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Group Psychoeducation for the Treatment of Subclinical Disordered Eating in Women with Type 1 Diabetes Mellitus

by

Stefanie Christine Wilson 

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

in

Nutrition and Metabolism

Department of Agricultural, Food and Nutritional Science

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The undersigned certify that they have read, and recommended to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled ***Group Psychoeducation for the Treatment of Subclinical Disordered Eating in Women with Type 1 Diabetes Mellitus*** submitted by ***Stefanie Christine Wilson*** in partial fulfillment of the requirements for the degree of *Master of Science* in *Nutrition and Metabolism.*

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ABSTRACT

The coexistence of type 1 diabetes and subclinical disordered eating is associated with poor blood glucose control and increased risk of long-term diabetic complications. We hypothesized that a 6-session group psychoeducation program would improve metabolic control, diabetes treatment adherence, eating disorder symptomatology and general psychopathology. Participants (average age 31.9 years) were assigned to the treatment group (n=8) or the wait-list control group (n=6) based on the dates that they were recruited. Subjects were assessed pre-, post-, 1 month post-, and 6 months post-intervention (6 month follow-up only in treatment group). There were no differences in how the treatment group and the control group changed over time, indicating that the psychoeducation program was no more effective than the wait-list control group. Further research is needed to determine if group psychoeducation is an effective treatment for adult women with co-existing diabetes and subclinical disordered eating.

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TABLE OF CONTENTS

LIST OF APPENDICES

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

ADA: American Diabetes Association

ANOVA: Analysis of Variance

APA: American Psychiatric Association

BDI: Beck Depression Inventory

BMI: Body Mass Index

BSI: Brief Symptom Inventory

CBT: Cognitive Behavioural Therapy

CVD: Cardiovascular Disease

DCCT: Diabetes Control and Complications Trial

DSM: Diagnostic and Statistical Manual of Mental Disorders

EDI: Eating Disorder Inventory

EDNOS: Eating Disorder Not Otherwise Specified

EAT: Eating Attitudes Test

GHb: Glycated hemoglobin

HbA_{1c}: Hemoglobin A_{1c}

MANOVA: Multivariate Analysis of Variance

MNT: Medical Nutrition Therapy

N/A: Not available

RSE: Rosenberg Self-Esteem Scale

SCI: Self-Care Inventory

SMBG: Self-Monitoring of Blood Glucose

DEFINITIONS

Metabolic Control

Metabolic control describes the degree to which chronic blood sugar values are within normal ranges. Other terms synonymous with metabolic control include glycemic control, diabetic control and blood sugar control.

Binge Eating

An episode of binge eating involves eating, in a discrete amount of time (within a 2 hour period) an amount of food that is definitely larger than what most people would eat during a similar period of time under similar circumstances. It also involves a loss of control over eating, indicating the inability to stop eating or control what or how much one is eating.

Definition adapted from American Psychiatric Association (APA), 1994

Anorexia Nervosa

Anorexia Nervosa is characterized by 1) the refusal to maintain weight above a minimally normal standard (weight < 85% expected) 2) an intense fear of gaining weight or becoming fat, even though underweight 3) a disturbance in the way one's body weight and shape are experienced, denial of seriousness of current low body weight or body weight having an undue influence on self-evaluation 4) in postmenarchal females, amenorrhea (absence of at least 3 consecutive menstrual cycles).

Definition adapted from APA, 1994

Bulimia Nervosa

Bulimia Nervosa is characterized by 1) recurrent episodes of binge eating 2) recurrent inappropriate compensatory behaviours (self-induced vomiting, misuse of laxatives, diuretics or other medications; fasting; or excessive exercising) 3) the binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for 3 months 4) body weight and shape having an undue influence on self-evaluation 5) the disturbance does not occur exclusively during episodes of anorexia nervosa

Definition adapted from APA, 1994

Eating Disorder Not Otherwise Specified (EDNOS)

Individuals with EDNOS have a disturbance very similar to bulimia nervosa or anorexia nervosa, but fail to meet the diagnostic criteria of those disorders because one of the essential diagnostic features is missing. Some examples EDNOS include the following:

1. For females, all of the criteria for anorexia nervosa are met except the individual has regular menses.
2. All of the criteria for anorexia nervosa are met except that, despite significant weight loss, the individual's current weight in the normal range.
3. All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms 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 behaviours by an individual of normal body weight after eating small amounts of food.
5. Repeatedly chewing and spitting out, but not swallowing, large amounts of food.
6. Binge eating disorder (see below definition)

Definition adapted from APA, 1994

Binge Eating Disorder

Describes an individual who has recurrent binges in the absence of the compensatory behaviours seen in people with bulimia nervosa.

Definition adapted from Fairburn and Walsh, 1995

Disordered Eating

The terms disordered eating or subclinical disordered eating are frequently used to describe the behaviours of an individual with an eating disorder. The term disordered eating is used to indicate that an individual suffers from the same symptoms of an individual with an eating disorder, but the frequency or severity of the symptoms have not been formally confirmed with a diagnostic interview. Similarly, the term subclinical disordered eating indicates that an individual demonstrates the symptoms of EDNOS without a formal diagnosis.

CHAPTER ONE
INTRODUCTION

A. RATIONALE

The coexistence of type 1 diabetes mellitus and subclinical disordered eating is associated with poor metabolic control, abnormalities in lipid metabolism, poor adherence to diabetes treatment regimen and increased risk of long-term diabetic complications (Affenito et al., 1997a; Affenito et al., 1997b; Rydall et al., 1997; LaGreca et al., 1990). There are several factors associated with type 1 diabetes that could increased the risk of disordered eating (Steel et al., 1990). Research demonstrates that up to one third of women with type 1 diabetes have some form of disturbance in eating behaviour, including using insulin reduction as a way of purging unwanted calories (Polonsky et al., 1994; Stancin et al., 1989; Fairburn et al., 1991a; Marcus et al., 1992). It is crucial that individuals with diabetes receive treatment for mild disordered eating in order to prevent life-threatening complications and the progression to more severe forms of eating disorders.

