EATING DISORDERS IN ADOLESCENT FEMALES WITH
TYPE 1 DIABETES MELLITUS:
A CONTROLLED THREE-SITE STUDY

By

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A thesis submitted in conformity with the requirements for the degree of
Doctor of Philosophy (PhD)
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ABSTRACT

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Body dissatisfaction and dieting have become increasingly common in young women in Western countries, and are often associated with a cycle of binge-eating, further dietary restriction, and purging behaviours. In females with type 1 diabetes mellitus, this cycle may be triggered or amplified due to several aspects of diabetes and its management. Eating disorders have been found to be relatively common in adolescent females with type 1 diabetes and are associated with impaired metabolic control and an increased risk of microvascular complications. However, controversy persists as to whether eating disorders occur more frequently in females with diabetes than in their non-diabetic peers.

A 3-site, 2-stage cross-sectional study was conducted to compare the frequency of eating disorders in 356 adolescent females with type 1 diabetes (mean age 15±2 years) to 1098 age-matched non-diabetic controls. Eating disorders were assessed using self-report screening measures followed by a standardized diagnostic interview for those scoring above predetermined cut-off levels. Metabolic control in diabetes subjects was assessed by hemoglobin A1c.

Eating disorders that met DSM-IV criteria were more prevalent in subjects with diabetes (10.1 percent) than in non-diabetic controls (4.5 percent, P<0.001). Sub-threshold eating disorders were also more common in those with diabetes (13.8 percent) than in controls (7.6 percent, P<0.001). Body mass index was higher in diabetes subjects (22.6±3.8 kg/m²) than controls (20.6±3.3 kg/m², P<0.0001), and diabetes subjects with an eating disorder were
older at diabetes onset than those without. At screening, insulin dosage manipulation to promote weight loss was reported by 11 percent of the subjects with diabetes, and by 42 percent of subjects diagnosed with a DSM-IV disorder. Mean hemoglobin A1c was higher in diabetes subjects with a DSM-IV eating disorder (9.4±1.8 percent) than in those without (8.6±1.6 percent, P=0.004).

DSM-IV and sub-threshold eating disorders were almost twice as common in adolescent females with type 1 diabetes as in their non-diabetic peers, and were associated with insulin manipulation for weight loss and impaired metabolic control.
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LIST OF ABBREVIATIONS

AN   Anorexia Nervosa
ANOVA Analysis of Variance
APA American Psychiatric Association
BMI Body Mass Index
BN   Bulimia Nervosa
CHEO Children's Hospital of Eastern Ontario
DCCT Diabetes Control and Complications Trial
DSED Diagnostic Survey for Eating Disorders
DSM-III Diagnostic and Statistical Manual of Mental Disorders (Third Edition)
DSM-III-R Diagnostic and Statistical Manual of Mental Disorders (Third Edition-Revised)
DSM-IV Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition)
EAT-26 Eating Attitudes Test (26-item version)
EDE Eating Disorder Examination (version 11.5 d revised)
ED-NOS Eating Disorder Not Otherwise Specified
EDI Eating Disorder Inventory
HbA1c Glycosylated hemoglobin A1c
HHSC Hamilton Health Sciences Corporation
HSC Hospital for Sick Children
OR Odds Ratio
PASS Power Analysis and Statistical Power
SES Socioeconomic Status
SPSS Statistical Package for the Social Sciences
CHAPTER ONE
INTRODUCTION AND LITERATURE REVIEW

HUMAN EATING BEHAVIOUR

The development of eating habits is a complex human process. Although all living organisms must eat food for growth and the maintenance of tissues, individuals and societies do not eat according to the availability, edibility, and nutritional value of food alone (Herman, 1996). There are many other reasons why people eat. In fact, "The history of man has been called the chronicle of his quest for food" (Bruch, 1973, pp. 9).

An individual's set of cultural, social, and psychological ties all dictate food habits and nutrition behaviour (Bruch, 1973). Food and eating have many meanings, which are intimately tied to our whole way of life. Our attitudes and beliefs about food develop from birth, and are based on our culture, society, and family. In fact, food habits are among the oldest and strongest aspects of many cultures and exert a great influence on the behaviour of the people (Bruch, 1973). They are also shaped by life experiences, knowledge about food, pleasure and fears about food, the beliefs held about food and health, and ethnic diversity. Therefore, food and eating are closely related to and intermingled with both physiological and psychological aspects of human behaviour.

Food habits in any setting are highly socialized. Modeling and social inhibition exhibit strong effects over eating behaviour (Conger et al., 1980; Clendenen et al., 1994), and in social relationships, food can represent sociability, warmth, and social acceptance. In family relationships, food habits are even more significant. Foods can trigger childhood memories,
emotional warmth and security, and be valued for reasons other than its nutritional value. In addition, family income, community sources of food, and market conditions influence food habits and food choices.

The science surrounding eating and nutrition began during the late 18th century when respiratory gases and metabolism were first discovered (Robinson et al., 1986). During the 19th century chemists and physiologists added to the knowledge of nutrition following the discovery of proteins, carbohydrates, fats, and a number of minerals (Robinson et al., 1986). However, it appears that the greatest discoveries regarding eating behaviour and nutrition were not made until this century (Robinson et al., 1986). Following World War II, it was discovered that there are two neural structures for eating: one which initiates eating and one which terminates eating (Stunkard, 1993). Before this time, hunger was viewed as a basic drive to eat and satiety was simply a passive process that occurred when the drive to eat ran out (Stunkard, 1993). These findings changed the way that researchers viewed what "hunger" is. Overeating was no longer simply accounted for in terms of hunger but could also be explained by decreased satiety. It is now generally believed that there are mechanisms that control both the onset and termination of eating, and that limit the amount of food eaten to appropriate quantities.

Most research on human eating behaviour continues to be based on animal models. Although the concepts of hunger and satiety continue to be emphasized, their functioning in humans are still not clearly understood (Herman, 1996). Human research is complicated further by the lack of agreement on the constructs of hunger and satiety, and the difficulties measuring them. In a review of the concepts of "hunger" and "satiety" presented at the American Dietetic Association Annual Meeting in 1998 (ADA, 1998), the most commonly agreed upon definitions of hunger
included the following: 1) the number of hours of deprivation; 2) a motivational state regarding the acquisition of food; 3) a subjective experience which is reported by the individual; and 4) the activation of specific physical mechanisms which motivate us to eat. Blundell and Hill (1986) have defined "appetite" or "hunger" as phenomena that link both physiology and environmental events. Hunger and appetite can probably be best understood using a systems approach, which involves the interrelationships between psychological events and eating behaviour, with the peripheral physiological events and the central neurochemical processes.

Satiety has been more clearly defined in the literature as the inhibition of hunger as the result of eating. This is differentiated from satiation, which is what stops eating. Satiety and satiation appear to act together to maintain a biologically-appropriate pattern of eating that relates to the needs of the body and thus determines the drive to eat and eating behaviour.

Most theorists now agree that physiologic factors alone do not explain human eating. Certainly, there appear to be numerous individual mechanisms that influence hunger. Eating is often a response to social and emotional pressures and influences, beyond the simple demands of hunger and satiety (Herman, 1996). For example, hunger in humans can be mediated by conscious and deliberate control of eating (e.g., dieting) (Herman, 1996). Further, other factors may upset the appetite control system and extrinsic forces can disrupt the conditioned process of eating and digestion. For example, consuming a food that has the taste which is identical to another food, but which has a different metabolic property (e.g., aspartame vs. sucrose), can disrupt the metabolic process (Lavin et al., 1997; Rogers et al., 1990). The most severe disruption occurs when a person deliberately adjusts the pattern of eating, which dysregulates the process of eating (Stunkard, 1993).
The disorders of eating are typically associated with the consumption of abnormal amounts of food, either by excessively eating or by restriction of food intake. Nutritional disorders, such as obesity, now occur at alarming rates and are associated with serious health risks such as diabetes and heart disease. In fact, obesity and type 2 diabetes (which may result from obesity) affect over 30% of North American adults (Kuczmarski, 1992; Bennett, 1994; Hanson et al., 1995). Further, eating disorders such as anorexia nervosa and bulimia nervosa are also increasing in young women (Streigel-Moore et al., 1986). Although, it has been argued that because we still do not know what is "normal" eating, and it is difficult to identify abnormal eating, most agree that the eating disorders such as anorexia nervosa and bulimia nervosa are pathological (Herman, 1996).

EATING DISORDERS IN THE GENERAL POPULATION

Historical Perspective on Eating Disorders

Eating disorders, including anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders- not otherwise specified (ED-NOS) are conditions that have only been recognized relatively recently and have seemingly arisen in the context of changing societal attitudes towards obesity.

The origins of AN have been traced by some historians to the fasting saints and cults in medieval times (Stunkard, 1993). However, others have argued that these fasts were motivated primarily by religious reasons and not necessarily for thinness, and therefore are different from contemporary anorexia nervosa (Lacey, 1982).
Two of the earliest descriptions of AN were presented in papers by Edward Morton in 1694 and Robert Whytt in 1764 (Vandereycken and van Deth, 1994). However, little else was written about AN until the late 1800's and early 1900's, when pathological responses to the rejection of obesity began to surface in reports of restricting AN (Silverman, 1995). It was at this time that Charcot wrote about patients who presented with a relentless pursuit of thinness and overconcern regarding their body image (Habermas, 1989). In addition, William Gull, in his address to the British Medical Association (BMA) emphasized the loss of appetite and the physiological presentation of this disorder, and on this basis chose the name "anorexia nervosa" (Gull, 1986; Vandereycken and van Deth, 1994; Silverman, 1995).

There was much confusion for the next several decades, regarding the nature of AN, during which time the classification of this disorder passed through many different phases (Crisp, 1965; Russell, and Treasure, 1989; Stunkard, 1993). Early in this century, the view of AN ranged from it being a disease of the pituitary gland to a nonspecific variant of numerous psychiatric disorders (Garfinkel et al., 1995). This was exemplified in the 1960 book on anorexia nervosa by Bliss and Branch, who defined AN to be any weight loss greater than 25 lbs that could not be explained organically. The current view of anorexia is that it is a specific disorder with its own clinical features and symptoms (Russell and Tresure, 1989). These features included a fear of being fat and a 'relentless pursuit of thinness', which were specified in the Diagnostic and Statistical Manual for Mental Disorders-III (DSM-III) in 1980 (American Psychiatric Association, 1980) and essentially remained the same for the next two versions of the DSM (DSM, 1987; 1994).
The history of *bulimia nervosa* is more complex. Although "*bulimia nervosa*", as we know it today, was not defined until 1979 by Russell, early references to bulimia date back as early as the 8th century BC and have continued throughout history. In ancient Greek, the word for hunger was "limos" and the prefixes "bou-" (meaning a great amount), or "boul-" (meaning ox or steer), were added to characterize a "ravenous hunger" (Parry-Jones and Parry-Jones, 1995). Ancient authors referred to "boulimos" as episodes of overeating that were triggered by unusual hunger, and variants of the Greek "boulimos" have also been noted in other languages, including medieval Latin "bulimus" and middle French "bolisme" (Parry-Jones and Parry-Jones, 1995). Later, both Greek and Roman authors used the term to refer to the weakness and faintness associated with hunger and the resultant overeating.

In 1743, James described "true boulimus" as being intensely preoccupied with food and overeating for very short intervals, followed by fainting (Stunkard, 1993). He also described a variant which he called "caninus appetitus" in which overeating was followed by vomiting. It was not until the end of the 18th century that the criterion of feeling faint was no longer central to the disorder. In 1772, Cullen described patients whose hunger was so intense that they would eat more than they could digest (Stunkard, 1993), and by 1797, bulimia was fairly well recognized as binge eating and was even included in the Encyclopedia Britannica (Stein and Laasko, 1988). Although bulimia began to be recognized as a distinctive form of behaviour and was documented in several textbooks of medicine (Stein and Laasko, 1988), it did not receive very much attention again until after WWII. Present day *bulimia nervosa* appears to be more clearly tied to society's changing views of obesity over the past several decades, and has seemingly evolved out of the changing conceptualization of *anorexia nervosa*. 
Society's dislike of obesity is not necessarily a new phenomenon and dates back to the Greeks and Romans (Bruch, 1973) and other cultures such as medieval Japan (Stunkard et al., 1998). During the Middle Ages and more contemporary times, attitudes towards obesity appear to have been more positive and the "reproductive" "maternal" figure was in favor. For example, during Victorian and Edwardian times, the ample mature figure was preferred, and "plumpness" was a sign of social status and wealth (Rudofsky, 1972). However, beginning in the late 1800's and early 1900's, obesity once again fell out of favor following the publication of several papers and books in which medical concerns about obesity and fatness, even in small degrees, were expressed (Bray, 1990). Obesity and fatness, which were relatively acceptable prior to this became unfavorable (Stunkard, 1993), thus setting the stage for the widespread degradation of fatness. Following WWII, the changing sociocultural expectations of young women promoted dieting in an effort to avoid fatness. At the same time, descriptions of patients with anorexia nervosa who were binge eating and purging (primarily through self-induced vomiting) started to surface (Stunkard, 1993). This binge-purge behaviour was not confined to patients with AN, and by the 1970's bingeing and vomiting were commonly reported in normal weight females and termed "bulimarexia" (Stunkard, 1993). This lead to the recognition of BN as a distinct and separate disorder in 1979 (Russell, 1979). Although some of the symptoms of 20th century BN are similar to historical accounts, the extreme concern of body weight and shape and the use of compensatory behaviours appear to be related more to present day pressures to be thin (Brumberg, 1985; Habermas, 1989).

In 1980, the DSM-III (APA, 1980) was published and included "bulimia" for the first time, based on the criteria presented by Wermuth et al. (1977). At that time, the main features of bulimia were binge eating, associated with loss of control, followed by self-deprecating
thoughts. However, it was not until 1987, with the advent of the DSM-III revised edition (DSM-III-R) (APA, 1987), that there was a major change in the conceptualization of bulimia. At this time, the term bulimia was replaced by "bulimia nervosa", following the case reports of bingeing and purging behaviours in the landmark paper written by Russell (1979) in which "bulimia nervosa" was described and defined. Following this, bulimia nervosa came to mean binge eating which was followed by purging (typically vomiting) and this was made distinct from anorexia nervosa.

**Current Description and Classification of Eating Disorders**

Over the past two decades, the classification and diagnosis of eating disorders have received much attention and there has been significant progress in both the understanding and treatment of such disorders (Stunkard, 1997). Currently there has been some debate as to whether eating disorders occur at the end-point of a continuum, or if they are categorically different from sub-threshold and non-disordered groups. Most literature supports the view that eating disorders and associated weight concerns and psychopathology occur along a continuum of clinical severity, and eating disorders are thought to result from extreme weight concern and dieting practices (Lowe et al., 1996; Cotrufo et al., 1998; Hay and Fairburn, 1998; Stice et al., 1998). The clinical eating disorders which would occupy the end-point of the continuum are diagnosed using the current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) of the American Psychiatric Association (APA, 1994), and includes two specific diagnoses for eating disorders, anorexia nervosa and bulimia nervosa. In addition, a new, non-specific diagnosis, eating disorder-not otherwise specified was included to account for disorders which do not meet the specific criteria for AN or BN, but which are of clinical severity.
TABLE 1.1

DSM-IV Eating Disorders

Anorexia Nervosa

A. Refusal to maintain body weight at or above a minimally normal weight for age and height.

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one's body weight or shape is experienced; undue influence of body weight and shape on self evaluation, or denial of the seriousness of the current body weight.

D. In postmenarchal females, amenorrhea (i.e. absence of at least 3 consecutive menstrual cycles).

Bulimia Nervosa

A. Recurrent episodes of binge eating.

B. Recurrent inappropriate compensatory behaviour to prevent weight gain, such as self-induced vomiting, misuse of insulin, laxatives, diuretics, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.
TABLE 1.1 (Cont.)

DSM-IV Eating Disorders

**Eating Disorders-Not Otherwise Specified**

1) For females, all of the criteria for AN are met except that the individual has regular menses.

2) All criteria for AN except that, despite significant weight loss, the individual's current weight is in the normal range.

3) All the criteria for BN are met except that the binge eating and inappropriate compensatory behaviours occur at a frequency of less than twice a week or for a duration of less than 3 months.

4) The regular use of inappropriate compensatory behaviour by an individual of normal weight after eating small amounts of food.

5) Binge Eating Disorder: recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviours characteristic of bulimia nervosa.

Anorexia Nervosa (Table 1.1)

Anorexia nervosa is characterized by the refusal to maintain a minimally acceptable body weight for age and height and is associated with an intense fear of gaining weight and a disturbance in the perception of the shape and/or size of the body (APA, 1994). In order to meet diagnostic criteria for AN, an individual must weigh less than 85% of that expected for their age and height (APA, 1994). Usually this low weight is achieved through extreme reduction in food consumption, and in some individuals, by extreme exercising and purging. During childhood or early adolescence, AN may be characterized not by weight loss, but by a failure to gain weight despite growth in height (Woodside, 1995; Robin et al., 1998). Anorexia nervosa often begins with the pursuit of thinness through the avoidance of certain foods such as sweets and high-fat and high-carbohydrate containing foods (Garfinkel et al., 1988). Foods often become labeled "good" or "bad". Although weight is often lost and the initial weight goal is usually achieved, some individuals continue to be dissatisfied with their weight and make new goals to achieve even lower weights (Garfinkel et al., 1988). This preoccupation and inflexibility with food, and the steady loss of weight are often the first warning signs of the disorder.

Patients with AN are intensely afraid of any amount of weight gain and fear getting fat (APA, 1994). This fear is usually not alleviated by weight loss. In fact, weight loss may actually intensify this fear. Although patients with AN are emaciated, they often deny the seriousness of their low weight or deny that they are too thin (Garfinkel et al., 1995). Some individuals with AN will acknowledge that they are thin, but will deny that they are malnourished or in any medical danger (APA, 1994). Often they continue to feel overweight despite being significantly underweight and they often employ different methods to estimate their body size.
and weight, including excessively weighing themselves, measuring parts of their body, checking the mirror, or trying on certain clothes to detect if they fit differently (Garfinkel et al., 1995). Weight-loss is viewed as a sign of accomplishment and self-control, and weight-gain is viewed as devastating and a failure to be self-disciplined.

There appear to be significant differences between patients with AN who lose weight only through restriction (Restricting Type) and those who do so through purging and bingeing (Binge-Eating/Purging Type) (APA, 1994). The restricting type accomplishes weight loss primarily through fasting, dieting, and/or excessive exercise, and does not engage in binge eating or purging behaviours. Patients with the bulimic type of AN (which represents up to 50% of patients with AN) often have a family history of obesity, a tendency to have been overweight at some point in their lives and often are not as emaciated as those with the restricting type of AN (Yager et al., 1993). These patients regularly engage in binge eating and/or purging (APA, 1994). Most patients with AN who binge will also purge by vomiting, ingesting large quantities of laxatives, enemas, or in those with type 1 diabetes, by omitting insulin (APA, 1994). However, some patients who do not engage in binge eating nevertheless purge even after consuming small amounts of food (Yager et al., 1993).

Patients with either type of AN may exercise for hours each day (Yates, 1991), and may have obsessive-compulsive behaviours, and bizarre food rituals and preferences. Those with the bulimic type of AN also have higher rates of impulse-control problems, substance abuse, and depression (although major depressive disorder is common in all eating disorder patients) (APA, 1994).
Physiologically, the weight loss associated with AN results in amenorrhea in postmenarchal females and can delay menarche in premenarchal females (Yager et al., 1993; Robin et al., 1998). In addition, patients with AN are at risk of developing several medical complications including lowered heart rates and blood pressure, pathological fractures (Rigotti et al., 1991), impaired cardiac functioning (Schocken et al., 1989), electrolyte imbalances and infertility (Stewart et al., 1990).

**Bulimia Nervosa (Table 1.1)**

The essential features of bulimia nervosa are recurrent episodes of binge eating and inappropriate compensatory behaviours, and excessive concern with body weight and shape (APA, 1994). However, unlike patients with the binge/purge type of AN, these individuals are at a minimally-appropriate weight for their height and age. BN is more common than AN (Whitaker et al., 1990; Garfinkel et al., 1995; Garfinkel et al., 1996).

The definition of a binge has changed over the successive revisions of the DSM and still occupies a considerable amount of attention in terms of both qualitative and quantative qualities (Garfinkel et al., 1995). The DSM-IV has specified a bulimic binge to be characterized by the ingestion of a very large amount of food in a discrete amount of time, and this must be associated with feelings of loss of control (APA, 1994). This is differentiated from subjective or objective overeating in terms of the amount ingested and in terms of the associated features. Although the DSM does not define what exactly a "large" amount of food is, it does specify that it must be more than most people would eat under similar circumstances and is partly a function of the context in which the eating occurred (Fairburn and Wilson, 1993). Objective binges, on average, consist of over 4000 kcal (Walsh et al., 1989) and therefore do not represent
merely eating more than one would normally eat (e.g., second helpings at dinner, or eating a forbidden food). Binge eating often continues until the individual feels painfully full. Binges usually occur in a very short period of time (under 2 hours) and the individual feels out of control and unable to stop (Fairburn and Wilson, 1993). This differs from compulsive overeating in which a person may consume large amounts of food over long periods of time, without eating rapidly or feeling out of control (Pincus and First, 1999). Patients with BN often describe binges occurring during a frenzied state, and some describe feelings of dissociation during and/or after a binge (Pincus and First, 1999). The types of food consumed during a binge can vary, but typically they include sweets and high-calorie foods. In this regard, binges are characterized more by an abnormality in the amount of food eaten and the experience of eating rather than the consumption of specific nutrients, such as carbohydrates (Pincus and First, 1999).

Patients with BN may employ several different methods to compensate for binges. Although vomiting has been reported in up to 90% of patients with BN (APA, 1994), community studies have found it to be present in only 30% of individuals with BN (Garfinkel et al., 1996). It appears that the most common forms of compensatory behaviour are exercise, and/or fasting. Exercise is considered to be excessive if it interferes with other important activities, if it occurs at inappropriate places or times, or if a person continues to exercise despite an injury or illness (APA, 1994). Less common methods of compensatory behaviours are laxative and diuretic misuse, which have been reported in up to 30% of patients with BN (APA, 1994). In patients with type 1 diabetes mellitus, misuse of insulin to reduce the metabolism of food following a binge also appears to be a common purging behaviour (Rodin et al., 1986; Fairburn et al., 1991; Rydall et al 1997). The DSM-IV (APA, 1994) distinguishes subtypes of BN based on the
purging methods utilized. Those who regularly engage in self-induced vomiting or the misuse of laxatives, diuretics, or other medications (e.g., insulin) fall into the "purging type" of BN. Individuals with BN who engage in fasting or excessive exercise, but who do not regularly engage in self-induced vomiting or the misuse of laxatives, diuretics, or other medications are considered to have the "nonpurging" subtype of BN.

Similar to patients with AN, individuals with BN may also fear gaining weight, have a strong desire to lose weight, and have a high dissatisfaction with their bodies. In addition, such individuals place an excessive emphasis on their body weight and shape, and these factors become the most important determinants of self-esteem (ADA, 1994). Patients with BN are usually of normal weight, but before the onset of BN, they are likely to be heavier compared to their peers (APA, 1994). In addition, there is increased incidence of mood disorders, personality disorders, anxiety disorders, and depressive symptoms among patients with BN, and for most individuals, the onset of these disorders antedated the onset of BN (Gartner et al 1989; Yager et al., 1989; APA, 1994; Woodside, 1995). Some studies have reported one-third to one-half of patients with BN also report substance abuse and dependence (Wilson, 1992; Yager et al., 1993). However, controlled community sample studies have indicated that substance abuse is not significantly increased in patients with eating disorders (Welch and Fairburn, 1996).

The medical consequences of binge eating and purging include nutritional deficiencies, fluid and electrolyte abnormalities, metabolic acidosis and alkalosis, permanent loss of dental enamel, menstrual irregularities, cardiac arrhythmias, esophageal tears and gastric rupture (Yager et al., 1993).
**Eating Disorders-Not Otherwise Specified (Table 1.1)**

In addition to AN and BN, the DSM-IV has included a category of eating disorders called *eating disorder-not otherwise specified* (ED-NOS) (APA, 1994). This category has been designed to include individuals who have an eating disorder which is of clinical severity but which does not meet specific criteria for AN or BN (Fairburn and Wilson, 1993; Shisslak et al., 1995). Unfortunately, this category often is overlooked although clinical experience has shown that a large proportion of patients belong in it (Clinton and Grant, 1992; Mitray, 1992; Beumont et al., 1995; Shisslak et al., 1995). Adolescent females with ED-NOS are at increased risk for serious health concerns such as pubertal delay, growth retardation, and impairment of bone-mineral acquisition (Bachrach et al., 1990). Examples of individuals who would meet criteria for ED-NOS would include those for whom: 1) all of the criteria of BN are met, except binge-eating or recurrent compensatory behaviours are occurring less frequently than the specified number of twice per week over a 3 month period; 2) the presence of inappropriate compensatory behaviours occurring in the absence of binge eating; or 3) regular binge-eating episodes without the regular use of compensatory behaviours (i.e., Binge Eating Disorder).

**Sub-threshold Eating Disorders**

Sub-threshold eating disorders are milder eating disturbances with a lower frequency and/or severity of symptoms than specified in the DSM-IV. Such disorders would therefore occupy a position closer to the middle of the eating disorder spectrum (Button and Whitehouse, 1981; Stein et al., 1997; Franko and Omori, 1999). Sub-threshold disorders have been widely defined by different researchers, and definitions have ranged from disorders which meet two of the diagnostic criteria for AN or BN, to girls who score above the recommended cut-off on the Eating Attitudes Test (Button and Whitehouse, 1981; Kriepe et al., 1989; Meluc and Baerheim,
previous research has suggested that a large proportion of dieting and disordered eating and weight-related behaviors in children and adolescents do not meet criteria for AN, BN, or ED-NOS (Button and Whitehouse, 1981; Woodside, 1995; Stein et al., 1997; Robin et al., 1998), but are nevertheless associated with significant health risks and with the risk of subsequent progression to full-syndrome eating disorders (Davies and Furnham, 1986; Childress et al., 1993; Robin et al., 1998). In young women with diabetes, sub-threshold disorders have also been associated with impaired metabolic control and an increased risk of diabetes-related medical complications (Wing et al., 1986; Affenito et al., 1997; Rydall et al., 1997).

**Epidemiology**

Although eating disorders are relatively common among adolescent and young adult women in North America, the reported incidence of AN, BN, and ED-NOS in the general population has varied widely depending on the population studied, the response rates obtained, the measures and methods of assessment employed, and the diagnostic criteria used (Hsu, 1996).

Eating disorders have traditionally been considered to be illnesses that affect adolescents and young adults. In that regard, the mean age at onset for AN has been reported to be 17 years, with bimodal peaks of risk at 14 and 18 years (Hsu, 1996). However, there has been a widening of these peaks since the 1960's, and the prevalence of AN has increased in women between the ages of 20-30 years, although it has remained relatively stable for adolescents (Kendler et al., 1991; Pawluch and Gorey, 1998;). In a recent review of eating disorders, AN was reported to occur in approximately 0.5% of young women in Western cultures (Hsu, 1996). Although the highest risk for BN was previously thought to be between 17-25 years of age
(Woodside, 1992), recently BN has become more frequent in the adolescent population (Kendler et al., 1991; Garfinkel et al., 1995). *Bulimia nervosa* is more common than AN, and has recently been reported to occur in 0.5%-2% of women in the general population (Fairburn and Belgin, 1990; Goldbloom and Garfinkel, 1993; Fairburn and Wilson, 1993; Woodside, 1995; Hsu, 1996; Dorian and Garfinkel, 1999).

The current version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (APA, 1994), defines BN as a more homogeneous and uniformly severe disorder, compared to that defined in the earlier DSM-III (APA, 1980). One of the most significant differences between the DSM-III and the current DSM-IV with regard to the diagnosis of BN is that the DSM-III did not require a minimum frequency for binge eating to occur. In both the DSM-III-R (APA, 1987) and the DSM-IV, binge eating is required to occur a minimum of 2 times per week over the past 3 month period to meet criteria for BN. The more rigorous criteria of the DSM-III-R, compared to the DSM-III, led to a 10-fold reduction in the reported prevalence of BN (Ben-Tovim, 1988). The introduction of the DSM-IV led to two further changes in the criteria for BN. While neither the DSM-III nor DSM-III-R specifies a minimum frequency for purging behaviours, the DSM-IV requires a minimum of 2 purges per week over the past 3 months for the diagnosis of BN. In addition, the earlier versions of the DSM did not include the misuse of medications, such as insulin, for the purpose of weight loss as an inappropriate compensatory behaviour. Consequently, the use of the DSM III and the DSM-III-R potentially may have led to a possible underestimation of the prevalence of eating disorders in special populations such as in those with type 1 diabetes. Intentional misuse of medication is now recognized in the DSM-IV criteria as a form of purging behaviour.
Due to its relatively new introduction in the DSM-IV and its somewhat ambiguous criteria, there are very little data on the prevalence of ED-NOS in the general population. In studies where ED-NOS has been operationalized, it has been reported in 1.3-5% of subjects (King and Mazey, 1989; Patton et al., 1990; Fairburn et al., 1991; Peveler et al., 1992; Garfinkel et al., 1995). The prevalence of eating disorders in children and young adolescents appears to be lower but no methodologically sound study has yet been conducted (Lask and Bryant-Waugh, 1993). Unfortunately, the DSM-IV criteria for AN and BN may not be sensitive to the special developmental features which characterize child and adolescent eating disorders (Robin et al., 1998). Strict application of these criteria could potentially overlook significantly disordered eating (Beumont et al., 1994; Sullivan et al., 1998). Many children and young adolescents who present with severely disordered eating will fail to meet DSM-IV criteria for AN or BN, but will fall within the ED-NOS category (Robin et al., 1998).

