk Factors

Mother's Preoccupation with Dieting: The scores for this variable were taken directly from the DSED. The actual question posed was "To what extent was your mother preoccupied with her own weight and dieting?" A high score on this variable indicated that the participant's perception of her mother was that she was extremely concerned with controlling her own weight and often engaged in dieting behaviours.

Mother's criticism of Daughters' weight: The scores for this variable were taken directly from the DSED. The actual question posed was "To what extent did your mother suggest that you need to diet?" A high score on this variable indicated that the participant's perception was that her mother was often critical of her weight and always encouraged her to diet.

Childhood Weight: The scores for this variable were taken directly from the DSED. The actual question posed was "Describe your weight as a child." A high score indicated that the participant believed that she was extremely overweight as a child.

Teenage Weight: The scores for this variable were taken directly from the DSED. The actual question posed was "Describe your weight as a teenager." A high score indicated that the participant believed that she was extremely overweight as a teenager.

Teasing by Peers: The scores for this variable were taken directly from the DSED. The actual question posed was "To what extent were you teased by your peers for being overweight?" A high score on this variable indicated that the participant felt that she was teased excessively by their peers because of her weight.

Dependent/Endogenous Variables

After comparing the two and three-factor models to answer the question, “Is the Family Environment a separate class of risk factors for BN?”, the extent to which the resulting factors would predict bulimic symptomatology was addressed. A measure of BN symptoms was regressed onto the factor classes. The dependent variable, bulimic symptomatology, was a combined score of three cardinal features of BN, drive for thinness, body dissatisfaction and bingeing/purging (Garner, 1991). An alpha reliability test was performed on this newly developed scale and revealed a moderate alpha level of 0.55.

Drive for Thinness: This scale was originally a construct derived from Bruch (1973) who described ‘drive for thinness’ as a cardinal feature of eating disorders. The scale was designed to measure the relentless pursuit of thinness through attitudes such as excessive concern with dieting, preoccupation with weight and a morbid fear of weight gain. A high score on this variable indicated that the participant had an extremely strong drive for thinness.

Body Dissatisfaction: This scale was designed to capture the participants feelings about their own body shape and weight and to assess the degree to which they judge themselves by these things. Body dissatisfaction measured the participant’s dissatisfaction with key areas of the body such as stomach, hips, thighs, and buttocks. It is important to recognize that some body dissatisfaction is common to most young North American women. However it is the extreme dissatisfaction that tends to be a central feature of eating disorders. Garner (1991) suggested that body dissatisfaction is “a major

factor responsible for initiating and then substantiating the weight controlling behaviours of those with eating disorders” (p. 5). A high score on this variable indicated that the participant was very unsatisfied with the weight and shape of her body.

Bingeing/Purging: This scale was designed to assess the degree to which the participant thought about and engaged in bingeing and purging behaviours. This was a critical feature because it differentiated an individual with BN from individuals with restricting types of eating disorders such as restricting type AN. It is important to note that this variable was heavily weighted in terms of measuring the extent to which the participant binged. Only one question from this scale related to the purging behaviour of the individual. This was a major limitation for the dependent variable because purging is a major diagnostic feature of BN. However, because the data source for this study was an archival database, this was the best measure of bulimic symptomatology that was available. A high score on this variable indicated that the participant believed that she engaged in an excessive amount of uncontrolled eating and as well spent a great deal of time thinking about a binge.

Chapter V

ResultsMissing Value Analysis

The data for this study came from an archival database belonging to the Regional Center for the Treatment of Eating Disorders (Ottawa Hospital - General Campus). As a result, a review of the data for missing values was a necessary first step in the statistical process. A review of each of the variables revealed that only variables taken or derived from the DSED had missing values. The variables that had missing values follow in Table 2 along with the number of missing values and the percent of the values missing.

Table 2

Missing Value Analysis

Variable	Total Sample Size	Number Missing	Percent Missing
Mom's Preoccupation with Dieting	92	3	3.3
Mom's Criticism of Daughters' Weight	92	5	5.4
Childhood Weight	92	2	2.2
Teenage Weight	92	3	3.3
Teasing by Peers	92	5	5.4
Abuse	92	3	3.3
Parent-Child Relationship	92	2	2.2

A pattern assessment of missing values indicated that there were no patterns of missing values within the data. The missing data were then estimated following guidelines presented in Stevens (1996) (see Appendix A for details). Overall the percentage of missing values were small with the largest percentage being 5.4%. It was therefore assumed that imputation would not significantly skew the true results for this study.

Confirmatory Factor Analysis

A first-order confirmatory factor analysis was used to test the hypothesis that the three-factor model (see Figure 1) would provide a better fit to the data than a two-factor model (see Figure 2). The three-factor model separated the three factor classes, the General Psychiatric factor, the Dieting and Obesity factor and the Family Environment factor. The two-factor model separated out only two factors, the General Psychiatric factor and the Dieting and Obesity factor. To compare the two models, the items making up the Family Environment factor in the two-factor model were combined with the items for the General Psychiatric factor. In this way, the same amount of information was used in both analyses. The main objective of this statistical step was to assess the conceptualization of the factor groupings in order to determine whether the family environmental factor was a separate factor class.

Assumptions

The primary assumption underlying structural equation modeling was the assumption of multivariate normality. Normality was assessed by examining the univariate skewness and kurtosis statistics (see Table 3), as well as the overall multivariate kurtosis statistic.

