5-2000

Eating Disorder Risk Factors: A Prospective Analysis

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EATING DISORDER RISK FACTORS:

A PROSPECTIVE ANALYSIS

by

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A dissertation submitted in partial fulfillment
of the requirements for the degree

of

DOCTOR OF PHILOSOPHY

in

Psychology

UTAH STATE UNIVERSITY
Logan, Utah

2000

ABSTRACT

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Utah State University, 2000

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The current study examined whether elevations on four variables (drive for thinness, depressed mood, maladaptive cognitions, and ineffectiveness) were related to increased risk of developing an eating disorder over a 4-year prospective interval. Subjects (N = 191) were female undergraduates who were assessed with the Anorexia-Bulimia Inventory (ABI), Eating Disorder Inventory (EDI), and a structured clinical interview.

Results indicated that individuals with elevated scores on each of the four variables at the initial assessment did not show higher absolute eating disorder incidence rates over the 4-year interval. However, initial scores on the four variables together explained approximately 13% of both anorexia and bulimia symptom severity variance at the final assessment. Changes over time in the four variables were more highly related to later symptom severity, explaining 34% of the variance in anorexic severity and 16% in bulimic severity.

Thus, the findings suggest that initial scores, and especially changes in scores, on the four variables were related to severity of symptoms 4 years later. However, a large proportion of the variance in eating disorder severity remained unexplained. Examination of the role of each risk variable individually revealed that initial elevations on maladaptive cognitions and drive for thinness subscales were related to higher anorexic symptom severity at the later assessment. Of interest was the absence of significant relationships between early scores on maladaptive cognitions and drive for thinness and subsequent bulimic symptoms, suggesting that anorexia and bulimia may have somewhat different risk pathways.

The pretest scores on the depressed mood and ineffectiveness subscales were not significantly correlated with symptom severity at the later assessment, and were not identified by the regression analyses as parsimonious or efficient predictors of eating disorder symptoms. This finding suggests that perhaps early difficulties with depression and low self-esteem are less related to onset of later eating disorders than previously believed.

Finally, the overall 4-year incidence rate of .6% found in the current study suggests that as women move through their college years, they are departing the developmental period of high risk for onset of eating disorders, and thus, new cases become increasingly rare.

(165 pages)

ACKNOWLEDGMENTS

I would like to thank my committee chairperson, David M. Stein, Ph.D., for his invaluable assistance with this dissertation. His continual encouragement and assistance guided my progress from the start of the project (by making the initial data set available to me) to its completion. I am also grateful for his consistent support and mentoring throughout my graduate studies, providing guidance for my developing clinical, research, and teaching skills.

I would like to express appreciation to my committee members, Mary Doty, Ph.D., Julie Gast, Ph.D., Kevin Masters, Ph.D., and Richard Gordin, Ed.D. Their insightful and honest criticism regarding issues related to rationale, methodology, and interpretation of results proved especially helpful in the completion of this project. I also would like to acknowledge the financial assistance I received from the Utah Psychological Association in support of my dissertation, which helped offset some of the expenses of the research.

I am grateful to my parents, Ferd and Linda Dobmeyer, and my siblings, Katie, David, and Rebecca, for their love, encouragement, and support throughout this process.

Finally, I give special thanks to my husband, Todd Uhlman, for the countless times he provided a willing ear, an insightful mind, and a supportive shoulder as I grappled with various issues and dilemmas that arose during this project. I deeply appreciate his love, companionship, and support.

Anne C. Dobmeyer

CONTENTS

Page

LIST OF TABLES

Table Page

Table Page

CHAPTER I

PROBLEM STATEMENT

The successful prevention and treatment of eating disorders remain largely elusive, despite over two decades of clinical observation and research on anorexia and bulimia nervosa. Reviews of treatment outcome studies have shown that even with the most promising forms of treatment for bulimia, approximately half of individuals with bulimia fail to show substantial improvement (Wilson & Fairburn, 1998). Likewise, the research on treatments for anorexia has revealed a lack of consistently positive findings (Vitousek, 1995). The fact that a large proportion of eating disorder patients are treatment nonresponders has at least two implications for future research. First, the limitations of existing treatments underscore the necessity for continued innovation and refinements of treatment approaches. More importantly, they highlight the need for basic knowledge about risk factors. The current lack of understanding of these predisposing factors makes it exceedingly difficult for prevention programs to confidently identify subsets of young women who may be at highest risk for developing an eating disorder. Furthermore, prevention program curricula presently "target" particular aspects of girls' psychological status (e.g., self-esteem, depression), despite considerable uncertainty that these variables represent actual risk factors.

Individuals with eating disorders have an increased likelihood of experiencing a variety of negative and potentially life-threatening physiological complications. This fact underscores the importance of identifying specific risk and etiological factors. Some of

the related health complications (e.g., osteoporosis, anemia, cardiac problems, death) are mainly due to the direct effects of starvation, while others (e.g., dental problems, electrolyte imbalances, gastrointestinal disorders) result primarily from the use of compensatory measures, such as self-induced vomiting or abuse of laxatives (American Psychiatric Association, 1994; Garfinkel & Garner, 1982). In addition to these physiological complications, individuals with eating disorders also frequently experience a host of secondary psychological symptoms and features. Researchers have found that between 30-50% of persons with eating disorders meet diagnostic criteria for comorbid major depression (Hudson, Pope, Yurgelun-Todd, Jonas, & Frankenburg, 1987; Swift, Andrews, & Barklage, 1986). Other psychological problems, such as substance abuse, personality disorders, and anxiety disorders, also occur with higher frequencies in eating disorder populations (Bulik, 1987; Halmi et al., 1991; Oldham et al., 1995). Thus, women with eating disorders experience not only the debilitating symptoms of their primary disorder, but also an increased risk of potentially severe health complications and a variety of comorbid psychological problems.

A large body of research focuses on the psychological, behavioral, biological, and environmental correlates of eating disorders. This research, combined with impressions gleaned from self-reports and clinical observations of women with eating disorders, has resulted in numerous hypotheses regarding purported factors that increase risk for developing abnormal eating patterns. Some of the more frequently cited factors thought to be influential include demographics (e.g., female gender, Anglo ethnicity, high socioeconomic status; Jones, Fox, Barbigan, & Hutton, 1980; Willi & Grossmann, 1983),

early puberty (Gross & Duke, 1980; Killen, Hayward, Wilson, & Taylor, 1994), poor body image (Cash and Brown, 1987), personality characteristics (e.g., undue perfectionism; Bruch, 1973; Kenny & Adams, 1994), affective disorder (Leung & Steiger, 1991), lack of interoceptive awareness (Bruch, 1962), above average weight (Garfinkel & Garner, 1982), early and chronic dieting (Polivy & Herman, 1993; Schlundt & Johnson, 1990), various family characteristics (including poor parent-child relationships and history of family psychopathology; Felker & Stivers, 1994; Rosenfield, 1988), and possible biological or genetic factors (Johnson & Maddi, 1986; Scott, 1986).

Many of these proposed risk factors have been found to correlate with patient or family reports and current or past symptomotology. Unfortunately, the majority of studies investigating these variables have used research methodologies that limit the conclusions that can be drawn regarding risk. In correlational studies, individuals with known eating problems are studied to determine their associated personal, biological, and environmental characteristics. This methodology, however, can tell us little about whether or not those characteristics were present prior to the development of eating problems, or were influential, predisposing factors. It is possible that a number of these assumed risk factors develop secondarily or concurrently with eating disorder symptoms, and therefore would be useless in the prediction of the development of eating disorders. Historical recall (retrospective) studies, whether in interview or questionnaire format, can be biased by current experiences, reconstruction over time due to life events, and simple memory errors. Finally, comparative studies (that contrast clinical with nonclinical

subjects) tell us only that these groups are presently different; the design tells us nothing about how they got to be that way.

Prospective methodologies, which involve analysis of the characteristics of nonsymptomatic subjects who subsequently develop eating problems, are required to accurately identify factors that are truly predictive of eating disorder onset. Interestingly, the available prospective studies examining risk factors lend support to several, but not all, variables identified by correlational, comparative, and historical report studies as predictive of eating disorder symptomotology. For example, although correlational and historical report studies have shown that perfectionistic tendencies and the setting of high achievement standards are often present in women with eating disorders (Bruch, 1973; Kenny & Adams, 1994), a number of prospective investigations failed to find a relationship between earlier levels of perfectionism and later development of eating problems (e.g., Attie & Brooks-Gunn, 1989; Killen, Taylor et al., 1994). Disparate findings such as these will be highlighted in the review of prospective studies in a subsequent chapter.

The present investigation sought to determine whether early elevations in key purported risk variables were related to a heightened likelihood of developing an eating disorder over a 4-year interval. The four variables selected for inclusion in the current study were: depressed mood, ineffectiveness (low self-esteem), drive for thinness, and maladaptive (eating) cognitions. In addition, this study assessed whether scores on the four factors decreased or remained at elevated levels in women who were in the process

of recovering from an eating disorder. A prospective methodology was used to avoid the external validity problems associated with retrospective and correlational designs.

A secondary aim of the current research was to assess both the overall prevalence and incidence rates of eating disorders in a female college population, as well as prevalence rates in women who were either high or low on the four hypothesized risk factors. Comparing differential prevalence rates in these latter two subgroups of women may reveal information about general and specific risk factors for certain women.

Finally, the study sought to remediate a gap in the current eating disorder risk factor literature. Nearly all longitudinal studies conducted to date have evaluated junior high or high school girls. Because the average age of onset for bulimia is late adolescence or early adulthood, research using somewhat older subjects is needed to identify risk factors, if any, relevant to this particular group. Therefore, subjects for the current study were female undergraduate students enrolled in a medium-sized university.

CHAPTER II

REVIEW OF THE LITERATURE

The review of the literature begins with a summary of the recent estimates of prevalence and incidence of eating disorders. Such statistics demonstrate that these disorders continue to be a problem for a significant proportion of adolescent girls and young women. This is followed by a discussion of relevant definitional and methodological issues, and an integration of general findings of published prospective studies. The review of the literature continues with a discussion of risk factor changes observed among recovered eating disorder subjects. Several models of eating disorder etiology are presented in order to draw parallels between theoretical models of eating disorder development and the existing prospective risk factor research. The review concludes with a discussion of the rationale for risk factor selection and the hypotheses that guided the current investigation.

Prevalence and Incidence Rates

Anorexia and Bulimia

The Diagnostic and Statistical Manual of Mental Disorders--Fourth Edition (DSM-IV; American Psychiatric Association, 1994) provides eating disorder prevalence rates based on research with adolescent and young adult women. Within this group, prevalence estimates for individuals meeting full criteria for anorexia range from .5% to 1%, and those for bulimia range from 1% to 3%.

In a review of 40 studies of the prevalence of bulimia in college women, Stein (1991a) reported that conservative methods to diagnose bulimia (i.e., adherence to strict criteria for binge and purge behaviors) yield minimum prevalence estimates of between 0.8% and 3.0%. When diagnostic criteria are more broadly defined, estimates increase to approximately 7% to 13% among college populations. Hsu (1996) conducted a recent, comprehensive review of epidemiological studies of eating disorder incidence and prevalence. Prevalence rates in young females living in Western cultures approximated 0.5% for anorexia and 2.0% for bulimia.

Eating disorder incidence estimates (emergent cases per 100 per year) vary widely, depending on the population under investigation. In samples of adolescent females, recent prospective studies have found incidence rates of 1.0/100/year (Killen et al., 1996), 3.6/100/year (Killen, Taylor et al., 1994), and 6.1/100/year (Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990), to a high of 10.1/100/year (Patton, 1988). An incidence rate for bulimia of 4.2/100/year was found in a female college sample (Drewnowski, Yee, & Krahn, 1988).

Hsu's (1996) review revealed much lower incidence rates (reported as emergent cases per 100,000 per year) in populations of young women (adolescents through young adults, both college and community samples). He found that in the more rigorous studies conducted since 1985, incidence rates ranged from 14.1/100,000/year to 43/100,000/year. The review indicated that although there has been a gradual increase in the incidence of eating disorders over the last decade, the increase has been small and may have reached a plateau. Finally, the author noted that despite the low incidence and prevalence rates of

anorexia and bulimia, they nevertheless remain "among the most common psychiatric disorders in young women" (p. 689).

Eating Disorder Not Otherwise Specified

The DSM-IV (1994) contains a diagnostic category for individuals who evidence disturbed eating patterns, but do not meet all the criteria for either anorexia or bulimia. Examples of symptom presentations that would be diagnosed as eating disorder "Not Otherwise Specified (NOS)" include: (a) a person who meets all criteria for a diagnosis of anorexia, with the exception of irregular menses or below normal weight; (b) a person who meets bulimia criteria except that the size of binges is small; or, (c) an individual who binge eats but does not use maladaptive compensatory strategies (i.e., binge eating disorder). The DSM-IV (1994) notes that prevalence estimates for eating disorder NOS are much higher than the 0.5% to 3.0% estimates for diagnoses of clinical anorexia or bulimia. However, no additional numerical estimates for this diagnostic category are provided. The 7% to 13% prevalence estimates for "broadly defined" bulimia, cited in Stein's (1991a) review, may actually reflect cases that would meet criteria for eating disorder NOS, rather than strict bulimia nervosa criteria.

Summary

In summary, the current literature on prevalence rates suggests that eating disorders continue to affect a substantial proportion of young women. Estimates of eating disorder prevalence and incidence rates vary substantially, however, depending on the population studied and methodology used. On average, studies that use stringent criteria

for diagnosis and include a clinical interview yield more accurate (and lower) prevalence and incidence rates. The results of these studies suggest that, although eating disorders have increased over the last decade, the rising incidence rates have not reached epidemic proportions, as has been frequently suggested by writers who quote studies using broad criteria and questionnaire methods of diagnosis.

Definitional Issues: Risk Factors, Etiological Factors, and Predictor Variables

Some confusion exists regarding the terminology used in the eating disorder risk factor literature. Different authors have used the terms "risk factor," "predictor," and "etiological factor" in both interchangeable and distinctive ways. These terms, however, do connote somewhat distinct concepts. For example, a variable that statistically predicts development of an eating disorder (a "predictor") may not actually be a causal variable (an "etiological factor"). In studies that primarily use correlational or multiple regression techniques, causal relationships cannot clearly be drawn. Therefore, the term "etiological factor" should not be used when referring to these results.

Similarly, a variable that increases an individual's risk for developing an eating disorder (a "risk factor") may not have good predictive value. For example, although many psychologists believe that dieting is a risk factor (i.e., dieting raises an individual's risk or probability of developing an eating disorder), dieting actually functions as a poor statistical predictor of eating disorder onset (This is probably because the rate of dieting is very high among young female populations in general, while eating disorder incidence

rates are quite low. In other words, predicting the development of an eating disorder based solely on knowledge of dieting behavior is relatively ineffective). Therefore, in the present study, the term "predictor" will be used only in the narrow context of actual statistical prediction of eating disorder development (for example, in discussing the results of a discriminant or regression analysis), while the term "risk factor" will be used more broadly.

Review of Prospective Studies

Correlational and comparative research, along with information gleaned from historical case report studies, has been relied upon heavily by researchers hoping to shed light on eating disorder risk factors. This body of literature has described the symptoms and features that often occur in women with anorexia and bulimia, some of which may prove to be true risk factors for these disorders. Unfortunately, these research methodologies are inadequate for addressing the question of risk for eating disorders, as they can only provide tentative hypotheses about which variables might be related to eating disorder onset. Researchers must use prospective methodologies to ascertain more accurately whether these purported risk factors actually function as accurate predictors of the development of eating disorders.

Unfortunately, correlational and comparative risk factor studies dominate the eating disorder literature, and relatively few prospective studies have been conducted to date. The purpose of this section is to identify the major risk themes that have emerged in prospective studies, as well as to discuss any significant contradictory results.

Taken together, the findings of the prospective studies conducted to date allow for only modest consensus regarding eating disorder risk factors. The following section attempts to integrate the findings of these studies by highlighting the areas of consensus and contradiction. Organization of the purported risk factors follows a cultural/familial/individual schema. Table 1 contains details regarding the subject characteristics (e.g., age, sample size, socioeconomic status, type of disorder) of each study; Table 2 reports the study characteristics (e.g., length of study interval, type of dependent variable, completion rate, risk variables measured). Table 3 contains a summary of results. Several proposed risk variables (temperament, EAT-26 scores, locus of control, and family religion) that were investigated by only one study and were not found to relate to onset of eating disorders are not included in the following narrative review, but are listed in Table 3 for reference purposes.

Cultural and Societal Factors

Leon, Fulkerson, Perry, and Early-Zald (1995) conducted the only prospective study that examined the impact of race on the development of eating disorders. Their finding that race was a statistically significant predictor of subsequent eating disturbances accords with the common opinion that eating disorders are more common in Caucasian populations than in other ethnic groups. Yet the occurrence of eating disorders does not fall along strictly racial or ethnic lines; rather, epidemiological evidence suggests that eating disorders primarily occur in societies and subcultures holding certain "Westernized" values and beliefs in which the ideal body image for women is restricted

Table 1

Prospective Studies of Eating Disorder Risk Factors: Subject Characteristics

Author and year	Mean age	N	SES estimate	Sample characteristics	Type of disorder
Attie & Brooks-Gunn, 1989	13.9	145	High	7th- to 10th-grade girls	NOS
Barnett, 1996	14.0	68	NR	Middle school girls	BN
Button et al., 1996	11.5	397	Mid to High	15- to 16-year-old girls	NOS
Cattarin & Thompson, 1994	12.5	210	Mid	10- to 15-year-old girls	NOS
Garner et al., 1987	13.0	35	NR	Ballet students	AN & BN
Graber et al., 1994	14.3	116	Mid	7th- to 9th-grade girls	NOS
Joiner et al., 1997	20.0	459	High	Harvard students	BN
Killen, Taylor et al., 1994	12.4	887	Mid	6th- to 7th-grade girls	NOS
Killen et al., 1996	14.9	877	NR	9th-grade girls	NOS
Leon et al., 1995	13.5	843	Mid	7th- to 10th-grade girls	BN
Leon et al., 1999	NR	726	Mid	7th- to 10th-grade students	AN and BN
Leung & Steiger, 1991	15.0	543	Mid	13- to 17-year-old girls	NOS
Marchi & Cohen, 1990	6.0	326	Mid	1- to 10-year-old girls	AN and BN
Patton, 1988, 1990	15.0	735	Low to High	15-year-old girls, London	AN and BN
Rosen et al., 1990, 1993	15.9	143	Mid	9th- to 12th-grade girls	NOS
Wlodarczyk-Bisaga & Dolan, 1996	15.3	747	Mid	14- to 16-year-old girls, Poland	NOS

Note. Age (years) = Mean age of subjects, in years, at the time of initial assessment. SES estimate = socioeconomic status estimate. Low = lower SES. Mid = middle SES. High = upper SES. NR = not reported (authors did not provide information on this variable). AN = anorexia nervosa. BN = bulimia nervosa. NOS = Eating Disorder NOS or subclinical subjects.

Table 2

Prospective Studies of Eating Disorder Risk Factors: Study Characteristics

Author and year	Study interval (mos)	Type of DV	Completion rate	Risk variables measured
Attie & Brooks-Gunn, 1989	24	Questionnaire	75%	weight, body fat, puberty, body image, ineffectiveness, affect/ impulse problems, perfectionism, family relations, aggression
Barnett, 1996	60	Interview	NR	depression, cognitive distortions, body image, ineffectiveness, age
Button et al., 1996	48	Questionnaire	67%	social class, self esteem, fear of fatness
Cattarin & Thompson, 1994	36	Questionnaire	41%	age, obesity, maturational status, body dissatisfaction
Garner et al., 1987	36	Interview	64%	EDI subscales, family relations, locus of control, feelings of inadequacy
Graber et al., 1994	96	Interview	NR	EAT-26 scores
Joiner et al., 1997	120	Questionnaire	57%	drive for thinness, maturity fears, perfectionism, interpersonal distrust
Killen et al., 1996	48	Interview	NR	weight concerns, EDI subscales, dietary restraint, temperament, height, weight, BMI, alcohol consumption
Killen, Taylor et al., 1994	36	Questionnaire	77%	weight concerns, EDI subscales, dietary restraint, pubertal timing, height, weight, BMI, aggression, social problems

(table continues)

Author and year	Study interval (mos)	Type of DV	Completion rate	Risk variables measured
Leon et al., 1995	36	Questionnaire	81%	class, race, weight, puberty, negative emotions, interoceptive awareness, body dissatisfaction, ineffectiveness, interpersonal distrust, maturity fears, perfectionism, constraint & positive emotionality, sexuality, autonomy (family relations), grade
Leon et al., 1999	36 - 48	Interview	NR	substance-related impulsivity, pubertal development, negative affect/ attitudes, psychopathology
Leung & Steiger, 1991	6	Questionnaire	79%	depressive symptoms
Marchi & Cohen, 1990	120	Interview	NR	early childhood problematic eating behaviors
Patton, 1988, 1990	12	Interview	69%	social problems, social class, family psy. history, sexuality, weight, personality, family background variables, dieting
Rosen et al., 1990, 1993	4	Questionnaire	88%	daily & major life stressors, psychological symptoms
Wlodarczyk-Bisaga & Dolan, 1996	10	Interview	84%	BMI, body dissatisfaction, sexuality, history of psy. problems, family religion, family psy. history, demographics, history of loss (stressful life events), class

Note. Study interval (mos) = Number of months between initial and final assessment. Type of DV = Type of dependent variable, either questionnaire only or clinical interview. Completion rate = % of subjects who were assessed at initial assessment who also were assessed at the final assessment. NR = not reported (authors did not provide information on this variable).