There are a number of different treatments available for individuals with clinical eating disorders, all being relatively easy to adapt for use with individuals with diabetes (Peveler and Fairburn, 1992). However, there are very few treatment options for those individuals suffering from subclinical disordered eating. Group psychoeducation is one type of treatment that has proven effective in mild and subclinical cases of eating disorders in individuals without diabetes (Olmsted et al., 1991; Kaminski and McNamara, 1989), but only one study to date has documented its effectiveness in individuals with co-existing subclinical disordered eating and type 1 diabetes (Olmsted et al., 1998). Further studies are required to evaluate group psychoeducation in this critical population.

B. PURPOSE

The purpose of this study is to evaluate the efficacy of a group psychoeducation program for individuals with type 1 diabetes and subclinical disordered eating.

C. HYPOTHESES

A group psychoeducation program will result in the following effects in individuals with type 1 diabetes and subclinical disordered eating:

a) increased metabolic control
b) increased diabetes treatment adherence
c) decreased eating disorder symptomatology
d) decreased general psychopathology

D. OBJECTIVES

1. Individuals with subclinical disordered eating will be identified by screening with the Eating Disorder Inventory (EDI) and the Eating Attitudes Test (EAT).

2. The hypotheses will be tested by assessing the following measures pre-, post-, and 1 month post-intervention in the treatment and control group, and 6 month post-intervention in the treatment group:
   a) metabolic control, using fructosamine and hemoglobin A_{1c} (HbA_{1c})
   b) diabetes treatment adherence, using the Self-Care Inventory (SCI)
   c) eating disorder symptomatology, using the EAT, the EDI and 2 questions concerning insulin omission
   d) general psychopathology, using the Rosenberg Self-Esteem Scale (RSE), Beck Depression Inventory (BDI) and the Brief Symptom Inventory (BSI)

CHAPTER TWO

LITERATURE REVIEW

Type 1 diabetes mellitus and eating disorders are two common diseases, each with dire physical and emotional consequences and complex treatment approaches. The coexistence of the two illnesses is particularly threatening, even when disordered eating is very mild. Treatment of subclinical disordered eating in individuals with type 1 diabetes is especially important in order to minimize risk of life-threatening complications, in addition to preventing progression to more severe and intractable forms of eating disorders.

A. DIABETES MELLITUS

Diabetes mellitus is a chronic metabolic disease characterized by hyperglycemia resulting from the body's inability to produce and/or respond to insulin. Insulin is a hormone produced by the pancreas that is required for the use and storage of body fuels. The chronic hyperglycemia of diabetes is associated with long-term damage to the small blood vessels of various organs, specifically the eyes, kidneys and nerves. Diabetes is also a strong risk factor for the development of atherosclerotic cardiovascular, peripheral vascular and cerebrovascular disease, as well as hypertension and abnormalities in lipoprotein metabolism. The emotional consequences of diabetes and its therapy can cause significant psychosocial problems for both individuals with diabetes and their families.

Diabetes mellitus is an important public health concern. It is a continually increasing cause of morbidity and premature mortality around the world (Harris et al., 1998). In the United States, diabetes is the leading cause of blindness, end-stage renal disease and lower-extremity amputation (Roman and Harris, 1997). Diabetes affects a relatively large proportion of the Canadian population as well. The prevalence of diabetes in Canada is approximately 0.12% and 5% in children under the age of 15 and adults over the age of 18 respectively (Blanchard et al., 1997; Tan and MacLean, 1995).

1. Classifications of Diabetes

The vast majority of cases of diabetes can be categorized into two main classifications: type 1 diabetes and type 2 diabetes. The classifications are based on clinical presentations and etiologic factors.

a) Type 1 Diabetes

Type 1 diabetes accounts for less than 10% of all known cases of diabetes mellitus. It results from a cellular-mediated autoimmune destruction of the β-cells of the pancreas (Atkinson and Maclaren, 1994). Both genetic predisposition and environmental factors play a role in causing the destruction. The β-cells are the insulin producing cells of the pancreas, therefore individuals with type 1 diabetes are dependent on exogenous insulin for survival. The speed of β-cell destruction varies, being rapid in some individuals (mainly infants and adolescents) and slow in others (mainly adults) (Zimmet et al., 1994). Type 1 diabetes most commonly occurs in childhood with most cases being diagnosed in people younger than 30 years of age (Jiwa, 1997). At diagnosis, individuals are usually lean and have experienced excessive thirst, frequent urination and significant weight loss, all due to a deficiency of insulin. The presence of obesity, however, is not incompatible with diagnosis (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1998).

b) Type 2 Diabetes

Approximately 90% of all known cases of diabetes are type 2 diabetes. Individuals with type 2 diabetes have insulin resistance and usually have a relative deficiency of insulin (Reaven et al., 1976). They are frequently unable to produce enough insulin to overcome concomitant insulin resistance. Although individuals with type 2 diabetes do not require exogeneous insulin for survival, approximately 40% will eventually require exogenous insulin for adequate blood glucose control. This form of diabetes often goes undiagnosed for many years because the hyperglycemia develops gradually and during its early stages is not severe enough to notice the classic symptoms of diabetes. There is no single cause of type 2 diabetes, although it has a very strong

genetic component (Newman et al., 1987). The risk of developing type 2 diabetes increases with age, positive family history of diabetes, certain ethnic backgrounds (Aboriginal people, and those of Hispanic, Asian and African descent), history of gestational diabetes, abdominal obesity and lack of physical activity (Rewers and Hamman, 1995; Zimmet, 1992; Wein et al., 1997). Abdominal obesity is a major cause of insulin resistance and although many individuals with type 2 diabetes are obese, this form of diabetes can occur in the non-obese (Kissebah et al., 1982; Bogardus et al., 1985). In non-obese individuals, insulin resistance is likely caused by an increased proportion of body fat distributed predominantly in the abdominal region (Kissebah et al., 1982).