Table 3

Descriptive Statistics of Exogenous Variables

Variables	Mean	SD	Median	Skewness Statistic	Kurtosis Statistic
Index of Related Disorders	75.15	12.05	73.75	-0.03	0.09
Index of Substance Abuse	52.78	10.90	50.75	1.66	3.31
Affective Instability	68.55	11.81	69.00	-0.51	-0.26
Self-Esteem	13.08	7.64	12.50	0.23	-0.86
Interceptive Awareness	12.23	6.93	10.50	0.47	-0.44
Impulse Regulation	5.50	5.16	4.00	1.31	1.010
Index of Self Harm	63.58	14.99	60.00	0.73	0.04
Suicidal Ideation	71.21	18.80	66.00	0.46	-0.80
Non-supportive environment	60.50	14.46	58.00	0.46	-0.30
Stress	61.65	11.22	62.00	0.21	-0.89
High Expectations	3.42	1.44	3.00	0.14	-0.87
Parent-Child Relationships	2.66	1.19	2.50	0.29	-0.83
Disruptive Life Events	0.27	0.19	0.25	0.50	-0.40
Abuse	0.43	0.41	0.50	0.29	-1.47
Negative Relationships	65.75	11.70	65.00	0.03	-0.71
Mom's Preoccupation Dieting	2.98	1.28	3.00	0.04	-1.10
Mom's Criticism of Daughter	2.59	1.78	2.00	0.83	-0.61
Childhood Weight	3.35	1.02	3.00	-0.44	0.38
Teenage Weight	3.89	0.93	4.00	-0.77	0.67
Teasing by Peers	2.51	1.56	2.00	0.41	-1.39

As indicated by the univariate skewness statistics in Table 3, several variables appeared to be positively or negatively skewed. However, an assessment of the multivariate normal distribution via Mardia's (1970) coefficient ($M=7.4255$) showed that the assumption of multivariate normality was upheld (Byrne, 1994). Because the assumption of multivariate normality was upheld it was understood that the estimates of the chi-square statistic and the Comparative Fit Index (CFI) would not be influenced by univariate skewness. This was confirmed by assessing the normalized estimate (Byrne, 1994) which was $Z=-1.2005$. This value was understood as the multivariate normality of a z-score. Bentler (1989) suggested that any normalized estimate that was at or close to zero would indicate a multivariate normality for the sample.

Outliers on each of these variables were assessed by examining the five most extreme cases of outliers with respect to multivariate normality. The five most extreme cases were all close in value. This indicated that outliers were not a concern for the confirmatory factor analysis (Byrne, 1994).

The sample size for this data set was deemed to be appropriate for the confirmatory factor analysis. Stevens (1996) found that most studies that have performed confirmatory factor analysis have included sample sizes between 100 and 350. Stevens (1996) noted the popular rule for defining sample size as a function of the number of variables being analyzed (usually 5 subjects per variable). Stevens (1996) also described a Monte Carlo study showing that a small sample size was appropriate as long as there were strong correlations between the variables and their factor classes (Guadagnoli & Velicer, 1988). An assessment of the factor loadings for each variable on its respective

factor class confirmed that most variables were strongly correlated with their factor class (See Table 4). Those variables that were not strongly correlated with their factor classes were not removed because this was a theoretically driven factor analysis (Stevens, 1996). This check confirmed that the sample size for this study was within the limits of acceptability.

It should be noted however that studies with small sample sizes can have a tendency to capitalize on chance if the model is respecified in a post-hoc fashion (MacCallum, 1986). As a result of this concern, no post-hoc model respecifications were performed. The goodness of fit indices were assessed for the original model only.

Table 4

Factor Loadings for Variables in the Three-Factor Model

Factor Class	Risk Factor Variables	Standardized Factor Loadings	R-squared
GP	Index of Related Disorders	0.88	0.77
GP	Index of Substance Abuse	0.14	0.02
GP	Affective Instability	0.54	0.29
GP	Self-Esteem	0.75	0.56
GP	Interoceptive Awareness	0.70	0.50
GP	Impulse Regulation	0.61	0.39
GP	Index of Self-Harm Behaviours	0.46	0.21
GP	Suicidal Ideation	0.62	0.39
FE	Non-Supportive Environment	0.67	0.45
FE	Stress	0.50	0.25
FE	High Expectations	0.29	0.08
FE	Parent-Child Relationships	0.72	0.52
FE	Disruptive Life Events	0.33	0.11
FE	Abuse	0.54	0.29
FE	Negative Relationships	0.71	0.51
DO	Mother's Preoccupation with Dieting	0.16	0.02
DO	Mother's Criticism of Daughters' Weight	0.37	0.13
DO	Childhood Weight	0.70	0.50
DO	Teenage Weight	0.74	0.55
DO	Teasing By Peers	0.81	0.65

Note: GP means General Psychiatric Factor, FE means Family Environment factor and

DO means Dieting and Obesity Factor.

Test of the Three-Factor Model

A confirmatory factor analysis was first performed for the three-factor model in which the twenty variables were loaded (non-zero loadings) onto one of the three factors, the General Psychiatric factor, the Family Environment factor, and the Dieting and Obesity factor (see Figure 1). An alpha level of 0.05 was used for all statistical tests. The three factors were allowed to co-vary. The General Psychiatric factor correlated with the Family Environment factor $r = 0.589$ ($p < 0.05$), and with the Dieting and Obesity factor by 0.149 ($p < 0.05$). The Family Environment factor correlated with the Dieting and Obesity factor by 0.233 ($p < 0.05$).

A variety of 'goodness of fit' indices were examined based on recommendations that using only one index of fit might fail to provide an adequate assessment of the model (Pedhazur, 1997; Stevens, 1996). These indices included the independence chi-square statistic (sensitive to sample size), and a set of practical indices of fit including the comparative fit index (CFI) (less sensitive to small sample sizes) and an analysis of the residuals. A threshold criteria value of 0.90 was selected a priori for the CFI based on recommendations by Bentler and Bonett (1980).

First, an overall assessment of fit was performed by comparing the difference in chi-square between the null model (in which independence of all variables was assumed) $\chi^2(190) = 793.19$ and the proposed three-factor model, $\chi^2(167) = 335.07$. The chi-square change for the proposed three-factor model was found to be significantly different from the independence (null) model, $\Delta\chi^2(23) = 458.12$, $p < 0.05$. A second assessment of

overall fit was performed using the comparative fit index (CFI). The CFI value for the three-factor model, $CFI = 0.721$, showed that this model did not provide an excellent fit to the data based on Bentler and Bonett's (1980) CFI index threshold of 0.90. A third assessment of overall fit was performed by examining the Average Off-Diagonal Absolute Standardized Residuals. The average residuals were found to be 0.09, which was considered a reasonable fit because this residual value was quite close to zero. Finally, the overall fit was deemed reasonable because a visual inspection of the standardized residuals did in fact show that they centered around zero, and that 73.82% of the standardized residuals were between -1 and +1. Overall, despite an unfavorable CFI value, the 'goodness of fit' of the three-factor model was deemed reasonable for these data.