The most common form of eating disturbance, which can be viewed as occupying a large area on the continuum of eating disorders, are sub-threshold variants of eating disorders. Approximately one-third of young women in the general population report engaging in binge eating as well as strict dieting to lose weight (Fairburn and Beglin, 1990). Sub-threshold eating disorders have been reported in 5-30% of young women in the general population, depending on the criteria applied (Garfinkel and Garner, 1982; Patton, 1988; Patton et al., 1990; Goldbloom and Garfinkel, 1993; Shisslak et al., 1995; Stein et al., 1997; Franko and Omori, 1999).

The prevalence of eating disorders in males is about one-tenth of that in females (Hsu, 1996). Further, eating disorders appear to be far more prevalent in industrialized societies including
Canada, United States, Europe, Australia, New Zealand, Japan, and South Africa, where there is ample access to food and where attractiveness is linked to thinness (APA, 1994). There has been little systematic research looking at the prevalence of eating disorders in other cultures. However, there is a growing body of literature that supports the view that there are comparable levels of eating disorders in Western-oriented countries and they are increasing in Third World developing countries (Pate et al., 1992). Examinations of eating disorders in immigrant populations and ethnic minority groups within the same country have led to varying rates. In one recent study looking at eating disorders in black, Asian, Hispanic, and white women, the authors reported similar levels of weight concerns and weight control behaviours in these groups (Le Grange et al., 1998).

**Course and Outcome**

The course and outcome for eating disorders are variable. However, there is general agreement that such disorders can become chronic, and may not remit spontaneously (Hsu and Holder, 1986; Russell, 1991). Additionally, there appears to be significant mortality and morbidity associated with severe eating disorders (Crisp et al. 1992; Woodside, 1995). For patients with severe AN, hospitalization is often required to restore weight and to treat electrolyte imbalances (Yager et al., 1993). Some individuals with anorexia recover fully following a single episode, while others exhibit weight gain which is followed by relapse, and others experience a chronically deteriorating course of the illness over many years. Long-term mortality from AN is higher than BN and has been reported in about 5-9% of cases within the first 5 to 8 years of the disorder (Garfinkel and Garner, 1982; Hsu, 1988); this increases to 13-20% after 20 years (Woodside, 1995). Rates of recovery vary from 32% (Russell, 1991) to 71% (Theander, 1985). In clinic samples of patients with BN, disturbed eating behaviour appears to persist for several
years and the course may be chronic or intermittent. The short and long-term outcome of BN has consistently shown relapse rates in the 30-50% range (Hsu and Holder, 1986; Woodside, 1995). Mortality has been reported in 5-20% of all patients with BN (Crisp et al., 1992), and in up to 6% of adolescent patients with BN (Steinhausen and Seidel, 1993).

Etiology

The research and theory surrounding the development and maintenance of eating disorders is a subject of much speculation with little consensus. No etiological factor postulated thus far is sufficient, on its own, to explain the development of eating disorders or to account for the variation found in those who develop eating disorders. Most likely, the etiology of eating disorders is multi-factorial in origin with developmental, cultural, and biological factors all playing a role (Garfinkel and Garner, 1982; Slade, 1982; Hsu, 1990; Tobin et al., 1991; Polivy and Herman, 1993; Streigel-Moore, 1993; Cooper, 1995; Woodside, 1995; Dorian and Garfinkel, 1999).

Whether or not a person will develop an eating disorder may depend on the presence of circumstances which activate vulnerabilities to specific factors which are a risk or which are protective (Cooper, 1995). In this regard, an individual must be exposed to certain predisposing cultural, familial, and biological factors prior to the onset of an eating disorder. These factors increase the likelihood that a person will diet and develop an eating disorder. Dieting is almost always present prior to the onset of an eating disorder (Johnson et al., 1984; Streigel-Moore et al., 1986; Shisslak et al., 1987; Herman and Polivy, 1988a, 1988b; Yates, 1989; Rosen et al., 1990a; Polivy and Herman, 1995; Polivy, 1996; Hsu, 1997). However, whether dieting in vulnerable individuals acts as a precipitating factor or whether it is
secondary to the psychological disturbance, and whether events such as stress, physical changes, negative comments or other events are necessary to trigger an eating disorder in already susceptible individuals, is unclear. It seems likely that individuals with certain predisposing vulnerabilities who begin to diet may develop an eating disorder, when there are other precipitating events. Further, certain physiological and psychological aspects of an eating disorder may perpetuate the disorder, forming a system that is highly self-maintaining (Slade, 1982), while other factors may be protective, resulting in a more transient disorder. Previous research has implicated social factors, individual factors, biological factors, and familial factors in the development of eating disorders, and these will be presented (see Figure 1.1, The Development and Maintenance of Eating Disorders).
FIGURE 1.1 Development and Maintenance of Eating Disorders

Cultural, Individual
Familial, Biological
Factors

Drive for Thinness,
Body Dissatisfaction,
Affect Dysregulation,
Impaired Self-Esteem

Dietary Restraint

Purging

Binge-Eating

Episodic Dieting
Environmental Risk Factors

Researchers have placed a particular significance on the role of environmental variables in the development of eating disorders. In fact, the role of sociocultural factors in the development of dieting and eating disorders is now generally accepted (Garner and Garfinkel, 1980; Garner et al., 1983; Dolan and Gitzinger, 1994).

i) Cultural Ideal of Thinness

Throughout history, the cultural ideals of beauty have changed and through each era women have conformed to these ideals by altering their bodies (Ehrenreich and English, 1978; Banner, 1983). An extreme example of such a cultural ideal of beauty was the binding of women's feet during the 12th and 13th centuries in Japan to prevent normal growth and make them appear very small. In this culture, small feet were thought to reflect beauty and sexuality and represented high social status, since this would not allow for manual labor (Wilfley and Rodin, 1995). The cultural pressures were so great that women bound their feet even though they were then no longer able to walk and were dependent on being carried around. In the 19th century, women wore tightly bound corsets in order to appear to have very small waists, with the incidental effects of indigestion, constipation and weakness. In the early 20th century, flat chests became fashionable and consequently, women would painstakingly bind their breasts to flatten their chests (Wilfley and Rodin, 1995).

Since at least 1960, the thin female body form has represented the ideal of beauty, and this has been widely distributed due to the advent of mass media (Rodin et al 1985; Streigel-Moore et al 1986; Becker and Hamburg, 1996). It is not clear why Western society has developed such an idealized view of thinness. Historically, when there were more differentiated social classes,
thinness might have indicated a particular heredity and the availability of high quality food which, in turn, indicated higher social status (Streigel-Moore et al., 1986). More recently, however, body weight and shape have come to represent achievement through control and are often associated with connotations of success, self-control and acceptance (Wilfley and Rodin, 1995). Wooley and Wooley (1980) observed that controlling one's weight can symbolize control in other areas of one's life, and weight loss has become valued as a visible accomplishment. Further, thinness and physical attractiveness have increasingly been equated with success and competence (Garner et al., 1983; Brownmiller, 1984), and concern with beauty and thinness is present in almost every social context (Pierce, 1990; Purcell and Stewart, 1990). Though women are now appearing in a wider variety of roles in the mass media, they are still most often represented by thin, physically attractive, young individuals (Davis, 1990; Signorielli, 1990, 1993). The mass media and industry have been relentless in their portrayal of the "ideal" woman and the "right" look, and women's looks are constantly being fabricated and their bodies objectified. Images of the "ideal woman" are shown, along with the promise that anyone can achieve the right look if they buy enough and that this, in turn, will make them happier, more successful, and result in a better life (Kilbourne, 1987). In a recently completed study by Pinhas et al. (1999), women who watched images of female models reported higher levels of depression and negative affect compared to controls. Taylor et al. (1998) showed that the wish to look like women on TV or in magazines significantly predicted weight concerns in adolescent girls. In this regard, increasing numbers of girls who consider themselves to be overweight and are dieting, appear to accept readily the societal messages about the importance of thinness (Streigel-Moore et al., 1986; Taylor et al., 1998).

The increased emphasis on thinness and beauty in Western culture has been associated with a higher degree of bodily preoccupation and an increased prevalence of eating disturbances.
among young women born after 1960 (Kendler et al., 1991; Garfinkel et al., 1995; Pawlish, 1998). Further, dieting behaviours and body dissatisfaction have reached almost epidemic levels (Polivy and Herman, 1985, 1987, 1993; Streigel-Moore et al., 1986; Rosen et al., 1987; Herman and Polivy, 1988a, 1988b; Polivy et al., 1990; Garfinkel et al., 1992; Woodside, 1995; Cooper and Goodyer 1997; Dorian and Garfinkel, 1999; Franko and Omori, 1999). Given that attractiveness appears to be important in achieving both professional and interpersonal success and that women seem to care more than men about the opinions of others and to seek their approval (McGuire and McGuire, 1982; Streigel-Moore et al., 1993), it is not surprising that women make weight and shape priorities in their lives (Pliner et al., 1990). This is especially true in women whose professions or sports require them to be thin, or who are under pressure to achieve. In this regard, researchers have identified groups of females who appear to be at increased risk for the development of eating disturbances. These groups include female medical students (Tordjman et al., 1994), females in boarding schools and colleges (Squire, 1983), and subcultures in which optimal weight is specified and lean body weight is rewarded. The latter include ballet dancers (Garner and Garfinkel, 1980; Abraham, 1996), models (Garner and Garfinkel, 1980), gymnasts (Rosen and Hough, 1988), skaters (Ziegler et al., 1998), and other competitive athletes (Crago et al., 1985; Davis, 1992; Davis et al., 1999).

Over the past several decades, the average weights for females less than 30 years of age have actually increased. This, in the context of the increasingly thinner "ideal", has resulted in a larger disparity between the "ideal" and the "real", and may be responsible for the high incidence of dieting among women in Western cultures (Garner and Garfinkel, 1980). Females who have higher body mass index (BMI) values compared to their peers, or who report themselves as overweight, often have increased body dissatisfaction, a higher drive for thinness
(Davis and Furham, 1986), and have been shown to be more likely to diet and to develop eating disturbances (Boskind-White and White, 1983; Fairburn and Cooper, 1983; Johnson-Sabine et al., 1988; Attie and Brooks-Gunn, 1989; Hill et al., 1989; Patton et al., 1990; Childress et al., 1993; Killen et al., 1994, 1996; Smolak and Levine, 1994; Fairburn et al., 1997; Strong and Huon, 1998). Adolescents who are obese or have higher than average BMI are often subjected to more teasing (Thompson et al., 1995), which may result in lowered self-esteem, body dissatisfaction, an increased drive for thinness, and initiation of weight loss practices. Further, females whose self-worth is determined by their appearance and who have higher BMI may be sensitized to this teasing and to other past humiliations which they believe were caused by their weight. These individuals may resort to dieting in an effort to avoid future embarrassment.

Polivy and Herman have suggested that familial and biological factors may combine to increase one's risk for impaired affect, self-esteem and self-efficacy. When social pressures to be thin are then added, these vulnerable girls often resort to dietary restraint and thus increase their risk for the development of an eating disorder (Polivy and Herman, 1993).

ii) The Socialization and Changing Role of Women

From early childhood, girls are taught by their families, friends, schools, books and the media to believe that physical appearance is very important (Streigel-Moore et al., 1986). Further, they are taught that being physically attractive is related to pleasing and serving others and will secure their love. There is evidence that girls internalize these messages about the importance of physical attractiveness and are more concerned about looking attractive than are boys (Douvan and Adelson, 1966). The development of self-concept in girls is based more on interpersonal and external forces than boys, and girls tend to refer to the views of others to
describe themselves (McGuire and McGuire, 1982). For young girls, even those in grade
school, weight and shape appear to be critical in the development of body image and self-
concept. Pre-pubertal girls report beginning to worry about becoming fat as early as 9 years of
age (Wardle and Marsland, 1990; Streigel-Moore et al., 1992). Negative self-perception has
been demonstrated to be a major contributor to weight concerns and dieting awareness in girls
(Hill and Pallin, 1998). In one large study of girls in grades 4-8, it was demonstrated that
weight concerns in the younger girls (grades 4-5) were most strongly related to peer pressure
and the desire to look like models and women in the media. In the older girls, low self-
confidence, higher BMI, and being teased about weight were also were important risk factors
for weight concerns (Taylor et al., 1998).

Adolescence presents young girls with physical and psychological changes that can amplify
crmons regarding weight and shape. This time also seems to be critical in the development of
many eating patterns that remain into adulthood. Certain amounts of unhealthy eating are
expected, and these appear to increase during adolescence (Wood et al., 1992). However, the
transition to middle school, which often coincides with the onset of puberty, has been noted to
be a time when many girls develop problematic eating behaviours (Attie and Brooks-Gunn,
1992; Levine and Smolak, 1992). Further, body dissatisfaction increases progressively through
the prepubertal years (Thelen et al., 1992), and by adolescence many normal weight girls report
being dissatisfied with their bodies and weight and want to be thinner (Garner et al., 1980;
Wardle and Beales, 1986; The McCreary Center Society, 1993; Killen et al., 1996). Body
dissatisfaction is a significant predictor of eating disorders (Attie and Brooks-Gunn, 1989; Leon
et al., 1993; Cattarin and Thompson, 1994). Physically, puberty results in the formation of fat
tissue and weight gain (Marino and King, 1980; Tanner, 1978). As body fat increases, girls feel
even farther away from the physical pre-pubertal thin “ideal” of beauty, resulting in increased body dissatisfaction, feelings of fatness, and lowered self-esteem (Simmonds and Rosenberg, 1975; Dornbusch et al., 1984; Gralen et al., 1990). In response to feelings of insecurity, and to avoid negative evaluation from others, adolescent girls become increasingly sensitive to and compliant with sociocultural mandates and the opinions of others, i.e., self-esteem becomes regulated externally.

In Canada, 50% of females report feeling “too fat” by the age of 18 years, even though 80% are actually at a normal weight (The McCreary Centre Society, 1993), and a large majority have dieted at least once in the past (Davies and Furnham, 1986). By middle school, 30-55% of girls report having dieted at some time (Maloney et al., 1989; Koff and Rierdan, 1990; Moreno and Thelen, 1995). The positive comments and attention which girls often receive from an initial weight loss resulting from a diet may reinforce continued dieting in those whose self-esteem is regulated externally and in whom there is little support of autonomy (Huon and Strong, 1998).

At the same time as the physical changes of adolescence are occurring, adolescents are presented with the developmental challenge of establishing their independence and autonomy. For female adolescents, this task of separation and individuation may be particularly difficult since conflicting social attitudes towards independence in young women can cause girls to become confused and insecure regarding their roles in society. Dorian and Garfinkel (1999) have suggested that social self-efficacy and effective interpersonal skills may be important in an adolescent's ability to deal with multiple pressures from peers, family, and the mass media.

The inability to relate to the world in an autonomous and independent manner, which has been associated with lower self-esteem, may cause some girls to focus on the body to achieve a sense of personal value (Dorian and Garfinkel, 1999). However, this, in turn, may increase the risk
for the development of an eating disorder (Wagner et al., 1987; Kenny and Hart 1992). Further, because dieting is viewed as a sign of maturity (Steele, 1980), weight loss efforts may reflect a girls' desire to demonstrate that she is growing up and becoming independent. Alternatively, dieting may be an effort to avoid the developmental challenges of adolescence and losing weight may represent an effort to delay pubertal changes that signal maturity and becoming autonomous (Streigel-Moore et al., 1986).

At the same time as dieting for weight loss and the drive for thinness have emerged in Western cultures, women's roles within our society have also changed dramatically. In addition to being expected to achieve the "ideal" body, women are now also expected to be successful not only as mothers and wives, but also as professionals. Unfortunately, these changing expectations have been superimposed upon, and not replaced by, traditional values. Consequently, this trend has not necessarily been associated with increased autonomy in interpersonal relationships and women continue to face ongoing discrimination, abuse, and substantial power imbalances (Dorian and Garfinkel, 1999). Although, women now feel more capable and empowered to achieve professionally, there remains a corresponding sense that they need to "have it all" and to live up to all the demands of society (Levine and Smolak, 1992). Consequently, being professionally successful does not relieve a woman of the expectations to be beautiful, nurturing, and a caregiver. The conflicting roles of today's "superwoman" can lead women to feel ineffective when they do not meet all the expectations; this has been linked to the risk of eating disorders (Streigel-Moore et al., 1986; Levine and Smolak, 1992). In this regard, girls who most internalize the "superwoman" notion, appear to be at a higher risk to develop an eating problem, compared to those girls who set more realistic goals (Steiner-Adair, 1986). The stress to achieve in all domains may cause some females to feel that their lives are out-of-
control and cause them to focus on their body weight and shape, since it is something that they can control. Focusing on weight and shape often becomes a major pre-occupation, which may affect one's ability to meet the challenges of life.

iii) Dietary Restraint

There is a well-established literature demonstrating an association between restrained eating, dieting, and overeating (Abraham and Beumont, 1982; Garner et al., 1985; Polivy and Herman, 1985, 1987, 1993, 1995; Herman and Polivy, 1988; Pyle et al., 1990; Rosen et al., 1990; Schlundt and Johnson, 1990; Polivy, 1996; Hsu, 1997). In fact, dieting is almost always a predisposing factor in the development of an eating disorder (Johnson et al., 1984; Streigel-Moore et al., 1986; Shisslak et al., 1987; Yates, 1989; Rosen et al., 1990; Hsu, 1997). In a large review conducted by Hsu (1997), dieting behaviour was found to be a major risk factor for the pathogenesis of eating disorders. Further, the prevalence of eating disorders in a given community was directly correlated with the prevalence of dieting behaviour in the community (Hsu, 1997). Although the nature of this relationship is correlational, there appear to be several biological, cognitive, and affective consequences of dieting which may increase the risk for the development of an eating disorder.

Dieters are forced to ignore hunger-driven eating and instead to follow a planned diet or to engage in dietary restraint. This often results in the disruption and dysregulation of normal caloric intake and may contribute to an insensitivity to external cues leading to possible overeating and bingeing (Garfinkel, 1974; Garfinkel et al., 1988). Further, low calorie diets have been associated with lower basal metabolic rates over time (Rauussin and Swinburn, 1992). Consequently, when calories are ingested they are more likely to be stored as fat and
result in weight gain making future weight loss even more difficult. This can contribute to recurrent weight loss and gain, popularly known as "yo-yo" dieting, and can lead to lowered self-esteem, and feelings of guilt over the failed attempts to lose weight (Wadden and Foster, 1992). It is these individuals who appear to be most vulnerable to attempt further weight loss strategies and purging behaviours thus increasing the risk of an eating disorder.

Polivy and Herman (1985) have suggested that dietary restraint may cause binge eating by altering one's reactivity to certain "forbidden" foods, making them more attractive, and/or by increasing the drive for hunger in response to caloric deprivation. In the conditioning model of binge eating suggested by Booth et al. (1990), dieters become conditioned to binge-eat in response to fattening foods. In this regard, dieters appear to be more vulnerable to loss of control after eating a "bad" food, and react with an "all-or-nothing" or "learned helplessness" response in which the person abandons all attempts to regulate food intake after eating or overeating of a forbidden food, and thereby overeats (Wilson and Fairburn, 1993).

Dieting and chronic caloric restriction have also been shown to have a destabilizing effect on mood and can lead to irritability and increased levels of anxiety and depression (Keys et al., 1950). This may lead to increased distress and lowered self-esteem. One interesting finding is that in non-dieters, distress can suppress eating whereas in dieters, it may actually increase eating (Baucom and Aiken, 1981; Wardel and Beales, 1988; Herman and Polivy, 1988a, 1988b; Heatherton et al., 1991, 1993; Polivy and Herman, 1999). Further, threats to one's ego are more likely than physical threats to increase food intake in restrained eaters (Heatherton et al., 1991), and some have postulated that this may be due to the distraction provided by eating (Herman and Polivy, 1988) or to the comfort provided by food (McKenna, 1972). However, these
speculations have not been strongly supported by empirical evidence (Polivy et al., 1994; Polivy and Herman, 1999).

Polivy and colleagues have suggested that overeating may actually be a defense strategy used when faced with a threat to the ego (Polivy et al, 1988). When a threat is perceived, the dieter will often overeat, and in an effort to preserve her emotional well being, will convince herself that the distress is due to the overeating and not the actual and original source. This was supported in a recent study by Polivy and Herman (1999) in which restrained and unrestrained college students were asked to complete a cognitive task and then told that they either failed or passed. Following this, subjects were then asked to complete a taste test for ice cream and were either given 3 spoonfuls of ice cream or as much ice cream as they wanted. Restrained eaters, who were given the "fail" label and who were allowed to eat as much ice cream as they wanted, attributed more stress to the ice cream and later reported that they felt helpless and that eating ice cream distracted them from the distress caused by the cognitive failure. In the process, they reattributed their distress from the real source (the cognitive failure) to the eating (Polivy and Herman, 1999).

The binge-purge cycle that results from dietary deprivation is often self-perpetuating and may also maintain and/or perpetuate eating disorders. Purging may reinforce the cycle because it allows one to eat and yet feel as though one has lost weight. It may also be a way to express emotion in those who cannot tolerate direct expression, since purging may relieve or displace negative affect (Abraham and Beumont, 1982; Fairburn and Cooper, 1987; Herman and Polivy, 1988; Schlundt and Johnson, 1990). Unfortunately, shame, guilt, and depression (Abraham and Beumont, 1982; Elmore and DeCastro, 1990; Schlundt and Johnson, 1990) quickly replace any
relief that may be experienced. The feeling of being out-of-control and the worry about gaining weight leads to dieting, thus setting up the next binge (Schlundt and Johnson, 1990).

Heatherton and Polivy have suggested a "spiral" model of restraint and bingeing, in which high drive for thinness and low self-esteem may cause an individual to diet. However, dieting is usually not successful and often results in episodes of overeating, and even weight gain. Dietary failures can lower self-esteem and result in negative self-affect that may increase restraint and result in further failures (Polivy et al., 1988; Heatherton and Polivy, 1991).

Although the cultural pressures on women to be thin, to diet and to be successful may indirectly be linked to eating disorders, cultural influences alone are not sufficient to explain the exact mechanism for the development of eating disorders. Further, though dieters have eight times the risk for developing an eating disorder compared to non-dieters, we know that dieting does not lead to eating disorders in the majority of girls (Patton et al., 1990). Consequently, environmental and other triggering factors alone, are insufficient etiological explanations for the development of an eating disorder. Instead, it seems likely that early vulnerabilities, developmental or genetic, interact with these environmental factors to place certain individuals at an increased risk to diet in an effort to improve self-esteem, achieve a sense of control and to appear more acceptable. In the absence of these environmental triggers, the individual vulnerabilities might have remained latent or manifested themselves in other forms (Dorian and Garfinkel, 1999).
Individual and Developmental Risk Factors

Although all females are subjected to pressures to be thin, and may diet, eating disorders appear to develop from an interaction between both the societal issues addressed above, with individual and familial factors.

Bruch (1979) suggested that eating disorders are not simply disorders of eating but are also the result of how one perceives and expresses oneself in the world. Bemporad et al. (1992) have argued that contemporary cultural ideals and values which reinforce attractiveness and the internalization of gender role messages may shape the course of eating disorders in women, but that all patients with an eating disorder have a history of dysfunctional early relationships which results in impaired psychological functioning. Further, they suggest that these individuals would have developed a disorder in any social climate though the presentation of the illness may be different (Bemporad et al., 1992). However, whether early developmental and individual factors predispose individuals to the development of an eating disorder remains an area of much speculation. Without prospective longitudinal studies of girls before the age of risk for the onset of eating disorders, it is difficult to know which features are predisposing and which are the result of the eating disorder.

Women who have difficulty separating from their families and achieving autonomy are not able to develop a sense of mastery over their lives (Bruch, 1979; Garfinkel and Garner, 1982). As a result, they often feel helpless and may use dieting to achieve a sense of personal control (Garfinkel and Garner, 1982). Further, a relative deficit in relating to the world in an autonomous manner may be associated with the later development of an eating disorder (Garfinkel and Garner, 1982; Bruch, 1973). Deficits in social functioning have been linked to
low self-worth, which can heighten the need to focus on appearance to gain feelings of personal self-worth (Frederick and Crow, 1996). When self-worth is not internally derived but is instead tied to pleasing others, self-esteem becomes dependent on external standards of performance and appearance. This has been supported by findings in which women with eating disorders score higher on measures of interpersonal sensitivity (Streigel-Moore, 1994), and have been shown to have an increased need for approval from others (Dunn and Ondercin, 1981; Katzman and Wolchik, 1984).

Hill and Pallin (1998) recently demonstrated that negative self-perception, including global self-worth and low social acceptance, was the major determinant of weight concern in 8-year-old girls. In this regard, girls who are not able to experience their "true self" become hypervigilant about how they appear to others and increase their focus on physical appearance (Solomon and Schopfler, 1982). Further, increased awareness of deficits may intensify appearance-related concerns and may increase social anxiety. Self-consciousness has been associated with greater self-criticism (Fenigstein, 1979), social withdrawal (Carver et al., 1979), lowered self-esteem and disordered eating (Streigel-Moore, 1993; Button et al., 1997). As discussed above, having low self-esteem, and a negative self-perception may also make an individual more vulnerable to the pressures to be thin and thus increase their desire to lose weight and diet (Herman and Polivy, 1988). This has been supported by the finding that girls with lower self-esteem have been found to have more body dissatisfaction and a higher drive for thinness (Attie and Brooks-Gunn, 1989). In addition, dieters and patients with eating disorders have been reported to have lower than average levels of self-esteem (Garfinkel and Garner, 1982; Herman and Polivy, 1988; Polivy et al., 1988).
Perceived social stress, in school life, family life or financial matters, has also been implicated/associated with an increase in dieting (Patton et al., 1990; Rosen et al., 1990). In one prospective epidemiological study of 1010 school girls, the only strong predictor of abnormal eating attitudes and behaviours was perceived stress in school and social life (Johnson-Sabrine et al., 1988). In a study conducted by Streigel-Moore et al. (1989), perceived stress was associated with worsened eating behaviours in a group of female college students. Stress and negative affect have been implicated as developmental precursors to eating disorders, interacting with other risk factors (Polivy, 1976; Davis et al., 1988; Elmore and DeCastro, 1990; Schotte et al., 1990; Heatherton and Baumeister, 1991; Heatherton et al., 1991; Polivy and Herman, 1993; Schmidt et al., 1993). These findings appear to support the additive stress hypothesis, which predicts that those females who encounter challenges and stresses simultaneously are at an increased risk for the development of an eating disorder (Levine and Smolak, 1992). Strober (1984) found that patients who developed BN, reported significantly more life events occurring 8 months before the onset of their eating disorder, compared to girls who did not have an eating disorder. Welch and colleagues (1997) also found that subjects with BN reported significantly more life events (especially the disruption of family or social relationships or threats to physical safety) occurring 1 year prior to the onset of BN compared to controls. There have also been a number of studies that have demonstrated that the more life changes a girl encounters within a short period of time, the more distressed she will become (Petersen and Hamburg, 1986; Simmonds and Blyth., 1987; Compas et al., 1989; Welch et al., 1997). Levine and Smolak (1992), showed that disordered eating attitudes, based on the EAT-26, increased in a non-clinical sample of girls when dating coincided with the onset of menarche, whereas one event alone did not result in elevated scores.
Dieting in middle school has been associated with menarche and dating (Gralen et al., 1990; Smolak et al., 1993). Further, the specific timing of puberty has been implicated in the development of eating disorders. The onset of puberty can often cause young women to focus more on body shape and weight and they may become particularly sensitive to other's evaluations of their appearance (Calam and Waller, 1998). Girls who develop secondary sexual characteristics earlier are vulnerable to a wide range of adjustment difficulties in adolescence (Stattin and Magnusson, 1990). Early-maturing females are less satisfied with their bodies and have more eating disturbances than later-maturing girls (Crisp, 1970; Brooks-Gunn and Warren, 1985; Killen et al., 1994; Fairburn et al., 1997). This may be due to the fact that early-maturing girls weigh more than on-time and late-maturing peers, and this difference remains after maturation is completed (Simmonds et al., 1983). In addition, early-developing girls are viewed as more mature, which may require them to cope with experiences that they are not developmentally ready to deal with, thus increasing stress and anxiety (Streigel-Moore, 1993). Further, early maturing girls begin dating earlier than later-maturing girls (Gargiulo et al., 1987). In this regard, girls who date during early adolescence report more history of binge eating (Dykens and Gerrard, 1986; Gralen et al., 1990). This may be due to an increased focus on attractiveness in girls who are dating (Gralen et al., 1990). Girls experience dating as more stressful than boys (Bush and Simmonds, 1987). They are also more likely to attribute failure of a relationship to a deficiency in themselves (Kaplan, 1986), which they may be tempted to correct by dieting. One of the biggest changes observed in adolescent behaviour over the past few decades has been in sexual activity. Adolescent girls have become sexually active at much younger ages than in earlier decades (Brooks-Gunn and Furstenberg, 1989). This is not necessarily a positive experience for them, since 61% of sexually active teens indicate that they initiated sexual relations in response to peer pressure (Udry and Billy, 1987).
Finally, there has been much speculation about the effect of sexual or physical abuse on psychological development, and whether or not such abuse is a risk factor for the development of an eating disorder (Pope and Hudson, 1992; Conners and Morse, 1993; Fairburn et al., 1997; deGroot and Rodin, 1999). To date, the research regarding childhood sexual and physical abuse has been inconclusive and controversial. The reported rates of sexual abuse in eating disorders have ranged from 20-50% (Bulik et al., 1989; Garfinkel et al., 1995; Dansky et al., 1997). Although the reported rates are generally elevated compared to that found in the general population, these rates are similar to those found in psychiatric populations (Stuart et al., 1990; Pope and Hudson, 1992; Palmer and Oppenheimer, 1992; Welch and Fairburn, 1994). Consequently, it can only be concluded that sexual abuse is associated with a number of different psychiatric disorders, including eating disorders (Welch and Fairburn, 1994).