2. Complications of Diabetes

The potential complications associated with diabetes are numerous and can significantly disrupt quality of life. Both the physiological and psychological consequences of diabetes are associated with poor metabolic control.

a) Physiological Complications

i) Acute Complications

Hypoglycemia, hyperglycemia and diabetic ketoacidosis are acute complications of diabetes. Hypoglycemia (blood glucose less than 3.9 mM) is a common side effect of insulin therapy and can be quite dangerous in its advanced stages. Symptoms of advanced hypoglycemia include headaches, confusion, lack of coordination, blurred vision, seizures and coma. Hypoglycemia can occur from medication errors, inadequate food intake, increased activity and alcohol intake and it is easily treated with carbohydrate intake.

Hyperglycemia is an acute complication of diabetes that can lead to diabetic ketoacidosis, a life-threatening complication that results from inadequate insulin for glucose utilization. Clinically, diabetic ketoacidosis is defined as blood glucose levels above 13.9 mM and the presence of ketones in the blood and urine. It almost always occurs in those with type 1 diabetes as individuals with type 2 diabetes usually have

enough insulin producing capacity to prevent the development of ketoacidosis. Symptoms of diabetic ketoacidosis include frequent urination, hyperventilation, hunger, dehydration, fatigue and eventually coma and death. While symptoms can be very severe, diabetic ketoacidosis is effectively treated with insulin, fluid and electrolyte replacement and medical monitoring, which is usually successful when done by an experienced clinician.

ii) Chronic Complications

In addition to acute complications of diabetes, there are several chronic complications of diabetes that are classified as microvascular and macrovascular complications. The microvascular complications include nephropathy, retinopathy and neuropathy, while the macrovascular complications include cardiovascular, cerebrovascular and peripheral vascular disease.

Many individuals with diabetes eventually develop microvascular complications. Diabetic nephropathy involves damage to the kidneys and is the most common cause of end-stage renal failure in the Canada (Meltzer et al., 1998). It is estimated that approximately 20-30% of people with diabetes will develop nephropathy (American Diabetic Association (ADA), 1998a; Andersen et al., 1983). Retinopathy is a chronic ocular disease that eventually develops, to some degree, in all people with diabetes. Diabetic retinopathy poses a serious threat to vision and is estimated to be the most frequent cause of blindness among adults in North America (ADA, 1998b). Diabetic neuropathy involves nerve damage that can affect peripheral nerves of the feet and hands, as well as the nerves that control various organs such the heart, sexual organs and the gastrointestinal tract. Approximately 40% to 50% of individuals with diabetes are affected by diabetic neuropathy (Meltzer et al., 1998).

Diabetes is associated with a marked increased risk of cardiovascular disease (CVD), cerebrovascular disease and peripheral vascular disease (Uusitupa et al., 1993; Folsom et al., 1997; Wilson et al., 1991). CVD is the main cause of mortality and morbidity in individuals with diabetes. The cause of the increased risk of vascular disease in diabetes is multifactorial. Individuals with type 2 diabetes tend to have

numerous risk factors for vascular disease including hypertension, hypertriglyceridemia, decreased high density lipoproteins, increased low density lipoproteins and obesity (Cowie and Harris, 1995; Uusitupa et al., 1990; Bierman et al., 1992). Additionally, insulin resistance, hyperinsulinemia and hyperglycemia are associated with elevated CVD risk either independently or by contributing to abnormalities in serum lipids (Klein, 1995; Despres et al., 1996; Uusitupa et al., 1993; Ronnemaa et al., 1991).

In summary, there are many physiological complications of diabetes. The chronic complications are a major cause of morbidity and mortality among individuals with diabetes. Research has clearly demonstrated that poor metabolic control is associated with the development and progression of microvascular complications in individuals with type 1 diabetes (Diabetes Control and Complications Trial (DCCT) Research Group, 1993). Research now suggests that the same association exists in people with type 2 diabetes (UK Prospective Diabetes Study Group, 1998; Klein, 1995; Ohkubo et al., 1995). The relationship between metabolic control and macrovascular complications does not seem to be as strong as that with microvascular complications, but evidence indicates that achieving optimal glycemic control will aid in the prevention and treatment of macrovasular complications (Klein, 1995; Wilson et al., 1991; Barrett-Connor, 1997).

b) Psychological Complications

Psychological complications of diabetes warrant attention as they are prevalent and associated with poor metabolic control as well as lack of adherence to diabetes self-care routines. Depression and anxiety disorders are markedly more common in people with diabetes than in the general population (Lustman, 1988a; Goonick et al., 1995; Lustman et al., 1992). Depression is also more common, however, in people with chronic medical illnesses, but diabetes seems to be on the high end of the spectrum of depression severity in the medically ill (Weyerer et al., 1989; Lustman et al., 1992). Eating disorders are another common psychological complication of diabetes, especially type 1 diabetes. Although the prevalence is not elevated in the type 1 diabetic population, the combination of eating disorders and diabetes can be extremely

destructive. The etiology of psychological complications in individuals with diabetes is complex, with evidence suggesting that poor metabolic control, psychological stress, chronic pain and daily treatment demands have etiologic roles (Geringer, 1990).

The association between psychological complications and diabetes may be mediated through poor metabolic control. Depression, anxiety, quality of life and disordered eating have all been shown to be inversely associated with metabolic control in individuals with diabetes (Mazze et al., 1984; Lustman et al., 1986; Rydall et al., 1997). Additionally, improvements in metabolic control are associated with improvements in anxiety, depression, quality of life and disordered eating. Further research is needed to determine if causality exists and if a one-way relationship exists between poor metabolic control and the various psychological problems seen in diabetes (Mazze et al., 1984).