Table 3

Eating Disorder Risk Factors: Results of Prospective Studies

Proposed risk factor	+: -	Positive findings	Negative findings
Age/grade	1: 2	Barnett (1996)	Cattarin & Thompson (1994), Leon et al. (1995)
Aggression/delinquency	0: 2		Attie & Brooks-Gunn (1989), Killen, Taylor et al. (1994)
Body dissatisfaction	2: 5	Attie & Brooks-Gunn (1989) Garner et al. (1987)	Barnett (1996), Killen, Taylor et al. (1994), Killen et al. (1996) Leon et al. (1995), Wlodarczyk-Bisaga & Dolan (1996)
Bulimia	0: 3		Killen, Taylor et al. (1994), Killen et al. (1996) Garner et al. (1987)
Childhood eating patterns	1: 0	Marchi & Cohen (1990)	
Class/parental occupation	0: 4		Button et al. (1996), Leon et al. (1995) Patton et al. (1988, 1990), Wlodarczyk-Bisaga & Dolan (1996)
Cognitive distortions	0: 1		Barnett (1996)
Depression	0: 4		Attie & Brooks-Gunn (1989), Barnett (1996), Leon et al. (1995) Leung & Steiger (1991)
Dietary restraint	0: 3		Killen, Taylor et al. (1994), Killen et al. (1996) Patton et al. (1988, 1990)

(table continues)

Proposed risk factor	+: -	Positive findings	Negative findings
Drive for thinness/ fear of fatness	3: 2	Button et al. (1996) Garner et al. (1987) Joiner et al. (1997)	Killen, Taylor et al. (1994), Killen et al. (1996)
EAT-26 scores	0: 1		Graber, Brooks-Gunn, Paikoff, & Warren (1994)
Family psychopathology	1: 1	Patton et al. (1988, 1990)	Wlodarczyk-Bisaga & Dolan (1996)
Family relationship disturbance	0: 3		Attie & Brooks-Gunn (1989), Garner et al. (1987) Leon et al. (1995)
Impulsive/hyperactive	0: 1		Attie & Brooks-Gunn (1989)
Ineffectiveness/ self esteem	1: 6	Button et al. (1996)	Attie & Brooks-Gunn (1989), Barnett (1996), Garnet et al. (1987) Killen et al. (1996), Killen, Taylor et al. (1994), Leon et al. (1995)
Interoceptive awareness	1: 3	Leon et al. (1995)	Garner et al. (1987), Killen, Taylor et al. (1994) Killen et al. (1996)
Interpersonal distrust	0: 5		Garner et al. (1987), Joiner et al. (1997), Killen et al. (1996) Killen, Taylor et al. (1994), Leon et al. (1995)
Introversion	1: 0	Patton et al. (1988, 1990)	
Locus of control	0: 1		Garner et al. (1987)

(table continues)

Proposed risk factor	+: -	Positive findings	Negative findings
Maturity fears	1: 4	Joiner et al. (1997)	Garner et al. (1987), Killen et al. (1996) Killen, Taylor et al. (1994), Leon et al. (1995)
Perfectionism	1: 5	Joiner et al. (1997)	Attie & Brooks-Gunn (1989), Garner et al. (1987) Killen, Taylor et al. (1994), Killen et al. (1996), Leon et al. (1995)
Psychopathology	0: 3		Leon et al. (1999), Rosen et al. (1990;1993) Wlodarczyk-Bisaga & Dolan (1996)
Pubertal timing	0: 5		Attie & Brooks-Gunn (1989), Cattarin & Thompson (1994) Killen, Taylor et al. (1994), Leon et al. (1995), Leon et al. (1999)
Race	1: 0	Leon et al. (1995)	
Religion (family)	0: 1		Wlodarczyk-Bisaga & Dolan (1996)
Sexuality	0: 3		Leon et al. (1995), Patton et al. (1988, 1990) Wlodarczyk-Bisaga & Dolan (1996)
Social problems	1: 1	Patton et al. (1988, 1990)	Killen, Taylor et al. (1994)
Stress	0: 2		Rosen et al. (1990, 1993); Wlodarczyk-Bisaga & Dolan (1996)
Substance use	0: 2		Killen et al. (1996); Leon et al. (1999)

(table continues)

Proposed risk factor	+: -	Positive findings	Negative findings
Temperament	0: 1		Killen et al. (1996)
Weight/body fat	1: 5	Patton et al. (1988, 1990)	Attie & Brooks-Gunn (1989), Cattarin & Thompson (1994) Killen et al. (1996), Killen, Taylor et al. (1994) Wlodarczyk-Bisaga & Dolan (1996)
Weight concerns	2: 0	Killen, Taylor et al. (1994) Killen et al. (1996)	

Note. +: - is the ratio of positive to negative findings in prospective studies. Positive findings are those that found that the risk factor was significantly related to later eating disorder symptoms. Negative findings are those that did not find a significant relationship between early risk factor scores and later eating disorder symptoms.

to a thin, tubular, underweight shape (Hsu, 1987; Pumariega, 1986).

Four longitudinal studies (Button, Sonuga-Barke, Davies, & Thompson, 1996; Leon et al., 1995; Patton, 1988; Wlodarczyk-Bisaga & Dolan, 1996) included social class (based on parental occupation) in their risk analyses. Contrary to clinical lore, none of these found that social class predicted eating disorder onset. These results, however, could be related to sample demographics. Although Patton's (1988) sample was representative of various social class backgrounds in London, Leon and others' (1995) sample drew from a midwestern, suburban population, and Button and others' (1996) study consisted primarily of middle- to upper-class girls. This may have resulted in a restricted range for this variable. Wlodarczyk-Bisaga and Dolan (1996), who used a Polish sample, noted that Polish socioeconomic status does not correspond to Western classifications. However, their sample consisted of 84% college-educated/professional parents, 15% working class parents, and less than 1% farming parents, again suggesting the possibility of a restricted range.

Thus, living in more affluent Western cultures that emphasize thinness in women probably constitutes a risk factor in and of itself for the development of eating disorders. Individuals constantly surrounded by the message that "thinner is better" are at risk for developing the belief that maintaining a thin physique is of the utmost importance for attaining the present standards of beauty, virtue, and feminine identity. These individuals, consequently, may also be at higher risk for developing unrealistic expectations and goals regarding their own body shape. However, the relatively low base rate of eating disorders, even in "Westernized" societies, obviously indicates that the mere presence of

societal pressures for attaining thinness is not sufficient for the development of eating disorders. Other factors must also interact with these cultural and societal influences to produce disturbed eating patterns in certain individuals.

Familial Factors

Parent-child relationships. Much has been written regarding the strong, concurrent association between family relationship and interactional styles, and the presence of eating disturbances (e.g., Felker & Stivers, 1994; Leon, Fulkerson, Perry, & Dube, 1994; Pike & Rodin, 1991; Rosenfield, 1988). Three prospective studies (Attie & Brooks-Gunn, 1989; Garner, Garfinkel, Rockert, & Olmsted, 1987; Leon et al., 1995) investigated family relationships in an attempt to discern whether this association was predictive. Results of Leon and others' (1995) study indicated that adolescents' perceptions of their autonomy in interactions with family members and parents were not predictive of the subsequent development of eating problems. Attie and Brooks-Gunn's (1989) research investigated the impact of a greater number of family relationship variables, including the child's perception of each parent and of the parent-child relationship, and both the mother's and child's perceptions of the family structure and the interpersonal relationships between all family members. None of these variables were found to discriminate between girls who subsequently developed eating disorders and those who did not. Finally, Garner and associates' (1987) findings revealed that scores on an instrument measuring adolescents' perceptions of family interactions were not predictive of future eating disturbances.

The results of these studies are consistent with a number of reasonable interpretations. First, disturbed family interactional styles may not necessarily be present before the appearance of eating problems; rather, they may be a consequence of the eating disorder itself, or may develop concurrently. Second, family interactional problems could indeed be present before the development of eating disorders, but do not effectively predict onset (perhaps due to high base rates of family relationship problems in families without eating disorders). Third, the measures used in prospective studies to date may not have adequately measured true risk phenomena (e.g., two of the studies limited their family measures to child self-reports, none included interview or observational methods). Future research is necessary to evaluate these various hypotheses and resolve the question of whether impaired parent-child relationships constitutes a risk factor.

History of familial psychopathology. The relationship between a history of family psychopathology and the presence of eating disorders has been generally supported by correlational research (e.g., Bulik, 1987; Pike & Rodin, 1991; Rivinus, 1984), with a few exceptions (e.g., Leon et al., 1994). Two prospective studies have investigated this relationship. Wlodarczyk-Bisaga and Dolan's (1996) research found that the psychological history of families of girls who developed eating problems did not differ significantly from those whose eating patterns remained nonproblematic. Patton's (1988) results, however, indicated that a positive family psychiatric history was an effective predictor of later eating disturbances. Unfortunately, no data were provided regarding the specific types or degree of psychopathology assessed in the diagnostic interview. Based

on this limited information, one cannot claim with assurance that a positive family history of psychopathology functions as a risk factor for the development of eating disorders.

Individual Biological Factors

Sex. Although being female places an individual at higher risk for developing an eating disorder, none of the prospective studies isolated biological sex as a predictor variable. Yet, prevalence rates indicating that females are approximately 10 times as likely as males to develop eating disorders provide overwhelmingly strong evidence that sex is an important risk factor (DSM-IV, 1994). The question of how much of the increased risk is due to the sociocultural ramifications of being female versus the biological consequences of one's sex (e.g., metabolic differences), remains unclear at the present time.

Weight. Prospective investigations of the role of body weight in the development of eating disturbances have yielded mixed results. Five studies (Attie & Brooks-Gunn, 1989; Cattarin & Thompson, 1994; Killen et al., 1996; Killen, Taylor et al., 1994; Wlodarczyk-Bisaga & Dolan, 1996) found that the level of premorbid body weight was not related to onset of eating disorders. However, Patton (1988) found that individuals possessing a higher body weight were at greater risk for the development of an eating disorder. In fact, percentage of standard body weight was the single best predictor of eating problems at 1-year follow-up. One interpretation of this finding is that women with higher body weights may be more likely to perceive a larger discrepancy between their own body image and their ideal shape, compared to women of lower weights. This

discrepancy could lead to dieting, as women strive to achieve the culturally esteemed body shape. As will be discussed later, dieting may be a risk factor for development of eating disorders in and of itself.

Pubertal timing (precocious development). Five prospective studies investigated the hypothesis that girls who undergo puberty earlier than their peers may be at greater risk for developing eating disorders (Attie & Brooks-Gunn, 1989; Cattarin & Thompson, 1994; Killen, Taylor et al., 1994; Leon et al., 1995; Leon, Fulkerson, Perry, Keel, & Klump, 1999). Attie and Brooks-Gunn (1989) concluded that early pubertal changes were associated with early eating problems but not predictive of those in later adolescence. The four remaining studies also failed to verify that early pubertal changes were related to higher rates of subsequent eating disorders. Thus, at this time, it remains unclear what aspects of early pubertal maturation function as risk factors for eating disorders.

Critical age periods. Epidemiological data overwhelmingly suggest that age is a risk factor for the development of eating disorders. According to the DSM-IV (1994), anorexia nervosa has a mean age at onset of 17 years, and bulimia nervosa typically begins in early adulthood or late adolescence (19 or 20). The precise mechanisms by which age interacts with cultural, developmental, and psychological factors to promote eating disorder onset remains unclear at this time.

Cattarin and Thompson (1994) found that age was not related to changes in scores on the bulimia subscale of the Eating Disorder Inventory (EDI; Garner & Olmsted, 1984). The research by Leon et al. (1995) indicated that grade level was not prospectively

associated with increased eating disturbances. However, Barnett (1996) found that age at initial assessment was the only early predictor of total eating disorder symptoms at a 5-year follow-up. In her investigation, subjects who developed eating disorders over the course of the study were, on average, one year younger than low- or high-risk subjects who did not develop an eating disorder. The author hypothesized that perhaps the younger subjects reported more symptoms than older girls at follow-up because during the course of the study, they passed through a variety of stressful, developmental risk periods thought to be most linked to the development of an eating disorder. For example, the youngest subjects were initially in early junior high and finished the study as they were completing high school. It is notable that little risk factor research exists on whether there are different risk factors for individuals with early versus late onset of eating disorders.

Childhood feeding problems. The results of Marchi and Cohen's (1990) prospective investigation of the relationship between childhood eating patterns and adolescent eating disorders indicated that the presence of pica and problem meals in early childhood were predictive of later bulimic symptoms, and the presence of picky eating and digestive problems were predictive of subsequent anorexic symptoms. The authors proposed that a biological substrate may underlie various eating problems, including the presence of both early childhood eating problems and adolescent anorexia nervosa. However, their research design did not eliminate confounding environmental variables that could account for the presence and stability of eating problems across childhood and adolescence. Thus, at this point in time, this argument for a biological "early feeding"

predisposition for disturbed eating behaviors is limited.

Psychological/Developmental Issues

Depression. Remarkably, none of the four prospective studies published to date that included measures of depression or negative affect found that this variable was predictive of future eating problems (Attie & Brooks-Gunn, 1989; Barnett, 1996; Leon et al., 1995; Leung & Steiger, 1991). A fifth study (Leon et al., 1999) found that "negative affect/attitudes" was a significant predictor of later disordered eating. However, their construct actually was comprised of a number of different variables including depression, negative emotionality, low self-esteem, poor interoceptive awareness, and body dissatisfaction. Hence, it is not possible to conclude that it was "depression" or "negative mood" per se that was responsible for the significant relationship to eating disorder onset.

The existing evidence does not support the position that depressive symptoms per se may help predispose one to an eating disorder. Rather, depression may develop subsequent to eating disturbances, or may, as proposed by Swift et al. (1986), be a part of an interactional and reciprocal relationship between the two disorders. Indeed, the view that affective disturbances follow the development of eating disorders is supported by the fact that in women with anorexia, severe weight loss and food restriction appears to produce physiological symptoms that are indistinguishable from symptoms of depression. Nevertheless, there exists strong clinical impressions and correlational evidence associating depression with eating disorders. Additionally, some current theoretical models of etiology implicate depression as an important causal factor. It may be

important, therefore, to investigate the relationship between depression and eating disorders further, perhaps using alternate measures of depression and/or samples of subjects drawn from different populations.

Psychopathology (general). Three investigations included general or nonspecific measures of psychopathology in their risk analyses. Rosen, Tacy, and Howell (1990) and Rosen, Compas, and Tacy (1993) found that initial scores on the Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982) did not predict eating disorder symptoms or dieting behaviors at follow-up. Similarly, Wlodarczyk-Bisaga and Dolan's (1996) and Leon and others' (1999) research indicated that a history of psychological problems or treatment did not predict which subjects would develop eating problems.

Sexuality. Three studies included various measures of "sexuality" as proposed risk variables, with all investigations concluding that this variable did not relate to increased risk for development of eating disorders. However, drawing conclusions about the function of sexuality as a risk indicator is made difficult by the different definitions used in the studies. For example, Leon et al. (1995) studied positive and negative attitudes toward sexuality, as assessed by a 12-item checklist. Patton et al. (1990) studied behaviors by including measures of contraceptive use and history of having a boyfriend or a sexual relationship. Wlodarczyk-Bisaga and Dolan (1996) also investigated history of having a boyfriend and sexual activity. It is notable that no prospective studies have examined history of sexual abuse as a possible risk variable, despite recent correlational studies investigating its possible relationship to eating disorders (Connors & Morse, 1993; Douzinas, Fornari, Goodman, & Sitnick, 1994).

Psychological stress. Two studies have addressed the question of whether stressful life events function as a risk factor for the development of eating disorders. Wlodarczyk-Bisaga and Dolan (1996) included a history of significant, stressful losses (e.g., divorce or death in the family) in their investigation. Subjects who developed eating disturbances did not have higher levels of these life events than subjects whose eating patterns remained within normal limits. Rosen and others' (1993) investigation of the relationship between the degree of daily and major life stress and onset of eating disorder symptoms did not find a predictive relationship between these two variables. It is possible, then, that psychological stress might more accurately be seen as a consequence of eating disorder symptoms, rather than a cause.

Drive for thinness. The high desire to be thin and intense fear of gaining weight, prototypical characteristics of women with eating disorders, were included in five prospective investigations. Garner et al. (1987) found that a strong desire to be thin (as evidenced by higher scores on the EDI's drive for thinness subscale) was predictive of subsequent development of eating disorders. A 10-year prospective study of college women revealed that the EDI's drive for thinness subscale was a significant predictor of later scores on the EDI's bulimia subscale (Joiner, Heatherton, & Keel, 1997). Similarly, Button and others' (1996) findings indicated that an item assessing fear of fatness made a significant, independent contribution to prediction of later eating disorder symptoms. However, Killen, Taylor and others' (1994, 1996) investigations did not find a high desire for thinness to be of predictive significance. These results, however, may be misleading due to the variables included in the analyses. Their measure of weight concerns (which

was the only variable found to significantly predict onset in both investigations) correlated to a moderately high degree with the EDI's drive for thinness subscale. Thus, the substantial overlap between these two constructs suggests that a high desire for thinness may be a risk factor for development of eating disorders.

Body dissatisfaction. Dissatisfaction with one's body shape, another prototypic characteristic of women with eating disorders, was prospectively studied in seven separate investigations, with conflicting results. The research by Attie and Brooks-Gunn (1989) and Garner et al. (1987) identified body dissatisfaction (as assessed by the EDI) as a factor predictive of subsequent eating problems. However, three other studies (Killen et al., 1996; Killen, Taylor et al., 1994; Leon et al., 1995), found that the EDI's body dissatisfaction subscale did not significantly predict onset. Barnett (1996) found that although changes over time in this variable were related to the development of bulimic symptoms, initial scores alone were not predictive. Finally, Wlodarczyk-Bisaga and Dolan's (1996) results showed that although dissatisfaction with one's body was higher in women who developed eating disturbances, it did not significantly predict onset of symptoms.

There are several possible explanations for these discrepant findings. First, the results of the studies by Killen, Taylor et al. (1994, 1996) could have been affected by the overlap between their construct of weight concerns and that of body dissatisfaction. Second, as noted by Leon et al. (1995), the predictive strength of body dissatisfaction could have been dissipated due to the high prevalence of dissatisfaction with one's body in populations of normal adolescent girls. The extended length of time between the two

assessments in Barnett's (1996) study (i.e., 5 years) may have obscured a predictive relationship between the variables. Finally, small clinical sample sizes in two of the studies (Barnett, 1996; Wlodarczyk-Bisaga & Dolan, 1996) make interpretation of some findings difficult. Given this information, it seems plausible to conclude that dissatisfaction with one's body shape may be a risk factor for the development of eating disorders.

Concerns about weight. Although Killen, Taylor and others' (1994, 1996) measure of weight concerns shares considerable conceptual overlap with the EDI body dissatisfaction and drive for thinness subscales, as well as with measures of dietary restraint, it does deserve separate mention, due to its success in predicting eating disorder symptom onset in two separate prospective investigations. At this time, it remains unclear whether any differences between weight concerns and the other, similar measures are great enough to warrant viewing this as assessing a distinct risk factor, rather than as assessing the same underlying risk vulnerability.

Lack of interoceptive awareness. Poor interoceptive awareness as a risk factor for eating disorders--although supported by correlational research and clinical opinion--has been associated with mixed results in the prospective studies conducted to date. Specifically, Killen, Taylor and others' (1994), Killen and others' (1996), and Garner and others' (1987) research indicated that interoceptive awareness, as measured by the EDI, was not predictive of later eating patterns. However, Leon et al. (1995), also using the EDI, found that poor interoceptive awareness was the best predictor of subsequent disordered eating. These authors theorized that the strict dieting, bingeing, and

compensatory measures often found in women with eating disorders may be a coping mechanism (learned through cultural and familial influences) that decreases anxiety over negative, confusing, and indistinct emotional states. They also noted that an underlying constitutional vulnerability could be associated with these deficits in emotional recognition and modulation. Nevertheless, in light of the conflicting findings regarding this predictive variable, future research is necessary to clarify the relationship between poor interoceptive awareness and development of eating disorders.

Dietary restraint. As with many of the other variables under investigation, the role that dietary restraint may play in the onset of eating disorders remains unclear. Despite strong correlational and observational data linking restraint to eating problems (Cooper, 1995; Polivy & Herman, 1995), none of the three prospective studies examining the relationship between dieting and development of eating problems found that dietary restraint per se was predictive of subsequent eating disorders. These findings, however, may not yield a completely accurate depiction of the relationship between dieting and disturbed eating patterns. Killen, Taylor and others' (1994, 1996) failure to identify restraint as a predictive variable could have been affected by the overlap between their concept of weight concerns and their measure of dietary restraint, as discussed earlier. Although restraint was not included in the risk analysis by Leon et al. (1995) due to substantial overlap with other, more powerful variables, the authors noted a significant correlation between initial presence and severity of dietary restraint and subsequent eating disorder symptoms. Finally, although dieting was not predictive of future eating problems in Patton's (1988) research, a high proportion of girls who developed eating

problems were dieting at the time of first assessment. In fact, Patton noted that "in demonstrating the progression from dieting to caseness over 12 months, this study gives some support [for the view that] dieting itself should be regarded as an aetiological factor, rather than simply a symptom of an eating disorder" (p. 583).

Despite the lack of hard prospective evidence, these results suggest the possibility of a causal relationship between restraint and eating disorders, and attest to the necessity of further investigation into the nature of this association.

Cognitive distortions. Only one prospective study has investigated the role of cognitive distortions in eating disorder onset. This lack of research is notable, given that unhealthy or irrational cognitions regarding eating and weight issues frequently assume central importance in the treatment of eating disorders. Barnett (1996) found that scores on the physical appearance subscale of the Bulimia Cognitive Distortions Scale (BCDS; Schulman, Kinder, Powers, Prange, & Gleghorn, 1986) did not predict onset of bulimic symptoms. However, changes in scores on this measure were related to subsequent development of eating disorder symptoms over time. Future studies could be enhanced by inclusion of more comprehensive measures of cognitive distortions related to eating and weight issues.

Ineffectiveness/low self-esteem. Seven prospective studies investigated whether poor self-esteem (or feelings of ineffectiveness) predicted onset of problem eating behaviors. The six studies that used the EDI's ineffectiveness subscale (measuring feelings of inadequacy, low self-worth, and insecurity) found no relationship between this variable and development of eating problems (Attie & Brooks-Gunn, 1989; Barnett,

1996; Garner et al., 1987; Killen et al., 1996; Killen, Taylor et al., 1994; Leon et al., 1995). However, Button and others' (1996) investigation using an alternate measure of self-esteem (Rosenberg's Self-Esteem Questionnaire; Rosenberg, 1965) revealed a significant contribution of self-esteem to the prediction of EAT-26 scores. The failure of the low self-esteem/ineffectiveness construct to predict eating disorder onset in six of seven studies is somewhat surprising, given the strong clinical sentiment that low self-esteem is a core feature of women with eating disorders. Alternatively, however, it is possible that the frustration over chronic failure to control one's dieting and eating disorder symptoms, along with the embarrassment over loss of impulse control (e.g., bingeing, purging), may lead to feelings of low self-esteem after women develop an eating disorder. Additional inquiry in this area is needed.