**Family Risk Factors**

Non-genetic family risk factors for eating disorders have been of particular interest to clinicians and researchers. Although there have been a large number of studies in which the quality of family functioning has been linked to eating disorders, most of the research has been cross-sectional and it is difficult to distinguish whether the specific patterns observed are antecedents or whether the family dysfunction is a consequence of the eating disorder (North et al., 1995). Some research has suggested that dysfunctional interactions may not be any more common in females with eating disorders, and that they are found at the same rate in families of depressed women (Vandereycken et al., 1989). Further, the studies are often difficult to compare because of differences in the assessment methods used and other methodological issues (Vandereycken, 1995).
Family interaction patterns of girls with eating disorders have been described as being enmeshed, hostile, intrusive, overprotective, and negating of the child's emotional needs (Humphrey et al., 1986; Humphrey, 1987; Kog and Vandercycken, 1989). Families of patients with bulimic symptoms tend to be less close and nurturing and more openly conflictual (Strober and Humphrey, 1987; Dare and Eisler, 1997; Fairburn et al., 1997), and families of girls with AN are viewed as having more difficulty with affective expression, communication and role performance (Minuchin et al., 1978; Garfinkel et al., 1983; Johnson and Flach, 1985; Humphrey, 1986; Dare and Eisler, 1997). In addition, mothers and fathers of girls with eating disorders have been noted to have a higher tendency to use their daughters to mirror their own needs and do not adequately validate the emotional experience of their daughters (deGroot and Rodin, 1994). The development of a child's sense of self is dependent on sufficient responsiveness and attunement from their parents (deGroot and Rodin, 1994). Consequently, parents who are not nurturing enough or who are not able to respond to the needs of their child may impair their child's sense of self. Further, the identity of girls who have not received enough support and attention and who have had little validation of their subjective experiences may become determined by external forms of approval and disapproval from both men and women (deGroot and Rodin, 1994). When associated with other risk factors, this may contribute to the development and maintenance of an eating disorder.

The role of the mother in the development of eating disorders has received the majority of the attention in family research. Again, it is important to remember that mothers are part of the larger cultural picture that is preoccupied with thinness and dieting. Girls whose mothers are more critical of weight, report higher scores on disordered eating measures compared to girls
whose mothers accept their daughters weight (Pike and Rodin, 1991). In one study of 148 female high school students, parent's direct pressure on their daughters to diet predicted dieting (Strong and Huon, 1998). In addition, mothers who exhibit their own shape and weight concerns and who diet are more likely to have pre-adolescents who diet (Hill and Franklin, 1998) and adolescents with abnormal eating behaviours (Attie and Brooks-Gunn, 1989; Pike and Rodin, 1991). It seems likely that the mother's concerns are relayed to their children and may lead to development of a disorder through modeling of their attitudes and behaviours. Recently, more attention has been given to the role of the father in the development of eating disorders. Patients with bulimia have reported having fathers who are absent due to work or divorce (Leibowitz, 1991). Fathers of females who suffer from anorexia have been reported to use their daughters to project their own feeling and to improve their own self-image (Bemporad and Ratey, 1985).

Other factors implicated in the development of eating disorders include a family history of alcoholism and affective illness (Herzog, 1982; Hudson et al., 1987; Kassett et al., 1989; Strober et al., 1990; Yager and Strober, 1995), obesity (Garfinkel et al., 1980; Strober, 1981; Fairburn and Cooper, 1984) and family magnification of cultural factors (Streigel-Moore et al., 1986). In addition, fights at meal times in early childhood have been shown to be predictive of some extreme symptoms of bulimia (Marchi and Cohen, 1990). This may suggest that certain aspects of parent-child rebellion and control issues around eating may be factors in the development of eating disorders (Marchi and Cohen, 1991). Finally, there have been a few prospective studies that have indicated that positive family relations and closeness to parents seem to be protective against the development of eating disorders (Graber et al., 1994; Swarr
and Richards, 1996). This finding supports the contention that the family does play some role in the etiology of eating disorders.

**Psychological Risk Factors**

Several epidemiological studies have attempted to document the association of a wide variety of psychological traits and psychiatric disorders with eating disorders. These have included depression and dysthymia (Fairburn and Cooper, 1982; Mitchell et al., 1985; Williamson et al., 1985; Richards et al., 1990; Halmi et al., 1991; Whitaker, 1992; Devaud et al., 1998), anxiety symptoms and disorders (Hudson et al., 1987; Laessle et al., 1987; Steere et al., 1990; Cooper and Goodyer, 1993), personality or trait disturbances (Garfinkel and Garner, 1982; Gartner et al., 1989; Yager et al., 1989; Patton et al., 1990; Zanarini et al., 1990), chemical dependencies (Hudson et al., 1987; Pyle et al., 1983; Mitchell et al., 1985; Krahn, 1991; Higuchi et al., 1993), bipolar disorder (Shisslak et al., 1991), and borderline personality disorder (Wonderlich and Mitchell, 1991).

There has been a considerable amount of research examining depression in patients with eating disorders. The rates of depression and/or dysthymia have consistently been found to be elevated, compared to the general population (Fairburn and Cooper, 1982; Mitchell et al., 1985; Williamson et al., 1985; Richards et al., 1990; Halmi et al., 1991; Whitaker, 1992; Devaud et al., 1998). However, it is unclear whether the presence of depression and/or dysthymia precedes the onset of the eating disorder or is, in fact, a consequence of it. This relationship is further complicated by the fact that the symptoms of depression and dysthymia overlap with anorexia and bulimia (Altschuler and Weiner, 1985; Streigel-Moore et al., 1986). Although further research is needed to understand the nature of this association, the findings to date suggest that
depressive symptoms commonly occur in patients with eating disorders, but are mostly a secondary consequence of the eating disorder.

A wide range of anxiety symptoms has also been reported in patients with eating disorders. Although rates of agoraphobia, simple phobias, and panic disorders in patients with eating disorders do not appear to be higher compared to non-psychiatric controls, social phobias have been reported to occur 10 times more frequently (Hudson et al., 1987; Laessle et al., 1987). However, it is unclear whether this finding is due to comorbidity or to the fact that the central features of eating disorders include fears of food and weight gain, social eating, and of being evaluated by others. In one prospective study conducted by Rosen et al. (1990), the authors found that dieting increased anxiety in participants. Further, anxiety and social phobias appear to resolve following the normalization of eating habits and body weight. These findings suggest that these anxiety symptoms may arise as a direct result of the eating disorder (Cooper, 1995).

Researchers have also been interested in the prevalence of obsessive-compulsive disorder (OCD) in patients with eating disorders (Kasuikis et al., 1986; Halmi et al., 1991; Hsu et al. 1993). Some have argued that eating disorders are actually a form of obsessive-compulsive disorder, but the evidence for this is not compelling (Hsu et al., 1993). Although there does appear to be a higher prevalence of OCD in patients with eating disorders, some have argued that the symptoms of OCD are secondary to the eating disorder. Furthermore, and the response to medications commonly used to treat OCD, such as selective serotonin reuptake inhibitors (SSRI’s), has been unsuccessful in the eating disorder population (Cooper and Goodyer, 1993).
Most of the research on personality disorders and the subsequent development of eating disorders has focused on borderline personality disorder (BPD) in patients with BN. Once again, the data are unclear and inconsistent, with rates of BPD ranging widely from 2-47% (Wonderlich and Mitchell, 1991). Some researchers argue that BPD is actually rare in patients with BN and that depression is often misdiagnosed as BPD, while others argue that symptoms of BN may mimic BPD (Swift and Wonderlich, 1988). The latter is supported by the finding that there is a decrease in BPD following short-term interventions for eating disorders, indicating that BPD is not independent of BN (Wonderlich and Mitchell, 1991).

**Biological Risk Factors**

There now appears to be a general consensus that there is a familial risk for eating disorders and that there may, in fact, be a genetic predisposition to such disorders (Hudson et al., 1987; Kassett et al., 1989; Gershon et al., 1990; Keck et al., 1990; Woodside, 1995). First-degree relatives have been shown to have higher rates of eating disorders (Strober et al., 1990), with approximately 6-10 times the rate compared to the background population (Woodside, 1995). Further, twin studies have shown concordance rates of 0.75 to 0.80 for AN and 0.45 to 0.55 for BN (Holland et al., 1984; Holland et al., 1988; Kendler et al., 1991). However, it is not clear whether genetic vulnerability operates by directly increasing the risk of AN and BN, or by predisposing individuals to obesity or certain personality types which in turn increases the risk for the development of an eating disorder. In addition, mood disorders and alcohol abuse in first degree relatives have been implicated as specific risk factors for eating disorders, especially BN, although the explanation for this association is unclear.
Researchers have also been investigating the relationship between several neurophysiologicai and endocrine abnormalities and the pathogenesis of eating disorders (Garfinkel et al., 1988; Fairburn and Beglin, 1990; Cowen et al., 1996). However, prospective studies have not been conducted and it seems likely that most of the abnormalities that have been reported are not precipitants of eating disorders but are secondary to them. Recently, serotonin activity in the brain has received a considerable amount of attention because it was observed that SSRI's are useful in treating patients with bulimia (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992). However, further research is needed to understand the role of serotonin in the onset of eating disorders.

The physiological consequences of eating disorders have also been implicated in the maintenance of eating disorders. Starvation associated with AN can perpetuate the eating disorder because it is associated with behavioural, cognitive and affective changes which depress mood, and can lower self-esteem, increase concerns about shape and weight, and result in further dietary restriction in an attempt to gain more control. Dieting-induced disruptions in brain functioning may affect appetite and dietary dysregulation. A further difficulty in patients with eating disorders is that their perception of satiety may be distorted; these patients often report bloating, nausea, and distention after eating even small amounts of food. Studies have demonstrated that delayed gastric emptying may contribute to altered feelings of satiety (Dubois et al., 1979; Russell et al., 1983). Such physical discomfort may contribute to feelings of "fatness" and may further reinforce dietary restriction and purging behaviours. Cognitive distortions, such as negative thoughts and distorted irrational thoughts about food and body shape, may also play an important role in the maintenance of and recovery from eating
disorders and this is supported by research which has demonstrated the effectiveness of a cognitive behavioural approach to treatment (Wilson and Fairburn, 1993).

Researchers have also investigated the prevalence of eating disorders in several different medical populations, in which weight, bodily preoccupation and dietary consumption are inherent aspects of the condition and/or its treatment regimen. These include cystic fibrosis, thyroid disease, and type 1 diabetes mellitus. Cystic fibrosis is a condition in which malnutrition is common, and in which there is pressure to eat. There is no evidence of an increased prevalence of eating disorders in patients with cystic fibrosis (Cowen et al., 1988; Streiner et al., 1990), suggesting that bodily preoccupation, in the absence of dietary restriction, may not be a risk factor for the development of eating disturbances. Some researchers have recently reported an increased prevalence of eating disturbances in patients with thyroid disease (Tiller et al., 1994), and have speculated on several possible mechanisms by which thyroid disease could predispose to the development of an eating disorder. These mechanisms include weight fluctuations associated with the onset of thyroid disease and its treatment, and routine weighing at clinic visits, which may sensitize patients to weight and appearance, thus increasing body dissatisfaction. Dietary restraint may then follow, triggering a cycle of further restraint, subsequent binges, and purging through overdosing with thyroxin. These studies were all small, and further research is needed in this potentially high risk group.
TYPE 1 DIABETES MELLITUS

Epidemiology and Clinical Description

There are several different types of diabetes, with the two most common being type 1 diabetes mellitus and type 2 diabetes mellitus. Type 2 diabetes accounts for approximately 85% of cases and, in these individuals, diabetes generally occurs after the age of 40 years (Bennett, 1994).

Type 1 diabetes mellitus is one of the most common chronic childhood illnesses, affecting 1 in 300 to 600 children or adolescents in North America before the age of 20 (Bennett, 1994), with no apparent sex preference. Type 1 diabetes is currently thought to be an auto-immune disorder that requires a genetic vulnerability and certain external or environmental factors for its expression (Drash, 1987; Bennett, 1994). This condition, which results when the beta cells of the pancreas no longer secrete insulin, is characterized by chronic hyperglycemia and disturbances of carbohydrate and lipid metabolism (Bennett, 1994). Insulin is necessary for the intracellular metabolism of glucose and regulation of the level of glucose in the blood. Individuals with untreated diabetes develop extreme elevations in blood glucose levels and metabolic disturbances leading to the formation of toxic ketones (diabetic ketoacidosis (DKA)). Type 1 diabetes is typically associated with the relatively abrupt onset of hyperglycemia, resulting in increased urination and thirst, enhanced appetite, and subsequent weight loss.

There is currently no cure for type 1 diabetes, and individuals with diabetes face major changes in their lifestyle and the possibility of serious debilitating and life-threatening complications. The goal of treatment is to allow the individual to lead a normal life and to normalize blood sugar levels, in an effort to decrease episodes of hyperglycemia and DKA and to minimize the risk of long-term vascular complications. These long-term complications are specific to
diabetes and affect the kidneys, the retina, and the peripheral nerves, and are the leading causes of significant morbidity and mortality in individuals with diabetes (Drash, 1987). The treatment for type 1 diabetes is life-long, and includes regular measurement of blood sugar levels, multiple daily insulin injections, and the following of a carefully planned meal plan which is predictable and regular, and restricted in simple carbohydrates. Preoccupation with food and dietary restriction is a common consequence of both the management of diabetes and the disorder itself.

Following initial diagnosis of diabetes, children and adolescents with diabetes are dependent on insulin injections to survive and must, with their families, take on the immense responsibility of treating their diabetes. They face the prognosis of possible future complications and the risk of premature death. The initial period after diagnosis has been noted to result in feelings of loss and a period of bereavement (Jacobson, 1996). Some children and adolescents have been noted to withdraw from social situations, feel anxious and sad, and become more dependent on their parents (Grey et al., 1995). The psychosocial characteristics of newly diagnosed children can often predict typical or average adherence to the diabetes regimen after 4 years (Jacobson et al., 1987, 1990). Following this initial period, most patients seem to adjust reasonably well and to view their future optimistically (Johnson, 1980). In most studies, diabetes has not been shown to be related to higher rates of gross long-term psychological or behavioural problems compared to the general population (Johnson, 1980; Marcus and Wing, 1990; Jacobson et al., 1997). However, in one recently completed prospective study, 92 adolescents with newly diagnosed type 1 diabetes were followed for 10 years and assessed for psychiatric disorders. In total, 47% of the sample reportedly met criteria for a psychiatric disorder at some point over the 10 year period, and a majority of these were during the first year of treatment (Kovacs et al.,
1997). Unfortunately, this study did not have a control group of non-diabetic peers, so it was not known if the prevalence is significantly higher in the diabetic population.

In some individuals, the stress of coping with diabetes may interfere with normal development and may result in impaired self-esteem and ego development, and exacerbate dependence/independence issues (Tatersall and Lowe, 1981; Hauser et al., 1983; Hillard and Hillard, 1984; Kovacs et al., 1985; Jacobson et al., 1986; Jacobson et al., 1997). In one recently completed large cross-sectional study of 3,300 adolescents, it was found that individuals with chronic medical conditions (including diabetes) had poorer body image and self-esteem compared to healthy peers (Wolman et al., 1994). Diabetes may also place considerable pressures and demands on the family, and may result in dysfunctional diabetes-related family interactions that do not facilitate the achievement of independence and individualization (Marcus and Wing, 1996).

Although most children with type 1 diabetes are healthy, long-term diabetes-related complications, eventually occur in up to 40% of adults with type 1 diabetes (Krolewski et al., 1985, 1987). The Diabetes Control and Complications Trial (DCCT) has clearly shown that the risk of developing diabetes-related complications is greatly increased when diabetes is poorly controlled and blood sugars are consistently elevated (DCCT Research Group, 1993, 1995). These findings strongly support the current approach to diabetes management, which emphasizes intensive efforts to normalize blood sugar levels.

It has been demonstrated that metabolic control worsens during adolescence (Ludvigsson, 1977; Daneman et al., 1981; Amiel et al., 1986; Mortensen et al., 1997). Although decreased
sensitivity to insulin may play a role (Amiel et al., 1986), worsening metabolic control may be explained largely by a decrease in compliance with the diabetes treatment regimen (Littlefield et al., 1992). Patients with poor metabolic control appear to have higher rates of psychiatric illness (Lustman et al., 1986; Lustman, 1988), are inconsistent with medical visits (Jacobson et al., 1991), and often live in families which are highly conflictual and rigid, with impaired communication and high levels of criticism of the diabetic child (Anderson et al., 1981; Hanson et al., 1989; Hauser et al., 1990; Wysocki, 1993; Jacobson et al 1994). Female adolescents have been found to have worse metabolic control (Daneman et al., 1981; Mortensen and Hougaard, 1997, 1998), significantly more episodes of DKA and hospitalizations (Brink, 1997; Cohn et al., 1997), and to be frequently less compliant with their type 1 diabetes treatment regimen (Littlefield et al., 1992) compared to their male counterparts. Although the reasons for this difference are unclear (Brink, 1997; Cohn et al., 1997), it has been suggested that this finding may be due, at least in part, to the higher rate of disordered eating attitudes and behaviours reported in females with diabetes compared to males (Rodin and Daneman, 1992).

EATING DISORDERS AND TYPE 1 DIABETES

Etiology of Eating Disorders in Females with Type 1 Diabetes

Eating disorders and type 1 diabetes are both relatively common conditions. However, whether or not their association is coincidental or whether diabetes and its treatment are triggering or perpetuating factors remains unclear and controversial. Like their non-diabetic peers, female adolescents with diabetes are frequently dissatisfied with their bodies and pursue weight loss through dietary restraint (Rodin and Daneman, 1992). However, there are several aspects of diabetes and its management that may place females at higher risk for the development of
disordered eating attitudes and behaviours (Rodin and Daneman, 1992; Neumark-Sztainer et al., 1996) (see Figure 1.2, Potentiation of Eating Disorders by Diabetes and its Management).
FIGURE 1.2
Potentiation of Eating Disorders by Diabetes and its Management
#### Dietary Restraint and Food Preoccupation

Traditionally, patients with diabetes have been required to follow standard nutritional counselling which emphasizes chronic dietary restraint, in terms of timing of meals and snacks, and the quantity and types of foods eaten (Rodin and Daneman, 1992). Individuals with diabetes are required to understand the principal of nutritional planning and to avoid excessive overeating or undereating that can cause hyper- or hypoglycemia respectively. In addition, they are required to limit particular foods, especially simple carbohydrates and/or fat, and must consistently time meals to coordinate with insulin injections and blood sugar levels. Consequently, patients with diabetes must eat according to externally-determined cues and criteria, requiring them to ignore internal cues regarding hunger and satiety (Rodin and Daneman, 1992). Thus, the physiological and social cues for eating may become replaced by the rituals/rigors of a more fixed diet. This is similar to the effect of Western cultures in which young women of normal weight are encouraged to diet and thus to ignore their perceptions of hunger to achieve a culturally-valued and rewarded thin body type. This disregard of internal cues for hunger and satiety may cause some vulnerable individuals to develop disinhibited eating and dietary dysregulation (Polivy and Herman, 1985). This may occur especially in those individuals who already have difficulties with identifying and responding to internal cues.

Although the nutritional plan for people with diabetes is adequate in terms of total caloric intake, it resembles a weight-loss diet in that it externally prescribes dietary rules and restrictions that can lead to a similar dysregulation of eating patterns in vulnerable individuals. Further, preoccupation with food and the need to limit carbohydrates may lead to "carbohydrate phobia" (Powers, 1983). As reviewed earlier, dieting and dietary deprivation, whether voluntary or involuntary, contribute to a cycle of binge eating, lowered self-esteem, further
dieting, and purging behaviours such as self-induced vomiting and laxative abuse (Polivy and Herman, 1985; Hsu, 1990; Woodside, 1995), and increase the risk of developing an eating disorder (Attie and Brooks-Gunn, 1989). In addition, the psychological deprivation associated with refraining from favorite foods, and the stress of not being allowed to eat what one wants may result in an increased susceptibility to excessive eating and bingeing (Polivy, 1996). The imposed and perceived dietary restraint associated with diabetes management may trigger or amplify this cycle of binge eating, dieting and purging behaviours in susceptible girls, who may already be preoccupied with food and their weight and shape. In this regard, lapses in adherence to the diabetes diet are often referred to as "cheating".

Many people with diabetes experience intense cravings for seemingly forbidden foods and sweets, and virtually all individuals with diabetes perceive themselves as "cheating" on their diabetes diet, some quite regularly (Hillard and Hillard, 1980). This cycle of craving foods, eating, and feeling guilty is very similar to that experienced by patients with BN. Hillard and Hillard (1984) have suggested that it may be valid to view "cheating" on the diabetes diet as occupying a continuum with BN. The intensive emphasis on weight, diet and food intake may cause individuals with diabetes to become particularly vulnerable to eating disturbances. Although both male and female children and adolescents with diabetes must follow the diabetes diet, females with diabetes, like their non-diabetic peers, are more likely to be affected by pressures to be thin and are at greater risk than their male counterparts to develop disordered eating patterns (Garner et al., 1980).
**Weight Fluctuations**

In the early stages of diabetes, before treatment is initiated, patients may experience marked weight loss due to malnutrition and dehydration associated with insulin deficiency and glycosuria. This weight is quickly regained following the onset of insulin therapy (Steel et al., 1990). In individuals who are already sensitive to body weight and shape, the combination of the initial weight loss, which may have been viewed positively, and subsequent weight gain can be distressing. In the general population, weight gain may result in increased body dissatisfaction and drive for thinness, leading to dieting and eating disturbances (Garfinkel et al., 1992; Goldbloom and Garfinkel, 1993).

Weight loss in individuals with diabetes is quite difficult to achieve when blood sugars are well controlled (DCCT, 1988). Further, efforts to improve glycemic control through appropriate insulin use and intensive diabetes management, may lead to further weight gain (Steel et al., 1990; DCCT, 1994). This tendency to be overweight may further sensitize patients to their shape and appearance and prompt dietary restraint (Steel et al., 1989), thus increasing the risk of developing an eating disorder such as BN (Fairburn and Cooper, 1983). In this regard, some studies have shown that females with diabetes have higher stable BMI values, compared to their non-diabetic peers (DCCT, 1988), and this is most likely due to the metabolic effects of insulin. Prospective studies in the general population have shown that having a higher percentage of body fat in the adolescent period is a risk factor for the subsequent development of an eating disorder (Attie and Brooks-Gunn, 1989). In females with diabetes, the tendency to be overweight either before or after the diagnosis of diabetes may be an important trigger in the development of an eating disorder in susceptible young women. In a study of 152 women
to 25 years of age) with diabetes for a median of 11 years, subjects who had higher BMI values were found to have increased scores on the Eating Attitudes Test (EAT) (Steel et al., 1989).

**Effects of Diabetes on Psychological Development**

Diabetes is not associated with overt psychological disturbances in adolescents and young adults (Simonds, 1977; Tebbi et al., 1990; Jacobson et al., 1997), and there is no single "diabetic personality" (Dunn and Turtle, 1981). It is not known whether having a physical illness is a non-specific stressor for susceptible individuals, or if having a chronic medical illnesses, such as diabetes, may actually be a more specific stressor due to the subtle but pervasive impact on most aspects of daily living, which may strain the adaptive resources of the individual and the family (Garfinkel et al., 1988). In some children and adolescents with diabetes, the stresses of having diabetes can impair ego development, affect body image and self-esteem, cause feelings of being different, and exacerbate conflicts about independence and dependence (Hauser et al., 1983; Jacobson et al., 1986; Jacobson et al., 1997).

The conflict between dependence and independence may be intensified in adolescents with diabetes because of parental worry, overinvolvement or underinvolvement and the special challenges involved in becoming independent (Powers et al., 1983). Powers and colleagues (1983) have suggested that the conflict between dependence and independence is an etiologic factor in the development of eating disorders. These conflicts regarding autonomy and dependence, and consequent low self-esteem, can increase the risk for the development of an eating disorder. Further, eating disorders may provide an opportunity to shun the responsibilities of diabetes, and conflicts about autonomy may be expressed through overeating and undereating.
Adolescents with diabetes have been shown to have more subtle disturbances in ego development and self-image complexity, compared to their non-diabetic peers (Hauser et al., 1983). Diabetes, like other chronic illnesses, can present challenges to adolescents in their development of self-esteem and positive body image, and can contribute to an increased risk for eating disorders (Neumark-Szainer et al., 1998). In a recently published 10-year follow-up study of young adults both with and without type 1 diabetes, subjects with diabetes did not report more psychiatric disturbances but did report having lower perceived competence, global self-worth, sociability and physical attractiveness (Jacobson et al., 1997). The authors concluded that adjustment to diabetes improved over the course of the 10-year follow-up, and young adults with diabetes appear to be as well-adjusted as their non-diabetic peers. However, the stress of coping with diabetes may interfere with the ability to develop a positive self-image and self-esteem and may interfere with the development of social relationships, which may in turn predispose these individuals to the risk of future depression or other adjustment difficulties (Jacobson et al. 1997). Disturbances in the sense of self have been regarded by some theorists as central to the development of eating disorders (Goodsitt, 1985; Geist, 1989), and have been reported to be a poor prognostic factor for the outcome of eating disorders (Sohlberg et al., 1989). It is possible that impaired self-esteem and ego function, that have been reported in some individuals with diabetes, in concert with other factors, may increase the risk of the development of an eating disorder and/or perpetuate eating disorders resulting in poor outcomes.

**Effects of Diabetes on Family Dynamics**

The diagnosis of diabetes in a child or adolescent may place considerable stresses and demands on a family and its members, and can cause some children and their parents to feel guilty,
anxious or depressed. These feelings can lead parents to become either over-protective and over-involved, or to distance themselves from their child (Pond, 1979; Marcus and Wing, 1990). In order to develop independence, adolescents need parents to provide them with both support and leeway to develop autonomy. Balancing these needs may be difficult for parents of adolescents with diabetes because they must also assure that good metabolic control continues to be achieved while still allowing the child to become independent (Koski, 1969; Minuchin et al., 1975). Consequently, normal conflicts about autonomy may be intensified by the presence of diabetes in a child. Further, the management of diabetes often requires the involvement of the entire family in terms of scheduling and daily routines. These added strains might impair adolescents’ abilities to separate from their parents and to develop their own identity, which includes, but is not dominated by, their disease.

The quality of family functioning appears to influence the adolescent's ability to adjust to the diagnosis and management their diabetes; poor family functioning may increase the adolescent's risk of developing an eating disorder. Low family cohesion, high conflict, impaired communication and problem solving skills, enmeshed family boundaries, and rejection of the child with diabetes, have all been associated with poor adjustment to diabetes, poor treatment compliance, and impaired metabolic control in adolescents with diabetes (Anderson et al., 1981; Bobrow et al., 1985; Hanson et al., 1989; Hauser et al., 1990; Wysocki, 1993; Jacobson et al., 1994; Kovacs et al., 1997). Similar disturbances in family functioning have also been documented in families of non-diabetic patients who suffer from eating disorders (Garfinkel et al., 1983; Johnson, 1985; Humphrey et al., 1986; Kog and Vandereycken, 1989; Stern et al., 1989). Minuchin et al. (1978) found similar maladaptive characteristics in patients with eating disorders and in those with poorly-controlled diabetes,
and concluded that dysfunctional diabetes-related interactions may contribute to an environment that may increase the risk of the development of eating disturbances. In a more recent study, Marahaj and colleagues (1998) assessed eating attitudes and behaviours and family interactions and functioning in 113 adolescent females with diabetes and their mothers. Eating disturbances were associated with a family environment that was perceived by the mother and daughter to be conflictual and inadequate in support and structure. The parents of girls with more highly disturbed behaviours were also perceived to set high standards and emphasize independence, but they failed to provide adequate support in fostering personal growth. The authors suggest that this may lead to self-blame and lowered self-esteem when expectations are not met and may increase the risk for the onset and/or persistence of an eating disorder (Marahaj et al., 1998). Unfortunately, as with other correlational and cross-sectional studies it cannot be determined from this study whether the observed family dynamics in diabetic families actually increase the risk of eating disorders or if they are a consequence of them.

**Insulin Manipulation**

Patients with type 1 diabetes have a potent and unique method of weight loss available to them, namely intentional insulin underdosing or omission to induce glucose loss in the urine (Rodin and Daneman, 1992). Since patients with diabetes commonly perceive that insulin use leads to weight gain (Szmukler, 1984), they also learn that they can omit or underdose their insulin to avoid weight gain or to lose weight. Underdosing or omitting insulin results in hyperglycemia, which induces glycosuria and leads to weight loss. Insulin manipulation for weight loss has been reported in 5-37% of young women with diabetes depending on the age of the sample studied (Birk and Spencer, 1987; Stancin et al., 1989; Fairburn et al., 1991; Rodin et al., 1991;
Peveler et al., 1992; Rydall et al., 1997), and is the most common weight control strategy used by patients with diabetes (Fairburn et al. 1991; Rodin et al., 1991; Rydall et al., 1997). This is most likely due to the fact that such a method is constantly available to them, is relatively simple and can be concealed. Insulin manipulation for the purpose of weight loss is now recognized as a purging method in the DSM-IV, included in the "misuse of medication" criterion (APA, 1994).