There are a number of explanations for the association between metabolic control and psychological disturbances. Lustman et al. (1988) has suggested that symptoms of depression and diabetes may exacerbate one another at a biological level. For example, hormonal dysregulation associated with depression may cause glycemic dysregulation and vice-versa. Another explanation for the association is a disruption in diabetes self-care tasks that could theoretically lead to poor metabolic control. LaGreca (1990) demonstrated that individuals with disordered eating are less adherent to diabetes self-care tasks. Littlefield et al. (1992) demonstrated a similar association between individuals with low self-esteem and depressive symptoms and adherence. It is unclear, however, whether psychological disorders cause a disruption in adherence, or whether psychological disorders result from not adhering to self-care routines; it is possible that both situations coexist, thus creating a cycle.

Psychological problems could also be caused by psychosocial hardships that commonly accompany diabetes, such as frequent hospitalizations, daily treatment demands and fear of hypoglycemia. Medical complications associated with diabetes, such as vision loss, sexual dysfunction, cardiovascular disease and kidney insufficiency, are often painful and can be quite traumatic, potentially leading to depression and anxiety disorders. Higher rates of depression have been shown in individuals who

exhibit diabetes-related complications compared to individuals who do not (Leedom et al., 1991; Lloyd et al., 1992); however, not all studies confirm these results (Lustman et al., 1988b).

There are several risk factors associated with diabetes that may precipitate the development of eating disorders in vulnerable individuals. One such factor is the continual focus on food and diet that is traditionally required for the management of the diabetes. Another factor is the weight gain that commonly follows the diagnosis of type 1 diabetes. The topic of eating disorders and diabetes will be discussed in detail in Section C.

Psychological disturbances are prevalent among individuals with diabetes. These disturbances may affect metabolic control directly through neuroendocrine effects or indirectly through self-care. The detrimental effects on metabolic control may exert a negative reciprocal effect on emotional and psychological states. In order to minimize psychological problems, management of diabetes should include a focus on achievement of optimal metabolic control while addressing psychological issues around diagnosis, complications, metabolic control goals and adherence to self-care routines.

3. Treatment of Diabetes

As diabetes is a chronic disease, its management requires lifestyle and behavioural changes that last a lifetime. Management of diabetes includes a) self-management education; b) medical nutrition therapy (MNT); c) medications; d) exercise; and e) self-monitoring of blood glucose. The goal of treatment is to provide the individual with the necessary tools to achieve optimal glycemic control in order to reduce the risk of microvascular complications, while minimizing hypoglycemia, excessive weight gain and cardiovascular risk and maximizing quality of life and overall sense of well-being.

a) Self-Management Education

Ultimately, it is the individual with diabetes who is responsible for their diabetes self-management. Diabetes self-management programs are available to provide

individuals with the motivation, information and skills to management their own diabetes. These programs are usually facilitated by a variety of health professionals specializing in diabetes, including a physician, nurse and dietitian who educate and empower individuals to implement self-management into their lifestyles.

b) Medical Nutrition Therapy (MNT)

MNT is an integral component of total diabetes care, although it is probably the most challenging component for individuals with diabetes to learn and adhere to. The goals of MNT include: 1) maintenance of as near normal blood glucose levels as possible by balancing food intake with insulin and/or oral glucose-lowering medications and physical activity; 2) achievement of optimal serum lipid levels; 3) provision of adequate calories for maintaining or attaining a reasonable weight; 4) prevention and treatment of acute complications and long-term complications; and 5) improvement of overall health though optimal nutrition (ADA, 1998c).

Depending on the medication used for treatment, there are slight differences in nutrition therapy for those with type 1 diabetes and those with type 2 diabetes. For individuals with type 1 diabetes or type 2 diabetes on insulin therapy, a meal plan based on the individual's usual food intake is determined and used as the basis for integrating insulin therapy into usual eating and exercise patterns. The meal plan is then used as a guideline for day-to-day eating choices. The plan should incorporate all of the recommendations for a healthy diet including appropriate levels of protein, fat and carbohydrate to allow the individual to maintain a reasonable body weight. Individuals with type 1 diabetes are advised to eat at consistent times synchronized with the time of action of the insulin used. Intensive insulin therapy allows for the most flexibility in terms of the amount of food eaten and the timing of meals and snacks.

Nutrition therapy for those with type 2 diabetes is slightly less complicated than for those on insulin therapy. A nutritionally adequate, individualized meal plan with a reduction in total fat, specifically saturated fat, accompanied with an increase in physical activity is recommended. Maintenance and/or attainment of a reasonable body weight is also recommended.

c) Medication

There are two types of medications used for controlling blood glucose in individuals with diabetes: oral glucose-lowering medications and insulin therapy. Approved oral glucose-lowering agents in Canada include sulfonylureas, biguanides, alpha-glucosidase inhibitors and thiazolidinediones. They are used for the management of type 2 diabetes in individuals who are not adequately controlled though medical nutrition therapy alone. Oral glucose-lowering agents are often used in combination or in combination with insulin therapy. All individuals with type 1 diabetes depend on insulin therapy for survival. Those individuals with type 2 diabetes who cannot successfully control blood glucose with oral glucose lowering medications and diet therapy must also take insulin therapy. The insulin regimen is usually tailored according to the individual's nutrition care plan and physical activity habits. Intensive diabetes management is the therapy of choice by most doctors for individuals with type 1 diabetes and involves taking multiple daily injections of insulin, monitoring blood glucose several times throughout the day, as well as carefully balancing food intake, physical activity and insulin doses.

d) Exercise

An active lifestyle has substantial benefits for individuals with diabetes, including cardiovascular disease prevention, improved mental outlook and improved glucose metabolism. An increase in physical activity may also improve glycemic control, reduce medication needs for individuals with type 2 diabetes and lower blood pressure. Individuals with diabetes, especially those with type 2 diabetes, are at elevated risk for developing CVD. There is a large amount of evidence to show that regular exercise and physical activity are associated with decreased CVD risk (Slattery et al., 1989; Blair et al., 1989; Wallberg-Henriksson et al., 1982; ADA, 1998d). Participation in physical activity can also improve self-esteem, emotional well-being and depression especially in those individuals who demonstrate psychological disturbances (Steptoe and Butler, 1996; Morgan et al., 1970). This is especially

important for individuals with diabetes, as research indicates these individuals have higher rates of depression and anxiety.