Once the overall fit had been assessed for the three-factor model, an assessment of parameter misspecifications was performed using the Lagrange Multiplier Test (LM test). The Lagrange Multiplier test (LM test) was performed to gage the appropriate placement of each of the variables on their corresponding factor. The fixed input parameters were assessed multivariately in order to identify any specific parameters that would cause a significant drop in chi-square if allowed to be freely estimated in a subsequent run. In this way, any of the fixed risk factors could be assessed for their appropriate placement in the predetermined risk factor classes.

It should be noted that any parameter misspecifications that indicated an improvement in the overall chi-square by allowing errors to co-vary were not considered as viable for model respecification. According to a variety of sources, allowing suggested

error covariances to run free would capitalize on chance such that the 'goodness of fit' indices would no longer be meaningful and replicability would be greatly reduced. (Gerbing & Anderson, 1984).

The results of the LM test - Cumulative Multivariate Statistics, indicated that no one variable would have been better associated with a different factor. The LM test revealed that only by allowing error terms to co-vary would the fit be improved. Therefore, overall, the fit of the three-factor model to the sample data was deemed to be reasonable, with no need for respecification of any variables on different factors.

Test of the Two-Factor Model

A confirmatory factory analysis was performed for two-factor model in which the twenty variables were loaded (non-zero loadings) onto one of the two factors, General Psychiatric factor, and the Dieting and Obesity factor (see Figure 2). An alpha level of 0.05 was used for all statistical tests. Again the two factors were allowed to co-vary. The General Psychiatric factor was correlated with the Dieting and Obesity factor by 0.184 ($p < 0.05$).

A variety of 'goodness of fit' indices were examined based on recommendations that using only one index of fit might fail to provide an adequate assessment of the model (Pedhazur, 1997; Stevens, 1996). First an overall assessment of fit was performed by comparing the difference in chi-square between the null model (in which independence of all variables is assumed) $\chi^2(190) = 793.19$ and the proposed two-factor model, $\chi^2(169) = 389.88$. The chi-square change for the proposed two-factor model was found to be significantly different from the independence (null) model, $\Delta\chi^2(21) = 403.31$, $p < 0.05$.

A second assessment of overall fit was performed using the comparative fit index (CFI). The CFI value for the two-factor model, $\text{CFI} = 0.634$, showed that this model did not provide an excellent fit to the data based on Bentler and Bonnet's (1980) CFI index threshold of 0.90. A third assessment of overall fit was performed by examining the Average Off-Diagonal Absolute Standardized Residuals. The Average Residuals were found to be 0.09, which was considered a reasonable fit because this residual value was quite close to zero. Finally, the overall fit was deemed reasonable because a visual inspection of the standardized residuals did in fact show that they centered around zero, and that 78.06% of the standardized residuals were between -1 and +1. Overall, despite the unfavorable CFI value, the 'goodness of fit' of the two-factor model was deemed reasonable for this data.

The Lagrange Multiplier test (LM test) was performed in order to gage the appropriate placement of each of the variables on their corresponding factor. An examination of the Cumulative Multivariate Statistics indicated that no one variable would have been better associated with a different factor. The LM test revealed that only by allowing error terms to co-vary would the fit be improved.

Therefore, overall, the fit of the two-factor model to the sample data was also deemed to be reasonable.

Comparison of the Two and Three-Factor Models

Both of the models were shown to provide a significantly better fit to the data than the null model in which all variables were assumed to be independent. A comparison of

the two and three-factor model was performed to assess which of the two models was the better for describing the data set.

Only the overall goodness of fit statistics were used to compare the two models. First the chi-square values were compared to generate a chi-square change statistic. The three-factor model was found to provide a significantly better fit to the data than the two-factor model, $\Delta\chi^2(2)=54.81$, $p<0.05$. Finally the Comparative Fit Indices were compared for the three-factor model (CFI=0.721) to the two-factor model (CFI=0.634). Again the three-factor model proved to be the better fit to the data.

The factor loadings for the two and three factor model were then examined for the Family Environment variables. For each variable the factor loadings were higher on the three-factor model than the two-factor model (See Table 5). This was a further indication that not only was the three-factor model more appropriate for these data, the Family Environment factor was best suited as a separate factor class.

Table 5

Comparison of the Factor Loadings for the Family Environment Variables on the Two and Three-Factor Models

Variables	Factor Loadings Two-Factor Model	Factor Loadings Three-Factor Model
Non-Supportive Family Environment	0.46	0.67
Stress	0.49	0.50
High Expectations	0.17	0.29
Parent-Child Relationships	0.44	0.72
Disruptive Life Events	0.26	0.33
Abuse	0.42	0.54
Negative Relationships	0.59	0.71

Following the confirmatory factor analysis, an alpha reliability test was performed for the three factors. The variable scores were first converted to z-scores because of the large differences in range of scores for each of the variables. The alpha reliability scores for the three factors were acceptable. General Psychiatric factor $\alpha=0.8219$, Family Environment factor $\alpha=0.7393$, Dieting and Obesity factor $\alpha=0.6928$. An examination of the alpha items for each variable within its factor class indicated that the removal of no one variable would have substantially improved the overall alpha for its factor class.

Multiple Regression Analysis

Following the confirmatory factor analyses, a multiple regression analysis was performed by regressing the dependent variable, a measure of BN symptoms, onto the three factor classes in the model. This analysis was done to examine the relationships between the three factor classes and bulimic symptomatology.

Both the dependent and independent variables were first converted to z-scores so that differences in ranges of scores would not be problematic. The independent variables were derived by calculating the average of the z-scores for each variable making up that factor. The dependent variable was the z-score from the bulimic symptomatology scale, created from the body dissatisfaction scale, the drive for thinness scale and the bingeing/purging scale. The regression analysis was performed in one step so that only the unique shared variability between the dependent and independent variables were assessed.