**Timing of Diabetes Diagnosis**

Type 1 diabetes is typically diagnosed during childhood or adolescence, and is associated with an initial period of adjustment (Grey et al., 1995). Although different individuals deal with the stress of coping with diabetes in different ways, the timing of diagnosis may be an important trigger in the development of an eating disorder. According to the additive stress hypothesis, the more stresses encountered simultaneously the greater the risk of developing an eating disorder (Petersen and Hamburg, 1986; Compas et al., 1989). In this regard, if diabetes diagnosis coincides with puberty, or the onset of dating, or other life events, the additive effect may be to trigger the onset of an eating disorder. This hypothesis is supported by findings that girls with diabetes and eating disturbances are significantly older and closer to puberty (ages 10-14 years) at diabetes diagnosis compared to girls with diabetes who do not have an eating disturbance (Rydall et al., 1997; Nielsen and Molbak, 1998).

**Metabolic Factors**

It has been suggested that metabolic disorders similar to diabetes may trigger eating disorders. There has been no evidence that hyperglycemia, DKA, insulin deficiency at diagnosis, or peripheral hyperinsulinemia from subcutaneous insulin injections are direct triggers for eating
It has been suggested that metabolic disorders similar to diabetes may trigger eating disorders. There has been no evidence that hyperglycemia, DKA, insulin deficiency at diagnosis, or peripheral hyperinsulinemia from subcutaneous insulin injections are direct triggers for eating disorders (Rodin and Daneman, 1992). However, Hillard and Hillard (1984) have noted that even mild levels of hypoglycemia or hyperglycemia may result in cognitive disturbances or psychiatric symptoms such as depression or agitation. Further, the symptoms of poorly controlled diabetes may include some of the symptoms of major depressive disorder. In individuals with diabetes, fluctuations in blood sugars may trigger depressed moods, and extreme thirst or hunger which can result in binge eating. Binge eating can also lead to hyperglycemia and often girls are reluctant to compensate with the appropriate amount of insulin because they fear weight gain and their sugars remain high. Conversely, purging through vomiting may result in excess insulin and hypoglycemia. Bingeing and purging in young women with diabetes results in fluctuations in blood sugar levels and may also result in cognitive disturbances, and this may trigger or perpetuate the cycle of binge eating and purging.

Additional research is needed to clarify the potential relationship of diabetes-specific factors to the onset and/or maintenance of eating disorders. The standard diabetes nutritional counselling, weight gain, availability of a weight loss method (insulin manipulation), and the stresses of coping with diabetes and their effects on the family and on the self and ego development may cause some individuals to become more vulnerable to the development of an eating disorder. The presence of some or all of these factors may predispose young women with diabetes to the development of disordered eating and may trigger/perpetuate, and maintain eating disorders.
Complications of Eating Disorders in Patients with Type I Diabetes

Among young women with type 1 diabetes, clinical and sub-threshold eating disorders have been associated with impaired metabolic control (Birk and Spencer, 1987; Steel et al., 1989; Fairburn et al., 1991; Peveler and Fairburn, 1992; Affenito et al., 1997; Rydall et al., 1997), and an increased risk of diabetes-related microvascular complications (Steel et al., 1987; Marcus and Wing, 1990; Colas et al., 1991; Rydall et al. 1997). Rydall et al. (1997) recently conducted a 4-year follow-up study of 91 adolescent females with diabetes and demonstrated that eating disorders were common, persistent, and associated with the early development of one diabetes-related complication. Eighty-six percent of girls defined as having highly-disordered eating at initial assessment (or baseline), were found, 4 years later, to have some degree of diabetic retinopathy compared to 43% of the moderately-disordered and 24% of the non-disordered eating groups (Rydall et al., 1997). These findings suggest that there are serious long-term consequences of disturbed eating behaviours, and the resultant impaired metabolic control may place young women at risk for earlier-than-expected diabetes-related morbidity and mortality.

Review of Previous Research

The first reported case of a patient with both an eating disorder and diabetes was in 1973 (Bruch, 1973), and it was not until the early 1980's that further studies were published (Fairburn and Steel, 1980; Garner, 1980; Gomez et al., 1980; O’Gorman and Eyre, 1980; Roland and Bhanji, 1982; Szmukler and Russell, 1983; Powers et al., 1983; Hudson et al., 1985). Since then, systematic studies have determined that eating disturbances, including sub-threshold disorders, are common in young women with diabetes and are associated with a poor course and outcome. In a review conducted by Marcus and Wing in 1990, 57 cases of diabetes and
eating disorders were identified. Ninety-five percent of the cases reported were female, and diabetes preceded the eating disorder in 90% of the cases. In 62% of the cases, intentional insulin omission for weight-loss was reported, and impaired metabolic control was noted in 75% of the cases.

In young women with diabetes, significant eating disturbances, usually involving binge eating and insulin omission have been reported in up to 30% of those studied. Clinical and sub-threshold eating disorders are associated with psychological and physical morbidity and mortality (Woodside et al., 1995). In patients with diabetes they can also result in impaired metabolic control and can significantly increase the risk of diabetes-related complications, which pose a particularly serious risk to the long-term health of young people with diabetes (Steel et al., 1987; Colas et al., 1991; Rydall et al., 1997). Unfortunately, it has been noted in some case reports that patients with diabetes and eating disorders may be less responsive to eating disorder treatment programs compared to their peers who do not have diabetes (Peveler and Fairburn, 1992). Although, there has not been a controlled study to confirm these reports, this finding may indicate that treatment is more challenging in this population and may potentially lead to increased morbidity and mortality rates.

There is little debate that eating disorders and diabetes can coexist and increase the risk of medical complications. However, the results of studies which have attempted to assess the prevalence of eating disorders in the diabetic population have varied considerably, depending on the sample studied, methodology, and diagnostic criteria for eating disorders used (Shaw and Garfinkel, 1990; Rodin and Daneman, 1992).
Although there have been a few studies which assessed the occurrence of diabetes in clinical eating disorder populations (Gomez et al., 1980; Powers et al., 1983; Neilson et al., 1987; Feieris, 1988; Emborg, 1996), a majority of studies have assessed eating psychopathology in diabetes samples. While some of these studies have shown similar rates of eating disturbances as in the non-diabetic population (Wing et al., 1986; Robertson and Rosenvinge, 1990; Powers et al., 1990; Marcus et al., 1992), others have reported rates that are higher than population based rates (Hudson et al., 1985; Rodin et al., 1985; Wing et al., 1986; Rosmark et al., 1986; Birk and Spencer, 1987; Lloyd et al., 1987; Rodin et al., 1987; Stancin et al., 1989; Rodin et al., 1991; Rydall et al., 1997). Unfortunately, most of these studies used unreliable self-report measures and did not include control groups. Further, all of the studies conducted to date have had relatively small samples of female subjects with diabetes within the age group at highest risk for eating disorders, raising serious questions about the power to detect observed differences and about the reliability and generalizability of the findings.

Another reason for the wide range of conflicting findings may be the criteria used to define an eating disorder. Prevalence rates have been affected by changes in the criteria for eating disorders specified by the American Psychiatric Association. In earlier studies using DSM-III and DSM-III-R criteria, the prevalence of eating disorders in young women with diabetes may have been underestimated since insulin omission to produce weight loss was not considered an inappropriate compensatory behaviour. The misuse of medication, including insulin, for weight loss was included in the DSM-IV criteria for BN and ED-NOS. In studies using the broad DSM-III criteria (APA, 1980), a diagnosis can be made in 17-35% of females with diabetes (Rodin et al., 1985, 1986; Birk and Spencer, 1987; Stancin et al., 1989). The rate lowers to 5-16% in studies using the stricter DSM-III-R criteria (APA, 1987) (Steel et al., 1987; Fairburn et
al., 1991; Rodin et al., 1991; Peveler et al., 1992; Cantwell and Steel, 1996; Affentino et al., 1997; Friedman et al., 1998). Finally, small studies using DSM-IV criteria (APA, 1994), have reported the prevalence to range from 7-11% (Herpertz et al., 1996; Engstrom et al., 1999) of females with diabetes. In all of these studies, BN and ED-NOS are much more common than AN.

Since 1990, there have been seven controlled, interview-based studies of the prevalence of eating disorders in type 1 diabetes (Fairburn et al., 1991; Peveler et al., 1992; Striegel-Moore et al., 1992; Vila et al., 1993; Mannucci et al., 1995; Vila et al., 1995; Engstrom et al., 1999) (Table 1.2). In all but one of these studies (Streigel-Moore et al., 1992), the frequency of eating disorders in females with diabetes is at least 50% greater than among controls. However, in only one of these studies (Engstrom et al., 1999) was the difference statistically significant, and all of the studies had very low statistical power. In addition, the small size of the samples limits the reliability of the estimated population parameter (prevalence).

Peveler and colleagues (1992) conducted a cross-sectional survey to determine the prevalence of eating disorders in 33 female and 43 male adolescents (aged 11-18 years) with type 1 diabetes and compared it to 76 matched non-diabetic control subjects. Features of eating disorders were not found among the male subjects. In the females, no subjects met criteria for AN or BN, and 9 percent of the adolescent girls with diabetes and 6 percent of the female controls met criteria for ED-NOS. This difference was not statistically significant, and the authors concluded that the prevalence is not raised in young women with type 1 diabetes. However, there are several problems with this study and its conclusions. Firstly, this study had a very small sample size of girls and therefore very little statistical power to detect differences
in prevalence rates. In fact, based on the sample size and prevalence rates found, this study had only a 7.5% chance of detecting a difference of this magnitude (i.e., 9 vs. 6 percent). A further difficulty was that twelve percent of the diabetic sample admitted to currently manipulating their insulin dose for shape and weight reasons. However, the authors based their diagnostic criteria on the DSM-III-R, which did include "other inappropriate compensatory behaviours" but did not yet include the manipulation of medication (including insulin) as an inappropriate compensatory behaviour. Thus, if the researchers did not consider insulin omission, it seems likely that the prevalence rate in the diabetic sample was underestimated, compared to the prevalence rate they might have detected had they used DSM-IV criteria.

In a similar study with females aged 18-25 years, DSM-III-R eating disorders were diagnosed in 11 percent of the subjects with diabetes and 7.5 percent of the controls. Again, this difference was not statistically significant, and the study had very low power (<20%) to detect the difference observed (Fairburn et al., 1991).

In the study by Streigel-Moore et al. (1992), 46 girls with diabetes (aged 8-18) and 46 controls were screened and interviewed using the Eating Disorder Examination (EDE) interview (Cooper and Fairburn, 1987). No cases of eating disorders were detected in either group. However, 6.5% of the diabetes subjects reported insulin misuse within the previous month. Only two of the control subjects, and none of the diabetes subjects, reported feeling a loss of control over eating and 33 percent of the girls with diabetes compared to 24 percent of the controls reported excessive weight control within the past month. The authors suggest that there does not appear to be an increased prevalence of eating disorders in the diabetes
population, but note that the relatively small sample size raises questions regarding the
generalizability of their findings (Streigel-Moore et al., 1992).

Vila and colleagues published two additional studies on the prevalence of eating disorders in young women with type 1 diabetes (Vila et al., 1993, 1995). In both of these studies all subjects were within the age at risk and were interviewed with a semi-structured interview, and diagnosis were made based on DSM-III-R criteria. In the diabetes subjects, 8 percent of the girls were diagnosed with an eating disorder (in both studies). In the control sample, no cases were detected in the study published in 1993, and only 2% of girls met eating disorder criteria in the 1995 study. These findings were not significant, although the authors conclude that girls with diabetes are at an increased risk for eating disorders (Vila et al., 1995).

Mannucci and colleagues (1995) conducted the largest of seven controlled interview-based study of eating disorders in type 1 diabetes. This study involved both men and women, aged 15-60 years. The average age of the subjects was 34 years for the diabetes subjects, which is well above the age of risk for eating disorders. Each subject with diabetes identified three people to act as controls. Consequently, the control sample was not randomly selected. The authors found that 1.6% of the diabetes subjects and 0.9 percent of the controls met DSM-III-R criteria for AN; BN was diagnosed in 1.6 percent of the diabetes subjects, and 2.7 percent of controls; and ED- NOS was diagnosed in 4.9 and 2.7 percent, respectively. An additional 33% of the diabetes subjects met criteria for sub-clinical disorders compared to 22.5 percent of controls. Unfortunately, although this was the largest controlled study looking at the prevalence of eating disorders in type 1 diabetes, the findings must be considered with caution considering the age of the sample and the method used to select the control group.
In a recently published population-based study by Engstrom and colleagues (1999), 89 adolescent females with type 1 diabetes and 89 matched controls were asked to complete an eating disorder screening package. Subjects who scored above the predetermined cut-off, were then asked to complete a semi-structured interview. Fifteen girls with diabetes and 2 control subjects scored above the cut-off and participated in the interview. Neither of the control subjects met criteria for a DSM-IV disorder. Six (7%) of the subjects with diabetes met DSM-IV criteria for ED-NOS, and there were no cases of AN or BN. However, due to the small sample of subjects who were actually interviewed (including only two control subjects), the validity and reliability of this study are questionable and these findings must be interpreted with caution.

There remains considerable controversy with respect to the prevalence of eating disorders in this population. Despite the assertion by Fairburn et al. (1993), that reported studies fail to confirm the impression that eating disorders are over-represented among patients with type 1 diabetes, there has been no adequate controlled study to date with a large enough sample size of females in the age of risk for eating disorders to answer this question definitively.
Table 1.2

Previous Controlled Interview Based Prevalence Studies

<table>
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<tr>
<th>AUTHOR</th>
<th>N</th>
<th>DSM Disorders</th>
<th>Sub-Threshold Disorders</th>
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<tr>
<td></td>
<td>Diabetes</td>
<td>Control</td>
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<td>Diabetes</td>
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<tr>
<td></td>
<td>Diabetes</td>
<td>Sub-Threshold Disorders</td>
<td></td>
</tr>
<tr>
<td>Fairburn et al. (1991)*</td>
<td>54 (67)</td>
<td>11%</td>
<td>7.5%</td>
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<td>(18-25 years)</td>
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<tr>
<td>Peveier et al. (1992)*</td>
<td>33 (33)</td>
<td>9%</td>
<td>6%</td>
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<tr>
<td>(11-18 years)</td>
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<tr>
<td>Streigel-Moore et al. (1992)*</td>
<td>46 (46)</td>
<td>0%</td>
<td>0%</td>
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<tr>
<td>(8-18 years)</td>
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<tr>
<td>Vila et al. (1993)*</td>
<td>52 (24)</td>
<td>8%</td>
<td>0%</td>
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<tr>
<td>(12-19 years)</td>
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<tr>
<td>Mannucci et al. (1995)*</td>
<td>62 (148)</td>
<td>8%</td>
<td>6%</td>
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<tr>
<td>(15-60 years)</td>
<td></td>
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<tr>
<td>Vila et al. (1995)*</td>
<td>52 (46)</td>
<td>8%</td>
<td>2%</td>
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<tr>
<td>(13-19 years)</td>
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<tr>
<td>Engstrom et al. (1999)**</td>
<td>89 (89)</td>
<td>7%</td>
<td>0%</td>
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<tr>
<td>(12-18 years)</td>
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</table>

* DSM-III-R criteria

** DSM-IV criteria
RATIONALE FOR THE PRESENT STUDY

There is clear evidence that clinical and sub-threshold eating disorders frequently coexist in adolescent females and young women with type 1 diabetes, and often lead to poor compliance, impaired metabolic control and an increased risk of diabetes-related complications.

It has been suggested that several features of type 1 diabetes and its treatment, such as weight gain, dietary restraint and food preoccupation, and the availability of a potent means of inducing weight loss (i.e., insulin manipulation), may increase the prevalence of eating disorders by lowering the threshold for the appearance of a clinical or sub-threshold disorder in females who are vulnerable.

Researchers have attempted to assess whether the prevalence of clinical and sub-threshold eating disorders is increased in young women with type 1 diabetes compared to young women in the general population. However, the results of these studies have been inconclusive and prevalence estimates have been unreliable due to the relatively small sample sizes used. Consequently, whether or not there is an increased prevalence of eating disorders in young females with type 1 diabetes remains unresolved.

The present study was designed to remedy the methodological limitations of earlier studies by providing a large scale interview-based estimate of the prevalence of clinical and sub-threshold eating disorders in three pediatric tertiary care diabetes centers and in a large three-site school-based population.
In addition, researchers have previously suggested that several factors including increased BMI values, higher socio-economic status, older age, and (in the diabetes sample) onset of diabetes close to puberty, may increase the risk for the development of disordered eating behaviours and eating disorders. Although this study is cross-sectional and not prospective, these factors will be examined to determine if they can predict eating disorder status in either the diabetes or control groups.

It is hoped that establishing the magnitude of the problem in both the non-diabetic and diabetic population, and identifying potential risk factors for the development of eating disturbances, will further our knowledge of the etiology of eating disorders in both the diabetes and control populations. In addition, these findings may aid in the development and implementation of early intervention strategies that may prevent the development of full-blown clinical eating disorders and decrease the risk of poor metabolic control and subsequent long-term complications in young women with type 1 diabetes.
CHAPTER TWO

OBJECTIVES AND HYPOTHESES OF THE PRESENT STUDY

OBJECTIVES

Primary Objective

The primary objective of this study was to conduct a large interview-based evaluation of eating attitudes and behaviours in adolescent females with and without type 1 diabetes mellitus and to compare the two groups in terms of the prevalence of:

1. DSM-IV eating disorders
2. Sub-threshold eating disorders
3. Self-reported disordered eating attitudes and behaviours

Secondary Objective

A secondary objective of this study was to examine the relationship between previously implicated risk factors for eating disorders and their association with DSM-IV and sub-threshold eating disorders in young women both with and without type 1 diabetes (e.g., age, BMI, SES, age at diabetes diagnosis, duration of diabetes).

HYPOTHESES

Compared to non-diabetic controls, adolescent females with type 1 diabetes will demonstrate:

1. More disturbed eating attitudes and behaviours
CHAPTER THREE

METHODOLOGY

STUDY DESIGN

Subjects and Recruitment

A total of 430 adolescent females, 11 to 19 years of age, who had been diagnosed with type 1 diabetes for at least one year, were invited to participate in a two-stage study (self-report survey and diagnostic interview) of eating attitudes and behaviour. These subjects were drawn from diabetes clinics at the Hospital for Sick Children in Toronto (HSC), the Children’s Hospital of Eastern Ontario (CHEO) in Ottawa, and the Children’s Hospital at Hamilton Health Sciences Corporation (HHSC) in Hamilton, Ontario. All three diabetes clinics are the major primary treatment centers in their geographic areas for children and adolescents with type 1 diabetes, providing care for approximately 70% of potential patients within their respective catchment areas.

Females aged 12 to 19 years were identified from diabetes clinic lists and were approached by mail and phone call or during clinic appointments. Informed written consent was obtained from each subject and from a parent/guardian for patients younger than 16 years of age. A comparison group of 2,494 female students without diabetes was identified at junior high and high schools in Toronto, Ottawa, and Hamilton. These students were introduced to the study during a regular class period and were given consent and demographic forms to be signed and completed by the students and by their parents, if they were under 18 years of age.
Procedure

All participants completed the self-report screening package, which included: the Eating Disorder Inventory (EDI) (Garner and Olmsted, 1984), the Eating Attitudes Test (EAT-26) (Garner et al., 1982), and the Diagnostic Survey for Eating Disorders (DSED-modified) (Johnson, 1985). Body mass index (BMI) was based on self-reported height and weight and calculated in kg/m². Following screening, three control subjects for each subject with diabetes were randomly selected to act as matched (based on city and age) controls. Control subjects who scored above pre-determined cut-off levels were subsequently asked to complete the Eating Disorder Examination (EDE ver. 1.5d-modified) (Cooper and Fairburn, 1987), a semi-structured diagnostic interview. An additional 15% of the subjects who did not meet the cut-off were randomly selected for the interview to ensure interviewer blindness. Interviewer blindness to diabetes status was not possible due to the nature of the EDE interview and the diabetes-specific questions (e.g., insulin omission for weight loss). Blood was obtained by finger prick from each of the subjects with diabetes for HbA1c measurement at the clinic visit closest in time to the screening assessment. To ensure uniformity, HbA1c was measured in all subjects using the BioRad Variant HPLC assay (normal range, 4 to 6 percent) (Gillery et al., 1995) in the Department of Pediatric Laboratory Medicine at the Hospital for Sick Children in Toronto (see Figure 3.1, Flow Chart of Study Design).
FIGURE 3.1: STUDY DESIGN

RECRUITMENT
Informed Consent
Demographic Info

SCREENING
EHQ(DI, BD, Bulma)
"FAST-20"
FD-SES
HbA1c - Diabetes Subjects

Matching of Diabetes and Control Subjects

Diagnostic Interview
An Interview Based on Diagnostic Interview Schedule

Diagnostic Interview
ETHICAL CONSIDERATIONS

The Scientific and Human-Subjects Review Committees at HSC, CHEO, HHSC, and the participating school boards (see Appendix A) approved the study protocol. All subjects were told that the results of the EDI, EAT-26, DSED, and the EDE interview were confidential and that their parents (and diabetes doctor for diabetes subjects and teachers for non-diabetes subjects) would not be informed of the results without their permission. Subjects diagnosed with an eating disorder based upon the EDE interview were offered a referral for eating disorder assessment and/or treatment. All subjects were informed that study participation, refusal or withdrawal would not affect the status or quality of their current or future care at HSC, CHEO, HHSC or their school.

MEASURES

Demographic information gathered included (see Appendix B): age, SES, ethnic background. Clinical information gathered included age of diabetes diagnosis, duration of diabetes, number of daily insulin injections.

Screening Measures to Assess Eating and Weight Psychopathology (see Appendix B):

Three sub-scales of the Eating Disorder Inventory (EDI) (i.e., Body Dissatisfaction, Drive for Thinness, and Bulimia), Eating Attitudes Test-26 (EAT-26), and the Diagnostic Survey of Eating Disorder (DSED) were used for screening, in combination, in order to detect disordered eating attitudes and behaviours. The EDI provides quantitative assessments of a number of disordered eating attitudes and behaviours while the EAT-26 provides a total score for
disturbed eating attitudes and behaviours. The DSED allows the frequency of disturbed
behaviours to be quantified.

(i) The Eating Disorder Inventory (EDI) (Garner and Olmsted, 1984): The EDI is a widely-
used, reliable and valid 64-item forced-choice self-report measure of specific attitudes related to
eating and weight psychopathology (Garner and Olmsted, 1984). It is organized around 8
subscales, of which 3 were included at screening: Drive for Thinness, Bulimia, and Body
Dissatisfaction. As in previous studies involving subjects with diabetes (Steel et al., 1989),
items that could relate to the dietary restrictions imposed by the diabetes regimen and which
may not reflect eating pathology perse (e.g., “I eat sweets and carbohydrates without feeling
nervous”) were removed during scoring and the sub-scales were pro-rated.

(ii) The Eating Attitudes Test (EAT-26) (Garner et al., 1982): The EAT-26 is an abbreviated
26-item version of the original 40-item measure (EAT-40) (Garner and Garfinkel, 1979). The
EAT-26 is a forced-choice self-report measure which consists of three subscales that measure
eating attitudes and behaviours related to dieting, bulimia, food preoccupation, and oral control.
It has been shown to be a reliable, valid, and objective measure of eating disorder symptoms in
both diabetic and non-diabetic populations (Garner et al., 1982; Rodin et al., 1986; Wing et al.,
1986). Items that may relate to the dietary restrictions normally imposed by the diabetes
regimen and which may not reflect eating pathology (e.g., “I am aware of the caloric content of
foods eaten”) were removed during scoring as in previous studies involving diabetes subjects
(Steel et al., 1989).
(iii) The Diagnostic Survey for Eating Disorders (DSED-modified) (Johnson, 1985): The DSED is a self-report questionnaire, validated for use in the diabetes population (Rodin et al., 1991; Rydall et al., 1997), which elicits detailed information about eating habits, binge eating and purging behaviours over the past three months. It has been modified to include diabetes-related items such as insulin omission for weight loss and bingeing during hypoglycemic episodes (Rodin et al., 1991; Rydall et al., 1997).

**Diagnoses of Eating Disorders based on DSM-IV criteria were determined based on:**

(i) The Eating Disorder Examination (EDE ver. 11.5d-modified) (Cooper and Fairburn, 1987): The EDE is a semi-structured diagnostic interview that quantifies symptoms, behaviours and specific psychopathology of eating disorders based on DSM-IV criteria. It includes sections related to binge eating, purging, and dietary restraint. Five subscale scores can be derived from the ratings: Restraint, Overeating, Eating Concern, Weight Concern, and Shape Concern. A modified version of the EDE was used for the diabetes population which included diabetes-specific concerns such as insulin manipulation or omission for the purpose of weight loss/control (Olmsted et al, 1997). The EDE has demonstrated good reliability and validity and is currently the gold standard for the diagnosis of clinical and sub-threshold eating disorders (Cooper et al., 1989; Wilson and Smith, 1989; Rosen et al., 1990).

**SCREENING CRITERIA**

Subjects were asked to participate in stage two (the semi-structured EDE interview) if they met one or more of the following criteria at screening:
1) Score ≥ 15 on the Drive for Thinness subscale of the EDI; this corresponds to the 88th percentile for girls aged 11 to 18 years and the 47th percentile for clinically-diagnosed eating disorder patients (Garner and Olmsted, 1984);

2) Score ≥5 on the Bulimia subscale of the EDI; this corresponds to the 86th percentile for 11 to 18 year old girls and the 21st percentile for eating disorder patients (Garner and Olmsted, 1984);

3) Total score ≥20 on the Body Dissatisfaction subscale of the EDI; this corresponds to the 87th percentile for 11 to 18 year old girls and the 60th percentile for eating disorder patients(Garner and Olmsted, 1984);

4) Score ≥20 on the EAT-26 total score, the recommended cut-off score for screening purposes (Garner et al., 1982);

5) Current or past history of binge eating, self-induced vomiting, laxative or diuretic use, insulin omission for weight loss, or current dietary restriction as assessed by the DSED;

6) History of eating disorder diagnosis and/or treatment as reported on the DSED; or

7) ≤ 5th percentile BMI value for age-matched females (Hammer et al., 1991).

**DIAGNOSIS OF EATING DISORDERS BASED ON DSM-IV CRITERIA**

For the purpose of the present study, both DSM-IV and sub-threshold disorders were operationalized and assessed based on data from the Eating Disorder Examination. Subjects were classified with 1) a full syndrome eating disorder, based on the Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV) criteria (APA, 1994) (including *anorexia nervosa* (AN), *bulimia nervosa* (BN), and "eating-disorder not otherwise specified" (ED-NOS)); 2) a sub-threshold disorder based on operationalized criteria, or 3) no eating disorder. Although eating disorders are generally believed to occur along a continuum, for the purposes of
the present research, these three categories were considered to be mutually exclusive (see Table 3.1 for DSM-IV and sub-threshold diagnostic criteria).

Based upon the DSM-IV, a minimum of four clinical symptoms was necessary for the diagnosis of anorexia nervosa (AN) and bulimia nervosa (BN). The third category of DSM-IV eating disorder, "eating disorder not-otherwise specified" (ED-NOS) included individuals with an eating disorder of clinical severity but which did not meet criteria for AN or BN (Fairburn and Wilson, 1993). The DSM-IV has provided broad suggestions for ED-NOS diagnoses. ED-NOS in the present study was operationalized more precisely based upon the DSM-IV and criteria employed in previous controlled prevalence studies (Peveler et al., 1992; Streigel-Moore et al., 1992). As discussed above, eating disorders are thought to occur along a continuum and previous research has suggested that a large proportion of children and adolescents do not meet criteria for AN, BN, or ED-NOS but instead suffer from sub-threshold disorders. Sub-threshold eating disorders are milder eating disturbances with a lower frequency and/or severity of symptoms over the past three-months than specified in the DSM-IV. However, such disorders may still be associated with significant health risks and with increased risk of the subsequent development of a full-syndrome eating disorder (Kreipe et al., 1989; Childress et al., 1993; Melve and Baerheim, 1994; Robin et al., 1998). In the diabetes sample, sub-threshold disorders may still be clinically significant (Wing et al., 1986; Affenito et al., 1997; Rydall et al., 1997), and differences in the prevalence of sub-threshold disorders may exist between the diabetes and control populations. Binge eating for all diagnoses was defined as the consumption of a large amount of food (definitely more than what would be considered normal under similar circumstances) in a discrete amount of time (usually <2 hours), and associated with a feeling of loss of control.
TABLE 3.1
STUDY CRITERIA FOR DSM-IV AND SUB-THRESHOLD EATING DISORDERS

ANOREXIA NERVOSA:

A. Refusal to maintain body weight at or above a minimally normal weight for age and height.

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one's body weight or shape is experienced; undue influence of body weight and shape on self-evaluation, or denial of the seriousness of the current low body weight.

D. In postmenarchal females, amenorrhea, i.e., absence of at least 3 consecutive menstrual cycles (a woman is considered to have amenorrhea if her periods occur only following hormone e.g., estrogen, administration).

BULIMIA NERVOSA:

A. Recurrent episodes of binge eating.

B. Recurrent inappropriate compensatory behaviour to prevent weight gain, such as self-induced vomiting, misuse laxatives, diuretics, or other medications (including misuse of insulin), fasting, or excessive exercise; and

C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months; and

D. Self-evaluation is unduly influenced by body shape and weight; and

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.
STUDY CRITERIA FOR DSM-IV AND SUB-THRESHOLD EATING DISORDERS

EATING DISORDER-NOT OTHERWISE SPECIFIED (ED-NOS):

(1) All the criteria for AN except ammenorrhea; or

(2) All the criteria for AN except the subject does not report a fear of weight gain or does not report a disturbance in the way in which their body weight and/or shape is experienced.