Exercise improves glucose metabolism by increasing insulin sensitivity (Landt et al., 1985; Wallberg-Henriksson et al., 1982; Devlin et al., 1987). This is especially important for individuals with type 2 diabetes since they suffer from insulin resistance. Improvements in glycemic control in response to an exercise program have been demonstrated in individuals with type 2 diabetes (Schneider et al., 1984), although a similar effect has been difficult to show in individuals with type 1 diabetes (Zinman et al., 1984; Landt et al., 1985; Wallberg-Henriksson et al., 1982). Nonetheless, exercise is considered an important component in diabetes management and is recommended for individuals with diabetes, although certain alterations to diet, insulin regimen and self-monitoring should be made to ensure satisfactory glycemic control (Meltzer et al., 1998).

e) Self- Monitoring of Blood Glucose

Self-monitoring of blood glucose (SMBG) is an integral component in the treatment of diabetes. As mentioned earlier, the overall goal of diabetes management is optimal blood glucose control. SMBG allows individuals and health care providers to evaluate the success in achieving this goal. SMBG data can also be used to identify and treat hyper- and hypoglycemia as well as to make necessary changes to diet, exercise and medications in order achieve optimal metabolic control. To receive the benefits of SMBG, accurate record keeping of blood glucose readings should accompany monitoring.

Research shows that the frequency of SMBG is positively associated with glycemic control (Dorchy et al., 1997). Individuals treated with insulin demonstrate a worsening of metabolic control when frequency of SMBG decreases to less than four times per day (ADA,1994). Individuals not treated with insulin require substantially less monitoring, but benefit from fasting and post-meal testing (Meltzer et al., 1998). Barriers to adherence to SMBG include cost of monitoring, lack of convenience of

monitoring, discomfort associated with blood sampling, and lack of education about the health benefits and how to use the information (ADA, 1998e).

4. Evaluation of Diabetes Management

a) Assessing Glycemic Control

Measuring glycemic control is vital in evaluating diabetes management. There are a number of ways to assess glycemic control, including day to day measures such as blood glucose testing and long-term measures such as glycated protein testing. Each protocol has its benefits and disadvantages.

In the past, physicians frequently used fasting or random laboratory blood glucose measurements to assess glycemia. These measurements are not accurate estimates of long-term glycemic control in people with type 1 diabetes since their blood glucose levels fluctuate widely from day to day (Nathan et al., 1984). Fasting plasma blood glucose determinations over intervals of weeks to months provide more reliable measures of longer-term glycemia for most individuals with type 2 diabetes (Singer et al., 1989). However, there is the potential for the patient to change their behaviours on the day of the laboratory blood glucose test, thereby influencing the accuracy of the test. The use of fasting or random laboratory blood glucose measurements has been questioned as the use of SMBG and glycated protein assessments have become increasingly more common (Goldstein et al., 1995).

SMBG is very useful in assessing immediate blood glucose control. If information about long-term glycemia is needed, multiple timed values must be obtained and recorded. Accurate record keeping is crucial if SMBG information is to be useful in assessing long-term glycemic control. Some researchers have demonstrated that many patients fabricate blood glucose values in record keeping diaries, thereby invalidating results (Mazze et al., 1984).

Although blood glucose testing is important in evaluating glycemic control, it only provides information about immediate glycemic control and may not always be an accurate estimate of long-term glycemia. A single test of glycated proteins, primarily hemoglobin and serum proteins, can quantify glycemia over weeks and months.

Glycated hemoglobin (GHb) describes a series of stable minor hemoglobin components formed slowly and non-enzymatically from hemoglobin and glucose. There are three GHb fractions including hemoglobin A_{1a} (HbA_{1a}), HbA_{1b}, HbA_{1c}. HbA_{1c} is the fraction that is best correlated with average blood glucose concentrations (Benjamin et al., 1994). Hemoglobin is found in erythrocytes, cells freely permeable to glucose. The rate of formation of HbA_{1c} is directly proportional to the ambient glucose concentration. Therefore, the level of HbA_{1c} in a blood sample provides an estimate of glycemic history over the previous 120 days, the average lifespan of an erythrocyte.

GHb testing is considered the most accurate indicator of chronic blood glucose control. Data from the DCCT (1993) demonstrate a strong inverse statistical relationship between glycemic control and GHb (as measured by HbA_{1c}) (Goldstein et al., 1995). Additionally, GHb can predict risk of developing chronic complications of diabetes (DCCT, 1993). The routine use of GHb is recommended for all individuals with diabetes to quantify glycemia over the previous 2 to 3 months (Meltzer et al., 1998). HbA_{1c} values below 7% are considered optimal and associated with the fewest complications, although these values may be difficult for some individuals to achieve without severe side effects (Meltzer et al., 1998). HbA_{1c} values between 7% and 8.4% are considered suboptimal and are attainable by the majority of individuals with diabetes, but may not be low enough to prevent complications (Meltzer et al., 1998).

Although GHb is considered the gold standard for assessing long-term glycemic control, it does have some limitations. Modan et al. (1988) found individual variations in the propensity for hemoglobin to be glycated that were unrelated to glucose control. Two individuals with the same GHb levels could have clinically significant differences in actual average blood glucose. Additionally, there are individual differences in renal threshold for glucose (Goldstein et al., 1995). The higher the renal threshold, the higher the steady-state blood glucose level and the greater the ease with which hemoglobin can be glycated. Certain clinical situations may alter GHb values. For example, hemolytic anemia will lower GHb (Golstein et al., 1995). It is also important to consider the rate of change in GHb in relation to a change in glycemia. GHb is actually a "weighted" measure of mean blood glucose over the preceding 120 days; more recent past events

contribute relatively more to the final result than earlier events (Goldstein et al., 1995). Further studies are needed to determine the relative importance of these factors in interpreting GHb levels.