Sample size was deemed to be appropriate for the purposes of regression analysis based on Stevens (1996) who suggested that an N to k (predictor) ratio of 15:1 would

provide sufficient reliability in the prediction equation. Since the N:k ratio for this study was about 30:1 the generalizability of the regression results was acceptable.

Assumptions

The assumptions for linear regression were first checked. An examination of the histogram of standardized residuals revealed that the normality assumption for linear regression had been met. This was confirmed upon examination of the normal probability plot of the standardized residuals. The assumptions of linearity and homoscedasticity were confirmed by examining a plot of standardized versus predicted residuals. An examination of outliers on the independent variables revealed that there were no significant outlier scores, Mahalanobis distance, $D(4)=14.138$, $p<0.05$, and no influential data points, Cook's distance, $C = 0.110$. However, one extreme outlier was found for the dependent variable. An assessment of the standardized residual for that case, $R=-0.3117$, revealed that this value was beyond three standard deviations of the mean. Calculation of the Weisberg statistic showed that this outlier was a significant deviator from the mean of the dependent variable, $t= 3.633$, $p<0.05$. A closer examination of this variable showed that this participant was 41 years of age, with a Body Mass Index of 20, and a eating disorder chronicity of 15 years. Because of the significant Weisberg outlier score and the age and weight information that was considerably different from the mean age and weight for the sample (sample mean age = 29, sample mean BMI=26.76), this case was excluded from further analyses.

Regression Analysis

A multiple regression analysis was first performed by regressing the dependent variable, Bulimic Symptomatology (mean=0.026 (SD=0.700)), onto the three factors, General Psychiatric factor (mean=0.001 (SD=0.671)), Family Environment factor (mean=0.007 (SD=0.624)) and the Dieting and Obesity factor (mean=0.000 (SD=0.674)). All statistical tests were based on an alpha level of 0.05. An examination of the collinearity statistic (VIF) showed that multi-collinearity was not a problem among the factors. See Table 6 for the simple correlations between the factors and bulimic symptomatology.

Table 6

Simple Correlations Between Factor Classes and Bulimic Symptomatology

	General Psych. Factor	Family Enviro. Factor	Dieting/Obesity Factor	Bulimic Symptoms
General Psych. Factor	1.000	---	---	---
Family Enviro. Factor	0.589*	1.000	---	---
Dieting/Obesity Factor	0.149	0.233*	1.000	---
Bulimic Symptoms	0.380*	0.076	0.229*	1.000

Note: * ($p < 0.05$)

Overall, the factors in the prediction equation were found to significantly predict bulimic symptomatology, $F(3,87)=7.144$, $p < 0.05$, with an overall R squared value of 0.198 ($R^2_{adj} = 0.170$). Therefore 19.8% of the variance in bulimic symptomatology was explained by the variability in the three factors.

A closer examination of the standardized beta weights revealed that only the General Psychiatric factor, ($\beta = 0.439$, $t(87) = 3.964$, $p < 0.05$, $\omega^2 = 0.160$), and the Dieting and Obesity factor, ($\beta = 0.203$, $t(87) = 2.032$, $p < 0.05$, $\omega^2 = 0.083$), uniquely predicted the bulimic symptomatology dependent variable (see Table 7). An examination of the effect sizes of the predictive ability of the factors revealed that the General Psychiatric factor contributed the most amount (16%) of variability with bulimic symptomatology, and that the Dieting and Obesity factor contributed considerably less at 8.3%.

The Family Environment factor was not found to be a significant unique predictor of bulimic symptomatology, $\beta = -0.196$, $t(87) = -1.737$, n.s., $\omega^2 = 0.049$ (see Table 7). However, upon closer inspection there were indications that the Family Environmental Factor was acting as a suppressor variable. This was confirmed by first comparing the zero-order correlation between the Family Environment factor and the dependent variable ($r = 0.076$) with the semi-partial correlation ($r = -0.183$). The semi-partial correlation had the opposite sign of the zero-order correlation and its absolute value was much larger, indicating a suppressor variable (Pedhazur, 1997). Furthermore, an examination of the standardized beta weight showed a negative regression coefficient ($\beta = -0.196$) which also indicated that the Family Environment factor was acting as a suppressor variable (Pedhazur, 1997).

In order to examine the effect of the suppressor variable, the Family Environment factor was removed from the prediction equation, and a regression analysis was performed again. The overall regression analysis indicated that the two remaining factors still significantly predicted bulimic symptomatology, $F(2, 88) = 9.002$, $p < 0.05$, but the

overall R squared was smaller ($R^2=0.17$ ($R^2_{adj}=0.15$)). An examination of the parameter estimates revealed that the Family Environment factor was acting as a suppressor on both the General Psychiatric factor and the Dieting and Obesity factor. See Table 7 and 8 for direct comparisons. This was first revealed when the standardized beta weights were examined for the two factors. The standardized beta weights for both the General Psychiatric factor ($\beta=0.348$, $t(88)=3.526$, $p<0.05$, $\omega^2=0.125$) and the Dieting and Obesity factor ($\beta=0.166$, $t(88)=1.679$, n.s., $\omega^2=0.082$) were smaller for this second regression analysis. Their effect sizes were also smaller as compared to the first regression analysis. The General Psychiatric factor still significantly predicted the dependent variable. However its effect size dropped to 12.5%. The Obesity and Dieting factor no longer predicted the dependent variable, and its effect size dropped slightly to 8.2%. Finally a comparison of the zero-order correlations and the semi-partial correlations of the two factors with the dependent variable revealed that the semi-partial correlations were smaller ((GP Factor) $r = 0.352$ and (DO Factor) $r = 0.176$) in this regression analysis than compared to the first regression analysis ((GP Factor) $r = 0.391$ and (DO Factor) $r = 0.213$). This indicated that when the Family Environment factor was a predictor variable it served to partial out unexplained noise variation from the two other factors so that they were stronger predictors of the dependent variable.