(3) All the criteria for BN except that the subject does not report self-evaluation being unduly influenced by shape and/or weight; or

(4) All the criteria for BN except that the frequency of binge eating and purging behaviour(s) occurred at least once per week for 3 months, or 2 times per week over the previous four weeks; or

(5) An individual regularly engages in inappropriate compensatory behaviour in the absence of binge eating (e.g., recurrent self-induced vomiting or insulin omission for shape and weight control at least 1 time per week for the past 3 months, or twice weekly over the previous 4 weeks); or

(6) An individual who engages in recurrent episodes of objective binge eating (at least 1 time per week for the past 3 months or twice weekly over the previous 4 weeks).

SUB-THRESHOLD EATING DISORDERS:

(1) An individual engages in occasional (a minimum of 3 times) binge eating, and/or purging over the past 3 months; or

(2) An individual repeatedly chews and spits out food to prevent weight gain (at least once per week over the preceding 3 months, or twice a week over the previous 4 weeks); or

(3) An individual who regularly engages in extreme dietary restraint (< 500 kcal/day) or excessive exercising (more than 120 min/per day, 5 times a week) for the purpose of weight-loss over the past 3 months.
STATISTICAL ANALYSIS

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS; Noursis, 1992), with the exception of power analyses and sample size estimates which were performed with the Power Analysis and Statistical Significance program (PASS, 1998). P-values <0.05 were considered statistically significant. All P-values are 2-tailed.

**Descriptive Statistics:** Basic descriptive statistics (i.e., frequencies, means, standard deviations, ranges, and odds ratios) were conducted to provide information regarding the characteristics of the samples, the prevalence of disturbed eating attitudes and behaviours, and eating disorders and sub-threshold disorders. Normally-distributed continuous variables were compared using Student's t-test (age, BMI, HbA1c, diabetes onset and duration) and ANOVA tests. Frequency data were compared using chi-square analyses.

**Hypothesis 1:** Comparisons of Self-Reported Eating Attitudes and Behaviours in Diabetic and Control Subjects:

Chi-square tests of independence were used to compare the frequency of abnormal eating behaviours (based on the DSED) between the three city sites and in the combined samples. Non-normal data (EDI and EAT-26 subscales) were transformed logarithmically and one-way ANOVA tests were used to compare means between the three sites; post-hoc tests (Scheffe's Method) were conducted when significant effects were found. Independent t-tests were used for overall two-group comparisons between the diabetes and control samples.
Hypothesis 2: Prevalence of Eating Disorders in Diabetic and Control Subjects:

The prevalence rates of clinical and sub-threshold eating disorders in the two groups were compared using 2x3 chi-square analysis. Comparisons between the three sites were made using 3x3 chi-square analyses. Odds ratios and corresponding confidence intervals were calculated to measure the main effect of diabetes on eating disorders (Rosner, 1995).

Predictors of Eating Disorder Status:

A series of stepwise discriminant function analyses (using the minimization of Wilk's Lambda criterion) were carried out to predict the presence of DSM-IV and sub-threshold disorders. Predictor variables included diabetes status, age, BMI, SES, age of diabetes onset, duration of diabetes.

Sample Size Estimation:

Power analyses and sample size estimates were calculated to determine the sample size needed for this study. Based on previously-published small prevalence studies, we estimated a 4 percent difference in the prevalence of eating disorders between the diabetes and control populations (approximately 8 percent vs. 4 percent) (Fairburn et al., 1991; Peveler et al., 1992; Streigel-Moore et al., 1992; Vila et al., 1993; Mannucci et al., 1995; Vila et al., 1995). Consequently, we determined we would need a sample size of 350 diabetic subjects and 1,050 non-diabetic controls to have 80 percent power to detect a difference of this magnitude at a significance level of 0.05 (2-tailed).
CHAPTER FOUR

CHARACTERISTICS OF THE SAMPLES

RECRUITMENT AND PARTICIPATION RATES

A total of 361 of 430 eligible females with type I diabetes (84 percent) agreed to participate in the study (HSC n=203; CHEO n=86; HHSC n=72); 69 (16 percent) refused or did not return their forms. Of the 2,494 eligible school girls without diabetes, 1840 (74 percent) returned signed consent forms and agreed to take part in the study; 301 refused to participate (12 percent) and 353 (14 percent) did not return their consent forms and were considered non-participants. One hundred and fifty-one subjects were absent or could not participate because of school tests or field trips on the day the self-report survey was administered. Screening data were collected on 1,689 subjects without diabetes (68 percent) (Toronto n=661; Ottawa n=639; Hamilton n=389). Following this, a sub-sample of 1,114 age and site-matched control subjects was randomly selected (approximately 3:1 ratio) (Toronto n=560; Ottawa n=304; Hamilton n=250). Three percent of both the control (n=16) and the diabetes (n=5) subjects who met the cut-off criteria did not participate in the interview and were excluded from the prevalence analysis. An additional 96 control subjects (17 percent) and 38 diabetes subjects (21 percent) who did not meet cut-off were randomly selected to participate in the interview. Two hundred and twenty (97 percent) subjects with diabetes and 616 (97 percent) control subjects who were invited to the interview completed the study. In total, prevalence data is reported on 1,098 controls and 356 diabetes subjects. (see Figure 4.1, Recruitment and Participation).
FIGURE 4.1: RECRUITMENT

**Diabetes Subjects n=361**
- HSC n=203
- CHEO n=86
- HHSC n=72

**Control Subjects n=1689**
- Toronto n=661
- Ottawa n=639
- Hamilton n=389

**SCREENING**
- EDE-D (D.1, D.2, Bulimia)
- BAT-ZC
- DSD

**Matching Controls n=578**
- Toronto n=202
- Ottawa n=208
- Hamilton n=268

**SCOPING**
- **Below Cut-Off**
  - Diabetes Subjects n=174 (48%)
  - Matched Controls n=578 (52%)

- **Above Cut-off**
  - Diabetes Subjects n=187 (52%)
  - Matched Controls n=536 (48%)

~15% of subjects below cut-off were invited to interview

\[
\begin{align*}
\text{Diabetes n=38} \\
\text{Control n=96}
\end{align*}
\]

All subjects above cut-off were invited to interview.
COMPARISON OF THE THREE STUDY SITES

In order to obtain the required sample sizes, study participants were recruited from three sites across Ontario. Demographic and clinical characteristics of the diabetes and control subjects in the three sites were compared using oneway ANOVAs and are presented in Tables 4.1 and 4.2.

Control and diabetes subjects did not differ in age or BMI between the three sites. However, in the control group, SES differed significantly between the three sites (Table 4.1). Post-hoc tests using Scheffe's Method were conducted and indicated that control subjects from Ottawa had significantly higher SES compared to subjects from both Toronto and Hamilton.

There were no significant differences between the three sites on self-report measures, or interview-based data. This justified combining all three sites into one diabetes and one control group with comparisons then made between the combined groups.
### Table 4.1

**Characteristics of Control Subjects in Three Sites**

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Toronto Mean (SD)</th>
<th>Ottawa Mean (SD)</th>
<th>Hamilton Mean (SD)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1114</td>
<td>14.7 (1.9)</td>
<td>14.9 (1.8)</td>
<td>14.9 (2.0)</td>
<td>1.40</td>
<td>0.25</td>
</tr>
<tr>
<td>BMI</td>
<td>1002</td>
<td>20.5 (3.2)</td>
<td>20.6 (3.5)</td>
<td>20.9 (3.3)</td>
<td>1.66</td>
<td>0.19</td>
</tr>
<tr>
<td>SES</td>
<td>982</td>
<td>3.6 (1.1)</td>
<td>4.2 (0.7)</td>
<td>3.5 (0.8)</td>
<td>55.53</td>
<td>0.001</td>
</tr>
</tbody>
</table>

### Table 4.2

**Characteristics of Diabetes Subjects in Three Sites**

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Toronto Mean (SD)</th>
<th>Ottawa Mean (SD)</th>
<th>Hamilton Mean (SD)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age - Onset of Diabetes</td>
<td>361</td>
<td>15.0 (2.1)</td>
<td>14.7 (1.8)</td>
<td>14.8 (2.0)</td>
<td>0.93</td>
<td>0.39</td>
</tr>
<tr>
<td>BMI</td>
<td>346</td>
<td>22.6 (3.4)</td>
<td>22.8 (4.3)</td>
<td>22.6 (4.0)</td>
<td>0.06</td>
<td>0.94</td>
</tr>
<tr>
<td>SES</td>
<td>352</td>
<td>3.4 (1.0)</td>
<td>3.7 (1.0)</td>
<td>3.5 (0.8)</td>
<td>2.20</td>
<td>0.11</td>
</tr>
<tr>
<td>Onset Of Diabetes</td>
<td>352</td>
<td>7.9 (3.7)</td>
<td>8.2 (3.6)</td>
<td>8.7 (3.3)</td>
<td>1.49</td>
<td>0.23</td>
</tr>
<tr>
<td>Duration Of Diabetes</td>
<td>352</td>
<td>7.0 (3.5)</td>
<td>6.5 (3.9)</td>
<td>6.1 (3.5)</td>
<td>1.73</td>
<td>0.18</td>
</tr>
<tr>
<td>HbA1c</td>
<td>286</td>
<td>8.7 (1.4)</td>
<td>9.1 (1.9)</td>
<td>8.8 (2.1)</td>
<td>0.89</td>
<td>0.41</td>
</tr>
</tbody>
</table>
COMPARISON OF DIABETES AND CONTROL SUBJECTS

Age

Table 4.3 shows the age of the 361 diabetes subjects and the 1,114 control subjects at screening. Since control subjects were matched based on age and site, no difference in age between the two groups was expected. The age range was from 11-19 years. The mean age for the diabetes subjects was 14.9 ± 2.0 years, and was similar to that for control subjects 14.8±1.9 years ( t (1473)= -0.73, p=0.47).

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Diabetes Subjects (n=361)</th>
<th>Control Subjects (n=1114)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>11-13</td>
<td>109 (30)</td>
<td>334 (30)</td>
</tr>
<tr>
<td>14-15</td>
<td>107 (30)</td>
<td>371 (33)</td>
</tr>
<tr>
<td>16-17</td>
<td>105 (29)</td>
<td>291 (26)</td>
</tr>
<tr>
<td>18-19</td>
<td>40 (11)</td>
<td>118 (11)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>14.9±2.0</td>
<td>14.8±1.9</td>
</tr>
</tbody>
</table>
**Body Mass Index (BMI)**

BMI was calculated based on self-reported height and weight. Table 4.4 shows the distribution of body mass index (BMI) in both the diabetes and control groups. Mean BMI was significantly higher in the diabetes sample (22.7±3.8 kg/m², range 12.2-37.4) compared to the controls (20.6±3.3 kg/m², range 10.1-40.7), (t (1346)= -9.55, p<0.01) (Table 4.4).

<table>
<thead>
<tr>
<th>Body Mass Index</th>
<th>Diabetes Subjects(n=346)*</th>
<th>Control Subjects(n=1002)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>&lt;20</td>
<td>88 (25)</td>
<td>477 (47)</td>
</tr>
<tr>
<td>20-27</td>
<td>223 (65)</td>
<td>484 (48)</td>
</tr>
<tr>
<td>&gt;27</td>
<td>33 (10)</td>
<td>41 (4)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>22.7 (3.8)</td>
<td>20.6 (3.3)</td>
</tr>
</tbody>
</table>

**NOTE:**

*BMI was not available for 15 diabetes subjects at screening.
†BMI was not available for 112 control subjects at screening.
Socioeconomic Status (SES)

Socioeconomic status was determined using Statistics Canada 1994 Small Family Data which is based on tax filer information and provided for each postal walk. Subjects were asked to provide their postal code and the corresponding median total income for each postal code was applied. Based on Statistics Canada SES groups, subjects' SES was coded as follows: I ($15,000+), II (25,000+), III ($35,000+), IV ($50,000+), V ($75,000+), VI ($100,000+). The distribution of SES is presented in Table 4.5. Control subjects had significantly higher SES levels compared to the diabetes group ($x^2(5)=20.69, p<0.01$). There was a significantly larger proportion of control subjects from higher socioeconomic groups (V/IV) compared to the diabetes sample (18 percent vs. 11 percent, $x^2(1)=7.93, p<0.05$).

In order to determine whether the difference in SES found between the diabetes and control groups might affect our outcome data, SES was compared using chi-square analysis for those above and below screening cut-off and eating disorder groups. Table 4.6 shows the distribution of SES for subjects above and below screening cut-off. Subjects above cut-off did not have significantly higher SES compared to subjects below cut-off, ($x^2(5)=5.34, p=0.38$). Table 4.7 shows the distribution of SES for eating disorder groups. SES was not significantly different between eating disorder groups ($x^2(10)=17.47, p=0.10$). Since SES did not appear to be related to the outcome measures, we did not control for it in subsequent analyses.
<table>
<thead>
<tr>
<th>Socioeconomic Group</th>
<th>Diabetes Subjects (n=352)*</th>
<th>Control Subjects (n=960)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>5 (1)</td>
<td>7 (1)</td>
</tr>
<tr>
<td>II</td>
<td>47 (13)</td>
<td>81 (8)</td>
</tr>
<tr>
<td>III</td>
<td>114 (33)</td>
<td>290 (30)</td>
</tr>
<tr>
<td>IV</td>
<td>148 (42)</td>
<td>416 (43)</td>
</tr>
<tr>
<td>V</td>
<td>34 (10)</td>
<td>140 (14)</td>
</tr>
<tr>
<td>VI</td>
<td>4 (1)</td>
<td>36 (4)</td>
</tr>
<tr>
<td>MEAN (SD)</td>
<td>3.5 (1.0)</td>
<td>3.7 (1.0)</td>
</tr>
</tbody>
</table>

NOTE:
*SES data was missing for 9 diabetes subjects.
†SES data was not available for 154 control subjects.
<table>
<thead>
<tr>
<th>Socioeconomic Group</th>
<th>Below Cut-Off (n=699)*</th>
<th>Above Cut-Off (n=623)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>6 (1)</td>
<td>6 (1)</td>
</tr>
<tr>
<td>II</td>
<td>65 (9)</td>
<td>64 (10)</td>
</tr>
<tr>
<td>III</td>
<td>211 (31)</td>
<td>186 (29)</td>
</tr>
<tr>
<td>IV</td>
<td>276 (41)</td>
<td>289 (45)</td>
</tr>
<tr>
<td>V</td>
<td>101 (15)</td>
<td>73 (12)</td>
</tr>
<tr>
<td>VI</td>
<td>19 (3)</td>
<td>21 (3)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>3.68 (0.96)</td>
<td>3.65 (0.95)</td>
</tr>
</tbody>
</table>

**NOTE:**
* Based on diabetes and control subjects combined.
<table>
<thead>
<tr>
<th>Socioeconomic Group</th>
<th>No Disorder (n=1125)*</th>
<th>Sub-threshold Disorder (n=122)*</th>
<th>DSM-IV Disorder (n=75)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>I</td>
<td>9 (1)</td>
<td>0 (0)</td>
<td>3 (4)</td>
</tr>
<tr>
<td>II</td>
<td>111 (9)</td>
<td>8 (6)</td>
<td>9 (12)</td>
</tr>
<tr>
<td>III</td>
<td>344 (31)</td>
<td>42 (34)</td>
<td>18 (24)</td>
</tr>
<tr>
<td>IV</td>
<td>482 (43)</td>
<td>52 (43)</td>
<td>30 (40)</td>
</tr>
<tr>
<td>V</td>
<td>148 (13)</td>
<td>13 (11)</td>
<td>13 (17)</td>
</tr>
<tr>
<td>VI</td>
<td>31 (3)</td>
<td>7 (6)</td>
<td>2 (3)</td>
</tr>
<tr>
<td><strong>Mean (SD)</strong></td>
<td><strong>3.66 (0.95)</strong></td>
<td><strong>3.75 (0.94)</strong></td>
<td><strong>3.63 (1.11)</strong></td>
</tr>
</tbody>
</table>

**NOTE:**
* Based on diabetes and control subjects combined.
Clinical Characteristics of Diabetes Subjects

Diabetes Onset and Duration

The mean age of diabetes onset was 8.1 ± 3.6 years, with a range from 0.1 - 17 years. The average duration of diabetes was 6.7 ± 3.6 years, with a range of 5.3-16.7 years.

Metabolic Control

The mean hemoglobin A1c (HbA1c) blood level, which reflects metabolic control over the preceding 8-12 weeks, was 8.8% ± 1.7 for the sample at screening. The range of HbA1c values was 5.3% to 16.7% (Table 4.8). Based on the DCCT (DCCT Research Group, 1993), good or acceptable HbA1c levels for diabetics are considered less than 8%, while values greater than 9% are considered poor control. Consequently, 39% of our sample was in relatively poor control.
<table>
<thead>
<tr>
<th>HbA1c (%)</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>6.1-6.9%</td>
<td>22</td>
<td>8</td>
</tr>
<tr>
<td>7.0-7.9%</td>
<td>60</td>
<td>21</td>
</tr>
<tr>
<td>8.0-8.9%</td>
<td>87</td>
<td>30</td>
</tr>
<tr>
<td>9.0 -9.9%</td>
<td>56</td>
<td>20</td>
</tr>
<tr>
<td>10.0-10.9%</td>
<td>30</td>
<td>10</td>
</tr>
<tr>
<td>&gt;11%</td>
<td>26</td>
<td>9</td>
</tr>
<tr>
<td>------------</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>8.8 (1.7)</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE:**
*HbA1c was not available for 75 diabetes subjects at screening.*
CHAPTER FIVE

RESULTS

PREVALENCE OF DISTURBED EATING AND WEIGHT LOSS BEHAVIOURS

Hypothesis 1

Hypothesis #1 predicted that subjects with diabetes would have more disturbed eating attitudes and behaviours compared to the matched controls based on the self-report measures.

Disordered Eating Attitudes

Disordered eating attitudes were assessed at screening using the EDI (BD, DT, and Bulimia subscales) and the EAT-26, and were compared using student's $t$-tests. The mean EAT-26 scores for the diabetes and control groups were not significantly different ($8.3 \pm 9.2$ vs. $8.2 \pm 8.4$); $t(1261) = -0.58$, $p=0.95)$. There were no significant differences between the diabetes (n=361) and matched control groups (n=1114) on any of the EDI subscales (Table 5.1).
### Table 5.1

**Eating Disorder Inventory Subscales**

<table>
<thead>
<tr>
<th>EDI Subscales</th>
<th>Diabetes Subjects n=361</th>
<th>Control Subjects n=1114</th>
<th>t</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Dissatisfaction</td>
<td>9.5 (8.3)</td>
<td>10.2 (8.5)</td>
<td>1.36</td>
<td>1471</td>
<td>0.18</td>
</tr>
<tr>
<td>Drive for Thinness</td>
<td>4.5 (5.5)</td>
<td>4.3 (5.4)</td>
<td>-0.78</td>
<td>1471</td>
<td>0.58</td>
</tr>
<tr>
<td>Bulimia</td>
<td>1.4 (3.0)</td>
<td>1.3 (2.4)</td>
<td>-0.70</td>
<td>1471</td>
<td>0.49</td>
</tr>
</tbody>
</table>
**Disordered Eating and Weight Loss Behaviours**

Table 5.2 presents the prevalence of disordered eating and weight loss behaviours (i.e., risk behaviours) based on the self-report DSED.

Based on chi-square analysis, diabetes subjects compared to controls reported engaging in significantly more binge eating (30 vs. 23 percent, \( p<0.004 \)) and less dieting for the purpose of weight loss (12 vs. 23 percent, \( p<0.001 \)). Self-induced vomiting and laxative use were both fairly common, and were not significantly different between the two groups.

Of particular significance, was the dangerous practice of intentional insulin omission or undertreatment to promote weight loss in subjects with diabetes. This was the most commonly reported weight loss behaviour, after dieting. At screening, 48 (13 percent) of the 361 subjects with diabetes reported on the DSED that they had taken less than their prescribed dose of insulin to lose weight at some time in the past, and 41 (11.5 percent) were currently doing so.
<table>
<thead>
<tr>
<th>Risk Behaviour</th>
<th>Diabetes Subjects</th>
<th>Control Subjects</th>
<th>( x^2 )</th>
<th>p</th>
<th>Odds-Ratio*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dieting</td>
<td>43 (12)</td>
<td>254 (23)</td>
<td>20.10</td>
<td>0.001</td>
<td>0.50 [0.3 - 0.6]</td>
</tr>
<tr>
<td>Binge Eating</td>
<td>108 (30)</td>
<td>251 (23)</td>
<td>8.08</td>
<td>0.004</td>
<td>1.47 [1.1 - 1.9]</td>
</tr>
<tr>
<td>Self-Induced Vomiting</td>
<td>25 (7)</td>
<td>95 (9)</td>
<td>0.94</td>
<td>0.333</td>
<td>0.79 [0.5 - 1.3]</td>
</tr>
<tr>
<td>Laxative Abuse</td>
<td>9 (2)</td>
<td>14 (1)</td>
<td>2.70</td>
<td>0.100</td>
<td>2.01 [0.9 - 4.7]</td>
</tr>
</tbody>
</table>

**NOTE:**
* Odds ratios are shown with 95% confidence intervals.
Screening Cut-off

Forty eight percent of the control subjects (n=536) and 52 percent of the diabetes subjects (n=187) scored above the cut-off criteria and were asked to participate in the EDE diagnostic interview (Table 5.3).

Table 5.3

Diabetes and Control Subjects Above and Below Cut-Off

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>Above Cut-Off n (%)</th>
<th>Below Cut-Off n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Subjects</td>
<td>187 (52)</td>
<td>174 (48)</td>
</tr>
<tr>
<td>(n=361)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>536 (48)</td>
<td>578 (52)</td>
</tr>
<tr>
<td>(n=1,114)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Chi-square
x²=1.5, df=1, p=0.223

Odds Ratio*
1.16, [0.91 - 1.5]

NOTE:
* Odds ratios are shown with 95% confidence intervals.
In summary, there is partial support for hypothesis 1. Although subjects with diabetes did not have more disturbed eating attitudes than the non-diabetic control group based on the EAT-26-Total Score, and the Body Dissatisfaction, Drive for Thinness, and Bulimia subscales of the EDI, they did report significantly more binge eating based on the DSED. In addition, although diabetes and control subjects did not differ in terms of vomiting and laxative misuse, 11.5 percent of diabetes subjects reported current insulin manipulation for the purpose of weight loss.
FREQUENCY OF DSM-IV AND SUB-THRESHOLD DISORDERS

Hypothesis 2

In hypothesis 2 it was predicted that subjects with diabetes would have more DSM-IV eating disorders and sub-threshold disorders compared to the control subjects.

DSM-IV and Sub-threshold Disorders

The prevalence of eating disorders in the diabetes and control groups was compared using chi-squared analysis and is presented in Table 5.4. Subjects with diabetes were significantly more likely to have a sub-threshold or DSM-IV disorder.

<table>
<thead>
<tr>
<th>Eating Disorder Group</th>
<th>Diabetes Subjects (n=356)</th>
<th>Control Subjects (n=1098)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>No Eating Disorder</td>
<td>271 (76.1)</td>
<td>965 (87.9)</td>
</tr>
<tr>
<td>Sub-threshold Disorder</td>
<td>49 (13.8)</td>
<td>84 (7.6)</td>
</tr>
<tr>
<td>DSM-IV Disorder</td>
<td>36 (10.1)</td>
<td>49 (4.5)</td>
</tr>
</tbody>
</table>

CHI-SQUARE

χ²=30.12, df=2, p<0.001

Table 5.4
Proportion of Diabetes and Control Subjects in Eating Disorder Groups
DSM-IV Eating Disorders

Subjects with diabetes had significantly more DSM-IV eating disorders compared to the controls (see Table 5.5). Subjects with diabetes were 2.4 times more likely than controls to have a DSM-IV eating disorder (95 percent CI: 1.5-3.7). In total, 36 (10.1 percent) of the 356 diabetes subjects who completed the study met criteria for DSM-IV eating disorders compared to 49 (4.5 percent) of the 1098 non-diabetic controls ($x^2 (1) = 15.63, p<0.001$). No cases of AN were detected in either the diabetes or control groups. ED-NOS was the most frequent DSM-IV diagnosis. The frequency of BN and ED-NOS are shown in Tables 5.6-5.7.

Table 5.5
Prevalence of DSM-IV Disorders

<table>
<thead>
<tr>
<th>SUBJECT GROUP</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Subjects</td>
<td>36</td>
<td>10.1</td>
</tr>
<tr>
<td>(n=356)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>49</td>
<td>4.5</td>
</tr>
<tr>
<td>(n=1098)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CHI-SQUARE
$x^2 = 15.63, df=1, p<0.001$

ODDS RATIO*
2.4 [1.5 - 3.7]

NOTE:
*Odds ratios are shown with 95% confidence intervals.
Table 5.6
Prevalence of Bulimia Nervosa

<table>
<thead>
<tr>
<th>SUBJECT GROUP:</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Subjects</td>
<td>5</td>
<td>1.4</td>
</tr>
<tr>
<td>(n=356)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>5</td>
<td>0.5</td>
</tr>
<tr>
<td>(n=1098)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CHI-SQUARE
\[ x^2 = 3.55, df=1, p=0.07 \]

ODDS RATIO*
3.1 [0.89 - 10.83]

NOTE:
*Odds ratios are shown with 95% confidence intervals.
### Table 5.7
Prevalence of Eating Disorder-Not Otherwise Specified (ED-NOS)

<table>
<thead>
<tr>
<th>SUBJECT GROUP:</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Subjects</td>
<td>31</td>
<td>8.7</td>
</tr>
<tr>
<td>(n=356)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>44</td>
<td>4.0</td>
</tr>
<tr>
<td>(n=1098)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Total Incidence of ED-NOS**

<table>
<thead>
<tr>
<th></th>
<th>CHI-SQUARE</th>
<th>ODDS RATIO*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \chi^2 = 12.2, \text{df}=1, p&lt;0.0001 )</td>
<td>2.3 [1.4 - 3.7]</td>
</tr>
</tbody>
</table>

**NOTE:**
*Odds ratios are shown with 95% confidence intervals.*
Sub-threshold Eating Disorders

As predicted, diabetes subjects were significantly more likely to have a sub-threshold eating disorder compared to their non-diabetic controls (13.8% vs. 7.6%, p<0.001) (Table 5.8). The subjects falling into this category were most commonly engaging in binge eating and/or purging behaviours but these did not meet the frequency required for a DSM-IV disorder.

Table 5.8
Prevalence of Sub-threshold Eating Disorders

<table>
<thead>
<tr>
<th>SUBJECT GROUP</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Subjects</td>
<td>49</td>
<td>13.8</td>
</tr>
<tr>
<td>(n=356)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control Subjects</td>
<td>84</td>
<td>7.6</td>
</tr>
<tr>
<td>(n=1098)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CHI-SQUARE
\[ x^2 = 12.13, \text{ df}=1, \ p<0.001 \]

ODDS RATIO*
1.9 [1.3 - 2.8]

NOTE:
*Odds ratios are shown with 95% confidence intervals.

In summary, there is strong support for hypothesis #2. Diabetes subjects were approximately two times more likely to have a DSM-IV eating disorder or a sub-threshold disorder compared to their non-diabetic peers.
PREDICTORS OF EATING DISORDER STATUS

The second objective of this study was to examine our data post-hoc to see if we could predict eating disorder status in the diabetes group, control group, and total sample based on one or more of the demographic variables at screening. These are variables that have previously been implicated in the development of eating disorders.

Diabetes Subjects

The association between eating disorder status (non-disordered, sub-threshold disorder, and DSM-IV disorder) and potential predictor variables was examined in our diabetes group using discriminant function analysis and is presented in Table 5.9. The following variables were included:

i) age
ii) BMI
iii) socioeconomic status
iv) age of onset of diabetes
v) duration of diabetes

Based on univariate analyses, no differences were found between the three eating disorder groups on body mass index, socioeconomic status, or diabetes duration. However, there was a significant difference in age between the eating disorder groups. Based on post-hoc comparisons, the subjects with DSM-IV disorders were significantly older (16.4±3.5 years) (p<0.05) than those with no eating disorder (15.0 ±2.3 years) and neither group differed from the intermediate sub-threshold group (15.4 ± 1.9 years). In addition, the mean age of onset of
diabetes was significantly different between the three eating disorder groups. Again, using post-hoc analyses, diabetes subjects with DSM-IV disorder were significantly older ($9.6 \pm 3.7$ years) ($p<0.05$) at the time that diabetes was diagnosed compared to subjects with no eating disorder ($7.8 \pm 3.6$ years). The sub-threshold group was in between the other two groups and was not significantly different from either ($8.8 \pm 3.2$ years).