Externalizing behaviors, social problems, and introversion. Externalizing behaviors were studied in two separate investigations. Attie and Brooks-Gunn (1989) included the variables of impulsivity/hyperactivity, and aggressive or delinquent behavior in their risk analysis; also, Killen, Taylor et al. (1994) included a measure of aggression. Both studies found that these externalizing behaviors did not appear to successfully predict onset of eating disturbances. Two investigations also examined the predictive relationship between perceived social problems and problematic eating, with mixed results. Although one study found no relationship between a measure of unpopularity and eating problems (Killen, Taylor et al., 1994), another found that perceived social problems was an effective predictor of diagnostic status upon follow-up (Patton, 1988). Thus, future studies in this area are necessary before conclusions regarding risk can be

drawn. Finally, introversion (as measured by the General Health Questionnaire; Goldberg & Hillier, 1979) was found to predict onset of eating disturbances in the one study that investigated this variable (Patton, 1988). Replication of this latter finding would certainly be of interest.

Substance use. Two studies examined the relationship between substance use and later development of eating disorder symptoms. Killen et al. (1996) found that frequency of alcohol consumption was not related to the likelihood of developing an eating disorder. Leon et al. (1999) studied the variable of "substance-related impulsivity," which included measures of smoking frequency, drinking frequency, and scores on a constraint and impulsivity scale. The results did not support the hypothesis that higher problems with impulsivity and substance use would be related to higher incidence of eating problems.

Additional EDI subscales: perfectionism, interpersonal distrust, maturity fears, and bulimia. Six prospective studies (Attie & Brooks-Gunn, 1989; Garner et al., 1987; Joiner et al., 1997; Killen et al., 1996; Killen, Taylor et al., 1994; Leon et al., 1995) assessed whether the perfectionism subscale (measuring excessive achievement demands placed on oneself) of the EDI predicted eating disorders at a later point in time. Only one of the five studies (Joiner et al., 1997) found that this measure predicted subsequent development of eating disorder symptoms (as measured by the EDI's bulimia subscale).

Five studies investigated whether the EDI's interpersonal distrust subscale was related to later eating disorder symptoms (Garner et al., 1987; Joiner et al., 1997; Killen et al., 1996; Killen, Taylor et al., 1994; Leon et al., 1995). The interpersonal distrust

subscale was not found to be related to development of eating disorder symptoms in any of these five studies.

The relationship between initial scores on the EDI's bulimia subscale and later eating disorders was studied in three investigations (Garner et al., 1987; Killen et al., 1996; Killen, Taylor et al., 1994). The inclusion of this variable in a prospective study of risk factors is questionable, due to the fact that there exists a large overlap between diagnostic symptoms of bulimia nervosa per se and items on the EDI's bulimia subscale. Nevertheless, the results of all three studies revealed that initial scores on this measure did not significantly predict the later development of eating disorders.

EDI's maturity fears subscale was included in five prospective investigations (Garner et al., 1987; Joiner et al., 1997; Killen et al., 1996; Killen, Taylor et al., 1994; Leon et al., 1995). Only Joiner et al. (1997) found a significant relationship between early elevations on maturity fears subscales and later development of eating disorder symptoms (as measured by the EDI's bulimia subscale).

Prospective Research Design Issues

The preceding review of prospective studies raises a number of relevant issues regarding optimal research methodology in studies investigating risk factors for eating disorders. Two main methodological weaknesses exist within the prospective studies conducted to date. First, many rely exclusively on data gathered from paper-and-pencil questionnaires for classifying or diagnosing eating problems. Self-report inventories do have a number of distinct advantages in initial screening for eating disorders: they do not

require a trained interviewer and are easy to administer, they are economical, and they rely on objective scoring procedures (Garner, 1995). However, self-report inventories have been criticized on the grounds that they may be less accurate than interview methods, especially when complex behaviors such as bingeing are being assessed (Fairburn & Belgin, 1994; Garner, 1995). For example, the wording of binge-related items in self-report questionnaires may connote simple overeating to some subjects, and the overendorsement of the bingeing or dieting items by nonclinical subjects is common. As a result, although questionnaires can serve as appropriate screening instruments, semi-structured clinical interviews are almost universally deemed necessary in the diagnosis of eating disorders (Crowther & Sherwood, 1997; Fairburn & Belgin, 1994; Garner, 1995). Only half of the prospective studies conducted to date, however, have incorporated clinical interviews into their design methodology (see Table 2).

The second methodological limitation found in many of the prospective studies involves confounding effects in the classification of "high risk" versus "eating disordered" individuals. It is possible that some risk factor themes identified at initial assessment are basically measuring existing eating disorder symptoms. Risk measures should, ideally, not center too much on issues that represent diagnostic criteria for eating disorders per se (e.g., bulimia, EAT-26 scores), because it could be argued that researchers' supposed "at risk" samples actually include persons who would already meet a number of DSM-IV (1994) criteria for an eating disorder. Individuals with subclinical or clinical eating disorders need to be differentiated from the "high risk" group and

removed from the risk sample, so they are not construed as "new" or "emergent" cases at the follow-up assessment.

The current study incorporated structured clinical interviews in its design. This should have reduced the number of false positive identifications often found with questionnaires, and allowed removal of possible eating disorder cases from the "at risk" sample.

Conclusions from Prospective Research

This examination of the literature on risk factors for eating disorders emphasized the importance of relying on prospective methodologies for identifying early characteristics predictive of subsequent abnormal eating patterns. The results of the literature review revealed that only three variables--body dissatisfaction, drive for thinness, and weight concerns--were found to be significantly related to eating disorder onset by more than one study (Tables 3 and 4). Even for these three variables, though, conflicting results were present. For example, five additional prospective studies found that body dissatisfaction was not related to eating disorder onset, and two found that drive for thinness was not predictive of development of eating problems.

Eleven variables were found to be significantly related to eating disorder onset by just one prospective investigation: age, childhood eating patterns, family psychopathology, ineffectiveness, poor interoceptive awareness, introversion, maturity fears, perfectionism, race, social problems, and weight. Despite this initial indication that these variables may increase risk for developing eating problems, it is difficult to draw

Table 4

Results of Prospective Studies: Risk Variables Significantly Related to Later Eating Disorder Onset

Criteria	Risk variable
Significant results found in more than one study	Body dissatisfaction
	Drive for thinness
	Weight concerns
Significant results found in only one study	Age
	Childhood eating patterns
	Family psychopathology
	Ineffectiveness
	Poor interoceptive awareness
	Introversion
	Maturity fears
	Perfectionism
	Race
	Social problems
	Weight

firm conclusions due to contradictory findings in other prospective investigations. In particular, at least one additional investigation found that the variables of age, family psychopathology, ineffectiveness, maturity fears, perfectionism, weight, social problems, and poor interoceptive awareness were not significantly related to later development of eating problems. The remaining three variables--childhood eating patterns, race, and introversion--have only been included in one investigation each. Obviously, there exists a great need for future replication studies to clarify these issues and to determine each variable's possible contribution to the development of eating disorders.

The direction of the results for the remaining variables included in prospective investigations appears more clear. Four variables (class, depression, interpersonal distrust, and pubertal timing) failed to significantly relate to eating disorder onset in four or more separate studies. Five variables (family relationships, psychopathology, restraint, sexuality, bulimia/bingeing) were not related to later eating problems in three or more investigations, and three proposed risk indicators (aggression/delinquency, stress, substance use) yielded negative results in at least two studies. However, despite the seemingly clear indications that these variables do not function effectively as risk factors for eating disorders, possible limitations in the literature should be addressed before any final conclusions can be drawn.

Models of Eating Disorder Etiology

Ideally, any study of risk factors should shed light on existing theoretical models of eating disorder etiology, either by supporting, contradicting, or expanding elements of

the model. This process promotes further refinements in existing theory by suggesting modifications that would make the model more consistent with empirical research findings. In this section, two theories of eating disorder etiology will be presented: (a) a tri-stage model developed by Cooper (1995), and (b) a cognitive model proposed by Fairburn, Marcus, and Wilson (1993). The purpose of this review is not to provide a comprehensive analysis or critique of all proposed models of etiology. Rather, it will allow a brief comparison of theoretical expectations from several models with empirical research findings and will provide a framework for later discussion of the results of the present study in light of empirical models.

Cooper (1995) proposed a tri-stage model of eating disorder development and maintenance. He emphasized that no one factor in isolation can account for the development of an eating disorder; rather, it involves a complex interaction over time between "the occurrence of circumstances that activate the individual's vulnerability to particular risk factors and on the operation of protective factors" (p. 199). According to his theory, the initial stage of eating disorder development encompasses events and factors arising between birth and the initial, behavioral precursors of the eating disorder (typically dieting). The second stage involves the period between the development of behavioral precursors to the onset of a frank eating disorder. The final stage encompasses the interaction between maintaining factors and protective factors in determining whether the eating disorder becomes chronic, or whether it remains transient and quickly resolves. The focus of the current study and of the literature reviewed in this chapter lies primarily within the realm of the first and second stages. Cooper identified a number of possible

early factors and events that may predispose an individual to developing behavioral precursors to an eating disorder. He hypothesized that a persistent history of depression may play a role in the progression from the first to the second stage, particularly in the development of bulimia. Other potential factors included possible abnormal serotonin pathway functioning, implicated in both depression and eating disorders; low levels of self-esteem, resulting in dieting to enhance one's appearance and sense of control; obsessive-compulsive (perfectionistic) personality traits, particularly in the onset of anorexia; premorbid obesity, leading to dieting; adverse life events, such as childhood sexual abuse (which would only lead to progression to the second stage through combination with other risk factors); family history of psychiatric problems; and internalization of societal pressures for thinness.

Cooper (1995) noted that only a minority of individuals who develop a behavioral precursor, such as dieting, actually progress to a complete eating disorder. Therefore, specific additional factors must combine with dieting to increase the risk for the development of a subsequent disorder. He indicated that little research evidence exists on this question. However, he hypothesized that potential factors could include cognitive distortions about shape and weight; negative life events, such as death of a close relative, illness, or negative comments about one's appearance; and normal developmental factors such as leaving home, beginning new relationships, or the onset of puberty. He noted that many of these stressors are not specific to eating disorders, and that little is known about how these factors may interact to predispose an individual to the development of an eating disorder. The timing of the events, as well as the interaction with other factors,

may be the crucial elements in explaining the progression from simple dieting to a fully developed eating disorder.

A cognitive view of the development and maintenance of bulimia nervosa has been described by Fairburn et al. (1993). This cognitive model emphasized the importance of low self-esteem, cognitive distortions, high concerns about body shape and weight, and strict dieting in the development of eating disorders. The authors argued that low self-esteem often forms the foundation of problems with bulimia. Extreme concerns about weight and shape combine with these poor levels of self-esteem, leading to strict regulations about food intake and subsequent dieting. Dieting alone, however, does not typically result in development of an eating disorder without the presence of significant cognitive distortions about eating and weight. For example, rigid beliefs about whether particular foods are "good" or "bad," in combination with dieting, can promote binge eating. In individuals with poor self-esteem, dissatisfaction with their bodies, and highly distorted beliefs about the importance of weight and shape, the binge eating often results in increased feelings of ineffectiveness and dissatisfaction with self and weight, leading to heightened efforts to control weight (through a return to strict dieting or through engaging in unhealthy weight control strategies such as vomiting or use of laxatives).

This cognitive model, then, accentuated the role of distorted cognitions, low self-esteem, dissatisfaction with weight and shape, and subsequent strict dieting behavior in the development of eating disorders. The authors did not emphasize or attempt to identify particular factors or dynamics that might cause individuals to fail to initially develop healthy self-esteem, body satisfaction, and attitudes and cognitions about eating

and weight issues. One needs to extrapolate from the broader literature to draw conclusions regarding the types of learning experiences, developmental factors, and biological underpinnings that might contribute to these initial difficulties that would then predispose an individual for later development of an eating disorder.

Selection of Risk Criteria

The preceding literature review suggested ample areas for future research on eating disorder risk. Although many of the purported risk variables deserve further investigation, the present study narrowed the risk criteria under primary investigation to four. Several rationales guided the selection of risk variables included in this study. First, variables had to be selected from those available in an extant data set on the subjects used in the present investigation. As will be described in detail in the methods chapter, the initial data on these subjects were collected as part of a prior research study. Hence, the selection of variables available for longitudinal analyses in this project was limited by the instruments and measures administered as part of the earlier study. Data collected in the prior study included the following: subscales of the ABI (anorexia, binge, purge, depressed mood, anxiety, maladaptive cognitions, parent conflict, anergia, and exercise; Stein, 1991b), subscales of the EDI (drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears; Garner & Olmsted, 1984), age, height, and weight. Thus, these measures constituted the pool from which the variables in the current study were chosen.

Second, it was decided that the risk criteria must be distinct from overt behavioral eating disorder symptoms and DSM-IV (1994) criteria per se. In risk studies it is critical to be able to differentiate individuals who were initially at risk but nonsymptomatic, from those who were actually in the early, premorbid stages of an eating disorder. Therefore, variables measuring such behavioral symptoms as bingeing, purging, or excessive dietary restraint (e.g., EDI's anorexia and bulimia subscales) cannot be used as risk variables.

Third, the present author decided that a risk variable could be included in the present study if results from prior longitudinal studies suggested that the variable might be related to onset of eating disorder symptoms. If prior studies did not suggest that a given variable increased the risk for developing an eating disorder, compelling reasons justifying further study of the variable had to exist to warrant inclusion of the variable in the current study. Such reasons might include significant limitations in prior studies investigating the variable, or strong clinical sentiment supporting the variable, despite initial longitudinal findings to the contrary.

Finally, it was decided that selected risk variables should be consistent with existing theoretical models of eating disorder etiology. This would allow the results of the current empirical study to contribute to the ongoing process of the development and refinement of theoretical models.

Based on these criteria, the four variables selected for primary investigation in the present study included: (a) drive for thinness, (b) feelings of ineffectiveness, (c) depressed mood, and (d) maladaptive (eating) cognitions. The following section

contains more specific descriptions of the rationale for the inclusion of these particular constructs as the risk factors under principal investigation.

Drive for Thinness

An excessive preoccupation and concern with body weight, shape, and dieting are cardinal features of both anorexia and bulimia, but its status as a predisposing risk factor has not been conclusively determined. Cooper (1995) posited that a strong internalization of societal pressures for thinness and a heightened desire for thinness increases the risk for developing an eating disorder. The cognitive model of eating disorder etiology also emphasized the role of high concerns about body shape and weight in eating disorder onset. The conflicting results found in existing prospective studies (Button et al., 1996; Garner et al., 1987; Joiner et al., 1997; Killen et al., 1996; Killen, Taylor et al., 1994), however, underscore the need for further investigation of this purported risk variable. A high desire for thinness was chosen for inclusion rather than dissatisfaction with body shape or size. Although both variables appear to have strong promise as potential risk factors, there exists a relatively high correlation (redundancy) between the two measures (EDI's body dissatisfaction and drive for thinness subscales, $r = .62$; Garner, 1991). Rather than include two measures with substantial overlap, only one measure was chosen for inclusion. Thus, the current study included the drive for thinness subscale of the EDI as a proposed risk measure.

Ineffectiveness

Garner (1991) stated that the EDI ineffectiveness subscale "assesses feelings of

general inadequacy, insecurity, worthlessness, emptiness, and lack of control over one's life," and that although it is conceptually similar to low self-esteem and self-efficacy, it also includes feelings of aloneness and emptiness (p. 5). Both the cognitive model and Cooper's (1995) tri-stage model of eating disorder etiology emphasize the role of low self-esteem in the development of eating disorders. The contradictory findings in the prospective literature, along with the fact that many theorists and clinicians continue to view low self-esteem as a predisposing factor for the development of eating disorders, underscores the need for further research into this proposed variable. Thus, the EDI ineffectiveness subscale was included from the extant data set as a variable in the current study.

Depressed Mood

Some theoretical models of eating disorder etiology have emphasized the role of depression in the onset of eating disorders (e.g., Cooper, 1995), while others have not seen this as a core factor (e.g., Fairburn et al., 1993). The review of the literature revealed that no prospective studies conducted to date have found a significant link between depression and eating disorder onset. The strong clinical sentiment that depression or difficulty in regulating unpleasant or negative moods may be related to eating disorder risk, along with the fact that some models of etiology do implicate the role of depression, provide justification for additional study of this variable. Most of the existing prospective studies measured the construct of depression using the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The

current study used the depressed mood subscale of the ABI to measure this construct. This measure was the only assessment of depression included in the original data collection; hence, the present study was restricted to using this instrument in studying the contribution of depression to eating disorder onset. However, as will be discussed in Chapter III, the ABI depressed mood subscale, based on DSM-IV (1994) criteria for Major Depression, has been shown to have adequate psychometric properties in measuring depressive symptoms. An unintended advantage of employing this instrument is that it expands the number of depression instruments that have been used in prospective eating disorder onset research.

Maladaptive Cognitions

Researchers have found that the presence of cognitive distortions regarding eating, body image, and weight are far more frequent in women with eating disorders than in nonclinical women (e.g., Schulman et al., 1986). The role of such cognitive distortions is central to the cognitive model of eating disorder etiology, and is also implicated in Cooper's (1995) tri-stage model of etiology. Barnett (1996) included a measure of cognitive distortions in her risk analysis (the physical appearance subscale of the Bulimia Cognitive Distortions Scale). This subscale, however, measured only a circumscribed range of cognitive distortions related to eating disorders (i.e., those associated with body image). No prospective studies have yet investigated the role of global, eating-related cognitive distortions on the development of eating disorders. In the current study, scores on the maladaptive cognitions subscale from the ABI (Stein, 1991b) were investigated as

a possible risk factor. This subscale includes items assessing a wide range of distorted cognitions often found in women with eating disorders (e.g., "Anyone can be overweight, but it takes someone special to be thin," "If I fail in my diet, I must be a weak person," "People who are overweight risk rejection by loved ones").

Changes in Risk Factors after Recovery

A final area of investigation in the proposed study involved assessing changes in risk factors that occur with full or partial recovery from an eating disorder. Specifically, do risk factors that predict eating disorder onset decrease when eating disorder symptoms decrease, or do the risk factor scores remain at elevated levels? Data on this question have implications for understanding treatment progress and the risk of relapse.

A recent review of eating disorder outcome studies concluded that a number of psychological symptoms may remain at elevated levels in women who have recovered from anorexia or bulimia (Mukai, 1996). Characteristics that tend to persist even after formal eating disorder behavioral symptoms are no longer present include dietary restraint; distorted attitudes toward food, weight, and eating; and high levels of body dissatisfaction. However, some psychological problems, such as depression, may be more likely to normalize after the reduction of overt, behavioral eating disorder symptoms.

Other research also suggests that some risk factors may not remain problematic in women whose eating disorder symptoms have improved. Two prospective studies that investigated changes occurring in various behavioral, psychological, and psychosocial

variables after treatment for an eating disorder reported improvement in variables that have been considered possible risk factors. A 10- to 15-year follow-up study of women who received inpatient treatment for anorexia revealed that recovery from an eating disorder was associated with improved psychosocial functioning (defined as work status, social relating, and life satisfaction; Strober, Freeman, & Morrell, 1997). Similarly, a six-year prospective study of women who received treatment for bulimia found significant decreases in scores on EDI subscales (including drive for thinness and ineffectiveness), as well as improvement in levels of depression, anxiety, and body image (Fichter & Quadflieg, 1997). Finally, a meta-analytic review of outcomes following cognitive behavior therapy for bulimia concluded that cognitive distortions were lower in women whose eating disorder had improved (Lewandowski, Gebing, Anthony, & O'Brien, 1997).

Thus, the current literature provides conflicting findings regarding changes in risk factors after recovery from an eating disorder. Although there is some evidence that the four variables included in this study may remain at high levels, other research suggests that scores on some of these variables should decrease in subjects who recover from an eating disorder. It is probable that scores on all four risk factors will decrease to some extent in recovered subjects. However, it is also possible that of the four hypothesized risk factors included in the proposed study, the ones that involve cognitive elements specifically related to eating disorders (drive for thinness and maladaptive cognitions) may be more resistant to change and therefore may show a less marked decrease over time. These may be more recalcitrant because they represent highly internalized values and beliefs that are pervasive in society, are modeled to girls at a very early age, and are

socially reinforced throughout development. Those factors that involve more general psychological functioning (ineffectiveness and depressed mood) may be more likely to normalize with improvement of the eating disorder.

Rationale and Research Hypotheses

All but one longitudinal study of eating disorder risk factors conducted to date have evaluated girls who were in junior high or high school at the time of initial assessment. Research on girls in this younger age group could be especially useful in identifying risk factors for anorexia, with its bimodal peaks of onset at ages 14 and 18 (DSM-IV, 1994). However, the average age of onset for bulimia is late adolescence or early adulthood. Research using subjects in their late teens or early twenties, therefore, could be helpful in identifying risk factors relevant to this particular older age group. The sample in the current study was drawn from a slightly older, college population, helping to remediate this gap in the literature. Furthermore, because the primary purpose of this study was to investigate factors that may place an individual at risk for the development of an eating disorder, subjects were drawn from a nonclinical (rather than clinical) university population. Males were not included in the current study due to the low prevalence rates of eating disorders in male populations, and because the factors which place males at higher risk for developing eating disorders may be very different than those for women. Thus, a study of risk factors for males would best be conducted in a separate investigation.

The following hypotheses guided the proposed study:

1. College women scoring, at initial assessment, at or above the median on each of the four variables (drive for thinness, ineffectiveness, depressed mood, and maladaptive cognitions) will evidence higher incidence rates of eating disorders (at the 4-year follow-up) than the low risk group (individuals who scored below the median on the risk indices).

2. Initial scores on the four risk factors together will explain a significant amount of variance in later eating disorder symptom severity.

3. Changes over time in each of the risk factor scores will be related to later severity of major eating disorder symptoms (because it would be expected for scores on risk factors to covary with symptom outcomes).

4. Subjects who recover from an eating disorder over a 4-year interval will show a decrease in scores on all four risk factors. Scores on the depressed mood and ineffectiveness subscales will have decreased more dramatically than scores on maladaptive cognitions and drive for thinness subscales.

5. The prevalence rates of anorexia and bulimia in this university sample will be similar to the recent conservative estimates (i.e., 0.5% to 3.0%) reported in well-controlled research studies. On the other hand, prevalence rates for eating disorder NOS will approximate 7 to 13%.

6. The 4-year incidence rate of eating disorders (anorexia, bulimia, and eating disorder NOS) will be relatively low (i.e., approximately 3% to 4%), similar to estimates found in other prospective research studies.

CHAPTER III

METHOD

Subjects

The initial (Time 1) subject selection and data collection were part of a prior eating disorder research project conducted at Utah State University during the 1992-93 academic year by David Stein, Ph.D. Subjects in this prior study (N = 457) were female undergraduate students enrolled in introductory psychology classes. The response rate in various classes ranged from 70% to 95%. Appendix A contains a copy of the human subjects review approval letter, and Appendix B contains the subject consent form for the current study.