The turnover of serum albumin is much shorter than that of hemoglobin; therefore, the degree of glycation of serum proteins (mainly albumin) can provide an index of glycemia over a shorter-term than GHb. Measurement of total glycated serum proteins correlates well with GHb (Baker et al., 1983, Hindle et al., 1985). One of the most widely used methods to quantify glycated serum proteins is the fructosamine assay, an easy and inexpensive method for assessing short-term glycemic control (Allgrove and Cockrill, 1988; Hindle et al., 1985). A single measurement of fructosamine can quantify glycemia over the preceding 1 to 2 weeks. There is some debate as to whether fructosamine assay results should be corrected for serum albumin concentrations (Lamb et al., 1991). Lamb et al. (1991) reported a significant relationship between fructosamine and serum albumin concentrations, while others have found fructosamine measurements to be independent of serum albumin if albumin is within the normal range (Baker et al., 1983; Allgrove and Cockrill, 1988; Schleicher et al., 1993). Currently fructosamine results are reported without reference to total serum protein; however the test is not recommended if serum albumin is less than 30 g/L (Benjamin and Sacks, 1994).

In summary, glycated hemoglobin is the best index of chronic glycemic control. Instances where short-term information is required, fructosamine is a valuable tool, although it should be used in conjunction with glycated hemoglobin for the most reliable results.

b) Measures of Diabetes Treatment Adherence

Individuals with diabetes are expected to follow a number of behavioural actions to care for their diabetes on a daily basis. Assessing adherence to these behavioural actions is an important part of assessing diabetes management. Adherence is defined as the extent to which an individual's behaviour coincides with what is recommended. It

is very difficult to assess in individuals with diabetes because of the complexity in the diabetes regimen and the variability in each individual's recommended regimen.

Currently, there are no widely accepted, reliable measures of adherence (McNabb et al., 1997). The diabetes 24-h recall interview is a reliable method of assessing compliance to the recommended diabetes self-care tasks if multiple interviews and multiple informants (example: mother and child) are used and if the questions are specific and time limited (Johnson, 1990; Johnson et al., 1986). The 24-hour recall interview is a very time consuming and expensive method of assessing adherence. Self-report questionnaires are also useful measures of regimen adherence and are frequently used in diabetes care. The Self Care Inventory (SCI) is a 14 item self-report inventory used to assess patient's perceptions of the degree to which they adhere to recommendations for diabetes self-care. An overall adherence score can be calculated as well as 4 subscales scores, including use of blood glucose testing, use of insulin, use of food and use of exercise. Polonsky et al. (1995) demonstrated good internal reliability for each of the 4 subscales, with Cronbach's alphas ranging from .53 to .81. Results from the SCI correlated well with results from the 24-hour recall interview ($r = .71$, $p < .001$) (Greco et al., 1990). The SCI and the 24-hour recall interview both demonstrate the same statistical relationship to metabolic control and mean adherence (Greco et al., 1990). Glucose testing frequency is significantly correlated with HbA1c as measured by the 24-hour recall interview ($r = -.52$, $p < .01$) or the SCI ($r = -.54$, $p < .005$) (Greco et al., 1990).

B. EATING DISORDERS

Eating disorders are disturbances in eating behaviours and attitudes that significantly impair physical health and psychological functioning. According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association (APA), 1994), there are two clinical eating disorders, anorexia nervosa and bulimia nervosa, as well as one subclinical or partial syndrome eating disorder, Eating Disorder Not Otherwise Specified (EDNOS). Clinical eating disorders represent only a small proportion of the people who engage in unhealthy weight control practices.

1. Classifications of Eating Disorders

The most recent diagnostic criteria for eating disorders is defined in the DSM-IV (APA, 1994). Anorexia nervosa is characterized by a refusal to maintain a body weight over or at a minimally normal weight for age and height, an intense fear of gaining weight and evidence of an endocrine disorder (amenorrhea in women and loss of sexual potency and sexual interest in men). The approximate prevalence of anorexia nervosa is 0.5% and 0.09% in females and males, respectively (Walters and Kendler, 1995; Rastam et al., 1989). Bulimia nervosa is characterized by recurrent episodes of binge eating, inappropriate compensatory behaviours and an intense fear of being overweight. The estimated prevalence of bulimia nervosa is 1% to 3% in females and approximately 0.5% in males (Garfinkel et al., 1995; Kendler et al., 1991).

Eating Disorder Not Otherwise Specified (EDNOS) describes disordered eating that is not severe enough to meet the full diagnostic criteria for anorexia nervosa or bulimia nervosa. Subthreshold, partial syndrome or subclinical eating disorders are other terms sometimes used in place of EDNOS. For example, an individual with EDNOS would be an individual who has all the clinical features of bulimia nervosa but bingeing and compensatory behaviours occur less than 2 times per week. Another example would be an individual who meets all of the criteria for anorexia nervosa except that, despite significant weight loss, the individual's current weight is within

normal limits. Binge eating disorder is another example of EDNOS. It is defined as recurrent episodes of binge eating in the absence of compensatory behaviours.

EDNOS deserves more attention then it actually receives. These subclinical disorders constitute a large proportion of the morbidity associated with eating disorders; approximately 25% of patients presenting with eating disorder pathology have EDNOS (Mizes and Sloan, 1998). The prevalence of subclinical eating disorders is approximately 5% - 10%, about 5 times that of clinical eating disorders (Dancyger and Garfinkel, 1995; Walters and Kendler, 1995; Kendler et al., 1991). Some researchers suggest that subclinical disordered eating represents the early stages before the onset of a full-blown eating disorder (Kaminski and McNamara, 1996). Individuals with EDNOS are not commonly treated because they are not considered to have a life - threatening disorder.