A stepwise discriminant function analysis was employed to predict the presence or absence of eating disorders and is presented in Table 5.9. Overall, this model accounted for 3.8% of the variance in predicting eating disorder status and correctly classified 47% of the cases. Age was the only significant contributor to the model, and accounted for the total 3.8% of the variance predicting eating disorder status. BMI, SES, age of diabetes onset, and diabetes duration did not add to the predictive ability of the model and consequently were not included.
Table 5.9
Predictors of Eating Disorder Status in Diabetes Subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>No-ED (n=271)</th>
<th>Sub-threshold (n=49)</th>
<th>DSM-IV (n=36)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>15.0 (2.3)</td>
<td>15.4 (1.9)</td>
<td>16.4 (3.5)</td>
<td>7.66</td>
<td>.001</td>
</tr>
<tr>
<td>BMI</td>
<td>22.5 (3.7)</td>
<td>22.9 (3.4)</td>
<td>23.5 (4.3)</td>
<td>1.89</td>
<td>.152</td>
</tr>
<tr>
<td>SES</td>
<td>3.5 (0.9)</td>
<td>3.6 (0.8)</td>
<td>3.5 (1.0)</td>
<td>0.71</td>
<td>.493</td>
</tr>
<tr>
<td>Onset of Diabetes</td>
<td>7.8 (3.6)</td>
<td>8.8 (3.2)</td>
<td>9.6 (3.7)</td>
<td>5.06</td>
<td>.007</td>
</tr>
<tr>
<td>Duration of Diabetes</td>
<td>6.8 (3.8)</td>
<td>6.1 (3.3)</td>
<td>5.9 (3.9)</td>
<td>0.65</td>
<td>.522</td>
</tr>
</tbody>
</table>

Predictor Variables at Screening

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Wilks' Lambda</th>
<th>F</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>.962</td>
<td>6.46</td>
<td>.002</td>
</tr>
<tr>
<td>BMI</td>
<td>.992</td>
<td>1.32</td>
<td>.268</td>
</tr>
<tr>
<td>SES</td>
<td>.995</td>
<td>0.88</td>
<td>.417</td>
</tr>
<tr>
<td>Onset of Diabetes</td>
<td>.969</td>
<td>5.26</td>
<td>.006</td>
</tr>
<tr>
<td>Duration of Diabetes</td>
<td>.995</td>
<td>0.84</td>
<td>.433</td>
</tr>
</tbody>
</table>
Summary Table:

<table>
<thead>
<tr>
<th>Step</th>
<th>Predictor Variable Entered</th>
<th>Cumulative Wilk's Lambda</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Age</td>
<td>.962</td>
<td>.002</td>
</tr>
</tbody>
</table>

This variable accounts for 3.8% of the variance.

Canonical Discriminant Function

<table>
<thead>
<tr>
<th>Eigenvalue</th>
<th>Canonical Correlation</th>
<th>Wilks' Lambda</th>
<th>Chi-square (df)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>.039</td>
<td>.194</td>
<td>.962</td>
<td>12.675 (2)</td>
<td>.002</td>
</tr>
</tbody>
</table>

Classification Results:

<table>
<thead>
<tr>
<th>Actual Group</th>
<th># Cases</th>
<th>Predicted Group Membership:</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No-ED</td>
<td>Sub-Threshold</td>
<td>DSM-IV</td>
</tr>
<tr>
<td>No-ED</td>
<td>271</td>
<td>145</td>
<td>27</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td></td>
<td>54%</td>
<td>10%</td>
<td>36%</td>
</tr>
<tr>
<td>Sub-Threshold</td>
<td>49</td>
<td>20</td>
<td>6</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td></td>
<td>41%</td>
<td>12%</td>
<td>47%</td>
</tr>
<tr>
<td>DSM-IV</td>
<td>36</td>
<td>9</td>
<td>5</td>
<td>22</td>
</tr>
<tr>
<td></td>
<td></td>
<td>25%</td>
<td>14%</td>
<td>61%</td>
</tr>
</tbody>
</table>

Percentage of cases correctly classified: 47%

NOTE: Missing data for one or more variables on 23 subjects.
Eating Disorder Status and Metabolic Control

Mean HbA$_{1c}$ was significantly different across the eating disorder groups (Table 5.10). Based on post-hoc comparisons, the mean HbA$_{1c}$ level in the DSM-IV group was significantly different at the 0.05 level, from the mean HbA$_{1c}$ level in the Non-Disordered group. The mean HbA$_{1c}$ value for subjects with a sub-threshold disorder did not differ significantly from the DSM-IV or non-disordered groups.

Table 5.10

Eating Disorder Status and HbA1c

<table>
<thead>
<tr>
<th>Eating Disorder Status:</th>
<th>Mean($%$) $\pm$SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Non-Disordered</td>
<td>8.6 $\pm$1.6</td>
</tr>
<tr>
<td>(n=214)</td>
<td></td>
</tr>
<tr>
<td>2. Sub-threshold Disorder</td>
<td>9.1 $\pm$1.8</td>
</tr>
<tr>
<td>(n=45)</td>
<td></td>
</tr>
<tr>
<td>3. DSM-IV Disorder</td>
<td>9.4 $\pm$1.8</td>
</tr>
<tr>
<td>(n=27)</td>
<td></td>
</tr>
</tbody>
</table>

Oneway ANOVA
F=4.02, p=0.02

*NOTE: Higher HbA1c levels reflects poorer metabolic control over the preceding 8-12 weeks.

*HbA1c was not available for 75 diabetes subjects at screening.
**Insulin Manipulation**

In subjects with diabetes, deliberate insulin omission or manipulation was the most common weight loss behaviour, after dieting. At screening, 48 (13 percent) of the 361 subjects with diabetes reported on the DSED that they had taken less than their prescribed dose of insulin to lose weight at some time, and 41 (11 percent) were currently engaging in this behaviour. Fifteen of the 35 diabetes subjects (43 percent) who were diagnosed with a DSM-IV disorder based on the EDE reported current insulin manipulation at the time of screening. This was significantly higher than the proportion of subjects who reported misusing insulin in the sub-threshold (18 percent) and non-disordered groups (6 percent, p<0.0001).
**Control Subjects**

The control group was examined separately using discriminant function analysis to determine the association between eating disorder status and the following variables: (Table 5.1)

i) age

ii) BMI

iii) socioeconomic status

Based on univariate analyses, no significant differences were found on age at screening, and socioeconomic status. There were significant differences in mean BMI between the eating disorder groups. Based on post-hoc analyses, control subjects with sub-threshold disorders were found to be significantly heavier (p<0.05) compared to the subjects with no disorder.

Stepwise discriminant function analysis was employed to predict the presence or absence of eating disorders. BMI was the only variable that significantly contributed to the model. Overall, this model accounted for approximately 1% of the variance in predicting eating disorder status and correctly classified 57% of the cases. Age and SES did not significantly add to the model and therefore were not included.
### Table 5.11

**Predictors of Eating Disorder Status in Matched Control Subjects**

<table>
<thead>
<tr>
<th>Variables</th>
<th>No-ED (n=966)</th>
<th>Sub-threshold (n=84)</th>
<th>DSM-IV (n=49)</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>15.3 (1.9)</td>
<td>15.6 (1.9)</td>
<td>15.5 (1.9)</td>
<td>1.12</td>
<td>.326</td>
</tr>
<tr>
<td>BMI</td>
<td>20.5 (3.3)</td>
<td>21.5 (3.5)</td>
<td>21.0 (3.0)</td>
<td>3.912</td>
<td>.020</td>
</tr>
<tr>
<td>SES</td>
<td>3.7 (1.0)</td>
<td>3.8 (1.0)</td>
<td>3.8 (1.2)</td>
<td>0.380</td>
<td>.684</td>
</tr>
</tbody>
</table>

### Predictor Variables at Screening

<table>
<thead>
<tr>
<th>Predictor Variables at Screening</th>
<th>Wilks' Lambda</th>
<th>F</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.00</td>
<td>0.215</td>
<td>.806</td>
</tr>
<tr>
<td>BMI</td>
<td>.990</td>
<td>4.437</td>
<td>.012</td>
</tr>
<tr>
<td>SES</td>
<td>.999</td>
<td>0.442</td>
<td>.643</td>
</tr>
</tbody>
</table>

**Summary Table:**

<table>
<thead>
<tr>
<th>Step</th>
<th>Predictor Variable Entered</th>
<th>Cumulative Wilk's Lambda</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BMI</td>
<td>.990</td>
<td>0.012</td>
</tr>
</tbody>
</table>

**Canonical Discriminant Function**

<table>
<thead>
<tr>
<th>Eigenvalue</th>
<th>Canonical Correlation</th>
<th>Wilks' Lambda</th>
<th>Chi-square (df)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>.010</td>
<td>0.101</td>
<td>.990</td>
<td>8.83</td>
<td>.012</td>
</tr>
</tbody>
</table>

This variable accounts for 1.0% of the variance.
Classification Results:

<table>
<thead>
<tr>
<th>Actual Group</th>
<th># Cases</th>
<th>Predicted Group Membership:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Non-ED</td>
</tr>
<tr>
<td>Non-ED</td>
<td>769</td>
<td>522</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60%</td>
</tr>
<tr>
<td>Sub-Threshold</td>
<td>65</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>46%</td>
</tr>
<tr>
<td>DSM-IV</td>
<td>37</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>48%</td>
</tr>
</tbody>
</table>

Percentage of cases correctly classified: 57%

**NOTE:** Missing data on one or more variables for 110 subjects at screening
**Total Sample**

In the total sample, diabetes status was included along with age, BMI, and SES in an overall stepwise discriminant function analysis to predict eating disorder status in order to see how well these could predict eating disorder status for the whole sample.

Overall, this model accounted for 3.2% of the variance in predicting eating disorder status, and classified 63% of the cases using diabetes status and BMI. Diabetes status was entered into the model at the first step and accounted for 2.4% of the variance. BMI was entered in the next step, and accounted for an additional 0.8% of the variance. Age and SES did not significantly add to the model, and were not included in the stepwise model (Table 5.12).
Table 5.12
Predictors of Eating Disorder Status in the Total Sample

<table>
<thead>
<tr>
<th>Variables</th>
<th>No-ED (n=1024)</th>
<th>Sub-threshold (n=113)</th>
<th>DSM-IV (n=70)</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>15.2 (2.0)</td>
<td>15.5 (1.9)</td>
<td>15.9 (2.0)</td>
<td>5.468</td>
<td>.004</td>
</tr>
<tr>
<td>BMI</td>
<td>20.9 (3.5)</td>
<td>22.1 (3.5)</td>
<td>22.2 (3.0)</td>
<td>9.871</td>
<td>.001</td>
</tr>
<tr>
<td>SES</td>
<td>3.7 (0.9)</td>
<td>3.8 (0.9)</td>
<td>3.6 (1.1)</td>
<td>0.513</td>
<td>.599</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictor Variables at Screening</th>
<th>Wilk's Lambda</th>
<th>F</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes Status</td>
<td>.975</td>
<td>15.25</td>
<td>.001</td>
</tr>
<tr>
<td>Age</td>
<td>.996</td>
<td>2.62</td>
<td>.073</td>
</tr>
<tr>
<td>BMI</td>
<td>.983</td>
<td>10.32</td>
<td>.001</td>
</tr>
<tr>
<td>SES</td>
<td>.999</td>
<td>0.57</td>
<td>.561</td>
</tr>
</tbody>
</table>
Table 5.12 continued:

Summary Table:

<table>
<thead>
<tr>
<th>Step</th>
<th>Predictor Variable Entered</th>
<th>Cumulative Wilk's Lambda</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Diabetes Status</td>
<td>.975</td>
<td>0.001</td>
</tr>
<tr>
<td>2</td>
<td>BMI</td>
<td>.967</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Canonical Discriminant Function

<table>
<thead>
<tr>
<th>Eigenvalue</th>
<th>Canonical Correlation</th>
<th>Wilk's Lambda</th>
<th>Chi-square (df)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>.034</td>
<td>0.182</td>
<td>.967</td>
<td>40.57</td>
<td>0.001</td>
</tr>
</tbody>
</table>

All together these variables account for 3.32% of the variance.

Classification Results:

<table>
<thead>
<tr>
<th>Actual Group</th>
<th># Cases</th>
<th>Predicted Group Membership:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No-ED</td>
</tr>
<tr>
<td>Non-ED</td>
<td>1024</td>
<td>791</td>
</tr>
<tr>
<td></td>
<td></td>
<td>70%</td>
</tr>
<tr>
<td>Sub-Threshold</td>
<td>113</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td></td>
<td>53%</td>
</tr>
<tr>
<td>DSM-IV</td>
<td>70</td>
<td>41</td>
</tr>
<tr>
<td></td>
<td></td>
<td>53%</td>
</tr>
</tbody>
</table>

Percentage of cases correctly classified: 63%

NOTE: Missing data on one or more variables for 124 subjects at screening.
**Predictive Value of the Screening Cut-Off**

One control subject and one subject with diabetes who scored below the cut-off and were randomly selected for an interview, met criteria for a DSM-IV disorder. In addition, one control subject and 7 diabetes subjects who did not score above the cut-off, but who participated in the interview, were diagnosed with a sub-threshold eating disorder.

The screening measures were found to have high negative predictive value (proportion of patients with negative results who do not have an eating disorder) but low positive predictive value (proportion of patients with a positive screen who are diagnosed with an eating disorder) in both the diabetes and control groups. In the control population, we can expect that 99% of subjects who scored below cut-off, do not have a DSM-IV eating disorder. In the diabetes group, 98% of the girls with no eating disorder would be expected to score below the screening cut-off. In both groups the screening tool cut-off was highly sensitive (proportion of true positives), and was able to capture ~90% of the girls with a DSM-IV disorder. However, in both the control and diabetes groups the positive predictive value of the screening cut-off was low (21% vs. 9% respectively). Consequently, large proportions of girls who scored above the screening cut-off were not diagnosed with a DSM-IV disorder based on the EDE interview.

In terms of sub-threshold disorders, we can expect that 99% of the control subjects who score below cut-off will not have a sub-threshold disorder. In the diabetes group, 84% of those who score below-cut-off would not be expected to have a sub-threshold disorder. In terms of sensitivity, 93% of the control girls who had a sub-threshold disorder scored above screening cut-off. However, in the diabetes population we can only expect to capture 60% of the girls who have a sub-threshold eating disorder based on the screening cut-off. Again, the screening
cut-off had low positive predictive value for the control and diabetes groups (16% and 25%, respectively). Consequently, a large proportion of the girls who scored above the cut-off are not expected to be diagnosed with a sub-threshold disorder based on the EDE.

Based on these data, there appears to be support for the screening tool in terms of identifying girls who are suffering from DSM-IV disorders and sub-threshold disorders. However, since the screening cut-off was designed to be very broad, in order to capture all cases, there is also a high proportion of girls who were identified as "high risk" based on the cut-off, but who did not have a disorder.

**Comparison of Present Prevalence Data to Previous Studies:**

We examined odds ratios to determine whether the apparent effects of diabetes on the occurrence of DSM-IV disorders in this study differed significantly from previous smaller prevalence studies (Fairburn et al., 1991; Peveler et al., 1992; Streigel-Moore et al., 1992; Vila et al., 1993; Mannucci et al., 1995; Vila et al., 1995; Engstrom et al., 1999). Neither the individual nor the overall aggregate odds ratio of the earlier prevalence studies (pooled OR= 2.14, 95 percent CI, 1.14-4.02) differed significantly from the present study (OR= 2.36, 95 percent CI, 1.51-3.77) (Table 5.13).
<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>Odds Ratio+</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fairburn et al. (1991)</td>
<td>1.55 [0.39 - 6.31]</td>
</tr>
<tr>
<td>2. Peveler et al. (1992)</td>
<td>1.55 [0.19 - 14.48]</td>
</tr>
<tr>
<td>3. Streigel-Moore et al. (1992)</td>
<td>1.00 [0.00 - 22026]</td>
</tr>
<tr>
<td>4. Vila et al. (1993)</td>
<td>4.55 [0.3 - 1000]</td>
</tr>
<tr>
<td>5. Mannucci et al. (1995)</td>
<td>0.49 [0.14 - 1.65]</td>
</tr>
<tr>
<td>6. Vila et al. (1995)</td>
<td>3.7 [0.37 - 91.59]</td>
</tr>
<tr>
<td>7. Engstrom et al. (1999)</td>
<td>12.94 (0.88-22026)</td>
</tr>
</tbody>
</table>

**Pooled OR‡:**

2.14 [1.14 - 4.02]

**Current OR**

2.36 (1.51 - 3.77)

**NOTE:**

*Individual odd ratios were calculated for the prevalence of DSM-IV disorders between diabetes and control subjects in each study.

\+Odds ratios are expressed with 95% confidence intervals

‡Pooled odds ratio (Mantel-Haenszel) for all seven studies.
CHAPTER SIX
DISCUSSION AND CONCLUSIONS

DISCUSSION

The present study was designed to determine whether the prevalence of disturbed eating and weight loss behaviours and eating disorders is higher in adolescent females with type 1 diabetes. Compared with matched controls in the general population. To date, this is the first interview-based controlled prevalence study with a large enough sample size to adequately determine whether the prevalence is increased in this group. The present study extends findings from previous smaller studies by quantifying the prevalence of both DSM-IV and sub-threshold eating disorders in a large population of adolescent females with diabetes compared to a large matched control group. In addition, potential risk factors for the development of eating disturbances were explored.

DSM-IV and Sub-threshold Eating Disorders

The most striking finding was that DSM-IV eating disorders were more than twice as prevalent in the diabetes sample compared to controls. Ten percent of the subjects with diabetes compared to 4.5 percent of control subjects met criteria for a DSM-IV eating disorder (i.e., BN and ED-NOS). An additional 13.8 percent of the diabetes group and 7.7 percent of the controls were engaging in disordered eating behaviours which met the operationalized criteria for a sub-threshold disorder. The prevalence rates of eating disorders in the diabetic and control groups in the present study are similar to those reported elsewhere (Fairburn et al., 1991; Peveler et al., 1992; Vila et al., 1993, 1995; Mannucci et al., 1995), although the authors of the latter studies concluded that eating disorders are not more common in young women with diabetes.
However, the previous studies lacked the sample sizes needed to achieve statistical significance for observed differences, and due to the small sample sizes have also had limited reliability to measure the prevalence of eating disorders. The larger sample sizes of both diabetes and control subjects in the present study provided the necessary power to detect a difference of this magnitude and the reliability to adequately address the issues of prevalence.

In both the diabetes and control groups, the most frequent DSM-IV diagnosis was ED-NOS, which is similar to other studies of diabetic and non-diabetic populations (Peveler et al., 1992; Vila et al., 1993; Mannucci et al., 1995; Bunnell et al., 1990; Mitray, 1992). Previous research has demonstrated that clinically significant eating disorders in female adolescents most often do not meet full DSM-IV criteria for AN and BN but do qualify for ED-NOS (e.g., purging behaviour but no bingeing, or with a lower frequency of bingeing and purging than specified in the DSM-IV) (Robin et al., 1998). The symptoms of ED-NOS overlap with AN and BN and these are clinically significant disorders. Adolescent females with ED-NOS are at increased risk for serious health risks such as pubertal delay, growth retardation, and impairment of bone mineral acquisition (Bachrach et al., 1990).

The finding that eating disorders are twice as common in young women with diabetes compared to their non-diabetic peers is particularly worrisome in terms of the potential medical consequences. Patients with type 1 diabetes and eating disorders, are at increased risk for non-compliance with diabetes treatment, poor metabolic control, hypoglycemic events, diabetic ketoacidosis, diabetes-related hospitalizations, and long-term diabetes-related micro-vascular complications (Rydall et al., 1997; Affenito et al., 1997). The combination of diabetes and disordered eating behaviours contributes to impaired metabolic control as reflected by increased
HbA1c levels (Fairburn et al., 1991; Peveler and Fairburn, 1992; Steel et al., 1989), as was confirmed in the present study. It has previously been suggested that the association between eating disorders and impaired metabolic control may be due to binge eating, insulin manipulation, and noncompliance with the diabetes treatment regimen (Rodin et al., 1991). Diabetes and eating disorders have also been associated with an increased risk of long-term diabetes-related complications such as retinopathy and neuropathy (Rydall et al., 1997; Colas et al., 1991; Marcus and Wing, 1990; Steel et al., 1987). Rydall and colleagues (1997) recently reported that the prevalence and severity of an eating disturbance at baseline evaluation predicted the development of early microvascular complications four years later in a cohort of 91 young women with type 1 diabetes. Some degree of diabetic retinopathy was detected in 86 percent of the females who were defined as having highly-disordered eating at baseline, compared to 43 percent of those with moderately-disordered behaviours, and 24% of those in the non-disordered group. Similar associations between eating disturbances and the earlier onset of diabetes-related microvascular complications have been reported by others (Steel et al., 1987; Colas et al., 1991). These data suggest that eating disturbances which commonly result in poor metabolic control, may place young women with type 1 diabetes at an increased risk for long-term diabetes-related complications. Consequently, early detection and intervention for eating problems are important in terms of helping to prevent the early onset and/or progression of long-term complications. Unfortunately, evidence from the literature suggests that psychiatric disorders including eating disorders are often undetected and under-treated in medical populations (Rodin and Daneman, 1992).

In the diabetes population, there is a small percentage of cases in which the signs of an eating disorder are obvious and diagnosis is made easily. More often, eating disturbances present in a
more occult manner, and manifest with impaired metabolic control, noncompliance with the diabetes regimen, weight fluctuations, and repeated unexplained episodes of hypoglycemia or diabetic ketoacidosis (DKA) (Rydall et al., 1997; Littlefield et al., 1992; Marcus and Wing, 1990; Stancin et al., 1989; Steel et al., 1987; Rodin et al., 1986, 1985). Consequently, the detection of an eating disorder within the diabetes clinic setting is dependent on a diabetes team with a high index of suspicion for eating pathology. The diabetes team plays an important role in detection and prevention of eating disorders and should provide young girls with a supportive and therapeutic environment in which there is an open dialogue regarding weight and shape concerns. Clinicians should routinely ask about dieting, bingeing, and insulin omission, and must be prepared to deal with these and other forms of noncompliance in a non-judgmental manner.

When an eating disorder is detected or suspected, there are a number of different approaches to treatment, depending on the severity. In cases of severe eating disorders, a referral to an eating disorder program is necessary. Unfortunately, the management of an eating disorder in individuals with type 1 diabetes can be difficult since each disorder can exacerbate the other and affect the course and outcome. Although there have been no controlled treatment studies of eating disorders in patients with diabetes, there have been a number of case reports which have indicated lower success rates than those found in the non-diabetic population (Fairburn and Cooper, 1987; Hillard and Hillard, 1984; Szmukler, 1984; Malone and Armstrong, 1985; Newman et al., 1988; Neilson et al., 1987; Peveler and Fairburn, 1989; Ramirez et al., 1990; Peveler and Fairburn, 1992).
In girls with diabetes, even mild forms of eating disturbances (such as our sub-threshold group) often present with impaired metabolic control (Affenito et al., 1997). Traditionally, the treatment for impaired metabolic control is to intensify diabetes management. Unfortunately, in young women with eating disturbances intensified treatment may actually potentiate the eating problem. Insistence on a strict diabetes regimen and metabolic control may result in weight gain and increased weight and shape concerns. Further, increased dietary restraint may result in more focus on food and lead to binge eating. Consequently, this approach may actually cause metabolic control to deteriorate. In these milder cases of eating disturbances, a clinic-based approach may be sufficient to change eating attitudes and behaviour. One approach that has been suggested has been to take a more flexible approach to the diabetes diet by promoting a more realistic and flexible approach to eating (Rodin and Daneman, 1992). De-emphasizing the focus on food and dietary restraint and allowing girls to eat more based on hunger and satiety may reduce binge episodes and insulin omission, and ultimately improve metabolic control (Lawson et al., 1994).

Group, family and/or individual counseling may be useful to prevent and treat young women with mild disorders. Diabetes and its management may affect family functioning and this may be exacerbated in patients with eating disorders. In some cases, a brief family intervention may help to improve diabetes compliance and to open communication and develop strategies to deal with the eating problem.

Prevention and early treatment programs, such as psychoeducation, have been shown to be useful in the non-diabetic population by changing eating knowledge, attitudes, and behaviours (Olmsted et al., 1991; Santonastaso et al., 1999; Springer et al., 1999), and may be most
effective for high risk individuals and individuals with sub-threshold disorders (Killen et al., 1993). Since eating disorders in adolescent females with diabetes may be difficult to treat and are associated with a high rate of medical complications, early treatment and prevention programs seem justified and necessary. In this regard, Olmsted and colleagues (1997) have developed and tested a brief psychoeducation program for high-risk adolescent females with type 1 diabetes. The six psychoeducation sessions covered the causes of eating disorders, sociocultural attitudes towards women's bodies, weight set point, the impact of eating disorders on diabetes, and the development of a non-deprivational approach to eating. Six months after the program, subjects in the treatment group showed a significant decrease in binge eating and dieting compared to the control subjects who did not receive the psychoeducation (Olmsted et al., 1997). Although, it is not known whether this approach has long-term effects in terms of improving metabolic control and preventing the onset of early microvascular complications, this type of approach may have the potential to provide a relatively simple and cost-effective early treatment and prevention approach for young women in the diabetes clinic setting.

Self-Reported Eating Attitudes and Behaviours

Based on the DSED, a self-report measure of eating behaviours, we found some interesting differences between the diabetes and control groups at screening. Binge eating was reported more frequently by the subjects with diabetes, whereas the control subjects reported more dieting. The frequencies of self-induced vomiting and laxative misuse were reported equally in both groups. An unexpected finding from this screening measure was that dieting for weight loss was reported by 23 percent of the control subjects compared to 12 percent of the diabetes subjects. It is not clear why these diabetes subjects reported less dieting. It is possible that girls with diabetes consider a "diet" to be more focused and severe, since a key component in the
treatment of diabetes is adherence to a prescribed dietary regimen or a "diabetes diet". In addition, the availability of insulin manipulation for weight control, together with the dietary restrictions already imposed by the diabetes regimen, may explain why the diabetes group reported less "dieting" to lose weight than the control group, even though they reported more binge-eating. Alternatively, since dieting can result in hypoglycemia, these girls may be less likely to restrict food intake and instead may omit insulin in order to lose weight. Further, the term "dieting" is ambiguous and may mean different things to different people. Although the term dieting is frequently used to refer to changes in eating for weight loss or maintenance of weight, knowing how adolescents understand this term is essential to interpret these and other findings regarding the prevalence of dieting.

Alarmingly high rates of dieting, ranging from 12- 60 percent, have been reported in adolescent females (Polivy and Herman, 1983; Davis and Furham, 1986; Streigel-Moore et al., 1986, 1993; Wertheim et al., 1992; Story et al., 1995; Neumark-Sztainer et al., 1995; Grigg et al., 1996). However, although these and other findings suggest that dieting is fairly ubiquitous amongst adolescent and latency-aged girls, it is not always clear what girls mean when they say that they are "dieting", nor is it clear what is considered to be outside of the range of "normal" dieting (Brownell and Rodin, 1994). In this regard, Neumark-Sztainer and Story (1998) conducted a large study of junior and senior high school students to assess what dieting and binge eating mean among adolescents. Subjects were divided into 25 focus groups and asked questions about dieting and binge eating behaviours. In a majority of the groups, healthy eating, such as eating more fruits and vegetables, was mentioned in reference to dieting. Dieting was frequently described as an "umbrella" term that included different behaviours (i.e., exercise) or non-behavioural terms (i.e., a desire to lose weight). The authors concluded that
self-reported rates of dieting should be interpreted with caution and it should not be assumed that the majority of adolescents who report dieting are actually engaging in unhealthful behaviours. It was for this reason, that dieting was not included in our screening criteria, and that we must interpret this self-report finding with some caution.

Like "dieting", the term "bingeing" is often misunderstood or interpreted in different ways. Beglin and Fairburn (1992) demonstrated that when young women are asked about binge eating on self-report measures, without being given a definition, their responses often refer to episodes of perceived overeating. These authors concluded that, "a clear, unambiguous definition must be provided." In order to decrease the confusion and potential for misinterpretation of the term "binge", all subjects in the present study were provided with a verbal definition and explanation of a binge when they were given the self-report measures. A "binge" episode was defined as eating a larger than normal amount of food in a short period of time with associated feelings of loss of control (i.e., not being able to stop eating or control what or how much one is eating). An example of both a binge episode and an episode of objective overeating were provided.

Subjects with diabetes reported significantly more binge eating with associated feelings of loss of control compared to the control group. Thirty percent of the diabetes subjects reported current binge eating with 7 percent reporting this more than 2 times per week. In the control group, 23 percent of the subjects reported current binge eating. The relatively high levels of reported binge eating in both the diabetes and control samples may be, in part, due to the fact that, even when subjects are provided with a definition, self-reported binges may be difficult to quantify without a clinical interview (Loeb et al., 1994). For individuals with diabetes, even objectively small amounts of food eaten during a binge may be associated with profound
psychological guilt and self-loathing, and use of inappropriate compensatory behaviours (Rodin and Daneman, 1992), and maybe associated with impaired metabolic control. Since, young women with diabetes may feel very guilty after consuming objectively small amounts of food, it is possible that the diabetes sample may over-report this behaviour more than controls. However, it has been suggested that in populations such as type 1 diabetes, the usual definition of a binge may not be appropriate, and may underestimate the problematic behaviour. Rodin and Daneman (1992) have suggested that judgments about the clinical severity of eating disorders (and binge eating) for patients with type 1 diabetes should take into account more of the psychological and metabolic consequences of the behaviour.

An alternative explanation for the increased prevalence of self-reported binge eating in girls with type 1 diabetes, is that it is a consequence of the dietary restrictions imposed on girls as a result of their diabetes. Compliance with the dietary recommendations of diabetes often requires girls to ignore internal cues of hunger and satiety that can result in episodes of overeating. It has previously been demonstrated that dietary restraint, which inhibits eating in response to normal hunger and satiety cues, may initiate a cycle of bingeing and purging (Polivy and Herman, 1993; Polivy, 1996). Further, the psychological deprivation of not being allowed to eat what one wants may trigger episodes of eating "forbidden" foods and lead to "all-or-none" thinking and bingeing. Binge eating may also be triggered by fluctuations in blood glucose and insulin concentrations in girls with poor metabolic control.