Table 7

Regression 1- Family Environment Variables Included in the Regression Equation

Independent Variables	Unstand. β Weight	Standard. β Weight	t value	Sig.	ω^2	ZeroOrder Corr.	Semipart Corr.
GP Factor	0.456	0.439	3.964	0.000	0.160	0.379	0.381
FE Factor	-0.220	-0.196	-1.737	0.086	0.049	0.076	-0.167
DO Factor	0.211	0.203	2.032	0.045	0.083	0.229	0.195

overall R squared = 0.190, Rsquare adjusted = 0.170

Note: GP (General Psychiatric), FE (Family Environment), DO (Dieting and Obesity)

Table 8

Regression 2- Family Environment Variables Excluded from the Regression Equation

Independent Variables	Unstand. β Weight	Standard. β Weight	t- value	Sig.	ω^2	ZeroOrder Corr	Semipart Corr
GP Factor	0.362	0.348	3.526	0.00	0.125	0.379	0.352
DO Factor	0.172	0.166	1.679	0.09	0.082	0.229	0.176

overall R squared = 0.170, Rsquare adjusted = 0.150

Note: GP (General Psychiatric), DO (Dieting and Obesity)

Multivariate Regression

In order to understand the relationship between each of the three factors and the components of bulimic symptomatology, the dependent variable was broken down into its original three scales, Bingeing/Purging (mean=0.008 (SD=1.002)), Body Dissatisfaction (mean=0.031 (SD=0.961)), and Drive for Thinness (mean=0.029 (SD=0.966)). A multivariate regression was performed in which the three bulimic symptomatology scales were regressed onto the three factors, the General Psychiatric factor (mean=0.026 (SD=0.700)), the Family Environment factor (mean=0.001 (SD=0.671)) and the Dieting and Obesity factor (mean=0.000 (SD=0.674)).

An assessment of the General Psychiatric factor revealed that it uniquely predicted all three of the bulimic symptoms. The General Psychiatric factor most strongly predicted Drive for Thinness, ($F(1, 87)=10.774, p<0.05, \omega^2=0.110$), followed by Bulimia, ($F(1,87)=7.814, p<0.05, \omega^2=0.082$), and finally Body Dissatisfaction, ($F(1,87)=3.995, p<0.05, \omega^2=0.044$). Overall, of the 22.6% variability that was shared between the General Psychiatric factor and bulimic symptomatology, 11% was shared with Drive for Thinness, 8.2% was shared with Bingeing/Purging, and 4.4% was shared with Body Dissatisfaction.

An assessment of the Dieting and Obesity factor revealed that this factor only uniquely predicted the bulimic symptom Body Dissatisfaction, ($F(1,87)=6.578, p<0.05, \omega^2=0.070$). The Dieting and Obesity factor did not significantly predict Bingeing/Purging, ($F(1,87)=0.908, n.s.$) or Drive for Thinness, ($F(1,87)=0.448, n.s.$). Overall, of the 8.5% of the variance shared between the Dieting and Obesity factor and

bulimic symptomatology, the Dieting and Obesity factor shared 7% with Body Dissatisfaction.

An assessment of the Family Environment factor revealed that it did not uniquely predict any of the variables that made up the bulimic symptomatology dependent variable. This was not surprising given the earlier findings of this factor's role as a suppressor variable.

Overall, when the Family Environment factor was allowed to play the role of a suppressor variable, the General Psychiatric factor was a strong predictor of Drive for Thinness and Bingeing/Purging. It was also a moderate predictor of Body Dissatisfaction. The Dieting and Obesity factor was found to be a good predictor for Body Dissatisfaction.

Path Analysis

Suppressor variables are often difficult to interpret. The effect of a suppressor can more easily be understood through a path analysis (McFatter, 1979) in which the suppressor acts on dependent variable indirectly through the other factors. To test the postulation that the Family Environment factor was acting indirectly on bulimic symptomatology through the General Psychiatric factor and the Dieting and Obesity factor, a path model was created (see Figure 3). The path analysis performed was designed to understand the direct and indirect relationships of the Family Environment factor on bulimic symptomatology through the General Psychiatric factor and the Dieting and Obesity factor.

First, path coefficients for the model were calculated. The path coefficients were derived according to Pedhazur (1997) by taking the standardized beta weights from the

regression of the dependent variable onto the three factors (direct-effects). Another set of path coefficients were derived from the standardized beta weights of two regression equations; one in which the General Psychiatric factor was regressed onto the Family Environment factor, and one in which the Dieting and Obesity factor was regressed onto the Family Environment factor. The indirect effects were calculated by multiplying the path coefficients along two indirect paths leading from the Family Environment factor to the dependent variable.

The path coefficients are shown on the path model in Figure 3.

Assumptions

The assumptions for path analysis were the same as for multiple linear regression. Therefore because the assumptions for linear regression had already been checked, the assumptions were assumed to hold true for the path analysis also. The second assumption of path analysis pertaining to unidirectional flow was also met as all the pathways led to the dependent variable - bulimic symptomatology. The maximum total sample size required for this path model was calculated to be 70 (based on 5 path coefficients and 2 residuals, with a required minimum of 5 to 10 subjects per coefficient (Pedhazur, 1997)). Since the total N for this sample was 92, the sample size for the path model was considered sufficient.

Figure 3: Path Model for Direct and Indirect Effects of the Family Environmental Factor

Legend

FE Factor: Family Environmental Factor

GP Factor: General Psychiatric Factor

DO Factor: Dieting and Obesity Factor

B. Symptoms: Bulimic Symptomatology

* $p < 0.05$

Direct Effects

GP Factor $\rho = 0.439$

FE Factor $\rho = -0.196$

DO Factor $\rho = 0.203$

Indirect Effects

Total Indirect Effects $\rho = 0.273$

Indirect Effect through GP Factor $\rho = 0.217$

Indirect Effect through DO Factor $\rho = 0.056$

Direct Effects

The direct effects were first calculated for all three of the factors on bulimic symptomatology. There was a significant direct effect for the General Psychiatric factor, ($\beta = 0.439$), $t(87) = 3.964$, $p < 0.05$. There was a significant direct effect for the Dieting and Obesity factor, ($\beta = 0.203$), $t(87) = 2.032$, $p < 0.05$. The direct effect for the Family Environment factor was not significant, ($\beta = -0.196$), $t(87) = -1.737$, n.s.