For the present investigation, these 457 subjects were recontacted approximately four years later and asked to participate in the current study. Of the initial subject pool, the author was able to trace and recontact 253 women (55%), despite the fact that the vast majority of subjects were no longer students at the university. Sixty-two subjects who were successfully contacted either declined to participate in this follow-up phase of the research project (n = 7) or initially agreed to participate but ultimately failed to complete the assessment materials (n = 55). Thus, of the 253 contacted subjects, 191 (75%) successfully progressed from the initial (Time 1) to the follow-up (Time 2) stage of data collection. The completion rate for all subjects who completed the prior study, however, was much lower (42%; 191 out of 457)--a loss of subjects primarily due to inability to locate subjects after the four-year interval. An examination of completion rates in the

prospective studies discussed in the preceding literature review revealed that completion rates were highly dependent on the length of elapsed time between the follow-up assessment (see Table 2). Studies with short-term intervals (under 1 year) had average completion rates of 84%. This rate dropped to 72% with intervals ranging from 1 to 2 years, and to 66% for intervals up to 3 years in length. Unfortunately, of the six studies with intervals of 4 or more years, only two provided information on completion rates (67% in a sample of middle-school girls, most of whom remained enrolled in the school throughout the 4-year interval; 57% in a college sample). The remaining four studies' failure to provide information about completion rates raises the question of whether authors omitted subject attrition rates due to seemingly low completion rates across these longer intervals. Regardless, it seems likely that completion rates in prospective studies of 4 or more years, especially when samples consist of subjects who are more likely to change residences (e.g., college-age young adults versus middle or high school-age youth), would be substantially lower than those found in studies with shorter intervals and younger subjects.

Instruments

Subjects completed the EDI (Garner & Olmsted, 1984) and the ABI (Stein, 1991b) in both the Time 1 and Time 2 assessment phases. At the follow-up assessment, all individuals also participated in a structured clinical interview focused on eating disorder symptoms as outlined in the DSM-IV (1994). Data from these interviews were

coded and scored with the Clinician's DSM-IV Checklist (Stein, 1991c), yielding both categorical diagnoses and numerical estimates of severity of eating disorder symptoms.

Eating Disorder Inventory

The Eating Disorder Inventory (EDI), a 64-item self-report questionnaire, assesses various behavioral and psychological features thought to be associated with bulimia and anorexia nervosa. The instrument provides eight subscale scores (rather than one overall, global score) for the following domains: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, and maturity fears. Although a more recent version of the EDI has been published (EDI-2; Garner, 1991), the current study used the original inventory due to questionable psychometric properties of the provisional subscales in the revised instrument (Eberenz & Gleaves, 1994).

Psychometric properties of the original EDI appear adequate to high. Test-retest reliability correlations for the EDI subscales ranged from .65 to .97 over a 3-week interval (Wear & Pratz, 1987) and from .41 to .75 over a 1-year interval (Crowther, Lilly, Crawford, & Shepherd, 1992). Garner and Olmsted (1984) reported adequate internal consistency of EDI subscales with eating disorder subjects (Cronbach's alpha coefficients ranged from .83 to .93). The concurrent validity of the EDI was supported by significant correlations between EDI subscales and another eating disorder screening measure (Garner, Olmsted, & Polivy, 1983). Garner and Olmsted (1984) reported significant correlations between various EDI subscales and other instruments measuring symptoms

associated with eating disorders, including depression, feelings of inadequacy, lack of energy, and interpersonal sensitivity. Concurrent validity of the instrument was further supported by significant correlations between EDI subscales and clinicians' severity ratings of individuals with eating disorders (r = .43 to .68; $p < .001$). Factor analyses of the EDI with both eating disordered and nonclinical college samples have yielded eight-factor solutions paralleling the subscales (Welch, Hall, & Norring, 1990; Williams, Schaefer, Shisslak, Gronwaldt, & Comerci, 1986). However, in a separate investigation with a college sample, only three interpretable factors emerged (Welch, Hall, & Walkey, 1988), suggesting that the EDI may measure slightly different features in clinical versus at-risk populations.

Drive for thinness subscale. The drive for thinness subscale of the EDI (one of the hypothesized risk variables in the current investigation) was designed to assess the excessive pursuit of thinness and the fear of weight gain seen in women with eating disorders (Garner et al., 1983). Concurrent validity of the drive for thinness subscale was supported by significant correlations with the Eating Attitudes Test (r = .71; $p < .0001$) and measures of dietary restraint (r = .48; $p < .0001$; Garner & Olmsted, 1984). Mean scores on the drive for thinness subscale differed significantly for anorexia, bulimia, and nonclinical college samples ($p < .05$; Garner & Olmsted, 1984). A Cronbach's alpha coefficient of .87 indicated adequate internal consistency (Garner & Olmsted, 1984), and estimates of test-retest reliability ranged from .72 (1-year interval; Crowther et al., 1992) to .92 (3-week interval; Wear & Pratz, 1987) with nonpatient samples.

Ineffectiveness subscale. As mentioned previously, the EDI's ineffectiveness subscale measures feelings of inadequacy, low self-esteem, emptiness, insecurity, and lack of control over life events. Cronbach's alpha coefficients ranged from .88 with a nonclinical sample to .93 with an eating disorder sample (Garner & Olmsted, 1984). Test-retest reliability over short intervals was adequate (r = .85 over a 3-week interval; Wear & Pratz, 1987) but was lower over a 1-year interval (r = .55; Crowther et al., 1992). Scores on the ineffectiveness subscale successfully differentiated between eating disorder patients, recovered anorexics, and nonclinical college populations (Garner & Olmsted, 1984). Significant correlations with measures of feelings of inadequacy (r = .73; $p < .0001$) and locus of control (r = .42; $p < .0001$) provided support for the concurrent validity of the ineffectiveness subscale (Garner & Olmsted, 1984).

Anorexia-Bulimia Inventory

The Anorexia-Bulimia Inventory (ABI; Stein, 1991b) is a 75-item self-report questionnaire designed to assess the DSM-IV (1994) diagnostic symptoms of both anorexia and bulimia, as well as the associated problem areas that are often the focus of eating disorder treatment. Its nine subscales consist of anorexia, binge, purge, depressed mood, anxiety, maladaptive cognitions, parent conflict, anergia, and exercise. The instrument does not provide a global score; rather, it yields nine individual subscale scores.

Stein (1991b) found that all ABI subscales successfully discriminated between nonclinical and eating disordered subjects. The anorexia and binge subscales

differentiated between women with anorexia and bulimia. Discriminant validity of the ABI was further supported by the finding that the ABI correctly classified 93% of subjects into either anorexia, bulimia, or dieting groups (Stein, 1991b). Key eating disorder symptom subscales of the ABI showed significant correlations with clinician severity ratings derived from structured clinical interviews (Stein, 1991b).

Stein (1991b) found significant correlations between scores on the anorexia and binge subscales and actual laboratory eating behaviors, providing evidence of construct validity. Additionally, significant correlations existed between two ABI subscales (anorexia and purge) and tests of memory/perceptual biases towards food-related stimuli frequently observed in individuals with anorexia. Concurrent validity of the ABI was supported by correlations between ABI subscales and a wide variety of other inventories measuring similar constructs (Stein, 1991b). Test-retest reliability correlations (7-week interval) using a combined clinical and nonclinical sample ranged from .63 to .80 ($p < .01$; Stein, 1991b). Levels of internal consistency were acceptable (Cronbach's alpha coefficients ranged from .64 to .94). The factor structure of the ABI was stable and closely paralleled the intuitively designed subscales (Dobmeyer, 1997).

Depressed mood subscale. This subscale was designed to assess some of the major symptoms of depression, including suicidal ideation. Scores on the depressed mood subscale significantly differed for nonclinical (high school and college students) and clinical samples ($p < .05$; Stein, 1991b). Significant correlations with the Symptom Checklist 90-Revised depression subscale ($r = .81$; $p < .001$; Derogatis, 1977) and the Beck Depression Inventory ($r = .68$; $p < .05$; Beck et al., 1961) provided evidence of

concurrent validity (Stein, 1991b). Internal consistency of this subscale was excellent (Cronbach's alpha = .91) and test-retest reliability was adequate (r = .65; p < .01; Stein, 1991b).

Maladaptive cognitions subscale. The maladaptive cognitions subscale includes items that assess expectations and beliefs about dieting's impact on interpersonal relationships, and the benefits of losing weight. It also assesses "rigid and irrational rules about eating" (Stein, 1991b, p. 8). Scores on this subscale were significantly higher for anorexia and bulimia groups compared to nonclinical college and high school subjects (p < .05; Stein, 1991b). Concurrent validity was supported by significant correlations with two subscales of the Bulimia Cognitive Distortions Scale (Schulman et al., 1986): physical appearance (r = .68; p < .05) and automatic behavior (r = .73; p < .05). Test-retest reliability estimates with a combined clinical and nonclinical sample yielded a .73 correlation coefficient (p < .05), and internal reliability estimates for the subscale were high (Cronbach's alpha = .91; Stein, 1991b).

Structured Clinical Interview and Clinician's DSM-IV Checklist

Structured Clinical Interview. The Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1992) is a widely used, comprehensive, semistructured interview designed to assist clinicians in deriving Axis I diagnoses according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R; American Psychiatric Association, 1987). For the current

investigation, only the eating disorder module was used. The interview was slightly modified to align more closely with current DSM-IV (1994) criteria (Appendix C).

Psychometric data on the SCID indicated test-retest reliability was adequate and comparable to those obtained with other published structured diagnostic instruments (Segal, Hersen, & Van Hasselt, 1994; Williams et al., 1992). Estimates of interrater reliability indicated good agreement between raters (kappa values averaging .74 for Axis I diagnoses; Segal et al., 1994).

The Clinician's DSM-IV Checklist. The Clinician's DSM-IV Checklist (Stein, 1991c) provides a method for converting information gathered from the SCID to numerical estimates of the number and severity of DSM-IV (1994) eating disorder symptoms (Appendix D). The checklist consists of 11 items representing DSM-IV (1994) diagnostic criteria for eating disorders. Each item is rated on a scale from 1 to 5 (1 = "Severity or frequency of symptom is extremely low; or symptom is not present" to 5 = "Severity or frequency of symptom is extremely or unusually high for treatment program"). The checklist can be used to provide ratings on three scales: Global Severity Index (GSI), Anorexia Severity Index (ASI), and Bulimia Severity Index (BSI). The checklist also can be used to generate (categorical) diagnoses of anorexia, bulimia, and eating disorder NOS.

For a diagnosis of anorexia nervosa, individuals must receive a score of 3 or higher on Criterion A ("Refusal to maintain body weight over a minimal normal weight for age and height"). Cutoffs for each score (1 to 5) were based on the Metropolitan Life Insurance Weight and Height table for women, with scores of 3 and higher being

equivalent to at least 15% underweight. Criterion B ("Intense fear of becoming obese, even when underweight") and Criterion C ("Disturbance in the way in which one's body weight, size, or shape is experienced") both must be rated as a 3 or higher. Finally, a subject must also receive a 3 or higher on Criterion D ("Absence of at least three consecutive menstrual cycles when otherwise expected to occur--primary or secondary amenorrhea") to qualify for a diagnosis of anorexia.

Bulimia nervosa can be diagnosed when the following criteria are met: Score of a 3 or higher on Criterion E ("Recurrent episodes of binge eating"), F ("During the eating binges there is a feeling of lack of control over the eating behavior"), and H ("Self-evaluation is unduly influenced by body shape and weight"). Subjects must also receive scores of 3 or higher on at least one component of Criterion G ("In order to counteract the effects of binge eating, the individual regularly engages in: (a) self-induced vomiting; (b) use of laxatives, diuretics, or diet pills; (c) rigorous dieting or fasting; or (d) vigorous exercise--defined as 2 or more hours of aerobic exercise per day"). Finally, an individual who meets the criteria for a diagnosis of anorexia cannot also be diagnosed with bulimia.

Eating disorder NOS can be diagnosed when most, but not all, of the criteria for anorexia or bulimia are met. There are three specific clinical presentations that would result in a person with subclinical or atypical symptoms of eating disorders receiving a diagnosis of eating disorder NOS. The first pattern, what might be termed "subclinical bulimia," occurs when an individual meets the same criteria for a diagnosis of bulimia, but at a lower severity or frequency. Specifically, individuals must receive at least a 2 (rather than a 3) or higher on Criteria A (low weight), F (lack of control over binges),

and H (self-evaluation tied to weight), as well as a 2 (rather than a 3) or higher on at least one of the components of Criteria G (the maladaptive compensatory strategies such as vomiting, fasting, use of laxatives or diuretics, or excessive exercise).

The second pattern, what might be termed "subclinical anorexia," occurs when an individual meets the same strict low weight criteria required for a diagnosis of anorexia (3 or higher on Criterion A), but has slightly lower levels of fear of becoming fat (2 or higher on Criterion B), less disturbance in body perception (2 or higher on Criterion C), and more regular menstrual periods (Criterion D can be 3 or lower).

The third symptom pattern yielding a diagnosis of eating disorder NOS occurs when an individual shows clinical levels of binge eating but the absence of maladaptive compensatory measures. Specifically, an individual who receives a 3 or higher on Criterion E (binge eating), F (lack of control over binges), and H (self-evaluation tied to weight), but receives a scores of 1 on all components of Criterion G (maladaptive compensatory strategies), would be diagnosed with eating disorder NOS under this third symptom pattern.

The GSI score is derived by multiplying an individual's scores on each symptom criterion by a respective weighted score, and then summing the products. The weighted scores reflect the degree of health threat each symptom poses, based on judgments of DSM-IV (1994) criteria provided by 16 national eating disorder experts. These professionals listed the following symptoms as posing the highest health threat to individuals with eating disorders (in descending order): weight loss leading to 15% below normal body weight (Criterion A), laxative abuse (Criterion G-2), self-induced

vomiting (Criterion G-1), and binge eating (Criterion E). Thus, the GSI provides an estimation of the overall ASI and BSI scores can also be computed using the Clinician's DSM-IV Checklist. The derivation of these scores is similar to that of the GSI. The ASI represents the overall severity of anorexia symptoms, and the BSI represents the severity of bulimia symptoms in terms of threats to health, as rated by the 16 eating disorder experts.

Procedure

Initial Assessment

Students enrolled in introductory psychology courses during 1992-93 had the opportunity to receive extra credit for their participation in the first phase of this research project, part of an earlier, separate study. Individuals who agreed to participate received (during one of their class periods) a research packet consisting of an informed consent form, the EDI, and the ABI. Subjects completed the inventories in the privacy of their homes and returned them to the investigator at the start of the following class period. To enhance confidentiality, the investigator instructed subjects to put their names and phone numbers on only the consent forms, which were turned in separately from the response forms. Each subject was assigned a special identification number on consent and response forms to allow the investigators to subsequently match up the response forms with identifying information. This was necessary in order to allow comparison of Time 1 and Time 2 data for each individual.

Determination of Initial Risk Status

The first step of the follow-up phase of the current investigation involved determining the risk status of all subjects who participated in the Time 1 data collection procedure. Subjects who scored at or above the median on all four hypothesized risk factors (drive for thinness, ineffectiveness, depressed mood, and maladaptive cognitions) at the initial assessment were designated as meeting the high-risk criteria (the median was selected as the measure of central tendency to avoid problems associated with more extreme scores). The remaining individuals in the sample constituted the low risk (or control) group.

Any subjects who evidenced overt eating disorder symptoms at the initial assessment were removed from risk or control groups (regardless of whether their group was high-risk or control) and placed in the "initially symptomatic" group. The presence of such overt eating disorder symptoms was determined by scores on four subscales: bulimia (from the EDI) and purge, binge, and anorexia (from the ABI). Subjects who scored at least two standard deviations above the group mean on any of the four symptom subscales were placed in this "initially symptomatic" group (for possible exclusion from the prospective risk analyses).

Recontacting Subjects

Follow-up contact with the individuals who participated in the initial assessment began approximately 4 years after the initial data were collected. The vast majority of subjects had either changed their phone number or place of residence, left or graduated

from the university, and/or changed their surname. Due to the inherent difficulty in tracking such individuals over an interval of this length, the following strategy was used to maximize the number of subjects who could be successfully recontacted: (a) gathering previous phone numbers listed on the original consent form, (b) accessing public university school directory records for subjects who were current students at the time of follow-up assessment, (c) examining university alumni records for directory information of graduates, and (d) searching local phone directories for remaining subjects who may have continued living in the area. This approach resulted in successful contact with 253 (55%) of the original 457 subjects.

The initial phone contacts with subjects included a brief reminder of the purpose of the study (to gain information about how college women change over time in terms of health, nutrition, and emotional issues) and an overview of the Time 2 assessment procedures (self-report questionnaires and a phone interview). They were told that all women who participated in this follow-up assessment phase would be eligible to win a drawing for a $50.00 savings bond.

Follow-up Assessment

Questionnaires. Subjects who agreed to participate provided their current addresses and received a research packet (consisting of a new informed consent form, the EDI, and the ABI) in the mail. Each subjects' response forms were labeled with the same unique ID number used in the initial assessment. A postage-paid return envelope was provided to increase the return rate. Researchers recontacted (up to three times) subjects

who initially failed to return their packets. Replacement research packets were mailed out as necessary. Fifteen subjects returned incomplete or unusable questionnaires; however, all of these subjects volunteered to fill out the inventories a second time.

Interview. Resource constraints made conducting and coding diagnostic phone interviews with all 191 subjects who returned questionnaires unfeasible (interviews lasted up to 45 minutes, and each interview was listened to and independently coded twice). Rather, the investigator selected subjects for interviews based on risk status. All subjects who were initially classified as either high-risk ($n = 22$) or symptomatic ($n = 27$) based on their Time 1 responses were selected for the interview. Similarly, those individuals whose Time 2 responses met either the high-risk or symptomatic group criteria ($n = 12$) also received interviews. The remaining interviews were conducted with subjects who were randomly selected from the Time 1 control group ($n = 41$). Thus, a total of 102 individuals completed the SCID interviews.

The principal investigator conducted the majority of the diagnostic phone interviews, but was assisted by three trained, volunteer research assistants. To enhance reliability, interviewers completed a training procedure that involved listening to sample interviews and conducting practice interviews that were audiotaped and critiqued. All interviewers were blind to the risk status of the subjects they interviewed. To enhance confidentiality, only first names were used during the taped interview, and only the subjects' identification numbers were written on the cassettes.

Coding. The audiotaped interviews were coded by the principal investigator and two trained, volunteer research assistants who were also blind to the subjects' group

membership. Each recording of an interview was reviewed and coded by two different raters to check for accuracy. No interview was coded by the same research assistant conducting the interview. Coding an interview typically took each rater between 30 and 60 minutes, depending on the length of the interview. Initial interrater reliability rates (calculated by dividing the number of identical, or "correctly" scored items by the total number of items) reached 92%. Interrater discrepancies that could be easily resolved through recoding the interview a third time (e.g., those due to minor clerical mistakes) were resolved by the principal investigator. Incongruity due to differences in clinical judgment (e.g., what constitutes a binge) were resolved by David Stein, Ph.D. These procedures resulted in a final interrater reliability rate of 100%.

Data Analysis

Group Comparisons

Given the substantial attrition rate between the initial and follow-up assessments, it was necessary to evaluate whether any notable differences existed between subjects who completed the study versus those who did not. Also of interest was whether any differences existed between subjects who were able to be recontacted but declined to participate in the second assessment, and those who completed the study. To accomplish these objectives, mean scores on all subscales were compared by using one-way analysis of variance procedures and calculation of mean standardized difference effect sizes.

Means and standard deviations of the four risk subscales, the four symptom subscales, and the three severity indices were computed for each group (high-risk,

symptomatic, control) using both Time 1 and Time 2 data. Mean standardized difference effect sizes were also calculated to determine the magnitude and relevance of any differences between group means at the initial and follow-up assessments. These comparisons allowed a number of questions to be answered. For example, examination of the Time 1 risk factor effect sizes showed whether or not the group assignment procedure separated subjects into groups with meaningful differences in risk factor scores in the expected manner. Time 2 effect sizes were useful in assessing such issues as whether individuals meeting the high-risk criteria at the initial assessment showed elevated symptom scores at the follow-up assessment compared to the control group (as would be expected). Additionally, zero-order correlation coefficients were calculated between each of the Time 1 risk factors and the Time 2 measures of eating disorder symptom severity. Also of interest was how the scores of each group changed over time. To clarify whether a group's scores increased, decreased, or remained the same over the 4-year interval, effect sizes for differences across time on the four risk scores, four symptom scores, and three severity indices were calculated for all three groups.

Prevalence and Incidence Rates

Prevalence rates for anorexia, bulimia, and eating disorder NOS were calculated for the entire sample at both the initial and follow-up assessments. Specifically, the total number of individuals in the follow-up sample ($n = 191$) was divided by the number of individuals diagnosed with each disorder. Four-year incidence rates were calculated for the total sample, high-risk group, and control group. The incidence rate for the total

sample was based on the 169 subjects who did not have an eating disorder at the initial assessment (the 22 subjects in the symptomatic group, who all had a diagnosable eating disorder at Time 1, were excluded from this analysis). The incidence rates of the two subgroups were computed by dividing the number of new cases in each subgroup by the total number of subjects in that group.

Comparisons of Recovered and Nonrecovered Subjects

Once likely diagnoses at both assessment periods were determined, it was possible to identify subjects who recovered (or did not recover) from an eating disorder over the period covered by the present study. Of interest was whether scores on each of the four risk variables changed over time in the recovered and nonrecovered groups. To examine the direction and magnitude of any changes in risk factor scores over time, mean standardized difference effect sizes were calculated for each group. Finally, effect sizes measuring the differences between recovered and nonrecovered subjects' risk factor scores at the final assessment were computed to assess the extent to which the two groups' final risk factor scores differed.

Prediction of Eating Disorder Severity

A number of methods were employed to assess the extent to which initial scores on the four risk factors related to subsequent eating disorder severity. Zero-order correlations between Time 1 risk factor scores and Time 2 severity index scores were examined to measure the relationship between early risk scores and later eating disorder

severity. Regression analyses were used to assess the percentage of variance in Time 2 eating disorder severity that was explained by initial scores on the four risk factors. Finally, regression analyses were used to determine the most parsimonious, linear combination of variables explaining symptom outcomes.

The extent that changes over time in the four risk factor scores related to severity of later eating disorder symptoms was also examined. Hierarchical regression analysis was used to assess how changes over time in the four risk factors as a group, as well as changes over time in each individual risk factor, related to subsequent severity of eating disorder symptoms. These analyses provided information about whether the scores on the risk variables covaried with eating disorder symptom changes.