One particularly worrisome finding in the present study was the relatively high rate of self-induced vomiting and laxative abuse reported by both the diabetes and control groups. These rates are not likely to be overestimates because laxative misuse and vomiting are discrete
behaviours with a distinct onset and termination. Consequently, the difficulties encountered with defining dieting and binge eating are largely avoided (Beglin and Fairburn, 1992). Although adolescent females with diabetes were not more likely to engage in these behaviours compared to controls, the dangerous practice of intentional insulin omission or undertreatment to induce glycosuria and promote weight loss was reported in the present study by 11.5 percent of the diabetes sample. Indeed, in the present study, insulin omission was the most common weight loss method employed, after dieting, and was reported by 40 percent of those subjects with a DSM-IV eating disorder. Our diabetes sample reported complete omission of insulin doses and in some cases reducing the amount prescribed to lose weight. Individuals with type 1 diabetes quickly become aware that they can manipulate their insulin dose to promote weight loss (Rodin et al., 1991). Unfortunately, insulin manipulation for weight loss is a dangerous behaviour which can result in impaired metabolic control, and likely contributes to an increased risk of diabetes-related complications.

Despite the differences on the self-report (DSED) data and interview-based (EDE) data, diabetes and control subjects did not differ in terms of EDI-subscale scores or the EAT-26 total score. Although it is not clear why there were no detectable differences, the differences may become lost when considering the mean score of the total samples. Further, disordered eating attitudes and behaviours occur along a continuum and at varying rates depending on how the problem is defined (Marchi and Cohen, 1990). Self-report measures including the EAT and the EDI have been developed to assess symptoms which can discriminate between patients with clinical eating disorders and subjects with no disorder. However, these measures may not be as good at identifying "cases" in the general population, since there is a much lower prevalence of eating disorders and a broader spectrum of eating disturbances in the general population,
particularly in Western cultures where there is a high prevalence of dieting, drive for thinness and body dissatisfaction (Williams et al., 1982; Patton et al., 1990). Further, although the EDI and the EAT-26 may allow us to detect similarities and differences between groups, they are not able to reveal a subject's motivation or psychopathology behind their responses.

**Predictors of Eating Disorder Status**

It was postulated that several aspects of diabetes and its management may lower the threshold for the expression of an eating disorder in girls who are already vulnerable. The fact that the prevalence of eating disorders is increased in the diabetes population suggests that there may in fact be something specific to diabetes and its management that is causing the increased expression of these disorders. However, it is not clear from the present findings what specifically leads to an increased expression of eating disorders in young women with type 1 diabetes.

In order to determine if any diabetes-specific variables would predict eating disorder status, a stepwise discriminant function analysis was conducted which included age, BMI, SES, diabetes onset, and diabetes duration as predictors. The only predictor variable that was significantly related to eating disorder status and was entered into the model was age of diabetes onset. Subjects with diabetes and DSM-IV disorders were significantly older at the time that diabetes was diagnosed, and this variable accounted for 3 percent of the variance. In girls, body image and eating concerns appear to start around the ages of 9 and 10 (Wardle and Marsland, 1990; Koff and Rierdan, 1991; Mellin, et al., 1992; Thelen et al., 1992). However, these concerns appear to increase dramatically during the transition to junior high school (around 12 to 13
years of age) (Maloney et al. 1989; Richards et al., 1990; Killen et al., 1992; Atkins and Silber, 1993).

Levine and Smolak (1992) recently proposed a cumulative stressor model which proposes that the cumulative effects of normal developmental stresses may increase the risk of eating disorders in susceptible young females. These stresses include the weight gain associated with onset of puberty, the onset of dating, and the intensification of academic demands. Although all of these developmental challenges normally occur over 3-4 years, they are each associated with increased dissatisfaction with appearance and weight (Streigel-Moore et al., 1986; Gralen et al., 1990; Richards et al., 1990). Levine and Smolak's (1992) model proposes that, when pubertal weight gains and the development of heterosexual relationships of puberty occur during a time of increased academic stress, girls who have a slender body ideal may be more likely to react in ways that promote fear of fatness and a drive for thinness which underlie eating disorders (Fairburn and Garner, 1988). These interact with the pressures on girls to attain a slender body ideal and determine the onset of dieting or pathological eating disturbances (Levine and Smolak, 1992). Some of the stressors which they found to be associated with eating disturbances included onset of puberty, onset of dating, academic stress, and pressures from peers, family, and media to attain the "ideal" thin body (Levine et al., 1994). In a study of 79 grade 6 students who were followed up two years later, Smolak et al. (1993) found that higher EAT scores were associated with the synchrony of the onset of menstruation and dating, and the transition to junior high school.

Girls who are diagnosed with diabetes at the same time as weight and shape concerns are emerging and pubertal development is occurring, may be at an increased risk for the
development of pathological dieting and eating disorders. This is compatible with the additive stress model, which predicts that young women who are faced with multiple challenges and stresses all within a short period of time will become more distressed and this may result in disordered eating attitudes and behaviours (Petersen and Hamburg, 1986; Compas et al., 1989; Levine and Smolak, 1992; Levine et al., 1994). At screening, our diabetes sample had significantly higher BMI values compared to the controls. The average BMI for girls with DSM-IV disorders in the diabetes sample was 23.5 compared to 22.5 for the non-disordered group. However, this difference was not statistically significant and BMI was not a significant predictor of eating disorder status. Control subjects came from higher SES groups compared to the diabetes sample. However, SES was not a significant predictor of eating disorder status and was not significantly different between those above and below screening cut-off and in the eating disorder groups.

In the control sample, BMI was the only predictor variable that was significantly related to eating disorder status and it only accounted for 1% of the variance. In the total sample, diabetes onset and BMI accounted for just over 3% of the variance in predicting eating disorder status.

Although it was postulated that certain variables (i.e., age, BMI, SES) might predict eating disorder status in the samples, the present evidence suggests that in both the diabetes and general population groups, demographic variables alone do not adequately predict who will have an eating disorder. Rather, as outlined earlier, eating disorders seem to be multidimensional in nature, and environmental, individual, familial, and biological factors cluster together to predispose an individual to the development of an eating disorder. However,
the contribution of each of these variables can vary from individual to individual (Woodside, 1995).

Although, the hypothesized predictor variables did not prove to have predictive value, the data are compatible with the hypothesis that certain aspects of diabetes and its management increase the risk for the development of disturbances in eating behaviours and eating disorders (Rodin and Daneman, 1992). Potential diabetes-related risk factors for the development of eating disorders include the weight gain often experienced at diabetes diagnosis and associated with proper insulin administration, the chronic dietary restraint imposed by usual diabetes treatment regimens, and the availability of insulin omission or underdosing to control weight. It has been shown in females in the general population that an elevated BMI is associated with more body dissatisfaction and dieting (Steel et al., 1989), particularly during puberty and adolescence (Gralen et al., 1990; Wardel and Marsland, 1990). In the present study, BMI values were higher in diabetes subjects than controls, and subjects with DSM-IV disorders were more likely than those without eating disorders to have developed diabetes closer to puberty and adolescence. The coinciding weight gain associated with diabetes treatment and with pubertal onset, and the stresses that diabetes places on the individual and the family during a particularly sensitive time of development may increase the risk for the development of pathological eating attitudes and behaviours and eating disorders. However, since this study was not conducted prospectively, the nature of the relationship between BMI and age of diabetes onset to eating disorders is not clear and future prospective research is needed to further clarify these relationships.
STRENGTHS OF THE PRESENT STUDY

Some strengths of the present study include the following:

i) **Sample Size:** To date there have been seven interview-based controlled prevalence studies which have assessed the prevalence of eating disorders in young women with type 1 diabetes (Fairburn et al., 1991; Peveler et al., 1992; Streigel-Moore et al., 1992; Mannucci et al., 1995; Vila et al., 1993; Vila et al., 1995; Engstrom et al., 1999). In all but one of these studies (Streigel-Moore et al., 1992), the frequency of eating disorders in females with diabetes is at least 50% greater than among controls. However, all of these studies were small (ranging from 33-89 diabetes subjects and 24-148 controls). Only one of these differences reached statistical significance (Engstrom et al., 1999), and all of the studies had very low statistical power to determine whether the differences observed were statistically significant. Furthermore, the small sample sizes provided very little reliability in terms of their ability to assess prevalence. Consequently, the question of increased prevalence remained unresolved and controversial. The greatest strength of the present study was our ability to recruit large enough numbers of both diabetes and control subjects in order to adequately assess and compare the prevalence rates of eating disorders in the both groups. Power analysis was conducted prior to data collection in order to determine the minimum sample sizes need for each group. to our knowledge, this is the largest interview-based controlled prevalence study of eating disorders in adolescent females with type 1 diabetes.

ii) **Matched Control Group:** The present study included a large age and site-matched control group of adolescent females without diabetes. Although there have been numerous studies of eating disorders in patients with type 1 diabetes, very few have
utilized an age-matched control group. In addition, many of the previous controlled studies have utilized inappropriate samples which have included males (Fairburn et al., 1991; Peveler et al., 1992) and women in age ranges that are not at high risk for eating disorders (Mannucci et al., 1995).

iii) **Measures:** The present study utilized standardized methods to diagnose eating disorder status. Three reliable and valid screening measures (EDI subscales, EAT-26, and DSED) were employed to identify subjects who were at risk for an eating disorder. Self-report measures such as the EAT-26 and EDI are useful for screening for potential eating disorders. Subjects who scored above the screening cut-off (and 15% of those who did not meet cut-off) were then asked to complete a structured diagnostic interview (EDE). The EDE includes sections related to binge eating, purging, and dietary restraint, and allows for a clearer distinction between diabetes-related concerns and symptoms of eating disorders. Furthermore, the EDE can more clearly distinguish pathological eating and weight and shape concerns from the restricted food intake and food preoccupation that are inherent in diabetes management. The EDE is considered the current "gold standard" for eating disorder assessment. All measures were modified to include diabetes-related questions.

**LIMITATIONS OF THE PRESENT STUDY**

The following methodological limitations should be noted in the interpretation of the results of the present study:

i) **Response Rates:** A total of 84 percent of eligible females with type 1 diabetes agreed to participate in the study and 16 percent refused or did not return their forms. Of the
2,494 eligible non-diabetic controls, 26% percent refused to participate or did not return their consent forms and were considered non-participants. Fairburn and Belgin (1990) have previously noted that it is often difficult to obtain high response rates for eating disorder studies in community samples, and reported response rates have varied widely, ranging from 34-100% participation. Several authors have attempted to assess the prevalence of eating disorders in non-responders or study refusers and the results have varied. Some studies have reported significantly higher rates of eating problems in non-responders (Johnson-Sabine et al., 1988; King, 1989; Beglin and Fairburn, 1992), whereas others have found no evidence to suggest that eating disorders are over-represented in non-participants (Surtees et al., 1983; Roman-Clarkson et al., 1988; Wade et al., 1997). In a recently published study of 27 women who refused to participate in a study of eating disorders, eating disorders were not over-represented in the group of women who refused to participate (Wade et al., 1997). Although it was not possible to obtain information on non-responders, comparison of subjects within the three sites in the present study indicated no significant differences (except for SES) on the demographic variables or psychological variables assessed suggesting no systematic bias.

ii) **Socioeconomic Status:** There was a higher proportion of control subjects in the upper socioeconomic group compared to the diabetes sample. However, we found no statistical relationship between socioeconomic status and screening measures or eating disorder status. Previous research has demonstrated that eating disorders and dieting occurred most frequently in females in the middle-upper classes. However, more recent studies have revealed a broader social class distribution. This is supported by the
findings that subjects who scored above cut-off did not have significantly higher SES values compared to those who did not meet cut-off. In addition, SES did not appear to be associated with eating disorder status.

iii) **Two-Stage Design:** Although this study utilized a structured diagnostic interview to diagnose eating disorders, only subjects who were identified as being "at risk" (and a sub-set of those who were not at risk) for an eating disorder were interviewed due to limited resources and time constraints in the school boards. Such a design admittedly can lead to an underestimation of the prevalence of eating disorders in both the diabetes and control groups. In terms of the predictive value of the screening tools, the screening cut-off achieved a high negative predictive value but had low specificity. Consequently, a large number of the girls who scored above the cut-off and took part in the diagnostic interview did not have an eating disorder. This finding is not surprising given that the cut-off was set at a very low threshold in an attempt to capture all girls with any form of eating concern. However, the screening tools had a very high negative predictive value, and only a small number of girls who scored below cut-off would be expected to have an eating disorder. The interview data suggest that most of the "missed" girls in the present study had only recently begun to engage in disordered behaviours, and were not doing so at screening. Although most of the girls were interviewed within 3-4 months of the screening, due to scheduling difficulties, some girls were interviewed up to 6 months following initial screening. This suggests that the use of this type of screening tool is best used very close to the time of the interview. In order to improve this type of screening tool for future research or to use within the diabetes or school setting, researchers and clinicians should consider adjusting the cut-off criteria in an effort to
decrease the number of false positive screens (specificity) while not missing cases and increasing false negatives (sensitivity).

iv) Representativeness of the Diabetes Sample: Data were collected in three primary care diabetes clinics each within large children's hospitals that serve large surrounding areas. These clinics provide care to 60-70% of potential patients within their catchment areas. The demographics, management, and incidence of complications are similar between the three sites and comparable to other North American centers. Although this diabetes population is representative of the general diabetes population, this was not a population-based diabetes sample. Consequently, we can only definitively conclude that there is an increased prevalence of eating disorders in the diabetes clinic setting. Furthermore, treatment non-compliers are frequently absent from clinic and therefore these individuals may not have been included. The fact that females with eating disorders are often non-compliant suggests that the prevalence rate may actually be an underestimate.

SUMMARY AND FUTURE DIRECTIONS

The primary objective of this study was to provide information about the prevalence of disturbed eating attitudes, behaviours, and disorders in young women with and without type 1 diabetes mellitus.

This study is the first to have a large enough sample size of females in the age of highest risk for eating disorders to address adequately the question of prevalence. Evidence from this study suggests that individuals with diabetes are at an increased risk for the development of eating
disorders. Disturbed eating behaviours including insulin omission, were found to be common in young women with type 1 diabetes, and eating disorders that met DSM-IV criteria and their sub-threshold variants were approximately twice as common in adolescent females with type 1 diabetes as in their non-diabetic peers. Further, it appears that the timing of diabetes diagnosis (i.e. age of onset) may play an important role in the development of eating and body image disturbances.

These findings lend further support to our belief that certain aspects of diabetes and/or its management may lower the threshold for the development of eating disorders in vulnerable young women. Furthermore, our finding have several implications for diabetes management. Health care practitioners need to be aware of the risk of eating disorders among their adolescent female patients, and need to regularly screen for eating disturbances. Early detection of problems may prevent the development of full-blown disorders and associated diabetes-related complications.

The present study has both theoretical and practical implications. Eating disorders in adolescent females with type 1 diabetes pose a particular health risk in that they are associated with an approximately three-fold increase in the risk of diabetic retinopathy (Rydall et al., 1997). The present data emphasize the need for routine screening for eating disorders in adolescent females with type 1 diabetes and for the implementation of appropriate prevention and treatment strategies. In addition, these findings may demonstrate the interaction between individual and environmental factors in the pathogenesis of eating disorders similar to that observed in other high risk groups such as competitive athletes (Davis et al., 1999), models (Garner and Garfinkel, 1980) and ballet dancers (Abraham, 1996).
Although we can conclude from these findings that young women with diabetes are at an increased risk for the development of an eating disorder, this study does not answer why this is the case. Since this was a cross-sectional study in which the main purpose was to determine the prevalence of eating disorders, prospective studies are now needed to identify the factors which may explain this increased risk. Such future prospective studies should assess the whole spectrum of eating disorders in young women with diabetes and should include young girls who have not yet reached the age at risk for disturbed eating attitudes and behaviours. This will allow researchers to identify potential etiological factors for eating disorders before they become confounded by secondary symptoms, and will allow for a fuller understanding of the natural history of eating disorders. Prospective studies can also help us to identify what protective factors may play a role. Identifying potential risk factors for the development of eating disturbances, will further our knowledge of the etiology of eating disorders, and help in the development of causal models. In addition, they will aid in the formation and implementation of early intervention strategies in hopes of preventing the development of full-blown clinical eating disorders and decreasing the risk of long-term complications for young women with type 1 diabetes.

Future research should also focus on developing and assessing prevention programs for young women with type 1 diabetes within the diabetes clinic setting. These could include psychoeducation, individual, family, and group interventions. In addition, replication of the present findings using DSM-IV criteria would add further support to these findings. Future prevalence studies would ideally include a population-based diabetes sample and an age-
matched control group. Screening measures should have high sensitivity and if possible, structured clinical interviews should be conducted with all subjects to confirm diagnoses.
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APPENDICIES

Appendix A

Appendix B
APPENDIX A: Consent Forms and Ethical Approval

Study Consent Forms

Approval from Ethics and Scientific Review Committees
A Comparative Study of Eating Attitudes and Behaviours in Young Women with and without Type I Diabetes

We are conducting a multi-centre study of eating attitudes and behaviours amongst teenaged girls with and without diabetes. The study is open to all females, 12-18 years of age, with Type I diabetes for more than 1 year who attend the diabetes clinic at Hamilton Health Sciences Corporation. This study is also being conducted at diabetes clinics at the Hospital for Sick Children in Toronto, and The Children’s Hospital of Eastern Ontario in Ottawa. In addition, adolescent females without diabetes are also being recruited from various high schools and junior high schools in the Toronto, Ottawa, and Hamilton area.

If you agree to participate in this study, you will be asked to complete a confidential questionnaire about eating attitudes and behaviours. It will take approximately 20 minutes to complete the questionnaire. After we review the questionnaire, there will be a 50% chance that we will ask you to return to the hospital at a time convenient for you to complete a confidential interview about your eating attitudes and behaviours. This interview takes approximately 45 minutes. There are no known risks or adverse effects from completing the questionnaire or interview. Finally, during your clinic visit with your doctor, when you have your blood drawn for the tests ordered by your doctor (Hemoglobin A1c or “average blood sugar”), we will draw an additional 500 microliters of blood using the same venous puncture if possible.

If you participate in this study, our research assistant will also review your medical chart to obtain information about your diagnosis of diabetes, metabolic control, height and weight.

The knowledge we gain from completing this study may not directly benefit you. However, it will tell us whether young women with diabetes have different or similar eating attitudes and behaviours compared to their nondiabetic peers. This information will then help us to provide better diabetes care for children and adolescents with diabetes.

All information gathered in this study will be kept strictly confidential and will not be discussed with your doctor/s or parent/s without your written consent. If in the course of this study, we determine that you have a health problem, you will be offered referral to a health professional for treatment of the specific health problem. However, referral and/or treatment may be refused. In any case, the information will be kept confidential. If you refuse to participate, this will in no way affect your future medical care at any of the participating hospitals.

This study is being conducted by Dr. John Vandermolen (905-521-2100), Ms. Jennifer Jones (416-340-4828), and Dr. Gary Rodin (416-340-3044). If you have any questions, please do not hesitate to call.
I give permission to be enrolled in this study. I have had this study and the consent form explained to me. I have been assured that personal records related to this study will be kept confidential. I understand that I am free to withdraw myself from this study at any time. I further understand that if the study is not undertaken or if it is discontinued at any time, the quality of medical care will not be affected. I understand that if any knowledge gained from this study becomes available that could influence my decision to continue in this study, I will be promptly informed.

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<th>Signature of Participant (if &gt;16 years of age)</th>
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<td>Name of Witness</td>
<td>Signature of witness</td>
<td>Date</td>
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I have explained this study to the person authorized to sign above and am satisfied that it is understood.

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<tr>
<th>Name of Investigator</th>
<th>Signature of the investigator</th>
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A Comparative Study of Eating Attitudes and Behaviours in Young Women with and without Insulin Dependent Diabetes Mellitus

We are conducting a multi-centre study of eating attitudes and behaviours amongst teenaged girls with and without Type I diabetes. The study is open to females ages 12-18 years. We are recruiting adolescent females without diabetes from various high schools and junior high schools in the Toronto, Ottawa, and Hamilton areas. In addition, adolescent females with Type I diabetes are being recruited from the diabetes clinics at the Toronto Hospital for Sick Children, The Children's Hospital of Eastern Ontario in Ottawa, and the McMaster University Medical Clinic in Hamilton.

If you/your child agree(s) to participate in this study, she will be asked to complete a confidential questionnaire about eating attitudes and behaviours. It will take approximately 20 minutes to complete the questionnaire. After we review the questionnaire, a small percentage of the students will also be asked to take part in a confidential interview about eating attitudes and behaviours with the researcher. This interview takes approximately 45 minutes. There are no known risks or adverse effects from completing the questionnaire or interview. The questionnaire and interview will be conducted at your child's school. The timing will be discussed with the school principal in order to minimize interference with the daily routine. We also request that you (the parent or guardian) answer the 10 questions enclosed in order to assist us with our research.

All information gathered in this study will be kept strictly confidential and will not be discussed with your child's teachers, school officials, or doctor without written consent. Information related to this study will not appear in any school record, nor will school records be examined.

The knowledge we gain from completing this study will tell us whether young women with diabetes have different or similar eating attitudes and behaviours compared to their nondiabetic peers. This information will then help us to provide better diabetes care for children and adolescents with diabetes. We will also be able to determine the frequency of abnormal eating attitudes and behaviours among teenage girls in the general population and this will assist us and others to direct health education accordingly.
Participation in this study is completely voluntary, and your child may withdraw from the study at any time. If you/your child refuses to participate in this study, this will in no way affect her standing at school or your/her future care at any hospital. If in the course of this study, we determine that your child has a health problem, she will be contacted directly and offered referral to a health professional for treatment.

Your cooperation would be greatly appreciated in helping make our research a success.

This study is being coordinated in Toronto by Ms. Jennifer Jones (416-340-4828), and Dr. Gary Rodin (416-340-3044). If you have any questions, please do not hesitate to call.
CONSENT FORM

Please complete this form whether or not you wish to participate, and return to the researchers at your school.

The information collected is confidential and protected under the Municipal Freedom of Information and Protection of Privacy Act, 1989.

I have read and understood the request for me/my daughter to participate in the study, A Comparative Study of Eating Attitudes and Behaviours in Young Women with and without Insulin Dependent Diabetes Mellitus. I have discussed it with my daughter and --

[ ] I give permission to participate

[ ] I do NOT give permission to participate

Name of Student: (please print) Signature of Student: Date:

________________________  ______________________________________

Name of Parent/Guardian* (please print) Signature of Parent/Guardian:

________________________  ______________________________________

*Note: If you are 18 years of age or over, parental consent is not required.
May 27, 1996

Dr. Margaret Lawson
Endocrinology
CHEO INTRA

Re: Proposal 96/17E- A Comparative Study of Eating Attitudes and Behaviours in Young Women with and without Insulin-dependent Diabetes Mellitus

Dear Dr. Lawson:

Thank you for your letter of May 13, 1996 responding to the recommended changes of the Research Ethics Committee in our letter of May 6, 1996.

Please accept this letter as written approval from the Research Ethics Committee for these changes.

Sincerely,

Alasdair Hunter, M.D.
Chairman
Research Ethics Committee

rw
DATE: 13 November 1997

TO: Drs. J. Van Der Meulen/J.M. Jones

FROM: D. Rosenbloom, Pharm.D.
Chair, Research Advisory Group (Ethics Board)

RE: A comparative study of eating attitudes, behaviours and disorders in young women with and without type I diabetes mellitus - Project #97-138

This is to confirm that the above project was reviewed and approved by the Research Advisory Group (Ethics Board) on October 22, 1997. The revised consent form has also been approved.

Please note that it is the responsibility of the researcher to ensure that evidence of consent is inserted in the patient's health record. In the case of invasive or otherwise risky research, the researcher might consider the advisability of keeping personal copies.

D. Rosenbloom, Pharm.D.
Chair, Research Advisory Group
THE HOSPITAL FOR SICK CHILDREN

RESEARCH ETHICS BOARD

Approval & Terms of Agreement

APPLICANT/S: Drs. G. Rodin, D. Daneman, M. Olmsted, M. Lawson, and Ms. A. Rydall

PROJECT TITLE: "The Effect of Psychoeducation on Adolescent Females with IDDM"

DATE OF APPROVAL: June 17, 1994

EXPIRY DATE: June 1995

MEMBERS OF THE BOARD*: Dr. S. Zlockin, Chair Ms. M. McAllister
Mrs. M. P. Armstrong Dr. A. Moore
Dr. S. Jacobson Prof. J. Mosher
Dr. P. Joshi Dr. K. Periman
Dr. M. Lovett

*Meeting may not have been attended by all members.

I agree to carry out the proposed research involving human subjects in accordance with the protocol passed by the scientific peer review committee and the Research Ethics Board and using the approved consent form/s (if applicable). I shall notify the department chief and the Research Ethics Board prior to implementing any significant changes in the protocol.

SIGNATURE (INVESTIGATOR) DATE 1994
SIGNATURE (DEPARTMENT HEAD) DATE 1994

The Research Ethics Board of the Hospital for Sick Children has reviewed and approved the above-named project.

Chair, Research Ethics Board DATE 1994
September 23, 1997

Jennifer Jones
The Toronto Hospital
200 Elizabeth Street
CW2 306
Toronto, Ontario
M5G 2L4

Dear Jennifer,

Thank you for your recent research application. Permission is granted to conduct your study, "A Comparative Study of Eating Attitudes, Type I Diabetes Mellitus (IDDM)", on the following conditions:

1. that the approval of the principal of the participating school(s) be obtained;
2. that the researcher take all necessary steps to ensure that the data gathered will remain anonymous and be treated with confidentiality;
3. that parental consent for student involvement in the study be arranged prior to commencing the study;
4. that the questionnaire be developed in cooperation with the Coordinator of Research and Evaluation Services prior to its distribution in the school;
5. that the consent form be reviewed by Research and Evaluation Services staff prior to its distribution in the school;
6. that a final summary report be sent to interested school staff members and the Coordinator of Research and Evaluation Services upon completion of the study.

Best wishes for success in your survey.

Yours sincerely,

Bill Hogarth,
Director of Education
and Secretary-Treasurer

CC: D. Hamilton, Coordinator of Research and Evaluation Services
Sharon MacFarlane, Markville SS
Jim Gilliland, Middlefield CI
Ted Broadstock, James Robinson PS
Sandy Arbuck, Ashton Meadows PS

The York Region Board of Education
York Region Schools Purposeful, Dynamic, Inviting
22 April 1997

G. Rodin, M. Lawson
D. Daneman, M. Olmsted
S. Bennett, J. Jones
200 Elizabeth St.
CW-2-306
Toronto, Ont.
M5G 2C4

Dear Drs. Rodin, Lawson, Olmsted, Bennett, Ms. Jones:

The Research Review Committee considered your proposal, “A Comparative Study of Eating Attitudes, Behaviours, and Disorders in Young Women with and without Type I Diabetes”, in its meeting of April 16, 1997. The proposal was approved. The Committee was interested in knowing:

A more specific definition of “Subthreshold Eating Disorder” as discussed in p. 3 of Appendix 2; and suggests that this concept be included as part of the presentation in the auditorium sessions.

Sincerely,

[Signature]

Rob Brown
Chair, Research Review Committee
Matiopolitan
Council
des
Écoles
du Grand
Toronto

Office of the Director of Education

August 26, 1997

Jennifer M. Jones
Study Coordinator
Children's Hospital of Eastern Ontario
401 Smyth
Ottawa, Ontario
K1H 8L1

Dear Ms. Jones:

RE: A Comparative Study of Eating
Attitudes, Behaviours and Disorders
in Young Women with and without
Type I Diabetes Mellitus (IDDM).

I am pleased to inform you that permission has been granted to
conduct the above-noted survey.

If you require any further assistance, please do not hesitate
to contact James D. Feeney, Assistant Superintendent, Research,
at (416) 222-8282, extension 5335.

Best wishes for success with your study.

Yours sincerely,

J. Stewart
Director of Education
October 15, 1997

Jennifer Jones
Research Coordinator
Department of Psychiatry
The Toronto Hospital
200 Elizabeth Street
Toronto, ON
M5G 2C4

Dear Jennifer Jones,

Your request to carry out a study entitled “A Comparative Study of Eating Attitudes, Behaviours, and Disorders in Young Women with and without Type I Diabetes” has been reviewed and approved by the Outside Research Committee.

The Committee recommends that the parental consent form be changed to include the following statement: “If you/your child refuses to participate in this study, this will in no way affect her standing at school or your/her future care at any hospital.”

Please contact Janice Dyer in the Research and Evaluation Department at 394-7319 to determine which schools will be participating in the research. In addition, please complete the attached Freedom of Information form and return it to the Research Department.

Sincerely yours,

[Signature]

Ken McGhee
Superintendent of Program
12 August 1996

Dr. Susan Bennett  
Department of Pediatrics  
Children's Hospital of Eastern Ontario  
401 Smyth Road  
Ottawa, ON

Re: Research Proposal: "A comparative study of eating attitudes and behaviours in young women with and without insulin-dependent mellitus"

Dear Dr. Susan Bennett:

This letter is to confirm that the Ottawa-Carleton Research Advisory Committee has approved your request for the cooperation of schools for your research project. Your request will be forwarded to schools in September. Before that time, several minor changes remain to be resolved and approval of the Research Ethics Review Committee at your institution is required.

Yours sincerely,

[Signature]

Linda K. Clarke, Ed.D.  
Research Consultant:
APPENDIX B: Measures

Demographic Form

Eating Attitudes Test (EAT-26)

Eating Disorder Inventory (EDI)

Diagnostic Survey for Eating Disorders (DSED)
  -modified for diabetes

Diagnostic Survey for Eating Disorders (DSED)
  -for nondiabetic subjects

Eating Disorder Examination (EDE) -modified
  -first five pages (full interview available)
**DIRECTIONS:** (To be completed by parent/guardian)

We are conducting this study in order to further understand the risk factors and prevalence of eating disturbances in young women. We are asking all parents/guardians to help by providing us with demographic information so that we may have a clear understanding of who has taken part in this study. We would appreciate if you would complete the following questions and return them with the attached consent form with your daughter. If you do not wish to answer any questions, please leave it blank. PLEASE BE ADVISED THAT ALL ANSWERS WILL BE KEPT STRICTLY CONFIDENTIAL.