Indirect Effects

There were two indirect effects in this path model (see Figure 2). Both of the indirect effects reflected the indirect effects of the Family Environment factor on bulimic symptomatology. The total indirect effect of the Family Environment factor was $\rho = 0.273$. The breakdown of the indirect effect of the Family Environment factor on bulimic symptomatology through the General Psychiatric factor showed that this indirect effect made up most of the total indirect effect ($\rho = 0.217$). The indirect effect of the Family Environment factor on bulimic symptomatology through the Dieting and Obesity factor was a much smaller proportion of the total indirect effect ($\rho = 0.056$). Overall, the proportion of the indirect effect through the General Psychiatric factor (proportion 80%) was much larger than the indirect effect through the Dieting and Obesity factor (proportion 20%).

Total Effect of the Family Environmental Factor

The total effect of the Family Environment factor on bulimic symptomatology was calculated by adding the direct Family Environment effect ($\rho = -0.196$) and the total

indirect Family Environment effect ($\rho=0.241$). The total Family Environment effect was 0.045. To calculate left over spuriousness, the zero-order correlation for the Family Environment factor with the DV, ($r=0.76$) was subtracted from the total Family Environment effect ($\rho=0.045$). The left over spuriousness was 0.038. This value was considered quite small and it was concluded that most of the effect of the Family Environment factor on bulimic symptomatology was accounted for by the direct and indirect effects in the path model.

Chapter VI

Discussion

Is the Family Environment a Separate Class of Risk Factors?

The family environmental risk factors were assessed as a separate class of risk factors by comparing two-factor and three-factor models using confirmatory factor analyses. It was hypothesized that a) the variables associated with the development and maintenance of BN would be multi-dimensional, and b) the three-factor model involving a separate factor for the family environmental variables would provide a better fit to the data than a two-factor model. Results showed that both the three-factor and two-factor models provided a better fit to the data than the null model. In addition, the three-factor model provided a better fit than the two-factor model, suggesting the Family Environment factor is best understood as a separate factor class.

These findings concur with previous research that has emphasized the need for a separate family environmental factor class. Fairburn et al. (1997), using a series of univariate chi-square analyses, showed that significantly more participants with BN had been exposed to risk factors such as high parental expectations, negative parent-child relationships and difficult life events, as compared to general psychiatric participants and normal controls. The current study was similar to Fairburn et al.'s (1997) in that most of the same risk factor variables were explored for the BN participants. In the current study, not all of the risk factor variables were available that had been included in Fairburn's

study, however the majority were included. The current study differs from the study by Fairburn et al. (1997) in its methodological approach. In the current study an overall model was tested to assess all the putative risk factors simultaneously for only individuals with BN. By using structural equation modeling, the structural relationships between the risk factors and bulimic symptoms were pictorially modeled and tested (Byrne, 1994). The risk factors associated with BN were examined for their relationships with the specific symptoms of BN. In spite of their methodological differences, the two studies converged in concluding that there is a need for a separate Family Environment factor.

Overall, the goodness of fit indices suggest that the three-factor model is a good model. However, the comparative fit index (CFI) was found to be less than threshold value of 0.90 as suggested by Bentler and Bonett (1980). This less-than-optimal CFI can indicate a poor model specification. However, this possibility was refuted by the results of the Lagrange Multiplier (LM) Test. Instead, the low CFI index is attributed to measurement issues (Stevens, 1996). In this study some of the risk factor variables were made up of only one or two items because of the archival nature of the database (See Table 1). Variables that are made up of a small number of items are known to be less reliable than variables that are made of many items (Allen & Yen, 1979). According to Pedhazur (1997), in confirmatory factor analysis, poor reliability within exogenous variables can lead to large error covariances which can in turn cause a reduction of the CFI index. Thus, fit improvement is not a matter of changing the model, but of measuring the variables with more reliable scales. It is concluded, as predicted, that the Family Environment factor is a separate factor class.

What are the relationships between the three factor classes and bulimic symptomatology?

Regression analyses were performed to explore the associations between the risk factor classes and general bulimic symptomatology as well as with three hallmark symptoms of BN. The second hypothesis proposed that each of the three factor classes would significantly predict BN symptomatology. Results showed that only the General Psychiatric factor and the Dieting and Obesity factor were significant unique predictors of bulimic symptoms. The Family Environment factor was not found to be a significant unique predictor of bulimic symptoms; however it did act as a suppressor variable for the other two factors. This led to a revision of the original model in which the Family Environment factor was considered to act on bulimic symptomatology indirectly through the other two factors.

General Psychiatric Factor

There was a significant relationship between the General Psychiatric factor and overall bulimic symptomatology. This finding concurs with much of the existing research that has shown that co-morbid depression and anxiety (Lilenfeld et al., 1997), substance abuse (Welch & Fairburn, 1996), affective instability and negative self-esteem (Leon et al., 1997), poor interoceptive awareness (Leon et al., 1993), impulse regulation difficulties and self-harm behaviours (Favaro & Santonastaso, 1998; Welch & Fairburn, 1996) are risk factors that contribute to the development and maintenance of BN. This study provides further support for the importance of these risk factors in BN by

demonstrating that the General Psychiatric factor is related to the actual features and symptoms associated with BN.

In the multivariate regression analysis the General Psychiatric factor significantly predicted the three key aspects of bulimic symptomatology including bingeing/purging, body dissatisfaction and drive for thinness, when the other factors were controlled. This finding concurs with theoretical suggestions that these psychiatric risk factors are uniquely related to the specific features and symptoms of BN. Polivy and Herman (1993) argued that psychological factors such as depression, low self-esteem and affective instability cause excessive distress within the individual. When these issues are paired with issues around over weight and dieting, they ultimately give rise to the binge-purge cycle (Polivy & Herman, 1993).

The psycho-biological impasse theory by Johnson and Connors (1987) helps to explain the relationships between the General Psychiatric factor and the specific features of BN for this study. This theory suggests that binge eating behaviour clashes with the socio-cultural pressure to be thin, thereby causing conflict within the individual. In an individual with personality vulnerabilities, this internal conflict can be expressed in the form of extreme body dissatisfaction and strong drive for thinness. Ultimately this internal conflict has the potential to lead to purging behaviours and the beginning of the binge-purge cycle.