Although it remains a controversial issue, eating disturbances seem to lie on a continuum, with normal eating at one end of the spectrum, clinical eating disorders at the opposite end and subclinical forms falling at intermediate points along the continuum (Scarano and Kalodner-Martin, 1994; Shisslak et al., 1995). Groups along the continuum share some underlying characteristics of eating disorders such as weight concerns and body dissatisfaction, but differ in the frequency and severity of eating problems. Some different points along the eating behaviour continuum in order of increasing severity are normal eaters, weight-preoccupied persons, chronic dieters, purgers, subclinical bulimia nervosa and anorexia nervosa and full-blown anorexia nervosa and bulimia nervosa.

A large amount of research has been done to characterize the groups along the continuum in relation to full syndrome eating disorders. By definition, all of these groups display disordered eating behaviours such as repeated dieting, binge eating and purging, but in increasing severity and frequency along the continuum (Katzman and Wolchick, 1984; Kendler et al., 1991). The groups along the continuum exhibit similar psychopathologies that also tend to be seen in increasing levels of severity along the continuum, with the clinical eating disorders exhibiting significantly higher levels (eg. depression, distorted interoceptive awareness, interpersonal distrust, dieting

preoccupation, ineffectiveness and low self-esteem) (Garfinkel et al., 1995; Garner et al., 1984; Laessle et al., 1989; Katzman and Wolchik, 1984; Dancyger and Garfinkel, 1995). Many of these psychopathologies contribute to the development and continuation of disordered eating behaviours, so it is reasonable that they would increase in severity along the continuum. A large number of dieters, weight-preoccupied individuals and individuals with subclinical eating disorders exhibit the same degree of body dissatisfaction as those with clinical eating disorders (Garner et al., 1984; Dancyger and Garfinkel, 1995). Garner et al. (1984) has proposed that there are two components to disordered eating: 1) extreme concerns with weight and body image and 2) general psychopathology. The first component is seen in all of the groups along the continuum, while the second component is only present to a significant degree in clinical eating disorders.

There is often progression along the spectrum of eating behaviours. Individuals with less severe eating pathologies are at significant risk for developing clinical eating disorders. Patton et al. (1990) found that 20% of young women dieters progress to subclinical or clinical eating disorders over a one-year time span. Several longitudinal studies demonstrated that 30% to 45% of subclinical eating disorders progress to full clinical eating disorders (Yager et al., 1987; Herzog et al., 1993).

In summary, many women have some form of disordered eating, ranging from the most severe disordered behaviours seen in anorexia nervosa and bulimia nervosa to the mild behaviours seen in weight-preoccupied people. All individuals with disordered eating share many of the same behaviours and psychopathologies, but in differing levels of severity. Women displaying subclinical variants of disordered eating are at increased risk for developing clinical eating disorders.

2. Etiology of the Eating Disorders

The etiology of eating disorders, like many other psychiatric disorders, is multifactorial. Personality, familial, genetic, biological and sociocultural factors all play a role in how these illnesses unfold. The same factors contribute to the development of subclinical eating disorders (Garfinkel et al., 1995; Walters and

Kendler, 1995; Kendler, 1995). Understanding the etiology behind eating disorders provides information useful for selecting appropriate treatments as well as designing prevention programs.

There are three stages to the development of an eating disorder. Initially, an individual is exposed to predisposing factors that increase vulnerability to a behavioural precursor; the behavioural precursor is almost always dieting. In the second stage, precipitating events combine with dieting to lead to the onset of an eating disorder. Finally, a number of perpetuating factors determine whether or not a disorder will become established and maintained.

a) Predisposing Factors

Factors that increase the risk of dieting or the development of a psychiatric disorder generally contribute to the development of an eating disorder. Research shows that dieting frequently precedes the onset of and is strongly associated with an increased risk of an eating disorder (Hsu, 1997). Approximately 10 times more women suffer from these disorders than men, making female gender an obvious risk factor (Hsu,1996). The lifetime prevalence of depression, anxiety disorders and obsessive-compulsive personality disorder is significantly higher in individuals with eating disorders (Halmi et al., 1991; Rastam, 1992). In most cases, these psychiatric disorders occur concurrently with the eating disorder, however in some cases they may predate the onset and contribute to its development (Rastam, 1992). Low self-esteem, perfectionism, a slim ideal body shape and a preoccupation with physical appearance have all been implicated as risk factors for eating disorder development (Fairburn et al., 1997; Walter and Kendler, 1995; Kendler et al., 1991; Rastam, 1992). Obesity is also a strong risk factor for bulimia nervosa (Fairburn et al., 1997). Diabetes mellitus may be a risk factor for eating disorder development for a number of reasons including the weight gain that commonly follows diagnosis (Steel et al., 1990). Sexual abuse has been implicated as a risk factor for eating disorder development, but recent research suggests that sexual abuse is no more common in eating disorders than it is in other psychiatric disorders (Fairburn et al., 1997). Most risk factors are linked to eating

disorder development by increasing the chances that an individual will experiment with dieting.

There is also a strong familial component to eating disorders. The lifetime risk of eating disorders among first-degree relatives of those eating disorders is significantly greater than that among relatives of normal controls (Lilenfeld et al., 1998). Both genetic and environmental factors have been implicated in the familial transmission of disordered eating (Kendler et al., 1995). Support for genetic factors comes from twin studies; concordance rates for bulimia nervosa among monozygotic twins are much higher than among dizygotic twins (Kendler et al., 1991). Similar results have been demonstrated for anorexia nervosa and subclinical eating disorders (Kendler et al., 1991; Holland et al., 1988).

Genetics may contribute directly through a specific vulnerability to an eating disorder or indirectly by predisposing to obesity or a specific personality trait that in turn predisposes one to an eating disorder. Certain inheritable personality traits such as anxious worry, extreme insistence of order and regularity in behavioural routines may predispose one to anorexia nervosa (Strober, 1995). Inheritable traits such as thrill seeking, excitability and less persistence may increase the risk of bulimia nervosa (Strober, 1995). Some studies demonstrate increased rates of psychiatric disorders and alcoholism among first and second-degree relatives of probands with either anorexia nervosa or bulimia nervosa (Halmi et al., 1991; Fairburn et al., 1997; Garfinkel et al., 1995). It is unclear how a family history of a psychiatric disorder could increase a person's risk of an eating disorder. It has been suggested that a family history of a psychiatric illness could predispose to depression or obsessive-compulsive disorder that could then increase vulnerability to an eating disorder.