CHAPTER IV

RESULTS

Likely Impact of Subject Attrition

As discussed in Chapter III, there was a loss of subjects from the initial to the follow-up assessment. This occurred primarily as a result of difficulty encountered in tracing the whereabouts of subjects and recontacting them, rather than from subjects' refusal to participate in the follow-up assessment. Nevertheless, if the subjects who were not included in the follow-up differed significantly from those that were assessed, the external validity of the current study could be questioned on the grounds that the final sample was not representative of the population under investigation. To address this issue, the mean EDI and ABI Time 1 scores of subjects who did not participate in the follow-up (n = 266) were compared with the pretest scores of subjects who did complete both assessment periods (n = 191) with a one-way analysis of variance and calculation of mean standardized difference effect sizes. Results indicated that the two groups did not differ significantly on any of the ABI or EDI initial subscale mean scores (alpha = .05). Effect sizes for the differences, presented in Table 5, range between .00 and .14. These results suggest that it is unlikely that meaningful differences existed between the subjects who did and did not participate in the Time 2 assessment.

Comparisons were also made between the initial ABI and EDI scores of subjects who completed the study (n = 191) and subjects who were able to be recontacted but declined to participate or failed to complete the follow-up assessment materials (n = 62).

Table 5

Drop-outs Versus Retained Subjects: Time 1 Means, Standard Deviations, and Effect Sizes

Subscale	Drop-out subjects (N = 266) Mean (SD)	Retained subjects (N = 191) Mean (SD)	Effect size
EDI			
Drive for thinness	6.05 (5.72)	5.79 (5.49)	.05
Bulimia	1.65 (2.92)	1.64 (2.88)	.00
Body dissatisfaction	12.70 (8.75)	13.32 (8.80)	-.07
Ineffectiveness	3.18 (4.58)	3.14 (4.67)	.01
Perfectionism	6.67 (4.21)	7.28 (4.47)	-.14
Interpersonal distrust	3.14 (3.61)	2.93 (3.59)	.06
Interoceptive awareness	4.71 (3.60)	4.63 (3.73)	.02
Maturity fears	2.77 (3.72)	2.31 (2.93)	.14
ABI			
Parent conflict	2.04 (.47)	2.02 (.50)	.04
Depressed mood	2.06 (.52)	2.03 (.57)	.06
Anxiety	2.24 (.52)	2.19 (.58)	.09
Maladaptive cognitions	2.24 (.63)	2.23 (.62)	.02
Anorexia	1.73 (.47)	1.68 (.45)	.11
Anergia	2.42 (.57)	2.41 (.59)	.02
Purge	1.29 (.39)	1.28 (.41)	.03
Binge	1.70 (.66)	1.69 (.62)	.02
Exercise	2.43 (.57)	2.35 (.56)	.14

Note. "Drop-out subjects" include those who were only assessed at Time 1. "Retained subjects" include those who were assessed at Time 1 and Time 2.

Standardized mean difference effect sizes for these comparisons are presented in Table 6. The effect size for perfectionism was -.30, indicating that subjects who refused or failed to complete the Time 2 assessment materials had lower scores on perfectionism at Time 1 compared to subjects who completed all assessments. Effect sizes for the remaining variables were quite small, ranging from .00 to .15.

For reference, descriptive statistics for the ABI, EDI, and symptom severity indices of the retained subjects are presented in Table 34 in Appendix E. Intercorrelations among the severity scores are listed in Table 35 in Appendix E.

Initial Group Assignments: Accuracy and Modifications

Using the criteria described in Chapter III, subjects were temporarily assigned to membership in either the high-risk (HR), symptomatic (S), or control (C) group based on their Time 1 scores on the four risk factor subscales (drive for thinness, ineffectiveness, depressed mood, maladaptive cognitions) and the four symptom subscales (bulimia, anorexia, purge, binge). Since prior research has shown that diagnosing eating disorders solely from paper-and-pencil questionnaires often results in elevated rates of diagnosis (usually in the direction of false-positives), this initial group assignment was considered temporary and was only used to select subjects for the clinical diagnostic interviews. The results of these interviews were then used to reassign subjects to permanent groups that more accurately reflected their initial diagnostic and risk status.

Results of these interviews revealed that 3 of the 22 individuals (14%) who had been placed in the high-risk group based on their risk factor scores actually had an eating

Table 6

Refusal Subjects Versus Retained Subjects: Time 1 Means, Standard Deviations, and Effect Sizes

Subscale	Refusal subjects (N = 62) Mean (SD)	Retained subjects (N = 191) Mean (SD)	Effect size
EDI			
Drive for thinness	6.35 (5.87)	5.79 (5.49)	.10
Bulimia	2.08 (3.01)	1.64 (2.88)	.15
Body dissatisfaction	13.51 (8.94)	13.32 (8.80)	.02
Ineffectiveness	3.18 (4.80)	3.14 (4.67)	.01
Perfectionism	5.98 (4.11)	7.28 (4.47)	-.30
Interpersonal distrust	2.96 (3.42)	2.93 (3.59)	.01
Interoceptive awareness	4.98 (3.88)	4.63 (3.73)	.09
Maturity fears	2.28 (3.73)	2.31 (2.93)	-.01
ABI			
Parent conflict	2.00 (.45)	2.02 (.50)	-.04
Depressed mood	2.03 (.57)	2.03 (.57)	.00
Anxiety	2.24 (.53)	2.19 (.58)	.09
Maladaptive cognitions	2.26 (.70)	2.23 (.62)	.05
Anorexia	1.65 (.50)	1.68 (.45)	-.06
Anergia	2.37 (.60)	2.41 (.59)	-.07
Purge	1.33 (.47)	1.28 (.41)	.11
Binge	1.74 (.72)	1.69 (.62)	.07
Exercise	2.36 (.57)	2.35 (.56)	.02

Note. "Refusal subjects" include those who were assessed at Time 1 and were successfully recontacted at Time 2, but either refused or failed to complete the Time 2 assessment materials. "Retained subjects" include those who were assessed at Time 1 and Time 2.

disorder at Time 1. These subjects were moved from the high-risk group to the symptomatic group for all remaining analyses. Further, 15 of 27 individuals in the initial symptomatic group (56%) endorsed behavioral eating disorder symptoms at an elevated degree but did not show sufficient number or severity of symptoms (during interviews) for a diagnosis of anorexia nervosa, bulimia nervosa, or eating disorder NOS. Therefore, they were shifted from the symptomatic group to either the high-risk group (if their scores on the four high-risk criteria met or exceeded the cutoff criteria) or the control group.

The revised group assignments thus reflect subjects' diagnostic categories and their risk status based on clinical interview, rather than questionnaire data alone. In summary, the final symptomatic group consisted of the 22 subjects who had a likely eating disorder diagnosis at Time 1. The final high-risk group was composed of 24 individuals who did not have an eating disorder, but met criterion cut-off scores on the risk variables. The remaining 144 subjects comprised the control group. These final group assignments were used for all of the remaining statistical analyses.

Risk Scores, Symptom Scores, and Severity Scores

Time 1

Means and standard deviations of the four risk subscales, the four symptom subscales, and the three severity indices were computed for each group (HR, S, C) using Time 1 data. Effect sizes were also calculated to determine the magnitude and relevance of any differences between group means. Examination of Time 1 effects sizes also

allowed evaluation of whether the criteria used to assign subjects to groups did differentiate the groups in the expected manner. Results of these analyses, as well as those for the Time 2 data are presented in Table 7.

Comparisons of Time 1 risk factor scores for the three groups indicated that subjects in both the high-risk group and the symptomatic group had substantially higher risk factor scores than subjects in the control group. High-risk subjects scored between 1.14 and 1.38 standard deviations above the control group mean on the four risk factor subscales, and the symptomatic subjects scored between .95 and 1.46 standard deviations higher than the control group. Thus, these effect sizes revealed that the group assignment procedures did separate subjects into groups with meaningful differences on risk factor scores, in the expected manner.

Scores on the four symptom scales also indicated that the group assignment procedures resulted in a separation of subjects in the anticipated manner. Subjects in the symptomatic group (i.e., those with eating disorder diagnoses) scored between .93 and 1.29 standard deviations above the control group on each of the four symptom subscales. Their scores were also elevated compared to the high-risk group (effect sizes ranged from .18 to .60), as would be expected. Although the high-risk subjects evidenced fewer eating disorder symptoms than the subjects in the symptomatic group, they did have elevated levels of symptoms compared to the control group (effect sizes from .56 to 1.11). The interview data, however, confirmed that any symptoms these individuals were experiencing and reporting on the paper-and-pencil questionnaires were either not severe or frequent enough to warrant a formal eating disorder diagnosis.

Table 7

Time 1 and Time 2 Risk Factor, Symptom, and Severity Scores: Means, Standard Deviations, and Effect Sizes for the High-Risk, Symptomatic, and Control Groups

Subscale	High-risk group (N = 24) Mean (SD)	Symptomatic group (N = 22) Mean (SD)	Control group (N = 144) Mean (SD)	Effect sizes HR/S	HR/C	S/C
Time 1 risk factor						
Drive for thinness	10.88 (3.52)	11.77 (5.38)	3.99 (4.51)	-.20	1.38	1.46
Ineffectiveness	7.76 (5.30)	6.32 (6.23)	1.85 (3.41)	-.25	1.38	1.07
Depressed mood	2.51 (.51)	2.41 (.63)	1.89 (.50)	-.18	1.14	.95
Mal. cognitions	2.78 (.34)	2.79 (.57)	2.04 (.56)	-.02	1.24	1.22
Time 1 symptom						
Bulimia	2.80 (3.00)	4.64 (5.65)	.98 (1.65)	-.41	.91	1.29
Anorexia	1.81 (.32)	2.12 (.64)	1.59 (.39)	-.60	.56	1.13
Purge	1.43 (.37)	1.58 (.64)	1.20 (.34)	-.29	.66	.93
Binge	2.14 (.50)	2.26 (.82)	1.53 (.51)	-.18	1.11	1.20
Time 1 severity index						
GSI	2.83 (1.50)	8.11 (2.52)	2.27 (1.75)	-1.58	.33	1.76
BSI	1.76 (1.99)	7.50 (3.89)	1.32 (2.01)	-1.37	.22	1.61
ASI	3.98 (2.05)	8.49 (2.74)	3.13 (2.14)	-1.37	.40	1.60

(table continues)

Subscale	High-risk group (N = 24) Mean (SD)	Symptomatic group (N = 22) Mean (SD)	Control group (N = 144) Mean (SD)	Effect sizes HR/S	HR/C	S/C
Time 2 risk factor						
Drive for thinness	8.64 (5.15)	9.36 (6.51)	2.67 (3.47)	-.12	1.39	1.46
Ineffectiveness	4.56 (5.10)	5.55 (6.54)	1.14 (2.35)	-.17	1.09	1.25
Depressed mood	2.19 (.61)	2.36 (.74)	1.75 (.47)	-.25	.86	1.11
Mal. cognitions	2.54 (.62)	2.60 (.69)	1.90 (.51)	-.09	1.13	1.20
Time 2 symptom						
Bulimia	1.52 (2.16)	4.14 (4.51)	.47 (1.38)	-.71	.67	1.52
Anorexia	1.56 (.33)	1.91 (.64)	1.42 (.33)	-.67	.42	1.17
Purge	1.24 (.30)	1.52 (.45)	1.13 (.22)	-.70	.46	1.32
Binge	1.66 (.50)	2.12 (.85)	1.30 (.42)	-.64	.79	1.44
Time 2 severity index						
GSI	2.12 (.77)	3.74 (2.50)	1.60 (1.12)	-.82	.50	1.14
BSI	.61 (1.03)	2.54 (3.36)	.56 (1.09)	-.74	.05	.91
ASI	3.28 (1.06)	4.25 (1.88)	2.37 (1.43)	-.62	.65	1.06

Note. Mal. cognitions = maladaptive cognitions; GSI = Global Severity Index; BSI = Bulimia Severity Index; ASI = Anorexia Severity Index. Effect sizes represent standardized mean difference effect sizes.

Comparisons of scores on the three symptom severity indices revealed that subjects in the symptomatic group received considerably higher scores on all three severity indices (GSI, BSI, and ASI) than subjects in either the high-risk or control groups. Effect sizes ranged from 1.37 to 1.76. The high-risk group showed slightly higher eating disorder severity scores than the control group; however, the relatively small effect sizes (GSI = .33, BSI = .22, ASI = .40) suggest that these differences may not be meaningful.

Time 2

To identify whether any differences existed between the three groups at the follow-up assessment, the aforementioned analyses were performed using the Time 2 data. The means, standard deviations, and effect sizes for the risk, symptom, and severity scores are also presented in Table 7. It was expected that individuals meeting the high-risk criteria at the initial assessment would show elevated symptom scores at the follow-up assessment compared to the control group. The Time 2 data suggest, however, that on average, subjects in the high-risk group did not have substantially higher levels of eating disorder symptoms (based on the four symptom subscales) than the control group. Effect sizes, based on comparisons with the control group, were not notably greater than those at the initial assessment. Specifically, high-risk subjects' scores on the anorexia and purge subscales were .42 and .46 standard deviations, respectively, above the control group. However, greater differences were seen in the bulimia (ES = .67) and binge (ES = .79) subscales. Similar patterns of results were found with the three (interview) symptom

severity scores. High-risk subjects evidenced moderately higher scores than control subjects on the GSI (ES = .50) and the ASI (ES = .65). There were virtually no differences, however, in BSI scores between the two groups (ES = .05). Hence, the group presumed to be at highest risk for developing an eating disorder showed, on average, only mild elevations in eating disorder symptoms and severity at the follow-up assessment, compared to the means of the control group.

On the other hand, while few mean group differences were found on formal eating disorder symptoms, the risk factor scores of high-risk subjects continued to remain quite elevated, compared to control subjects, at the follow-up assessment. Specifically, differences between the high-risk and control groups on drive for thinness were manifested in an effect size of 1.39. Effect sizes for ineffectiveness (ES = 1.09) and maladaptive cognitions (ES = 1.13) were also quite large. The effect size for depressed mood, .86, was lower than that of the other three variables, but was still substantially higher than the control subjects' scores on that subscale.

Comparisons between subjects in the symptomatic group and those in the other two groups revealed that subjects in the symptomatic group continued to show elevated scores on all four symptom subscales relative to both the control and high-risk groups. Subjects in the symptomatic group had scores that were between .64 and .71 standard deviations higher than the high-risk group, and 1.17 to 1.52 standard deviations above the control group.

Comparisons of the Time 2 eating disorder severity scores (see Table 7) indicated that subjects in the symptomatic group had substantially higher scores than both the high-

risk and control groups on all three of the Time 2 (interview) severity scores (GSI, BSI, and ASI). Effect sizes ranged from .62 to 1.14. This suggests that these individuals, all of whom had eating disorder diagnoses at the initial assessment, continued to evidence eating disorder symptoms at higher levels of severity than the control or high-risk subjects four years later.

Changes Over Time in Risk, Symptom, and Severity Index Scores

The preceding analysis investigated how the mean scores of the three different groups compared to each other at two separate points in time: the initial and follow-up assessments. Another important issue involves how the scores of each group changed over time. For example, although the results described thus far have shown that subjects in the symptomatic group continued to have higher scores at the follow-up assessment than those in the control group, these comparisons cannot clarify whether the symptomatic group's scores increased, decreased, or remained the same over the 4-year interval. Thus, effect sizes for differences across time on the questionnaire and interview data were calculated for all three groups. Results are presented in Tables 8 (HR group), 9 (S group), and 10 (C group).

A consistent theme is that irrespective of group membership, women tended to show decreases in risk, symptom, and severity scores between college and their post-college years. This theme is detailed below.

Table 8

Changes in Risk Scores, Symptom Scores and Severity Index Scores Over Time: High Risk Group (N = 24)

Subscale	Time 1 Mean (SD)	Time 2 Mean (SD)	Effect size (T2/T1)
Risk factor			
Drive for thinness	10.88 (3.52)	8.64 (5.15)	-.52
Ineffectiveness	7.76 (5.30)	4.56 (5.10)	-.62
Depressed mood	2.51 (.51)	2.19 (.61)	-.57
Maladaptive cognitions	2.78 (.34)	2.54 (.62)	-.50
Symptom			
Bulimia	2.80 (3.00)	1.52 (2.16)	-.50
Anorexia	1.81 (.32)	1.56 (.33)	-.76
Purge	1.43 (.37)	1.24 (.30)	-.56
Binge	2.14 (.50)	1.66 (.50)	-.96
Severity index			
GSI	2.83 (1.50)	2.12 (.77)	-.62
BSI	1.76 (1.99)	.61 (1.03)	-.76
ASI	3.98 (2.05)	3.28 (1.06)	-.45

Note. GSI = Global Severity Index; BSI = Bulimia Severity Index; ASI = Anorexia Severity Index.

Table 9

Changes in Risk Scores, Symptom Scores and Severity Index Scores Over Time: Symptomatic Group (N = 22)

Subscale	Time 1 Mean (SD)	Time 2 Mean (SD)	Effect size (T2/T1)
Risk factor			
Drive for thinness	11.77 (5.38)	9.36 (6.51)	-.41
Ineffectiveness	6.32 (6.23)	5.55 (6.54)	-.12
Depressed mood	2.41 (.63)	2.36 (.74)	-.07
Maladaptive cognitions	2.79 (.57)	2.60 (.69)	-.30
Symptom			
Bulimia	4.64 (5.65)	4.14 (4.51)	-.10
Anorexia	2.12 (.64)	1.91 (.64)	-.33
Purge	1.58 (.64)	1.52 (.45)	-.11
Binge	2.26 (.82)	2.12 (.85)	-.17
Severity index			
GSI	8.11 (2.52)	3.74 (2.50)	-1.74
BSI	7.50 (3.89)	2.54 (3.36)	-1.37
ASI	8.49 (2.74)	4.25 (1.88)	-1.84

Note. GSI = Global Severity Index; BSI = Bulimia Severity Index; ASI = Anorexia Severity Index.

Table 10

Changes in Risk Scores, Symptom Scores and Severity Index Scores Over Time: Control Group (N = 144)

Subscale	Time 1 Mean (SD)	Time 2 Mean (SD)	Effect size (T2/T1)
Risk factor			
Drive for thinness	3.99 (4.51)	2.67 (3.47)	-.33
Ineffectiveness	1.85 (3.41)	1.14 (2.35)	-.25
Depressed mood	1.89 (.50)	1.75 (.47)	-.29
Maladaptive cognitions	2.04 (.56)	1.90 (.51)	-.26
Symptom			
Bulimia	.98 (1.65)	.47 (1.38)	-.34
Anorexia	1.59 (.39)	1.42 (.33)	-.47
Purge	1.20 (.34)	1.13 (.22)	-.25
Binge	1.53 (.51)	1.30 (.42)	-.49
Severity index			
GSI	2.27 (1.75)	1.60 (1.12)	-.47
BSI	1.32 (2.01)	.56 (1.09)	-.49
ASI	3.13 (2.14)	2.37 (1.43)	-.42

Note. GSI = Global Severity Index; BSI = Bulimia Severity Index; ASI = Anorexia Severity Index.

High-Risk Group

The high-risk group showed moderate to substantial decreases over time in all risk factor scores, symptom scores, and severity index scores. Scores on the four risk factors fell approximately 1/2 to 1/3 of a standard deviation over the 4-year interval (effect sizes ranged from -.50 to -.62). Scores on the four (questionnaire) symptom subscales also decreased substantially over time for the high-risk group. Specifically, purge and bulimia scores dropped roughly 1/2 SD, anorexia scores decreased 3/4 SD, and binge scores declined approximately 1 SD. The change in severity index (interview) scores were -.45 SD (ASI), -.62 SD (GSI), to -.76 SD (BSI).

Symptomatic Group

For subjects in the symptomatic group, scores on the severity indices decreased quite dramatically over the 4-year interval. The effect size involving GSI scores was -1.74, BSI scores showed an effect size of -1.37, and ASI scores evidenced a -1.84 effect size. Thus, the severity of eating disorder symptoms, as reported in a diagnostic interview, showed a marked decrease over time.

Interestingly, symptomatic subjects' self-report of most symptoms on the paper-and-pencil questionnaires, however, showed little change over time. Scores in changes on the bulimia, purge, and binge subscales had effect sizes of -.10, -.11, and -.17, respectively. Only the anorexia subscale showed a larger decrease, with an effect size of -.33. These differences between the questionnaire and interview data again suggest the

limitations of relying solely on questionnaire data for accurate assessment of eating disorder symptoms.

The various risk factor scores showed differential results across time for the symptomatic group. Two of the risk factor scores showed little change over the 4-year interval (ineffectiveness, ES = -.12; depressed mood, ES = .07). The remaining two risk factors, however, showed modest decreases (drive for thinness, ES = -.41; maladaptive cognitions, ES = .30). Thus, concomitant with the decreases in symptom severity was a decrease in some, but not all, risk factor scores. Scores on the ineffectiveness and depressed mood subscales appeared to be quite resistant to change in subjects whose eating disorder symptoms improved, while scores on the drive for thinness and maladaptive cognitions subscales did show a notable, but modest, decline.

Control Group

By definition, the control group had lower scores on risk factor, symptom, and severity index scores at the initial assessment than either of the other groups. Thus, it was expected that their scores on any of the risk, symptom, or severity measures would not increase much over time. Examination of the effect sizes for this group revealed that not only did their scores not increase, they actually showed a decline over the 4-year interval. These decreases were modest in size, with effect sizes ranging from -.25 to -.49. Thus, although the control group's risk factor and symptom scores were not high to begin with, they nevertheless still decreased over the course of the study.

Prevalence and Incidence Rates

Prevalence rates were calculated for the entire sample at both the initial and follow-up assessments. The SCID and the accompanying Clinician's DSM-IV Checklist provided the basis for the diagnoses, using the procedures and cut-off criteria described previously. Prevalence rates for Time 1 and Time 2 are presented in Table 11. The results revealed that 11.5% of subjects had an eating disorder of some type at the time of the initial assessment. This percentage fell to 4.2 by the follow-up assessment 4 years later.

Table 11

Prevalence Rates for Total Sample: Time 1 and Time 2

	Time 1	Time 2
Disorder	Percent (N)	Percent (N)
Anorexia nervosa	2.6 (5/191)	0.0 (0/191)
Bulimia nervosa	3.7 (7/191)	0.5 (1/191)
Eating disorder NOS	5.2 (10/191)	3.7 (7/191)
Total	11.5 (22/191)	4.2 (8/191)

Four-year incidence (new case) rates were calculated for the high-risk and control groups. The incidence rates for the total sample were based on the 169 subjects who did not have an eating disorder at the initial assessment (the 22 subjects in the symptomatic group, who all had diagnosed eating disorders at Time 1, were excluded from this analysis). The results, presented in Table 12, reveal an overall incidence rate of .6%. Thus, during the 4-year interval, only one individual developed an eating disorder. This subject, who was diagnosed with bulimia nervosa at the second assessment, was initially a member of the control group.