While the results of this study are consistent with the psycho-biological impasse theory, the correlational nature of the analyses and the absence of prospective data in which causal links can be mapped out, the psycho-biological impasse theory can not be

explicitly confirmed or refuted. Other models might be equally useful for explaining the results (Stevens, 1996). The psycho-biological impasse is a useful theory however, because it provides a framework for explaining and understanding the relationship between the General Psychiatric factor and the symptoms of BN.

Dieting and Obesity Factor

The regression analysis also revealed a significant unique relationship between the Dieting and Obesity factor and overall bulimic symptomatology. This finding supports the research that suggests that obesity (Fairburn & Cooper, 1982), teasing by peers (Thelen, Lawrence & Powell, 1992), criticism and dieting behaviours by family members (Moreno & Thelen, 1990; Pike & Rodin, 1991) are risk factors that contribute to the development and maintenance of BN. However, the multivariate regression analysis showed that the Dieting and Obesity factor was only significantly related to body dissatisfaction, when the other factors were controlled. This finding concurs with several studies in literature that have specifically focused on body dissatisfaction. Fabian and Thompson (1989) found that negative attention around shape and weight was significantly related to negative body image in premenarcheal girls. Brown, Cash and Lewis (1989) found a link between childhood teasing about appearance and negative body image in women.

There was no significant relationship between the Dieting and Obesity factor and either bingeing/purging or drive for thinness. These findings suggest that although an individual may have a history of overweight and criticism, these factors are not necessarily related to drive for thinness and bingeing and purging. The results of the

regression analysis suggest that drive for thinness and bingeing and purging are more related to psychopathological traits such as personality vulnerabilities and coping difficulties as seen in the General Psychiatric factor.

Johnson and Connors (1987) suggested that extreme body dissatisfaction is an important first step in the development of the binge-purge cycle that ultimately leads to BN (Johnson & Connors, 1987). The current results suggest that the General Psychiatric factor plays an important role in influencing the development of drive for thinness and bingeing and purging, while the Dieting and Obesity factor influences body dissatisfaction. These results suggest that it is the combination of the risk factors from both the Dieting and Obesity factor and the General Psychiatric factor that is critical in the development and maintenance of BN.

Family Environmental Factor

The Family Environment factor acted as a suppressor variable when it was included in the regression analysis. Therefore, although the second hypothesis is not directly supported because the Family Environment did not significantly predict bulimic symptomatology, this finding does indicate that the Family Environment factor is an important factor in the development and maintenance of BN. This finding concurs with research that has suggested that family environment risk factors such as negative parent-child relationships (Johnson & Flach, 1985), non-supportive family environments (Norman & Herzog, 1984), disruptive life events (Schmidt et al., 1993) and sexual and physical abuse (Welch & Fairburn, 1996) are important risk factors for the development and maintenance of BN.

Suppressor variables are the source of a great deal of confusion in research. They are often a serendipitous find, but can lead to difficulties in understanding and interpreting the results (McFatter, 1979; Pedhazur, 1997). A suppressor variable is one that is seemingly useless in terms of predicting the criterion variable, and yet its inclusion in the regression equation is important because it suppresses, or controls for, the unexplained variance in the other predictors so that they more strongly predict the criterion variable (Pedhazur, 1997). The suppressor variable in this study, the Family Environment factor, did not significantly predict the criterion variable, bulimic symptomatology. However its inclusion in the regression equation was important because it improved the predictive relationship between the General Psychiatric factor, the Dieting and Obesity factor and bulimic symptomatology.

The suppressing effect of the FE factor also suggested that it was acting on bulimic symptomatology indirectly through the other two factors. This suggested a possible developmental pathway for bulimic symptom development (McFatter, 1979). It was postulated that the Family Environment factor was affecting bulimic symptomatology indirectly through both the General Psychiatric Factor and the Dieting and Obesity factor.

What are the direct and indirect effects of the Family Environmental Factor?

In order to understand the possible developmental sequence between the risk factors in the regression analysis, a path model was developed (McFatter, 1979). The path model was designed to assess the direct and indirect effects of the Family

Environment factor on bulimic symptomatology through General Psychiatric factor and the Dieting and Obesity factor.

According to Pedhazur (1997), one of the problems with interpreting a suppressor variable is that all too often the role of theory is ignored. In order to ensure that the suppressor variable was being appropriately evaluated and interpreted, the developmental theories were first reviewed to develop a path model for the Family Environment factor. Attachment theory stresses the importance of Family Environment risk factors in the development of BN (Blatt & Shichman, 1983; Chassler, 1998). It suggests that when the family environment is tumultuous and the relationships are weak, distrustful and inconsistent, the individual will be more likely to develop personality vulnerabilities and psychological disorders, such as low self-esteem and depression (Chassler, 1998). This approach suggests that the Family Environment factor has an indirect effect on bulimic symptomatology through the General Psychiatric factor. Attachment theory also describes, although less clearly, the possible indirect effects of the Family Environment factor on bulimic symptoms through the Dieting and Obesity factor. A family environment that is highly critical in general would also likely be highly critical of shape and weight issues (Chassler, 1998).

Based on attachment theory's notion of the development of BN (Blatt & Shichman, 1983; Chassler, 1998) a path model was specified (see Figure 3) and an overall test of the model was performed. An assessment of the left-over spuriousness indicated that this path model accounted for most of the effect of the Family Environment

factor on bulimic symptomatology through the direct and indirect pathways. This suggests that the path model is a good one overall.

The overall path model indicated that the Family Environment factor acts on bulimic symptomatology through the General Psychiatric and Dieting and Obesity factors. These results support a postulation by Johnson and Connors (1987) that various family environments influence the nature of the eating disorder. For example they suggested that individuals coming from hostile and chaotic families are more likely to develop BN while individuals coming from highly strict and interfering family environments are more likely to develop AN. The finding within this study, that the Family Environment factor influences BN symptoms, provide some support for this postulation.