---

1. **What is the highest level of education that you have completed?**
   
   *(Please check ONE box in each column)*

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<th>1. Less than eighth grade</th>
<th>2. Junior high school (grade 9)</th>
<th>3. Some high school (grade 10 or 11)</th>
<th>4. High school graduate (grade 12 or 13)</th>
<th>5. One or two years of college, university, or specialized training</th>
<th>6. University graduate (bachelor's degree)</th>
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2. **What is your present employment status?** *(Please check all relevant boxes)*

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3. What is father's/guardian's usual occupation?
   Title: 
   Kind of work: 

4. What is mother's/guardian's usual occupation?
   Title: 
   Kind of work: 

5. What is father's/guardian's country of birth?

6. What is mother's/guardian's country of birth?

7. What is your child's country of birth?

8. If your child was not born in Canada, how long has your child lived in Canada?
   [] Months
   [] Years

9. What is the main language spoken in child's primary residence?

10. What is the postal code for your child’s primary residence?

   Thank you for your cooperation.
**INSTRUCTIONS:**

This is a scale which measures a variety of attitudes, feelings and behaviours. Some of the items relate to food and eating. Others ask you about your feelings about yourself. THERE ARE NO RIGHT OR WRONG ANSWERS SO TRY VERY HARD TO BE COMPLETELY HONEST IN YOUR ANSWERS. RESULTS ARE COMPLETELY CONFIDENTIAL. Read each question and put a dark “X” under the column which best applies to you. Please answer each question very carefully. Thank you.

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<td>1</td>
<td>I eat sweets and carbohydrates without feeling nervous.</td>
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<td>2</td>
<td>I think that my stomach is too big.</td>
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<td>3</td>
<td>I eat when I am upset.</td>
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<td>4</td>
<td>I stuff myself with food.</td>
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<td>5</td>
<td>I think about dieting.</td>
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<td>6</td>
<td>I think that my thighs are too large.</td>
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<td>7</td>
<td>I feel extremely guilty after overeating.</td>
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<td>8</td>
<td>I think that my stomach is just the right size.</td>
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<td>9</td>
<td>I am terrified of gaining weight.</td>
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<td>10</td>
<td>I feel satisfied with the shape of my body.</td>
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<tr>
<td>11</td>
<td>I exaggerate or magnify the importance of weight.</td>
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<tr>
<td>12</td>
<td>I have gone on eating binges where I have felt that I could not stop.</td>
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<tr>
<td>13</td>
<td>I like the shape of my buttocks.</td>
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<tr>
<td>14</td>
<td>I am preoccupied with the desire to be thinner.</td>
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<tr>
<td>15</td>
<td>I think about bingeing (overeating).</td>
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<tr>
<td>16</td>
<td>I think that my hips are too big.</td>
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<tr>
<td>17</td>
<td>I eat moderately in front of others and stuff myself when they're gone.</td>
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<tr>
<td>18</td>
<td>If I gain a pound, I worry that I will keep gaining.</td>
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<tr>
<td>19</td>
<td>I have the thought of trying to vomit in order to lose weight.</td>
<td></td>
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<tr>
<td>20</td>
<td>I think that my thighs are just the right size.</td>
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<tr>
<td>21</td>
<td>I think my buttocks are too large.</td>
<td></td>
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<tr>
<td>22</td>
<td>I eat or drink in secrecy.</td>
<td></td>
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<tr>
<td>23</td>
<td>I think that my hips are just the right size.</td>
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</tbody>
</table>
EATING ATTITUDES TEST

Present weight: [ ] Pounds    Height: [ ] Inches

Highest weight at present height: [ ] Pounds    At what age: [ ]

Lowest weight at present height: [ ] Pounds    At what age: [ ]

INSTRUCTIONS:

Please place an "X" in the box which applies best to each of the statements. All of the results will be strictly confidential. Most of the questions directly relate to food or eating, although other types of questions have been included. Please answer each question carefully. Thank you.

(Please check ONE box for each statement)

<table>
<thead>
<tr>
<th>Statement</th>
<th>Always</th>
<th>Usually</th>
<th>Occas.</th>
<th>Sometimes</th>
<th>Rarely</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Am terrified about being overweight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2. Avoid eating when I am hungry</td>
<td></td>
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<tr>
<td>3. Find myself preoccupied with food</td>
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<td>4. Have gone on eating binges where I feel that I may not be able to stop</td>
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<td>5. Cut my food into small pieces</td>
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<tr>
<td>6. Aware of calorie content of foods that I eat</td>
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<tr>
<td>7. Particularly avoid foods with a high carbohydrate content. (e.g., bread, rice, potatoes, etc.)</td>
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<td>8. Feel that others would prefer if I ate more</td>
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<td>9. Vomit after I have eaten</td>
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<tr>
<td>10. Feel extremely guilty after eating</td>
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<tr>
<td>11. Am preoccupied with a desire to be thinner</td>
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</tbody>
</table>
(Please check ONE box for each statement)

<table>
<thead>
<tr>
<th></th>
<th>Always</th>
<th>Usually</th>
<th>Often</th>
<th>Sometimes</th>
<th>Rarely</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>12.</td>
<td>Think about burning up calories when I exercise</td>
<td></td>
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<tr>
<td>13.</td>
<td>Other people think that I am too thin</td>
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<tr>
<td>14.</td>
<td>Am preoccupied with the thought of having fat on my body</td>
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<tr>
<td>15.</td>
<td>Take longer than others to eat my meals</td>
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<tr>
<td>16.</td>
<td>Avoid foods with sugar in them</td>
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<tr>
<td>17.</td>
<td>Eat diet foods</td>
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<tr>
<td>18.</td>
<td>Feel that food controls my life</td>
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<tr>
<td>19.</td>
<td>Display self-control around food</td>
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<tr>
<td>20.</td>
<td>Feel that others pressure me to eat</td>
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<tr>
<td>21.</td>
<td>Give too much time and thought to food</td>
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<tr>
<td>22.</td>
<td>Feel uncomfortable after eating sweets</td>
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<tr>
<td>23.</td>
<td>Engage in dieting behaviour</td>
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<tr>
<td>24.</td>
<td>Like my stomach to be empty</td>
<td></td>
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<tr>
<td>25.</td>
<td>Enjoy trying new rich foods</td>
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<tr>
<td>26.</td>
<td>Have the impulse to vomit after meals</td>
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</tbody>
</table>

Thank you for your cooperation
DIRECTIONS:

There are no right or wrong answers to the following questions. Many of the questions relate to personal information and may be answered differently by different people. Please take your time and try to answer all of the questions carefully. Please answer the questions on your own. Remember, all of your answers will be kept strictly confidential. If you have trouble with any of the questions, please do not hesitate to call Colleen Shaw at 340-4319 between 8 a.m. and 4 p.m., Monday to Friday.

1. How old are you?
   □ Years □ Months

2. How old were you when you developed diabetes?
   □ Years □ Months

3. How many insulin injections do you now take per day?
   □ Number

4. Do you have any medical problems other than diabetes? (Please check ONE box)
   □ 1. No
   □ 2. Yes, what are they?
      a. 
      b. 
      c. 
      d. 

5. Are you on any medications other than insulin? (Please check ONE box)
   □ 1. No
   □ 2. Yes, Which one(s)?
      a. 
      b. 
      c. 
      d. 

6. What is your current height?
   □ Feet □ Inches

7. What is your current weight?
   □ Pounds

8. What was your maximum weight ever?
   □ Pounds

9. What weight would you most like to be?
   □ Pounds

10. How do you see yourself NOW, at your current weight? (Please check ONE box)
    □ 1. Very thin
    □ 2. A bit thin
    □ 3. Normal weight
    □ 4. A bit overweight
    □ 5. Very overweight

11. How do you FEEL about yourself at your current weight? (Please check ONE box)
    □ 1. Very unhappy
    □ 2. A bit unhappy
    □ 3. A bit happy
    □ 4. Very happy
12. How good is your metabolic control most of the time?  
(Please check ONE box)
- [ ] 1. Terrible
- [ ] 2. Not very good
- [ ] 3. Good
- [ ] 4. Very good

13. How satisfied are you with your current level of metabolic control?  
(Please check ONE box)
- [ ] 1. Not at all satisfied
- [ ] 2. A little satisfied
- [ ] 3. Satisfied
- [ ] 4. Very satisfied

14. How often do you do each of the following things:  
(Please check ONE box for each question)

<table>
<thead>
<tr>
<th></th>
<th>Everyday</th>
<th>Several times weekly</th>
<th>About once each week</th>
<th>Several times each month</th>
<th>Once in a while</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. How often do you weigh yourself or measure your body size</td>
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<tr>
<td>b. Reduce portions of meals to control your weight</td>
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<tr>
<td>c. Skip meals to control your weight</td>
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<td>d. Completely stop eating to control your weight</td>
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<tr>
<td>e. Reduce calories below diabetic diet to control your weight</td>
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<tr>
<td>f. Exercise to control your weight</td>
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<tr>
<td>g. Omit or decrease insulin dose to control your weight</td>
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<tr>
<td>h. Use diet pills to control your weight</td>
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<tr>
<td>i. Use diuretics (water pills) to control your weight</td>
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<td>j. Other, please specify:</td>
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</table>

15. Are you currently on a diet to control/lose weight (aside from a diet to control your diabetes)?  
(Please check ONE box)
- [ ] 1. No
- [ ] 2. Yes
16. Have you ever had an episode of eating a lot more food than other people your age would eat in a similar situation (binge eating)? *(Please check ONE box)*

- [ ] 1. No *(skip to question 22)*
- [ ] 2. Yes

17. How frequently over the past 3 months have you had such an eating binge? *(Please check ONE box)*

- [ ] 1. Never
- [ ] 2. Less than once a month
- [ ] 3. About once a month
- [ ] 4. 2-3 times a month
- [ ] 5. Once a week
- [ ] 6. Several times a week
- [ ] 7. Every day
- [ ] 8. More than once a day

18. What would you typically eat during a binge?

19. When does bingeing usually occur? *(Please check ONE box)*

- [ ] 1. Only when I feel hypoglycemic
- [ ] 2. Sometimes when I feel hypoglycemic and sometimes not
- [ ] 3. Only when I do not feel hypoglycemic

20. How do you feel about your bingeing? *(Please check ONE box)*

- [ ] 1. Very unhappy
- [ ] 2. A bit unhappy
- [ ] 3. A bit happy
- [ ] 4. Very happy

21. Please check the number to show how often you do any of the following things when you are binge eating:

*(Please check ONE box for each question)*

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Sometimes</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. I eat very rapidly</td>
<td>[ ]</td>
<td>[ ]</td>
<td>[ ]</td>
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<tr>
<td>b. I feel out of control when I eat</td>
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<tr>
<td>c. I feel miserable or annoyed after a binge</td>
<td>[ ]</td>
<td>[ ]</td>
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<tr>
<td>d. I get uncontrollable urges to eat until I feel physically ill</td>
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<td>[ ]</td>
<td>[ ]</td>
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<tr>
<td>e. I binge eat in private</td>
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<td>[ ]</td>
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</tbody>
</table>

22. Have you ever vomited (thrown up) after eating in order to get rid of the food eaten? *(Please check ONE box)*

- [ ] 1. No *(skip to question 24)*
- [ ] 2. Yes
23. How frequently over the past 3 months have you made yourself vomit to get rid of food? (Please check ONE box)

☐ 1. Never
☐ 2. Less than once a month
☐ 3. About once a month
☐ 4. 2-3 times a month
☐ 5. Once a week
☐ 6. 2-3 times a week
☐ 7. Every day
☐ 8. More than once a day

24. Have you ever taken less insulin than you should have taken because you thought it would control your weight? (Please check ONE box)

☐ 1. No (skip to question 26)
☐ 2. Yes

25. How frequently over the past 3 months have you taken less insulin than you should have taken as a method of controlling your weight? (Please check ONE box)

☐ 1. Never
☐ 2. Less than once a month
☐ 3. About once a month
☐ 4. 2-3 times a month
☐ 5. Once a week
☐ 6. 2-3 times a week
☐ 7. Every day
☐ 8. More than once a day

26. Have you ever used laxatives because you thought they could control your weight? (Please check ONE box)

☐ 1. No (skip to question 28)
☐ 2. Yes

27. How frequently over the past 3 months have you used laxatives as a method of controlling your weight? (Please check ONE box)

☐ 1. Never
☐ 2. Less than once a month
☐ 3. About once a month
☐ 4. 2-3 times a month
☐ 5. Once a week
☐ 6. 2-3 times a week
☐ 7. Every day
☐ 8. More than once a day

28. Have you ever used any other methods of controlling your weight? (i.e., diet pills, diuretics, extreme exercise, other) (Please check ONE box)

☐ 1. No
☐ 2. Yes, please list everything you have used:

   a. 
   b. 
   c. 
   d. 

895 - 06
29. Have you ever competed or seriously trained in any of the following physical activities? (Please check all that apply)

- Yes
- No

- Distance running
- Track and field
- School team (e.g., volleyball, field hockey, etc.)
- Lifting weights
- Dancing
- Gymnastics
- Tennis, squash, racquetball
- Swimming
- Modelling
- Other sport, please specify

30. Have you ever been told by a doctor that you have an eating disorder? (Please check ONE box)

- No (skip to question 32)
- Yes

31. Have you ever had treatment for an eating disorder?

- No
- Yes

32. Have you started having your periods? (Please check ONE box)

- No
- Yes, at what age?

33. When did you have your last period? (Please check ONE box)

- Within the past 1 month
- Within the past 3 months
- More than 3 months ago

34. Who do you currently live with? (Please check ONE box)

- Both parents
- One parent
- Partner
- Roommate(s)
- Other, please specify

35. Total number of people in your household (including you)?

Number

36. What grade are you in at school?

Grade

37. What is the highest level of education that your parents have completed? (Please check ONE box in each column)

<table>
<thead>
<tr>
<th>Your mother</th>
<th>Your father</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Less than seventh grade</td>
<td>1. Less than seventh grade</td>
</tr>
<tr>
<td>2. Junior high school (grade 9)</td>
<td>2. Junior high school (grade 9)</td>
</tr>
<tr>
<td>3. Some high school (grade 10 or 11)</td>
<td>3. Some high school (grade 10 or 11)</td>
</tr>
<tr>
<td>4. High school graduate (grade 12 or 13)</td>
<td>4. High school graduate (grade 12 or 13)</td>
</tr>
<tr>
<td>5. One or two years of college, university, or specialized training</td>
<td>5. One or two years of college, university, or specialized training</td>
</tr>
<tr>
<td>6. University graduate (bachelor’s degree)</td>
<td>6. University graduate (bachelor’s degree)</td>
</tr>
<tr>
<td>7. Graduate training (e.g., M.D., M.B.A., M.A.)</td>
<td>7. Graduate training (e.g., M.D., M.B.A., M.A.)</td>
</tr>
</tbody>
</table>
38. What is the present employment status of your parents? (Please check ONE box in each column)

<table>
<thead>
<tr>
<th>Status</th>
<th>Since when (mother)</th>
<th>Since when (father)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Unemployed due to illness</td>
<td></td>
<td></td>
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<tr>
<td>2. Unemployed for other reasons</td>
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<tr>
<td>3. Employed full-time</td>
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<tr>
<td>4. Employed part-time</td>
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<tr>
<td>5. Full-time homemaker</td>
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<tr>
<td>6. Student</td>
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<tr>
<td>7. Retired</td>
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</tbody>
</table>

39. What is your father's usual occupation?
Title: __________________________
Kind of work he does: __________________________

40. What is your mother's usual occupation?
Title: __________________________
Kind of work she does: __________________________

41. How do you identify yourself? (Please check ONE box)

- [ ] 1. White
- [ ] 2. Black
- [ ] 3. Asian
- [ ] 4. Indian
- [ ] 5. Other, please specify: __________________________
**DIRECTIONS:**

There are no right or wrong answers to the following questions. Many of the questions relate to personal information and may be answered differently by different people. Please take your time and try to answer all of the questions carefully. Please answer the questions on your own. Remember, all of your answers will be kept strictly confidential.

1. **How old are you?**
   - [ ] Years
   - [ ] Months

2. **Do you have any medical problems?** *(Please check ONE box)*
   - [ ] 1. No
   - [ ] 2. Yes, what are they?
     - [a. ]
     - [b. ]
     - [c. ]
     - [d. ]

3. **Are you on any medications?** *(Please check ONE box)*
   - [ ] 1. No
   - [ ] 2. Yes; Which one(s)?
     - [a. ]
     - [b. ]
     - [c. ]
     - [d. ]

4. **What is your current height?**
   - [ ] Feet
   - [ ] Inches

5. **What is your current weight?**
   - [ ] Pounds

6. **What was your maximum weight ever?**
   - [ ] Pounds

7. **What weight would you most like to be?**
   - [ ] Pounds

8. **How do you see yourself NOW, at your current weight?** *(Please check ONE box)*
   - [ ] 1. Very thin
   - [ ] 2. A bit thin
   - [ ] 3. Normal weight
   - [ ] 4. A bit overweight
   - [ ] 5. Very overweight

9. **How do you FEEL about yourself at your current weight?** *(Please check ONE box)*
   - [ ] 1. Very unhappy
   - [ ] 2. A bit unhappy
   - [ ] 3. A bit happy
   - [ ] 4. Very happy
10. How often do you do each of the following things:

(Please check ONE box for each question)

<table>
<thead>
<tr>
<th></th>
<th>Everyday</th>
<th>Several times weekly</th>
<th>About once each week</th>
<th>Several times each month</th>
<th>Once in a while</th>
<th>Never</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. How often do you weigh yourself or measure your body size</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Reduce portions of meals to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c. Skip meals to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d. Completely stop eating to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e. Reduce calories to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>f. Exercise to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>g. Use diet pills to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>h. Use diuretics (water pills) to control your weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>i. Other, please specify:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

11. Are you currently on a diet to control/lose weight? (Please check ONE box)

☐ 1. No  ☐ 2. Yes

12. Have you ever had an episode of eating a lot more food than other people your age would eat in a similar situation (binge eating)? (Please check ONE box)

☐ 1. No (skip to question 17)  ☐ 2. Yes

13. How frequently over the past 3 months have you had such an eating binge? (Please check ONE box)

14. What would you typically eat during a binge?

15. How do you FEEL about your bingeing? *(Please check ONE box)*

- [ ] 1. Very unhappy
- [ ] 2. A bit unhappy
- [ ] 3. A bit happy
- [ ] 4. Very happy

16. Please check the number to show how often you do any of the following things when you are binge eating:

*(Please check ONE box for each question)*

<table>
<thead>
<tr>
<th>Option</th>
<th>Never</th>
<th>Sometimes</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. I eat very rapidly</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. I feel out of control when I eat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>c. I feel miserable or annoyed after a binge</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>d. I get uncontrollable urges to eat until I feel physically ill</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>e. I binge eat in private</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

17. Have you ever vomited (thrown up) after eating in order to get rid of the food eaten? *(Please check ONE box)*

- [ ] 1. No *(skip to question 19)*
- [ ] 2. Yes
18. How frequently over the past 3 months have you made yourself vomit to get rid of food? (Please check ONE box)

☐ 1. Never
☐ 2. Less than once a month
☐ 3. About once a month
☐ 4. 2-3 times a month
☐ 5. Once a week
☐ 6. 2-3 times a week
☐ 7. Every day
☐ 8. More than once a day

19. Have you ever used laxatives because you thought they could control your weight? (Please check ONE box)

☐ 1. No (skip to question 21)
☐ 2. Yes

20. How frequently over the past 3 months have you used laxatives as a method of controlling your weight? (Please check ONE box)

☐ 1. Never
☐ 2. Less than once a month
☐ 3. About once a month
☐ 4. 2-3 times a month
☐ 5. Once a week
☐ 6. 2-3 times a week
☐ 7. Every day
☐ 8. More than once a day

21. Have you ever used any other methods of controlling your weight? (i.e., diet pills, diuretics, extreme exercise, other) (Please check ONE box)

☐ 1. No
☐ 2. Yes, please list everything you have used:
   a.
   b.
   c.
   d.

22. Have you ever competed or seriously trained in any of the following physical activities? (Please check all that apply)

☐ 1. Distance running
☐ 2. Track and field
☐ 3. School team (e.g., volleyball, field hockey, etc.)
☐ 4. Lifting weights
☐ 5. Dancing
☐ 6. Gymnastics
☐ 7. Tennis, squash, racquetball
☐ 8. Swimming
☐ 9. Modelling
☐ 10. Other sport. please specify
   a.
   b.
   c.
   d.

☐ 11. I have never been seriously involved in any of the above activities or any other sports.

23. Have you ever been told by a doctor that you have an eating disorder? (Please check ONE box)

☐ 1. No (skip to question 25)
☐ 2. Yes
24. Have you ever had treatment for an eating disorder?

☐ 1. No
☐ 2. Yes

25. Have you started having your periods?
(Please check ONE box)

☐ 1. No
☐ 2. Yes, at what age?

☐ ___ Years old

26. When did you have your last period?
(Please check ONE box)

☐ 1. Within the past 1 month
☐ 2. Within the past 3 months
☐ 3. More than 3 months ago

27. Who do you currently live with?
(Please check ONE box)

☐ 1. Both parents
☐ 2. One parent
☐ 3. Partner
☐ 4. Roommate(s)
☐ 5. Other, please specify

a.

b.

28. Total number of people in your household (including you)?

☐ ___ Number

29. What grade are you in at school?

Grade ☐ ___
EDE (Modifie-.,
(Diagnostic Version)
(Edition 11.5 D, 1992)

Oxford University Department of Psychiatry

Rate 7 if the question is ‘not applicable’ to this subject.

<table>
<thead>
<tr>
<th>Severity ratings</th>
<th>Frequency ratings (based on a 28-day month)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - Absence of the feature</td>
<td>0 - Absence of the feature</td>
</tr>
<tr>
<td>1 - Feature almost, but not quite, absent</td>
<td>1 - Feature present on 1 to 5 days</td>
</tr>
<tr>
<td>2 -</td>
<td>2 - Feature present on 6 to 12 days</td>
</tr>
<tr>
<td>3 - Severity midway between 0-6</td>
<td>3 - Feature present on 13 to 15 days</td>
</tr>
<tr>
<td>4 -</td>
<td>4 - Feature present on 16 to 22 days</td>
</tr>
<tr>
<td>5 - Feature present to a degree not quite severe</td>
<td>5 - Feature present almost every day (23</td>
</tr>
<tr>
<td>6 - Feature present to an extreme degree</td>
<td>to 27 days)</td>
</tr>
</tbody>
</table>

If it is difficult to choose between two ratings, the lower rating (i.e., less symptomatic) should be used.

**INTERVIEW SCHEDULE**

**INTRODUCTION**

[It is best to start the interview proper by asking a number of introductory questions designed to obtain a general picture of the subject’s eating habits. Suitable questions are suggested below.]

To begin with I should like to get a general picture of your eating habits over the last four weeks.

Have your eating habits varied much day-by-day?

Have weekdays differed from weekends?

What about the previous two months?
Rate 7 if the question is 'not applicable' to this subject.

<table>
<thead>
<tr>
<th>Severity ratings</th>
<th>Frequency ratings (based on a 28-day month)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - Absence of the feature</td>
<td>0 - Absence of the feature</td>
</tr>
<tr>
<td>1 - Feature almost, but not quite, absent</td>
<td>1 - Feature present on 1 to 3 days</td>
</tr>
<tr>
<td>2 -</td>
<td>2 - Feature present on 6 to 12 days</td>
</tr>
<tr>
<td>3 - Severity midway between 0-6</td>
<td>3 - Feature present on 13 to 15 days</td>
</tr>
<tr>
<td>4 - Feature present to a degree not quite severe</td>
<td>4 - Feature present on 16 to 22 days</td>
</tr>
<tr>
<td>enough to justify a rating of 6</td>
<td></td>
</tr>
<tr>
<td>5 -</td>
<td>5 - Feature present almost every day (23 to 27 days)</td>
</tr>
<tr>
<td>6 - Feature present to an extreme degree</td>
<td>6 - Feature present every day</td>
</tr>
</tbody>
</table>

If it is difficult to choose between two ratings, the lower rating (i.e., less symptomatic) should be used.

**INTERVIEW SCHEDULE**

**INTRODUCTION**

[It is best to start the interview proper by asking a number of introductory questions designed to obtain a general picture of the subject's eating habits. Suitable questions are suggested below.]

To begin with I should like to get a general picture of your eating habits over the last four weeks.

Have your eating habits varied much day-by-day?

Have weekdays differed from weekends?

What about the previous two months?
PATTERN OF EATING

I would like to get an idea of your pattern of eating. Over the past 28 days, on how many days have you eaten the following meals.

[Rate each meal separately, usually accepting the subject's classification. Meals should be rated only if they did not lead on to a 'binge' and were not purged. 'Brunch' should generally be classified as lunch. Rate 00 if no meals taken, and 28 if meals taken on each day.]

PRELIMINARY DIABETIC QUESTION:
PATTERN OF EATING: (Details of usual weekday pattern)

<table>
<thead>
<tr>
<th>No/never (0)</th>
<th>Sometimes (1)</th>
<th>Yes/always (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A) Breakfast</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B) Mid-morning snack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C) Lunch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D) Afternoon snack</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E) Evening meal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>F) Evening snack</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

G. STABILITY OF PATTERN

☐ 0 - Highly erratic
☐ 1 - Somewhat erratic
☐ 2 - Regular (regular with 2 exceptions)

H. EFFECT OF MEAL OMISSION - (1 HOUR)

☐ 0 - None
☐ 1 - Slight
☐ 2 - Marked
☐ 3 - Not applicable; does not omit meals

I. ACTUAL MEAL OMISSION - (IN PAST 4 WEEKS) (Rate 999 if not applicable)

☐ Number of times meal omitted (not postponed)

SPECIFY EFFECT IF MEAL OMITTED - (IN PAST 4 WEEKS)
RESTRAINT OVER EATING - Restraint Subscale

Over the past four weeks have you been **consciously trying** to restrict what you eat whether or not you have succeeded?

Why?

Has this been to influence your shape or weight?

*Rate the number of days on which the subject has **consciously attempted** to restrict his or her food intake, whether or not he or she has succeeded. The restraint should have been intended to influence shape, weight or body composition, although this may not have been the sole or main reason. It should have consisted of planned attempts at restriction, rather than spur-of-the-moment attempts such as the decision to resist a second helping."

4. □ 0 - No attempt at restraint
   □ 1 -
   □ 2 - Attempted to exercise restraint on less than half the days
   □ 3 -
   □ 4 - Attempted to exercise restraint on more than half the days
   □ 5 -
   □ 6 - Attempted to exercise restraint every day

REASONS FOR RESTRAINT

<table>
<thead>
<tr>
<th>Reason</th>
<th>No (0)</th>
<th>To some extent (1)</th>
<th>Definitely (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4a) Diabetes</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>4b) Good health</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>4c) Shape/weight</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>
AVOIDANCE OF EATING - Restraint Subscale

Over the past four weeks have you gone for periods of eight or more waking hours without eating anything?

Why?

Has this been to influence your shape or weight?

[Rate the number of days on which there has been at least eight hours abstinence from eating food (soup and milk shakes count as food whereas drinks in general do not) during waking hours. It may be helpful to illustrate the length of time (e.g., 9:00 a.m. to 5:00 p.m.). The abstinence must have been at least partly self-imposed rather than being due to force of circumstances. It should have been intended to influence shape, weight or body composition, although this may not have been the sole or main reason.]

5. □ 0 - No such days
   □ 1 -
   □ 2 - Avoidance on less than half the days
   □ 3 -
   □ 4 - Avoidance on more than half the days
   □ 5 -
   □ 6 - Avoidance every day

EMPTY STOMACH - Restraint Subscale

Over the past four weeks have you wanted your stomach to be empty?

Why?

[Rate the number of days on which the subject has had a definite desire to have an empty stomach for reasons to do with dieting, shape or weight. This should not be confused with a desire for the stomach to feel empty or be flat.]

6. □ 0 - No definite desire to have an empty stomach
   □ 1 -
   □ 2 - Definite desire to have an empty stomach on less than half the days
   □ 3 -
   □ 4 - Definite desire to have an empty stomach on more than half the days
   □ 5 -
   □ 6 - Definite desire to have an empty stomach every day
**FOOD AVOIDANCE - Restraint Subscale**

Over the past four weeks have you **attempted** to avoid eating any foods which you like, whether or not you have succeeded?

What foods have you attempted to avoid?

Why?

Has this been to influence your shape or weight?

(Rate the number of days on which the subject has actively **attempted** to avoid eating specific foods (which he or she likes) whether or not he or she succeeded. The avoidance should have been intended to influence shape, weight or body composition, although this may not have been the sole or main reason. Note the type of food avoided.)

7. □ 0 - No attempt to avoid food
   □ 1 -
   □ 2 - Attempted to avoid food on less than half the days
   □ 3 -
   □ 4 - Attempted to avoid food on more than half the days
   □ 5 -
   □ 6 - Attempted to avoid food every day

**REASONS FOR AVOIDANCE**

<table>
<thead>
<tr>
<th>Reason</th>
<th>No (0)</th>
<th>To some extent (1)</th>
<th>Definitely (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7a) Diabetes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7b) Good health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7c) Shape/weight</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>