Risk Factor Scores of Recovered and Nonrecovered Subjects

Evaluation of changes in diagnoses over time revealed that in this sample, development of a new eating disorder over the 4-year interval was a rare occurrence. On

Table 12

Four-Year Incidence Rates for High-Risk and Control Group

Group	Group rate	Incidence rate
High-risk	0/25	0.0%
Control	1/144	0.7%
Total	1/169	0.6%

the other hand, examination of diagnoses at the initial and follow-up assessments revealed that recovery from an existing eating disorder over the 4-year interval was quite common. Of the 22 subjects initially diagnosed with anorexia, bulimia, or eating disorder NOS, 14 participants (64%) had recovered at least somewhat from their eating disorder by the second assessment (i.e., they did not receive an eating disorder diagnosis at Time 2). Eight subjects (36%) remained symptomatic and were diagnosed with an eating disorder at both the initial and follow-up assessments.

Also of interest in the current study was which, if any, of the risk factor scores would change over time in the recovered and nonrecovered groups. Comparison of Time 1 and Time 2 means on the four risk factor scores indicated that the maladaptive cognitions and drive for thinness scores decreased substantially over time in the recovered subjects (ES = -.72 and -.77, respectively). Scores on the other two risk factors also decreased, although to a lesser degree. Recovered subjects' ineffectiveness scores declined by .49 SD over the 4-year interval, and depression scores decreased by .28 SD over time. These results are summarized in Table 13.

Further analyses revealed, however, that although all risk factor scores declined in the recovered subjects, their risk factor scores continued to remain at significantly elevated levels compared to control subjects. The effect sizes for differences in Time 2 risk factor scores between the recovered subjects and control subjects were as follows: maladaptive cognitions (ES = .94), depressed mood (ES = 1.17), ineffectiveness (ES = 1.18), and drive for thinness (ES = 1.26). These results indicated that although the scores on risk factors did not decline to the level found among subjects who had

Table 13

Recovered and Nonrecovered Subjects: Effect Sizes for Changes in Risk Factor Scores

Risk factor	Time 1 Mean (SD)	Time 2 Mean (SD)	Effect size (T2/T1)
Recovered subjects (n = 14)			
Drive for thinness	12.36 (5.42)	7.79 (6.44)	-.77
Ineffectiveness	7.29 (6.23)	4.50 (5.05)	-.49
Depressed mood	2.53 (.64)	2.35 (.67)	-.28
Maladaptive cognitions	2.83 (.60)	2.40 (.60)	-.72
Nonrecovered subjects (n = 8)			
Drive for thinness	10.75 (5.52)	12.13 (6.03)	.24
Ineffectiveness	4.63 (6.26)	7.38 (8.65)	.37
Depressed mood	2.21 (.61)	2.40 (.89)	.25
Maladaptive cognitions	2.73 (.54)	2.94 (.73)	.33

never had an eating disorder diagnosis.

Changes in the Time 1 and Time 2 risk factor scores of the eight nonrecovered subjects were also analyzed, and are presented in Table 13. Examination of changes in the mean scores of the nonrecovered subjects revealed a modest increase over time in risk factor scores (1/4 to 1/3 SD higher at Time 2). This concurred with expectations

suggesting that risk factor scores would remain elevated in individuals who continued to experience significant problems with eating disorder symptoms.

Prediction of Eating Disorder Severity Based on Early (Time 1) Risk Factor Scores

To what extent were initial scores on the four risk factor subscales related to eating disorder severity after an interval of 4 years? This question was addressed by examining the results of several statistical analyses: zero-order correlations to assess the relationship between the Time 1 risk factor scores and the Time 2 severity index scores; regression analyses assessing the percentage of variance in eating disorder severity that was explained by the four risk factors; and regression analyses to determine the most parsimonious, linear combination of variables explaining symptom outcomes. This section provides an overview of the results of each of these procedures.

Pearson product-moment correlation coefficients were computed to assess the relationship between the Time 1 risk factor scores and the Time 2 eating disorder severity scores. Only subjects who were in either the high-risk or control group were included in this analysis; subjects who were diagnosed with eating disorders at the initial assessment (members of the symptomatic group) were excluded. These correlations are presented in Table 14.

The results revealed that initial scores on depressed mood and ineffectiveness were not significantly related to later scores on any of the three eating disorder severity indices. Time 1 scores on the maladaptive cognitions subscale correlated significantly

Table 14

Zero-Order Correlations Between Time 1 Risk Factors and Time 2 Eating Disorder Severity Scores: Control and High-Risk Subjects (N = 80)

Risk factor	ASI	BSI	GSI
Drive for thinness	.27 **	.08	.19
Ineffectiveness	.14	-.10	.11
Depressed mood	.06	-.21	.00
Maladaptive cognitions	.30 **	.14	.28 **

Note. ASI = Anorexia Severity Index; BSI = Bulimia Severity Index; GSI = Global Severity Index. ** $p < .01$.

with the ASI ($r = .30$, $p < .01$) and the GSI ($r = .28$, $p < .01$). A significant correlation also was found between drive for thinness scores and the ASI ($r = .27$, $p < .01$). Thus, it appears that subjects' initial maladaptive attitudes and beliefs about achieving thinness, and irrational beliefs about perceived effects of dieting most strongly related to eating disorder (particularly anorexia) severity 4 years later.

Simultaneous regression analysis was conducted to assess the extent to which Time 1 scores on the four risk factors (entered as a single block) predicted Time 2 scores on each of the three severity indices. Subjects who had an eating disorder diagnosis at the initial assessment were again excluded from these analyses. Table 15 contains a summary of the results of the regression analyses using the Time 2 ASI as the dependent

Table 15

Summary of Simultaneous Regression Analysis for Time 1 Risk Factors Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor	B	SE B	β
Drive for thinness	.03	.04	.10
Ineffectiveness	.06	.05	.23
Depressed mood	-.70	.44	-.28
Maladaptive cognitions	.64	.38	.28

Note. Subjects include high-risk and control groups. Total $R^2 = .13$ ($p < .05$).

variable. The results indicated that scores on the four risk factors at the initial assessment explained 13% of the variance in severity of anorexia symptoms at the follow-up assessment. Similar analyses were conducted using the BSI (Table 16) and the GSI (Table 17) as the dependent variables. These analyses revealed that together, the four risk factors also accounted for approximately 13% of the variance in both the BSI and the GSI. Although all three of these results reached levels of statistical significance ($ps < .05$), a large percentage of the variance in the ASI, BSI, and GSI scores remained unexplained.

One method of assessing the contribution of each individual risk factor to explaining the variance in later eating disorder severity is to examine the relative values and significance levels of the standardized beta coefficients (β) obtained from the

Table 16

Summary of Simultaneous Regression Analysis for Time 1 Risk Factors Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor	B	SE B	β
Drive for thinness	-.02	.03	-.07
Ineffectiveness	.03	.04	.15
Depressed mood	-.91	.34	-.48 *
Maladaptive cognitions	.64	.29	.36 *

Note. Subjects include high-risk and control groups. Total R^2 = .13 (p <.05). * p < .05.

Table 17

Summary of Simultaneous Regression Analysis for Time 1 Risk Factors Predicting Time 2 Global Severity Index (N = 80)

Risk factor	B	SE B	β
Drive for thinness	-.01	.03	-.03
Ineffectiveness	.06	.03	.27
Depressed mood	-.70	.33	-.37 *
Maladaptive cognitions	.66	.29	.38 *

Note. Subjects include high-risk and control groups. Total R^2 = .13 (p < .05). * p < .05.

previous analyses (the results when all four risk variables were entered simultaneously into the regression equations). These standardized beta coefficients, listed in Tables 15 through 17, indicated that when the four Time 1 variables were included in the equation together, depressed mood and maladaptive cognitions explained statistically significant proportions of the variance in the BSI and the GSI. The relative sizes of the standardized beta weights suggest that these variables are of equal importance in accounting for variance in the dependent variables. On the other hand, no one variable explained a significant percentage of the variance in the ASI. Of note was the finding of an inverse relationship between initial depressed mood scores and later BSI and GSI scores; that is, higher initial levels of depression were related to lower bulimic and global eating disorder symptoms at the final assessment. Finally, it should be noted that one problem with relying on this method to determine the relative contribution of individual risk factors is that the magnitude and direction of any given risk factor's relationship with the dependent variable (severity index) is moderated by the other independent variables also included in the equation.

A final method of exploring the relative contributions of each risk factor is to identify the linear combination of variables that explains the largest percentage of variance in the severity indices in the most parsimonious manner. This was accomplished through a series of stepwise regression equations. Independent variables (risk factors) were entered into the equation if $p < .05$ and were removed from the equation if $p > .10$. The results indicated that, when using the ASI as the dependent variable, Time 1

maladaptive cognitions scores alone most efficiently explained variance in subsequent severity of anorexic symptoms, accounting for 8.7% of the variance in the ASI, F (1,78) = 7.46, $p < .01$ (Table 18). Similarly, inclusion of the Time 1 maladaptive cognitions variable alone was the most parsimonious explanation of variance in later global eating disorder symptoms, F (1,78) = 6.38, $p < .01$ (Table 18). Finally, as would be expected, the results indicated that none of the four risk factors reached the inclusion value of $p < .05$; thus, no variables were entered into the equation when the dependent variable was the BSI. These results suggested that, overall, maladaptive cognitions scores alone provided the most efficient method of explaining variance in the ASI and GSI, and that none of the variables alone effectively accounted for a meaningful proportion of variance in the BSI. Although drive for thinness showed a statistically significant zero-order correlation with the ASI (and explained 7.3% of its variance), the results of this analysis indicated that drive for thinness did not contribute a meaningful proportion of variance above and beyond that obtained from the inclusion of maladaptive cognitions alone.

Relationship Between Changes in Risk Factors and Subsequent Eating Disorder Severity

Another method used to determine the extent to which the four variables functioned as risk factors for eating disorder symptoms was examination of how changes over time in the four risk factor scores related to severity of eating disorder symptoms at the follow-up assessment. To address this issue, regression analysis was used to examine

Table 18

Summary of Three Stepwise Regression Analyses for Most Efficient Linear Combination of Risk Factors Predicting Time 2 Severity Indices (N = 80)

Risk factors	B	SE B	β
Analysis 1: Dependent variable = ASI			
Time 1 maladaptive cognitions	.68	.25	.30 **
Analysis 2: Dependent variable = GSI			
Time 1 maladaptive cognitions	.48	.19	.27 **
Analysis 3: Dependent variable = BSI			
No variables were entered or removed for this analysis (no variable reached $p = .05$).			

Note. Subjects included individuals in high-risk and control groups. This table depicts the results of three separate regression analyses using three different dependent variables (ASI, GSI, and BSI). Results for ASI: $R^2 = .09$ ($p < .01$). Results for GSI: $R^2 = .08$ ($p < .01$). ** $p < .01$.

how changes in the four risk factors as a group, as well as changes in each individual risk factor, related to symptom severity. Subjects who had eating disorders at the initial assessment were excluded from these analyses. Results are presented in Tables 19 through 21.

Table 19

Summary of Hierarchical Regression Analysis for Changes in Risk Factors Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor blocks	B	SE B	β
Step 1			
Time 1 drive for thinness	.03	.04	.10
Time 1 ineffectiveness	.06	.05	.23
Time 1 depressed mood	-.70	.44	-.28
Time 1 maladaptive cognitions	.64	.38	.28
Step 2			
Time 2 drive for thinness	.15	.04	.52 **
Time 2 ineffectiveness	-.04	.05	-.11
Time 2 depressed mood	-.11	.41	-.04
Time 2 maladaptive cognitions	.47	.32	.21

Note. Subjects include individuals in high-risk and control groups. Total R^2 = .13 for Step 1 ($p < .05$) ; ΔR^2 = .34 for Step 2 ($p < .001$). ** $p < .001$.

With the ASI as the dependent variable, Time 1 scores on all four risk factors were entered in Block 1 of a hierarchical regression analysis, followed by the Time 2 scores on the risk factors in Block 2 (see Table 19). Results indicated that the change in R^2 value for Block 2 was 34%, ΔF (8,71) = 11.21, $p < .001$, overall F (8,71) = 7.68,

Table 20

Summary of Hierarchical Regression Analysis for Changes in Risk Factors Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor blocks	B	SE B	β
Step 1			
Time 1 drive for thinness	.02	.03	-.07
Time 1 ineffectiveness	.03	.04	.15
Time 1 depressed mood	-.91	.34	-.48 **
Time 1 maladaptive cognitions	.64	.29	.36 *
Step 2			
Time 2 drive for thinness	.04	.03	.17
Time 2 ineffectiveness	-.03	.05	-.09
Time 2 depressed mood	.40	.36	.20
Time 2 maladaptive cognitions	.38	.29	.22

Note. Subjects include individuals in high-risk and control groups. Total R^2 = .13 for Step 1 ($p < .05$) ; ΔR^2 = .16 for Step 2 ($p < .01$). * $p < .05$. ** $p < .01$.

overall $p < .001$. In other words, the changes in the risk factor scores explained an additional 34% of the variance in the ASI, above the Time 1 risk factor scores alone.

Hierarchical regression analysis using the BSI as the dependent variable indicated that changes in the four risk factor scores were significantly related to Time 2 severity of

Table 21

Summary of Hierarchical Regression Analysis for Changes in Risk Factors Predicting Time 2 Global Severity Index (N = 80)

Risk factor blocks	B	SE B	β
Step 1			
Time 1 drive for thinness	-.01	.03	-.03
Time 1 ineffectiveness	.06	.03	.27
Time 1 depressed mood	-.70	.33	-.37 *
Time 1 maladaptive cognitions	.66	.29	.38 *
Step 2			
Time 2 drive for thinness	.10	.03	.45 **
Time 2 ineffectiveness	-.05	.04	-.16
Time 2 depressed mood	.16	.30	.08
Time 2 maladaptive cognitions	.45	.24	.26

Note. Subjects include individuals in high-risk and control groups. Total R^2 = .13 for Step 1 ($p < .05$) ; ΔR^2 = .35 for Step 2 ($p < .001$). * $p < .05$. ** $p < .01$.

bulimic symptoms (see Table 20). The change in R^2 value for Block 2 was 16%, ΔF (8,71) = 3.99, $p < .01$, overall F (8,71) = 3.59, overall $p < .01$, indicating that changes in the four risk factors over time were significantly related to bulimic symptoms at the follow-up assessment. However, the lower R^2 change value compared to that obtained

for the ASI (16% vs. 34%) suggests that changes in the four risk factors may function as more effective predictors of anorexia severity than bulimia severity.

Similar analyses were performed to assess the relationship between changes in the risk factors and the GSI at the final assessment (Table 21). Hierarchical regression analysis using the GSI as the dependent variable yielded a change in $\underline{R}^2$ of 35% for the four risk factors change measures over time, $\Delta\underline{F}$ (8,71) = 11.63, $\underline{p} < .001$, overall $\underline{F}$ (8,71) = 7.96, overall $\underline{p} < .001$.

Thus far, these analyses have assessed the relationship between the severity indices and changes in risk factors as a group, rather than independently. Also of interest is the relationship between the severity index scores and changes in each risk factor independently (i.e., when no other risk factors have been included). Thus, hierarchical regression analyses were conducted using the severity indices as the dependent variables, entering the Time 1 scores for a given risk factor in Block 1 and the Time 2 scores for the same risk factor in Block 2. This procedure revealed the extent to which changes in an individual risk factor alone were related to eating disorder severity at the final assessment.

The relationships between the Time 2 ASI scores and changes in each risk factor over time were examined first and are presented in Tables 22 through 25. Changes in both the drive for thinness and the maladaptive cognitions scores were significantly related to ASI outcomes. The Step 2 change in $\underline{R}^2$ value for drive for thinness was 31%, $\Delta\underline{F}$ (2,77) = 38.60, $\underline{p} < .001$, overall $\underline{F}$ (2,77) = 23.75, overall $\underline{p} < .001$. Changes in maladaptive cognitions explained an additional 19% of the variance, above and beyond the Time 1 maladaptive cognitions scores alone, $\Delta\underline{F}$ (2,77) = 20.24, $\underline{p} < .001$,

Table 22

Summary of Hierarchical Regression Analysis for Changes in Drive for Thinness Scores Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 drive for thinness	.07	.03	.27 *
Step 2			
Time 2 drive for thinness	.18	.03	.60 **

Note. Subjects include individuals in high-risk and control groups. R^2 = .07 for Step 1 ($p < .05$) ; ΔR^2 = .31 for Step 2 ($p < .001$). * $p < .05$. ** $p < .01$.

overall F (2,77) = 14.77, overall $p < .001$. Changes in the remaining two risk factors, however, were not significantly related to Time 2 severity of anorexic symptoms. Specifically, changes in ineffectiveness scores did not result in a statistically significant increase in explained variance in the ASI, ΔR^2 = .01, ΔF (2,77) = .98, p = .33, overall F (2,77) = 1.25, overall p = .29. Changes in depressed mood scores yielded a change in R^2 value of only 3%, ΔF (2,77) = 2.17, p = .14, overall F (2,77) = 1.21, overall p = .30. These results suggested that when examining the risk factors individually, changes in the drive for thinness and maladaptive cognitions subscales functioned as better predictors of subsequent severity of anorexic symptoms than depressed mood or ineffectiveness scores.

Table 23

Summary of Hierarchical Regression Analysis for Changes in Ineffectiveness Scores Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 ineffectiveness	.04	.03	.14
Step 2			
Time 2 ineffectiveness	.05	.05	.12

Note. Subjects include individuals in high-risk and control groups. R^2 = .02 for Step 1 (p = .22) ; ΔR^2 = .01 for Step 2 (p = .33).

Follow-up scores on the BSI were also found to be associated with changes in several of the risk factor subscale scores over time (see Tables 26 through 29). Changes in drive for thinness scores explained approximately 10% of additional variance in the BSI, ΔF (2,77) = 8.93, $p < .01$, overall F (2,77) = 4.74, overall $p < .01$; and changes in maladaptive cognitions scores accounted for an additional 12% of the variance in the BSI, ΔF (2,77) = 10.34, $p < .01$, overall F (2,77) = 6.01, overall $p < .01$. These results paralleled those found for the ASI, although the percentage of explained variance was of a smaller magnitude. Contrary to results found with the ASI, however, was the finding that changes in depressed mood scores were significantly associated with subsequent BSI scores, ΔR^2 = .06, ΔF (2,77) = 5.09, $p < .05$, overall F (2,77) = 4.38, overall $p < .01$.

Table 24

Summary of Hierarchical Regression Analysis for Changes in Depressed Mood Scores Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 depressed mood	.14	.28	.06
Step 2			
Time 2 depressed mood	.52	.35	.20

Note. Subjects include individuals in high-risk and control groups. R^2 = .00 for Step 1 (p = .62) ; ΔR^2 = .03 for Step 2 (p = .14).

Finally, changes in ineffectiveness scores consistently failed to explain variance in symptom severity, ΔR^2 = .01, ΔF (2,77) = .52, p = .48, overall F (2,77) = .62, overall p = .54.

The same pattern of results was found when changes in each individual risk factor were used to predict Time 2 scores on the GSI (Tables 30 through 33). Changes in scores on the drive for thinness, maladaptive cognitions, and depressed mood subscales were found to be associated with statistically significant increases in the percentage of explained variance in the GSI. Specifically, the explained variance of the GSI was increased by 30% with inclusion of changes in drive for thinness scores, ΔF (2,77) = 34.16, p < .00, overall F (2,77) = 19.19, overall p < .001; by 22% with

Table 25

Summary of Hierarchical Regression Analysis for Changes in Maladaptive Cognitions Scores Predicting Time 2 Anorexia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 maladaptive cognitions	.68	.25	.30 **
Step 2			
Time 2 maladaptive cognitions	1.09	.24	.48 **

Note. Subjects include individuals in high-risk and control groups. R^2 = .09 for Step 1 ($p < .01$) ; ΔR^2 = .19 for Step 2 ($p < .001$). ** $p < .01$.

Table 26

Summary of Hierarchical Regression Analysis for Changes in Drive for Thinness Scores Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 drive for thinness	.02	.02	.08
Step 2			
Time 2 drive for thinness	.08	.03	.35 **

Note. R^2 = .01 for Step 1 ($p = .48$) ; ΔR^2 = .10 for Step 2 ($p < .01$). ** $p < .01$.

Table 27

Summary of Hierarchical Regression Analysis for Changes in Ineffectiveness Scores Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 ineffectiveness	-.02	.02	-.10
Step 2			
Time 2 ineffectiveness	.03	.04	.09

Note. R^2 = .01 for Step 1 (p = .40) ; ΔR^2 = .01 for Step 2 (p = .47).

Table 28

Summary of Hierarchical Regression Analysis for Changes in Depressed Mood Scores Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 depressed mood	-.39	.21	-.21
Step 2			
Time 2 depressed mood	.58	.26	.29 *

Note. R^2 = .04 for Step 1 (p = .07) ; ΔR^2 = .06 for Step 2 ($p < .05$). * $p < .05$.

Table 29

Summary of Hierarchical Regression Analysis for Changes in Maladaptive Cognitions Scores Predicting Time 2 Bulimia Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 maladaptive cognitions	.24	.20	.14
Step 2			
Time 2 maladaptive cognitions	.65	.20	.38 **

Note. R^2 = .02 for Step 1 (p = .22) ; ΔR^2 = .12 for Step 2 (p < .01). ** p < .01.

Table 30

Summary of Hierarchical Regression Analysis for Changes in Drive for Thinness Scores Predicting Time 2 Global Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 drive for thinness	.04	.02	.19
Step 2			
Time 2 drive for thinness	.13	.02	.59 **

Note. R^2 = .04 for Step 1 (p =.09) ; ΔR^2 = .30 for Step 2 (p < .001). ** p < .01.

Table 31

Summary of Hierarchical Regression Analysis for Changes in Ineffectiveness Scores Predicting Time 2 Global Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 ineffectiveness	.02	.02	.11
Step 2			
Time 2 ineffectiveness	.03	.03	.10

Note. R^2 = .01 for Step 1 (p = .33) ; ΔR^2 = .01 for Step 2 (p = .44).

Table 32

Summary of Hierarchical Regression Analysis for Changes in Depressed Mood Scores Predicting Time 2 Global Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 depressed mood	.01	.21	.00
Step 2			
Time 2 depressed mood	.54	.26	.27 *

Note. R^2 = .00 for Step 1 (p = .98) ; ΔR^2 = .05 for Step 2 ($p < .05$). * $p < .05$.