A closer examination of the indirect effect suggested that the indirect impact of the Family Environment factor is much stronger through the General Psychiatric factor than through the Dieting and Obesity factor. The importance of the General Psychiatric pathway is exemplified by these results. The path model suggests that negative family environments influence the development of psychiatric disorders in general. This vulnerability, in combination with exposure to dieting and obesity risk factors, puts an individual at risk to develop the symptoms of BN.

This finding supports Chassler's (1998) approach to BN. She suggested that impoverished relationships between the individual and the family, develop as a result of an insecure attachment style. This attachment style then leads to the development of poor interpersonal relationships, low self-esteem and poor self-image. The negative self-

esteem and the inability to develop strong support systems have the potential to give rise to a host of psychiatric disorders. This disturbance in psychological well-being, in a society that promotes extreme thinness in women, then has the potential to give rise to an eating disorder such as BN.

It would seem, according to attachment theory and the results of the path analysis, that the major role of the Family Environment factor is its link with risk factors for a psychiatric disorder in general. Family environment risk factors such as poor parent-child relationships, sexual and physical abuse and disruptive life events predispose an individual to general psychiatric disorders and the personality vulnerabilities that accompany them (Blatt and Shichman, 1983). The path model then suggests that if this individual is also faced with difficulties around weight management and dieting, the individual will be at a high risk to develop and maintain the symptoms seen in BN.

It is interesting to note that the results from the path model and the regression analysis suggest that there is something about a difficult family environment that is related only to the General Psychiatric factor and not bulimic symptoms themselves. However when this variability is removed from the General Psychiatric factor, the General Psychiatric factor becomes a stronger predictor of bulimic symptoms. According to these results there appear to be two aspects of the General Psychiatric factor, one that is related to the family environment and one that is related to the psychopathological symptoms of BN. Future research will be needed to explore a more detailed breakdown of this factor and how it serves as a mediator between the family environment and bulimic symptoms.

This developmental path model provides an explanation of the relationship between the Family Environment factor and the specific symptoms of BN. In the absence of prospective data in which causal links between risk factors and bulimic symptoms can be mapped out, this particular path model can not be explicitly confirmed or refuted. However this preliminary work is important because it provides a working developmental model involving the Family Environment factor and BN.

Limitations of this Study and Future Research

The major limitation of this study was its retrospective correlational approach. The 'risk factors' in this study were measured and assessed based on the participant's evaluation of their exposure to these factors. Therefore temporal relationships between these risk factors were not available to be analyzed. In the absence of prospective data that examines which risk factor came first, causation can not be inferred.

The concept of cause and the methods of its study have been the subject of considerable debate (Pedhazur, 1997). According to Kempthorne (1978) in order to truly study cause, one must use manipulation and perform measures of the elicited response in a well controlled setting. In social science research this is often difficult, particularly with human subjects. Structural equation modeling and path analyses are often the alternative solutions for non-experimental research (Pedhazur, 1997). Although these approaches do not allow the researcher to directly infer cause, they do test a model and provide an opportunity to refute the feasibility of that model. In this study the three-factor model, with the factors in parallel, was too simple. Instead, a sequential model was developed that better described the path of influence between these factor classes and BN symptoms.

Therefore although 'cause' could not be inferred in this study, a more sophisticated model was developed that provides a direction for future research.

The path analysis was also performed as a post-hoc follow up on the same data that the suppressor variable was discovered. This post-hoc approach demands very strict limitations of the generalizability of these findings. In the future, prospective methodological approaches and replication of results will be required to confirm these preliminary results.

As mentioned previously another important future modification will be to establish more consistently reliable measures of the risk factors. In this study, using archival data, several risk factor variables were made up of only one or two questions. This led to poor reliability for those risk factor variables and reduction in the overall goodness of fit for the three-factor model. In the future, studies with more reliable measures of the same variables will need to be done to confirm that the three-factor model was the best approach for BN risk factor data.

The single item variables were derived mainly from the DSED. From this study a set of recommendations will be made to the Regional Center for the Treatment of Eating Disorders, Ottawa Hospital (General Campus) regarding improvements and additions to the DSED questionnaire that will provide a more complete knowledge of family and environmental risk factors for eating disorders.

Finally, the sample size for this study was just within the limits of acceptability for the confirmatory factor analyses. In the future, these results need to be replicated with larger sample sizes.

Summary and Conclusions

This study showed that the Family Environment factor is an important factor class for understanding and predicting bulimic symptoms. The original three-factor model with the General Psychiatric factor, the Family Environment factor and the Dieting and Obesity factor was found to be the best model as compared to a two-factor model and a null model. However, further analysis indicated that this model was too simple. A regression analysis revealed that the Family Environment factor was acting indirectly on bulimic symptomatology through the General Psychiatric factor and the Dieting and Obesity factor. A path analysis revealed a more sophisticated model that described a possible developmental sequence for BN symptoms. It suggested that a negative family environment predisposes an individual to develop a psychiatric disorder in general. Exposure to general psychiatric risk factors in combination with risk factors for dieting and weight management difficulties leaves an individual at high risk for the development of the symptoms of BN.

Future research will be needed to examine other possible interpretations and pathways for the causal effects of the risk factors on BN. Further research will also need to explore the reciprocal relationships between these risk factors and bulimic symptoms. Although the proposed path model is not the only interpretation for this data and can not provide the last word on this issue, it was important because it provided a starting point for future developmental research in BN.

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Appendix A

Imputation of Missing Values

The first step in imputing these missing values was to apply logic where possible. Two variables were candidates for this approach. The first, teasing by peers, was modified by making the assumption that if there was a missing value it was because the participant had never been teased for being overweight. For this variable therefore, a score of 1, indicating that the participant had never been teased was imputed. The second variable, abuse, was modified by making the assumption that if there was a missing value it was because the participant had never been sexually or physically abused. Therefore a score of 1, indicating that the participant had never been abused, was imputed. The remaining variables required that an alternative approach be taken. The following variables were imputed by calculating their regression means; Mom's preoccupation with weight, Mom's criticism about daughters' weight, Childhood Weight, and Teenage Weight. These variables were grouped together because their focus was weight related. A separate regression mean was imputed for the parent-child relationship because of its focus on family relationships.