Table 33

Summary of Hierarchical Regression Analysis for Changes in Maladaptive Cognitions Scores Predicting Time 2 Global Severity Index (N = 80)

Risk factor	B	SE B	β
Step 1			
Time 1 maladaptive cognitions	.48	.19	.27 **
Step 2			
Time 2 maladaptive cognitions	.89	.18	.52 **

Note. R^2 = .08 for Step 1 ($p < .01$) ; ΔR^2 = .22 for Step 2 ($p < .001$). ** $p < .01$.

inclusion of changes in maladaptive cognitions scores, ΔF (2,77) = 24.67, $p < .00$, overall F (2,77) = 16.50, overall $p < .001$; and by 5% by inclusion of changes in depressed mood scores, ΔF (2,77) = 4.23, $p < .05$, overall F (2,77) = 2.11, overall $p = .13$. Additional variance explained by changes in ineffectiveness scores remained quite small, $\Delta R^2 = .01$, ΔF (2,77) = .60, $p = .44$, overall F (2,77) = .78, overall $p = .46$.

CHAPTER V

DISCUSSION

Are the four psychological variables examined in this study--depressed mood, maladaptive cognitions, ineffectiveness, and drive for thinness--related to increased risk for developing an eating disorder in a college population? The answer to this question was sought through multiple approaches. Relationships between initial scores and changes in scores on the risk factors and later symptom severity were examined. Incidence rates found in the high-risk group were compared with those of the control group. Changes in recovered subjects' scores were examined to see if decreases in eating disorder symptoms were accompanied by lowered scores on the four proposed risk factors. This chapter contains a discussion of the main findings and implications of these approaches to understanding the role of these four variables in the development of eating disorders.

How Early Risk Factor Scores Relate to Later Symptom Severity

Hypothesis 2 stated that "initial scores on the four risk factors together will explain a significant amount of variance in later eating disorder symptom severity." The results of the current study suggested that initial scores on the proposed risk factors were indeed related to subsequent severity of eating disorder symptoms. Early scores on these measures together accounted for 13% of the variance in subsequent eating disorder

severity scores. Although this percentage was statistically significant, a large proportion of the variance (87%) remained unexplained.

An examination of the role of each risk variable individually revealed that initial elevations of maladaptive thoughts and beliefs about eating and weight issues (maladaptive cognitions), and a preoccupation with body weight and a desire to be thinner (drive for thinness) were related to higher severity of symptoms at the later assessment. Specifically, the results revealed statistically significant zero-order correlations between initial drive for thinness scores and subsequent ASI scores ($r = .27$), and between initial maladaptive cognitions scores and later GSI ($r = .28$) and ASI ($r = .30$) scores (see Table 14). Thus, the Time 1 drive for thinness scores explained 7% of the variance in Time 2 ASI scores, and the Time 1 maladaptive cognitions scores explained 8% and 9% of the variance in Time 2 GSI and ASI scores, respectively. Regression analyses designed to identify the most parsimonious linear combination of variables in explaining the variance in symptom severity similarly revealed that initial maladaptive cognitions scores alone were the most efficient predictor of variance in subsequent severity of global symptoms. However, in explaining the variance in later anorexic symptom severity, the results showed that drive for thinness did not account for a significant increase in percentage of explained variance above and beyond that explained by maladaptive cognitions alone. In sum, it appears that although both initial maladaptive cognitions and drive for thinness scores are significantly related to later severity of anorexic symptoms, maladaptive cognitions alone provides the most parsimonious explanation of variance in the ASI.

It should be noted that although the aforementioned relationships reached levels of statistical significance, the amount of explained variance in eating disorder symptom severity was modest. They constituted one of many other, unidentified variables that influenced subsequent development of eating disorder symptoms.

Also of interest in the above findings was the absence of statistically significant correlations between the maladaptive cognitions, drive for thinness subscales, and the BSI. Although early scores on these two risk factors were related to later severity of anorexic symptoms, they were not significantly related to later symptoms of bulimia. This again points to the conclusion that anorexia and bulimia likely involve different risk factors, and that early elevations in maladaptive cognitions and drive for thinness may be more predictive of the developmental course of anorexia than bulimia.

Finally, it is notable that early scores on the depressed mood and ineffectiveness subscales were not significantly correlated with any of the symptom severity indices at the later assessment, and were not identified by the regression analyses as parsimonious or efficient predictors of eating disorder symptoms. These findings suggest that perhaps initial difficulties with depression and self-esteem are less related to onset of later eating disorders than previously believed.

Another possible interpretation of the failure to identify depression as a risk factor could be related to the nature of this mood construct. Perhaps it is not symptoms of clinical depression per se (as measured by the ABI depressed mood subscale in the current study, or by the BDI in prior prospective studies), but more global problems in negative mood modulation and management that is the key affective risk factor. Negative

affect modulation and management might involve difficulty coping with negative mood states, including not only depression, but also anger, frustration, and boredom. Such a construct has not been investigated in prospective eating disorder risk factor studies to date.

Additional research into the role of depression as a risk factor appears warranted, given unanticipated findings in the current study. Although the zero-order correlation between Time 1 depressed mood and Time 2 BSI (r = -.21) was not statistically significant, the direction of the correlation coefficient was negative (i.e., there was a trend for individuals with higher depression at Time 1 to have lower bulimic symptoms at Time 2). Similarly, although depressed mood did not reach statistically significant levels in the stepwise regression analyses determining the most parsimonious combination of variables in explaining symptom outcomes, the standardized beta coefficients were consistently negative. Finally, although there exist problems with using the simultaneous regression analyses (in which all four variables were entered together as block) to determine the contributions of individual variables, the standardized beta coefficients of depressed mood were again consistently negative in these results (Tables 15 through 17).

It is unlikely that these unexpected findings resulted from psychometric problems with the depressed mood subscale, given its very high correlations with other, well-validated measures of depression (e.g., BDI, diagnoses from structured clinical interviews). It also seems improbable that early problems with depression function as a protective factor against the development of later eating problems, particularly in light of the current finding that decreases (or increases) in depression over time were related to

lower (or higher) levels of eating disorder symptoms at the later assessment. Further speculation regarding the cause of this potential inverse relationship would be premature, especially in light of the fact that the majority of the results showing the inverse relationship did not reach levels of statistical significance. Future research using larger sample sizes and more frequent, intermediate assessment periods (e.g., yearly) might help to determine whether a true, robust inverse relationship between early depression and later eating disorder symptoms exists.

How Changes in Risk Factor Scores Relate to Later Symptom Severity

Hypothesis 3 predicted that "changes over time in each of the risk factor scores will be related to later severity of major eating disorder symptoms." This hypothesis was supported by the present results. A consistent and significant relationship was found between changes over time in the risk variables and subsequent eating disorder severity. Specifically, changes in risk factor scores over the 4-year interval explained 35% of the variance in global eating disorder symptoms, 34% of the variance in severity of anorexic symptoms and 16% of the variance in bulimic symptoms. In other words, increases over time in scores on the risk factors were related to higher Time 2 severity scores. The greater percentage of explained variance in anorexic symptoms, as opposed to bulimic symptoms, again indicated that these risk factors may be more relevant to the onset of anorexic than bulimic symptoms.

An examination of changes in each risk factor individually revealed that drive for thinness and maladaptive cognitions again appeared to be more relevant to eating disorder outcomes than either depressed mood or ineffectiveness. Specifically, changes in drive for thinness over time explained 31% of the variance in the ASI, 10% of the variance in the BSI, and 30% of the variance in the GSI. Changes in maladaptive cognitions explained 19%, 12%, and 22% of the variance in the ASI, BSI, and GSI, respectively. Thus, increases over time in these two risk factors (not just the initial scores alone) were related to higher severity of both bulimic and anorexic symptoms at the later assessment. Additionally, the trend seen earlier of stronger relationships between the risk predictors and anorexic outcomes (rather than bulimic outcomes) also remained in this analysis. Changes over time in both drive for thinness and maladaptive cognitions scores were more strongly related to anorexic outcomes than to bulimic outcomes.

Changes in depression over time also were related to eating disorder outcome, though to a lesser degree than that seen with maladaptive cognitions or drive for thinness. The results revealed that changes in depressed mood scores explained 6% of the variance in the BSI and 5% of the variance in the GSI. Contrary to the results found with the other two risk factors, there was no relationship between changes in depression over time and subsequent anorexic symptoms. It appears, therefore, that subjects who experienced a worsening of depression over the course of the study tended to have higher severity of bulimic, rather than anorexic, symptoms at the final assessment.

Changes in ineffectiveness scores over time were not significantly related to the subsequent scores on any of the symptom severity indices. This accords with the earlier

finding that initial scores on the ineffectiveness subscale were not correlated with later eating disorder outcomes. Thus, not only did initial problems with self-esteem and feelings of worth fail to predict later development of eating problems, but even increases over time in difficulties with self-esteem did not relate to higher severity of eating disorder symptoms at a later date.

Changes in Risk Factor Scores in Recovered Subjects

The majority of subjects who were diagnosed with an eating disorder at the initial assessment recovered from their disorder during the 4-year study interval (64%, $\underline{n} = 14$). Of interest was whether some or all of the risk factor scores declined in this subcategory of subjects. Hypothesis 4 predicted that "subjects who recover from an eating disorder over a 4-year interval will show a decrease in scores on all four risk factors." Scores on the depressed mood and ineffectiveness subscales will have decreased more dramatically than scores on maladaptive cognitions and drive for thinness subscales," as the latter may have been chronically internalized attitudes and beliefs.

Recovered subjects' scores on all four of the risk factors decreased over the 4-year interval, but still remained elevated compared to the control group scores. This contrasted with the scores of nonrecovered subjects, which showed a modest <u>increase</u> in risk factor scores over the same time period. Thus, as individuals recovered from problems with eating disorders, their scores on the four proposed risk variables declined, but did not reach levels as low as those seen in groups of individuals who had never had an eating disorder.

Contrary to the hypothesis, drive for thinness and maladaptive cognitions scores decreased more substantially over time than did the ineffectiveness and depressed mood scores (Table 13). This result makes sense, however, in light of the current findings about the relationship between these variables and the development of eating disorder symptoms. Several of the analyses have indicated that drive for thinness and maladaptive cognitions are the two variables most related to later symptom severity, while depressed mood and ineffectiveness have a weaker or even nonexistent relationship with subsequent eating disorder symptoms. Hence, if drive for thinness and maladaptive cognitions are more closely linked to the behavioral symptoms of eating disorders, it is not surprising that scores on these subscales decrease more dramatically when the eating disorder remits.

Differences in Risk Factors for Anorexia and Bulimia: A Summary of Findings

Few prior studies have attempted to discern differences between risk factors for anorexia nervosa and bulimia nervosa. The findings of the current study suggest that the risk factors for the two disorders likely differ. First, initial elevations on the drive for thinness and maladaptive cognitions subscales were related to subsequent heightened severity of anorexic, but not bulimic, symptoms. Changes in the four risk factors together accounted for a higher percentage of explained variance in later anorexic symptoms (34%) than bulimic symptoms (16%). Changes over time in drive for thinness and maladaptive cognitions scores also explained a greater percentage of the variance in the

severity of anorexic symptoms than bulimic symptoms, although both relationships reached levels of statistical significance. Finally, changes in depressed mood scores over time had a weak, but statistically significant, relationship with the subsequent severity of bulimic, but not anorexic, symptoms.

Together, these results suggest that anorexia nervosa and bulimia nervosa may indeed involve a somewhat different constellation of risk factors. Furthermore, the psychological variables under investigation in the current study may be, in general, more relevant to anorexia than to bulimia outcomes. Initial elevations and changes over time in maladaptive cognitions and drive for thinness, in particular, appeared to be more strongly related to the development of anorexic symptoms than to bulimic symptoms. Increases in depression, however, were more related to higher severity of later bulimic symptoms. These findings underscore the importance of differentiating between these two disorders in future risk factor research.

How Well the Risk Factors Predict the Development of New Eating Disorders

Although the aforementioned results provided ample evidence that at least several of the variables under investigation in the current study are related to later severity of eating disorder symptoms, the combination of these four variables did not provide a particularly effective means of screening which college women would develop an eating disorder over the 4-year prospective interval. This limited success may have been due to limitations in the number and type of risk factor measures used, and the general problems

inherent in screening for low base-rate disorders. The first hypothesis of this study predicted that "college women scoring, at initial assessment, at or above the median on each of the four risk indices (drive for thinness, ineffectiveness, depressed mood, and maladaptive cognitions) will evidence higher incidence rates of eating disorders (at the 4-year follow-up) than the low-risk group (individuals who scored below the median on the risk indices)." This hypothesis was not supported in the current study. The 4-year incidence rates were 0% for the high-risk group (0 out of 25 subjects) and .7% for the control group (1 out of 144 subjects). Obviously, an individual's initial scores on the four risk factors did not provide an accurate method of assessing whether a subject would develop an eating disorder over the course of the study.

This finding may partially be due to the extremely small number of emergent cases in the sample (1 out of 169 subjects; .6%), which could have decreased the reliability of comparisons between the incidence rates of the high-risk and control groups. Replication of the study with a larger sample size could be helpful in determining whether these results are stable. However, it is certainly the case that accurate prediction of eating disorder onset in this population would be extremely difficult even with a larger sample, due to the low base rates of the disorder in this age group. Regardless, the current results strongly show that using cut-off scores on these four measures as a method of early identification of college-age women who may be at risk for developing an eating disorder is not particularly useful.

The low incidence (new case) rate of .6% was unexpected and deserves further comment. Hypothesis 6 predicted that "the 4-year incidence rate (new cases) of eating

disorders (anorexia, bulimia, and eating disorder NOS) will be relatively low (i.e., approximately 3% to 4%), similar to estimates found in other prospective research studies." The finding of such a low incidence rate may suggest that risk of developing an eating disorder decreases across the 4-year period of college. By later college years--that is, by the latter and early post-college years--many women may be passing out of the high-risk period for developing problems with anorexia or bulimia. This finding, if confirmed by future research, has implications for eating disorder prevention programs. Prevention efforts may be most successful if they primarily target a younger age group (i.e., early teen-age girls, rather than college-age women), to address risk issues before individuals enter the period of significant high risk for eating disorders.

Obtained Prevalence Estimates

Hypothesis 5 predicted that the prevalence rates of anorexia and bulimia in this university sample will be similar to the estimates (i.e., 0.5% to 3.0%) reported in the most well-controlled research studies. It was expected that prevalence rates for eating disorder NOS in the current study would approximate 7 to 13%, the figures often reported in the literature. The current study provided partial support for this hypothesis. Prevalence rates for anorexia nervosa and bulimia nervosa at the initial assessment fell, in general, within the expected range (see Table 11). Of the sample, 2.6% were diagnosed with anorexia nervosa, and 3.7% with bulimia nervosa. The percentage of subjects diagnosed with eating disorder NOS was slightly lower than expected (5.2). Also, these results lend additional support for the view that diagnoses based on clinical interviews provide more

conservative estimates of eating disorder prevalence than those based solely on paper-and-pencil questionnaire data.

Surprising results were found, however, with regard to the prevalence rates at the final assessment. At Time 2, none of the subjects were diagnosed with anorexia nervosa, .5% were diagnosed with bulimia nervosa, and 3.7% were diagnosed with eating disorder NOS. These prevalence rates were all lower than expected. This finding may have resulted from two factors: first, the extremely low number of new cases that developed during the study, and second, the high percentage (64) of individuals who recovered from an eating disorder over the 4-year interval, with postassessment coming at the late college and early post-college years.

Implications for Existing Models of Eating Disorder Etiology

It is important that results of empirical studies of eating disorder risk factors be examined in reference to existing models of eating disorder etiology, in order to suggest further refinements in these theories. The results of the current study provide support for some, but not all, elements of the etiological models discussed in the preceding literature review.

Cooper's (1995) tri-stage model of etiology emphasized the critical role of depression in predisposing an individual to developing an eating disorder. The model held that this factor would be particularly relevant in the risk pathway for bulimia. The results of the current investigation revealed that worsening of depressive symptoms over

the course of the study was modestly related to higher severity of bulimic symptoms at the final assessment. This relationship did not hold true for anorexic symptoms. These findings offer some support for Cooper's model. However, the results also indicated that initial elevations in major symptoms of depression were not significantly related to later onset of symptoms of anorexia or bulimia. Therefore, the relationship between changes over time in depression and later severity of bulimic symptoms could simply represent worsening depression as bulimia develops, rather than depression as a causal factor in the etiology of bulimia.

The results of the current study also do not lend support for the view that low levels of self-esteem or efficacy are implicated in the development of eating disorders, a tenet of both the tri-stage and the cognitive models of etiology. The findings indicated that initial problems with self-esteem did not predict later development of anorexia or bulimia. Even individuals who experienced a worsening of self-esteem across time did not show higher eating disorder severity at the later assessment. These results, along with those from several other prospective studies examining the role of self-esteem in eating disorder etiology, suggest that poor global self-esteem may be less important in the developmental course of eating disorders than was previously thought. Future research efforts aimed at differentiating global self-esteem from more specific appearance- or body-related esteem in the development of eating disorders may be fruitful.

Both the tri-stage and the cognitive models of etiology theorize that cognitive distortions about shape and weight, along with heightened desires for thinness, increase the risk for developing an eating disorder. The results of this study provide support for

these elements of both theoretical models. The findings that initial scores on maladaptive cognitions and drive for thinness subscales were related to later eating disorder symptoms, and that changes over time in these two variables were related to subsequent eating disorder symptom severity, indicate that the inclusion of these elements in models of eating disorder etiology is consistent with empirical research findings.

Summary of Major Findings

In summary, the results of the current study revealed the following main points:

1. Initial scores on the four risk factors together explained approximately 13% of eating disorder symptom severity at the final assessment four years later. Although this percentage is statistically significant, a large percentage of the variance in symptom severity remained unexplained.

2. Changes in the four risk factor scores over time were more highly related to later symptom severity, explaining 34% of the variance in anorexic symptom severity and 16% of the variance in bulimic symptom severity.

3. Initial maladaptive cognitions and drive for thinness scores were more strongly related to later eating disorder symptoms than were depressed mood or ineffectiveness scores. Furthermore, maladaptive cognitions scores alone explained the variance in later severity of anorexic symptoms in a more parsimonious manner than drive for thinness alone, or than the two variables together.

4. Increases in maladaptive cognitions and drive for thinness scores over time were related to higher severity in bulimic and anorexic symptoms. Increases over time in

depressed mood scores were related, to a lesser degree, to higher severity in bulimic symptoms.

5. There was some indication that early depression may be inversely related to later severity of eating disorder symptoms. This trend must be confirmed by future research.

6. In recovered subjects, scores on all four risk factors decreased over time, but still remained at elevated levels compared to control subjects. Maladaptive cognitions and drive for thinness scores showed a more marked decline than scores on ineffectiveness or depressed mood.

7. The individual risk variables had different patterns of relationships with later bulimic and anorexic symptoms, suggesting that the two disorders may have somewhat different risk pathways.

8. Individuals with elevated scores on the four risk variables at the initial assessment did not show higher absolute eating disorder incidence rates over the interval than subjects in the control group. Thus, initial scores on these four risk factors do not provide an accurate means of predicting the development of an eating disorder over a 4-year interval.

9. The 4-year incidence rate in this sample was unexpectedly low (.6%), suggesting that as women move through their college years they are leaving the developmental period of high risk for onset of eating disorders.

Limitations and Future Directions

The current study has several limitations that may have impacted the obtained results. First, although the original sample size was large, the sample size obtained at the final assessment was relatively small. This made the study of a disorder with low incidence rates more difficult because fewer subjects experienced worsening of symptoms or development of a new disorder over time. The high attrition rate between the two assessment periods, although not resulting in any identified meaningful differences between retained and nonretained subjects on the EDI or ABI subscales, may have produced a final sample which differed from the population in other, unidentified ways. Thus, future studies would benefit from beginning with a larger original sample and taking additional measures to enhance subject retention over time. To reduce subject attrition rates over such an extended interval, researchers might benefit from more frequent contacts (e.g., yearly) with subjects to ascertain their whereabouts more often, thereby reducing the number of subjects who are untraceable after a 4-year interval. Furthermore, conducting clinical interviews with all subjects (as opposed to all high-risk and symptomatic subjects, but only a random sample of control subjects) would provide a larger final sample size for the regression analyses. Of course, implementation of these recommendations would require substantial additional resources.

The external validity of the current study is limited to female undergraduate students. Future research using other populations would be beneficial in expanding the knowledge of risk factors for eating disorders in alternative groups of individuals (e.g.,

non-college women, males, etc.). Finally, since most eating disorder risk factor research continues to be conduced with preadolescent and adolescent females, it is recommended that future researchers extend and replicate this current study with populations of women in their early 20s.

Some unexpected findings in the present study need confirmation by future research. In particular, the low incidence rate seen in this sample, and the suggestion of an inverse relationship between initial levels of depression and later severity of bulimic symptoms, should be corroborated by findings in other studies before high levels of confidence are placed in these results.

The current findings, along with those of other prospective studies conducted to date, should be used to guide selection of risk variables included in future studies. Our knowledge of eating disorder risk factors would be enhanced if future studies would not only investigate variables that are consistent with those found to be related to eating disorder onset by existing studies (e.g., maladaptive cognitions, drive for thinness), but also continue to study new variables. Given the limited success that current research has had in finding variables that account for large proportions of the variance in symptom outcomes (e.g., 13% in the current study), inclusion of new variables is warranted. Such variables might include history of trauma or sexual abuse, family eating patterns/family interpersonal functioning, biological or genetic factors, additional personality variables, and a general vulnerability towards poor coping with environmental or developmental stressors. Finally, future studies should avoid selecting independent variables that have consistently functioned as poor predictors of eating disorder onset (e.g., ineffectiveness).

It appears unwise for researchers to continue to assess the role of clinical depression in eating disorder onset. It is speculated, however, that future researchers may more profitably assess related constructs, such as problems with controlling and managing negative mood states (including sadness, anger, irritability, etc.) rather than focusing solely on clinical depression per se.

Finally, the results of the current research provided evidence that risk pathways for bulimia and anorexia may differ. Most past research has either focused solely on one disorder, or has grouped the symptoms of both disorders together in the analyses. Future studies would be enhanced by methodologies that allow separate analyses of risk factors for bulimia and anorexia, whenever possible.

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APPENDICES

Appendix A

Human Subjects Approval Letter

Utah State UNIVERSITY

VICE PRESIDENT FOR RESEARCH OFFICE
Logan, Utah 84322-1450
Telephone: (801) 797-1180
FAX: (801) 797-1367
INTERNET: [pgerity@champ.usu.edu]

April 2, 1996

MEMORANDUM

TO: David Stein
Anne Dobmeyer

FROM: True Rubal, Secretary to the IRB

SUBJECT: A Prospective Analysis of Risk Factors for the Development of Eating Disorders

The above-referenced proposal has been reviewed by this office and is exempt from further review by the Institutional Review Board. The IRB appreciates researchers who recognize the importance of ethical research conduct. While your research project does not require a signed informed consent, you should consider (a) offering a general introduction to your research goals, and (b) informing, in writing or through oral presentation, each participant as to the rights of the subject to confidentiality, privacy or withdrawal at any time from the research activities.

The research activities listed below are exempt from IRB review based on the Department of Health and Human Services (DHHS) regulations for the protection of human research subjects, 45 CFR Part 46, as amended to include provisions of the Federal Policy for the Protection of Human Subjects, June 18, 1991.

2. Research involving the use of educational tests (cognitive, diagnostic, aptitude, achievement), survey procedures, interview procedures or observation of public behavior, unless: (a) information obtained is recorded in such a manner that human subjects can be identified, directly or through the identifiers linked to the subjects: and (b) any disclosure of human subjects' responses outside the research could reasonably place the subjects at risk of criminal or civil liability or be damaging to the subjects' financial standing, employability, or reputation.

Your research is exempt from further review based on exemption number 2. Please keep the committee advised of any changes, adverse reactions or termination of the study. A yearly review is required of all proposals submitted to the IRB. We request that you advise us when this project is completed, otherwise we will contact you in one year from the date of this letter.

Appendix B

Example of Study Consent Form

CONSENT FORM

The purpose of this follow-up research study is to increase our understanding of how college women change over the course of 3 to 4 years in terms of their feelings and attitudes about health, nutrition, and emotional issues in their lives. The reason we are inviting your participation is because 3 to 4 years ago you agreed to complete a screening packet as part of an extra credit project in a psychology or related course. Your contribution of follow-up information at this time would be extremely valuable for two reasons: first, because there are almost no long-term studies of this kind being conducted in the US; and second, because collecting long-term follow-up data is extremely difficult due to the many individuals who graduate, move away, change their name, get married, etc. Thus, the overall number of available subjects is small. Your contribution of responses would likely be representative of the many subjects whom we were unable to locate; therefore, the information you would provide is extremely important. By participating in the study, you will be eligible to win a drawing for a $50 savings bond.

Completing the enclosed packet of inventories will take about 30-45 minutes of your time. The postage on the return envelope has already been paid. A small number of subjects may be recontacted for short phone interviews. All of your responses would be kept strictly confidential. This means that other administrators, parents, instructors, etc., could never have access to this information in the future. Each participant will be assigned a special ID code so that only the directors of this project will ever be able to match up responses for a given individual. All identifying information about subjects will be discarded at the end of the study. We are interested in group data, that is, responses across many subjects, and not information regarding individual subjects. Your participation is strictly voluntary, and you may withdraw your consent to participate in this study at any time, without any consequence.

Please fill out the enclosed questionnaires and return them as soon as possible in the postage-paid return envelope. **If your return mailing is postmarked by TUESDAY, MARCH 12th, we will include your name in an additional drawing for a $25 savings bond**. Do not put your name or any identifying information on ANY response forms. If you have questions please do not hesitate to contact Anne Dobmeyer at 797-2027 or 752-0124, or Dr. David Stein, USU Psychology Department, at 797-3274.

I have read the above and agree to participate: Name: _____

Signature: _____ Date: _____

Appendix C

Structured Clinical Interview for DSM-III-R (SCID)
(Modified Version for DSM-IV)

I. Anorexia Nervosa

The majority of women have been on various diet programs. Do you ever control what you eat to help you lose or maintain your weight?

Tell me about some of the ways of losing weight you have tried (skipping meals; fasting for 24 hours or more; exercise; how much, how often?)

What is your current height and weight?

What is the most weight you have ever lost? When was that?

What weight did you start out at?
How tall were you then?
What was your weight goal then?
What was the weight you finally got down to?

Have there been other times when you've lost weight?
When was this?
What weight did you start out at?
What weight did you get down to?

What has your average weight been over the past 5 years or so?

Are you trying to right now to lose weight?
What weight did you start out at?
What is your weight goal right now?
How long have you been working on the current weight goal?

Do you ever find that you tend to regain the weight you have worked hard to lose?

When you were dieting or were losing weight, did your periods ever become irregular or stop altogether for a few months in a row?

IF YES:

How many months did you skip in a row? (How irregular did they get?)

Were you pregnant at the time?

Have you usually been more regular when you weren't dieting or losing weight?

Have your periods ever started again?

How many have been missed in past 6 months?

Have you ever argued with anyone, because they were trying to convince you that you needed to eat more & gain weight?

Has anyone ever threatened to take you to the doctor or a treatment program because they were worried that your weight was too low?

If YES to either of the above: ask the following in PRESENT TENSE if the person may currently be anorexic (based on responses or appearance); otherwise, use PAST TENSE:

When did this disagreement over your weight happen? (Has this been quite recent?)

Were you trying to diet and lose weight at the time?

Did (do) you ever feel that others were (are) a bit jealous of the success you had with dieting?

Did/do you feel that losing weight is something you are quite good at, compared to most other people?

Tell me how much you weigh(ed) at the time people were most concerned about your weight.

What was the lowest weight you reached (during the period when others were trying to get you to gain weight)?

When people are/were trying to talk you into gaining weight, did you basically ignore them and quietly go on with your diet, losing weight as YOU saw fit?

Do you ever have the sensation of feeling fat, even through friends or relatives say you aren't fat at all?

IF YES:

Tell me more about what they say, and how you try to judge how fat or overweight you are

How do you feel overall about your body shape?

Do you ever feel that particular areas of your body are fat or are problem areas, and that you should diet to deal with these areas?

How concerned are you about the shape of your body or the size of different parts of your body? Would you say you are concerned an extreme amount, a moderately high amount, a normal amount, very little, or not at all?

How would you have rated this in the past?

What is it about the shape of your body you especially dislike; what would make your body more "ideal"; what would make you less worried about your weight?

I'm wondering about the degree that you worry about your weight...

Would you say that you worry an extreme amount, a moderately high amount, an average amount, very little, or not at all about your weight?

How would you have rated this in the past (Note: refer to a specific year or dieting episode if possible)

Do you feel that you worry too much during the day about your weight; or wish you weren't so worried about it?

Have you found that worry about your weight distracts you from doing other things that you should be doing?

Does worry about your weight interfere with your daily routines or activities?

Are you fearful of gaining weight?

On a scale running from 1 to 10, where 1 is no fear at all, and 10 is being absolutely terrified with fear, what number represents your fear of getting fat?

How would you have rated this in the past? (Note: refer to a specific year or dieting episode if possible)

II. Bulimia Nervosa

A survey of women found that a number experience "eating sprees" in which they ate large amounts of food in a relatively short amount of time.

Have you ever had the experience of suddenly consuming enough food to satisfy the hunger of several people?

Have you ever gone on eating binges; that is, eating a great deal more food than you had intended, in a short period of time?

If "NO" to both of the above, go to **********.

If "YES" to either of the above, continue with:

When were you having these binges? Have you had any within the past 6 months?

If you were to have any eating spree today, what kind of food would you eat, and how much?

Can you tell me what a typical binge is/was like for you--what types of food and how much food you eat/ate?

Give me a listing of what you eat and how much of it you eat during a typical binge

What causes you to stop eating?

Sometimes people's thought or feelings help them to stop eating; for others, something happens--a roommate or spouse interrupts their eating binge. What happens in your situation?

What is the most you have ever consumed during one of these sprees? (have them list types and amounts)

Do you find that you tend to have these binges at certain times of the day, more than other times?

I need to have some idea of how long it takes for you to go through an eating spree or binge, from start to finish. (Give me a specific example that you recall)

On a scale from 1 to 10, where "1" is being totally in control, and "10" is completely without control, what number represents the amount of control you feel over your eating during an eating binge? (Note: get both past and present ratings, if they have acknowledged bingeing in the past & present)

Do you feel you could stop eating at any time you wanted, or do you feel like you are mechanically eating and can't easily stop?

For how many months have you had (did you have) eating binges?

For current episodes (within past 6 months), ask: If you think back to the worst month in the last 6 months, how many times a week were you having these binges?

For past episodes, ask: When you think back to the worst month during the time you were having these binges, how many times a week did they occur?

****
****

From time to time, dieters experiment with a number of methods to lose weight. I want to list some common ways of losing weight. I'm interested in whether you have experimented with any of them...

a. taking laxatives, water pills, or diet pills?

b. fasting for 12 to 24 hours or more?

c. significantly cutting back on the amount of food you eat?

d. feeling that you want to throw up?

(if "YES": Do you ever throw up?)

e. exercise or trying to burn calories by physical activity?

NOTE: If subjects acknowledge one of the symptoms (a-e below), have subject elaborate as needed:

WHEN DID THIS OCCUR, HOW OFTEN, HOW MUCH, DETAILS OF AN EXAMPLE OF A SYMPTOM.

Get the details of either their worst month in the last six months (current episode), or for their worst month during the time they were using the method (past episode).

Example of follow-up questions: When were you using laxatives? Have you used any within the past 6 months? During your worst month of using laxatives, how many were you taking each week? For how many months did you take the laxatives?

****
****

When we think about self-esteem, we know that it can be tied to many things, such as personality, relationships, how people feel about their career, or how they feel about their weight or body shape.

On a scale from 0% to 100%, what percentage of your self-esteem would you say is tied to the way you feel about your body shape or weight?
(e.g., 10%, or 50%, or 90%)

Do you think this percentage is lower, the same, or higher than that of most other women?

Can you put a number on that, from 1 to 10, where 1 is much lower than most women, 5 is about the same, and 10 is much higher than most women?

(also get a past rating--either for a specific dieting episode, or a general rating over the past several years)

Have you ever had treatment regarding eating or weight issues?

If "YES":

Who did you see (physician, psychologist, therapist, nutritionist, etc.)?

When did this occur?

How many times did you see this person?

How long did you see this person (weeks, months, etc.)?

Any other details? (particular issues worked on)

Appendix D

Clinician's DSM-IV Checklist

Subject ID number _____ Current Episode___ Past Episode___ Height_____

Weight_____ Interviewer_____ Rater_____

Rate the severity or frequency of each symptom below based on the worst month in the last six months. The typical or usual patient should be assigned a rating of "3" on a symptom. If a letter has two ratings, mark only the category that is appropriate. Ratings range from "1" (Severity or frequency of symptom is extremely low; or symptom is not present) to "5" (Severity or frequency of symptom is extremely or unusually high for treatment program)

_____ **A. Refusal to maintain body weight over a minimal normal weight for age and height**:
(Record: lowest reported weight_____ height_____)

Weight loss leading to maintenance of body weight 15% below expected (use weight chart) OR
Failure to make expected weight gain during period of growth, leading to body weight 15% below expected (use weight chart)

_____ **B. Intense fear of becoming obese, even when underweight**:
Rating 1 - 10: 1-4 = 1; 5-6 = 2; 7 = 3; 8 = 4; 9-10 = 5)

_____ **C. Disturbance in the way in which one's body weight, size, or shape is experienced**:
Rating based on body dissatisfaction subscale of the Eating Disorder Inventory

_____ **D. In females, absence of at least three consecutive menstrual cycles** when otherwise expected to occur (primary or secondary amenorrhea).
List the number missed in the past 6 months (or in past episode): _____.
Rating: 0 missed = 1; 1 missed = 2; 2-3 missed - 3; 4-5 missed = 4; 6 missed = 5

_____ **E. Recurrent episodes of binge eating**
(rapid consumption of a large amount of food in a discrete period of time, usually less than two hours; at least 1200 calories).

List the average number of binges during her worst month: _____.
(1-2 episodes = 1; 3-5 episodes = 2; 6-8 episodes = 3; 9-12 episodes = 4; >12 episodes = 5).
List the types and amounts of foods consumed during an average binge:

_____ **F. During the eating binges there is a feeling of lack of control over the eating behavior.**
Rating 1-10: 1-4 = 1; 5-6 = 2; 7 = 3; 8 = 4; 9-10 = 5

G. In order to counteract the effects of binge eating, the individual regularly engages in:

_____ Self-induced vomiting. List the average number during the worst month in the last 6 months: _____.
(rating: less than monthly or never = 1; 1-4/month = 2; 5-9/month = 3; 10- 15/month = 4; >15/month = 5)

_____ Use of laxatives, diuretics, or diet pills. Rate highest frequency during the worst month in the last 6 months: _____.
(rating: less than monthly or never = 1; 1-4/month = 2; 5-9/month = 3; 10- 15/month = 4; >15/month = 5).

_____ Rigorous dieting or fasting. Rate frequency of 12 to 24 hour fasts during the worst month in the last six months: _____.
(rating: less than monthly or never = 1; 1-4/month = 2; 5-9/month = 3; 10-15/month = 4; >15/month = 5).

_____ Rate frequency of vigorous exercise (at least 2 hours per day aerobic exercise) during worst month in the last 6 months: _____. List types of (aerobic) exercise engaged in: _____.
(rating: less than monthly or never = 1; 1-8/month = 2; 9-16/month = 3; 17-24/month = 4; >24/month = 5).

_____ **H. Self-evaluation is unduly influenced by body shape and weight.**
List reported number 1-10: _____.
(rating: 1-4 = 1; 5-6 = 2; 7 = 3; 8 = 4; 9-10 = 5).

Appendix E

Tables

Table 34

Means and Standard Deviations of ABI and EDI Subscales (N = 191) and Symptom Severity Index Scores (N = 102)

Subscale	Time 1 Mean (SD)	Time 2 Mean (SD)
Eating Disorder Inventory (EDI)		
Drive for thinness	5.79 (5.49)	4.22 (4.96)
Bulimia	1.64 (2.88)	1.03 (2.38)
Body dissatisfaction	13.32 (8.80)	11.86 (8.44)
Ineffectiveness	3.14 (4.67)	2.09 (3.88)
Perfectionism	7.28 (4.47)	6.85 (4.58)
Interpersonal distrust	2.93 (3.59)	2.04 (3.15)
Interoceptive awareness	4.63 (3.73)	3.53 (2.81)
Maturity fears	2.31 (2.93)	1.68 (2.74)
Anorexia-Bulimia Inventory (ABI)		
Parent conflict	2.02 (.50)	1.90 (.57)
Depressed mood	2.03 (.57)	1.88 (.57)
Anxiety	2.19 (.58)	1.98 (.56)
Maladaptive cognitions	2.23 (.62)	2.06 (.61)
Anorexia	1.68 (.45)	1.50 (.41)
Anergia	2.41 (.59)	2.27 (.58)
Purge	1.28 (.41)	1.19 (.30)
Binge	1.69 (.62)	1.44 (.57)
Exercise	2.35 (.56)	2.26 (.58)
Symptom severity indices		
GSI	3.66 (3.01)	2.18 (1.68)
BSI	2.76 (3.54)	1.00 (1.98)
ASI	4.49 (3.10)	2.99 (1.64)

Note. GSI = Global Severity Index; BSI = Bulimia Severity Index; ASI = Anorexia Severity Index.

Table 35

Intercorrelations Between Severity Index Scores (N = 80)

Severity index	ASI	BSI	GSI
Time 1			
ASI	--		
BSI	.72	--	
GSI	.90	.92	--
Time 2			
ASI	--		
BSI	.58	--	
GSI	.87	.84	--

Note. ASI = Anorexia Severity Index, BSI = Bulimia Severity Index, GSI = Global Severity Index. Only high-risk and control subjects were included in this analysis; subjects who were symptomatic at Time 1 were excluded.

CURRICULUM VITAE
June, 2000

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EDUCATION

Ph.D. 2000	Utah State University, Logan, Utah Combined Clinical, Counseling, and School Psychology Program Full APA Accreditation Dissertation: Eating Disorder Risk Factors: A Prospective Analysis Committee Chair: David M. Stein, Ph.D. Anticipated graduation: August 2000 GPA: 3.99
M.S. 1997	Utah State University, Logan, Utah Counseling Psychology Thesis: Factor Structure of the Anorexia-Bulimia Inventory Committee Chair: David M. Stein, Ph.D. GPA: 4.00
Visiting scholar 1996-1997	Rutgers University, New Brunswick, New Jersey Clinical Psychology Ph.D. Program GPA: 4.00
B.A. 1993 *summa cum laude*	St. Olaf College, Northfield, Minnesota Major: Psychology GPA: 3.86

ACADEMIC AWARDS AND HONORS

1998-1999	Walter R. Borg Scholarship Recipient, Utah State University
1996	Utah Psychological Association Thesis/Dissertation Award
1994-1998	School of Graduate Studies Honor Roll, Utah State University

1994-1995	Recipient of Research Vice President's Fellowship, Utah State University
1993	Elected to Phi Beta Kappa
1992	Elected to St. Olaf Senior Women's Honor Society
1991	Elected to Psi Chi
1989-1993	Recipient of National Merit Scholarship

CLINICAL EXPERIENCE

Clinical Internship

1999-2000 **Malcolm Grow Medical Center, United States Air Force**
Andrews Air Force Base, Maryland
Provided intake assessments, individual and group therapy, and crisis intervention to individuals, couples, and children. Completed rotations in outpatient mental health, behavioral health psychology, primary care, prevention/consultation, and neuropsychology. Conducted psychological assessments, security clearance evaluations, and neuropsychology evaluations. Led groups for depression, anxiety, stress management, smoking cessation, weight management, and chronic pain. Participated in prevention activities, including suicide awareness and workplace violence outreach programs.
Supervisors: John M. Beery, Ph.D., Linda Broeckl, Ph.D., and Kevin Mulligan, Psy.D.
2000 hours (anticipated)

Clinical Practica

1999 **Bear River Community Mental Health Center**
Logan, Utah
Conducted intake assessments and comprehensive psychological evaluations. Provided individual, family, and couples therapy to adults, adolescents, and children.
Supervisor: Skip Winger, Ph.D.
150 hours

1997 - 1998 **Utah State University Counseling Center**
Logan, Utah
Provided individual, couples', and group therapy to university students. Conducted intake assessments and reports.
Supervisors: Mark Nafziger, Ph.D., and Mary Doty, Ph.D.
300 hours

1996 -1997 **Rutgers Psychological Clinic**
Piscataway, New Jersey
Provided individual outpatient therapy to adults.
Supervisors: Susan Arbeiter, Psy.D., and Jean Balinky, Ed.D.
65 hours

1995 -1996 **Clinical Services, Center for Persons with Disabilities**
Logan, Utah
Conducted comprehensive evaluations and psychotherapy for children and adolescents. Provided behavioral consultation to parents and teachers. Areas of emphasis included assessment of ADHD, learning disorders, and behavior disorders.
Supervisors: Phyllis Cole, Ph.D., and Patricia Truhn, Ph.D.
300 hours

1995 -1996 **Psychology Community Clinic, Department of Psychology**
Logan, Utah
Provided individual psychotherapy for adults and adolescents .
Conducted intake interviews and diagnostic evaluations.
Consulted with schools regarding eating disorder issues.
Supervisors: David M. Stein, Ph.D., Susan Crowley, Ph.D., and Kevin Masters, Ph.D.
400 hours

Program-Approved Training Experience

1998 - 1999 **Therapist, Bear River Community Mental Health Center**
Logan, Utah
Provided individual, family, and couples therapy to client population consisting primarily of low income adults, children, and adolescents, including the seriously mentally ill. Provided crisis intervention. Conducted comprehensive psychological evaluations and written reports.
Supervisor: Skip Winger, Ph.D.
550 hours

1997 - 1998 **Psychological Assessment and Treatment Specialists, Inc.**
Logan, Utah
Provided outpatient therapy to children, adults, and families. Conducted comprehensive psychological evaluations. Consulted with probation officers and school counselors.
Supervisor: Steven Gentry, Ph.D.
255 hours

1997 - 1998 **Diagnostician, Weber School District**
Ogden, Utah
Administered, scored, and interpreted psychoeducational assessment instruments.
Supervisor: Peggy Regl, MS.
150 hours

1998 **Psychometrician**
Logan, Utah
Conducted intellectual and psychological assessments as part of comprehensive vocational rehabilitation evaluations.
Supervisor: David M. Stein, Ph.D.
22 hours

1996 **Eating Issues Group Therapist**
Logan, Utah
Conducted intake assessments and facilitated cognitive-behavioral eating issues therapy group.
Supervisor: David M. Stein, Ph.D.
35 hours

1995 -1996 **Psychological Specialist, Clinical Services**
Center for Persons with Disabilities, Logan, Utah
Conducted comprehensive child and adolescent evaluations. Provided psychological treatment and behavioral consultation to children, families, and adults. Co-facilitated child social skills group. Supervised practicum students.
Supervisors: Phyllis Cole, Ph.D., and Patricia Truhn, Ph.D.
900 hours

RESEARCH EXPERIENCE

1996 - 2000 Dissertation research. A longitudinal investigation of risk factors for the development of eating disorders.
Major professor: David Stein, Ph.D.

1997 Research Assistant to David Stein, Ph.D., Utah State University
Drug and alcohol meta-analysis and prevention manual.

1996 -1997	Research Assistant, Institute for the Study of Child Development, University of Medicine and Dentistry of New Jersey Longitudinal study of links between children's reactivity to stress, objective self-awareness, and self-conscious emotions. Responsibilities included data analysis, coding of infant and child behavior, and data entry. Supervisors: Michael Lewis, Ph.D., and Douglas Ramsay, Ph.D.
1996 - 1997	Thesis research. Factor Structure of the Anorexia-Bulimia Inventory. Major Professor: David Stein, Ph.D.
1995	Child clinical research with Ken Merrell, Ph.D. Gender differences in internalizing disorders in elementary-age children.
1995	Norming project for PPVT-III. Administered assessment s to preschool children as part of national, multi-site norming project for the PPVT-III. Supervisor: Patricia Truhn, Ph.D.
1991	Research Assistant, St. Olaf College Psychology Department Conducted research trials in study investigating the effects of parent presence on gender and emotional display rules.

PUBLICATIONS

Dobmeyer, A. C. (1997). Factor structure of the Anorexia-Bulimia Inventory (Masters thesis, Utah State University, 1997). Masters Abstracts International.

Merrell, K. W., & Dobmeyer, A. C. (1996). An evaluation of gender differences in self-reported internalizing symptoms of elementary-age children. Journal of Psychoeducational Assessment, 14, 196-207.

PRESENTATIONS

Candy, C., & Dobmeyer, A. (1999, April). Primary prevention programs for eating Disorders: What do we know and where should we go? Paper presented at the meeting of the National Association of School Psychologists, Las Vegas, NV.

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