There are certain patterns of family interactions that are commonly observed in families of individuals with eating disorders; however; it is unclear whether these patterns predispose to eating disorders or are a consequence of them. Family characteristics such as parental overprotectiveness, rigid family organization, avoidance of open discussion of disagreements are associated with anorexia nervosa (Vandereycken, 1995; Walters and Kendler, 1995). Conversely, low parental contact,

parental criticism, parental high expectations and noncohesiveness are associated with bulimia nervosa (Fairburn et al., 1997; Vandereycken, 1995). These family dynamics could foster disordered eating in a number of ways including attention seeking, perfectionism, or low self-esteem.

Western culture's continual focus on beauty and an excessively thin beauty ideal can also contribute to the development of eating disorders. Societal pressures can cause individuals, especially young girls, to turn to extreme weight-loss methods trying to meet an unrealistic beauty ideal. Stice and Agras (1998) demonstrated that adolescent girls' perceived pressure to be thin, their thin-ideal internalization, and body dissatisfaction predicted the onset of binge eating and compensatory behaviours. Research has consistently demonstrated an increased rate of eating disorders in subcultures that particularly emphasize thinness. For example, female elite athletes in sports that emphasize leanness, such as gymnastics, are at significant risk for disordered eating (Sundgot-Borgen, 1994). Society's extremely thin beauty ideal and the idea that the human body is infinitely malleable can lead to disordered eating in vulnerable individuals.

There may also be a biological component to eating disorder predisposition. For example, individuals with anorexia nervosa often show an increase in serotonin activity even after long-term weight restoration, suggesting that abnormal serotonin activity is an intrinsic disturbance (Kaye, 1995). Serotonin pathways inhibit appetite and may be associated with certain characteristics such as rigid, inhibited, anxious and obsessional behaviours. Thus, it has been suggested that a disturbance in serotonin activity may contribute to the pathological eating behaviour and weight loss that leads to anorexia nervosa. The opposite may be true for bulimia nervosa. It has been proposed that decreased serotonin activity and increased peptide YY (PYY) may predispose to bulimia nervosa (Pirke, 1995; Kaye et al., 1990). Increased PYY and decreased serotonin would strongly stimulate feed intake and could predispose to binge eating. These findings do not imply that eating disorders are "predetermined" by one's biology. However, they do suggest that some individuals have specific intrinsic vulnerabilities

for certain behaviours that may be exaggerated into an illness by certain stressors or psychosocial factors.

b) Precipitating Factors

Dieting is the behaviour precursor to an eating disorder; however, not all individuals who diet develop eating disorders. Dieting is considered a necessary, but not sufficient cause of eating disorders. There are certain factors that, when combined with dieting, result in an eating disorder in vulnerable individuals. It is not known exactly what factors act as precipitating factors. The risk factors mentioned above can act to precipitate an eating disorder once dieting has started. For example, a girl has low self-esteem so she begins a diet to receive approval from her peers at school. She is also a perfectionist. She loses a small amount of weight, but is not happy with a small weight loss and keeps striving for more and more weight loss, resulting in severe weight loss and anorexia nervosa.

Additionally, certain life stressors may act to precipitate an eating disorder in individuals who do not have adequate coping skills or support to deal with crises. Research has demonstrated a linear trend between the number of traumatic life events or stressors and eating disorder case status (Welch et al., 1997; Schmidt et al., 1997). The precipitating events shown to be associated with eating disorders are those involving a disruption in family or social structure such as a change in residence or change in family structure, and those involving a threat to physical integrity such as pregnancy or sexual or physical abuse. Troop and Treasure (1997) demonstrated that the onset of disordered eating is associated with fewer coping skills and less support in response to severe crises. Thus, individuals who lack adequate coping skills may turn to extreme dieting as a coping mechanism in the event of a major life stressor.

c) Perpetuating Factors

Some eating disorders are transient while others become firmly established and may even become chronic. The effects of starvation as well as certain cultural, family and psychological factors can serve to maintain an eating disorder once it has started.

In anorexia nervosa, starvation induces physiological changes that can perpetuate the disorder. Research has demonstrated that underweight anorexic patients have elevations in cerebrospinal fluid concentrations of corticotropin-releasing hormone, neuropeptide Y, vasopressin, and reductions in beta-endorphin and oxytocin levels (Demitrack et al., 1990; Kaye, 1995; Kaye et al., 1990). The correction of these disturbances by weight restoration implies that they are secondary to the malnutrition and/or weight loss. These alterations likely contribute to many of the psychological disturbances of the acutely ill anorexic such as depression, obsessional thoughts about food, cognitive distortions about the consequences of eating and obsessive distortions (Demitrack et al., 1990; Kaye, 1995; Kaye et al., 1990). These psychological disturbances can reinforce maladaptive eating behaviours and perpetuate the anorexia nervosa. Some research suggests that individuals with bulimia nervosa may demonstrate some of the same endocrine derangements (Brewerton et al., 1992). Patients with anorexia nervosa commonly experience delayed gastric emptying resulting in an increased and prolonged sensation of fullness after eating which can further discourage a patient from eating (Stacher et al., 1992).

There are also psychological and cultural factors than can maintain an eating disorder. Depression and anxiety commonly occur secondary to disruptive eating habits (Steere et al., 1990). Research indicates that these feelings can precipitate binge eating which can then result in more depression and anxiety, thus creating a self-perpetuating cycle (Stice and Agras, 1998; Elmore and Castro, 1990). Additionally, eating disorder symptoms can produce positive feedback in family systems or they can preserve the equilibrium in the family system by deflecting away from other problem areas (Garner, 1993). Cultural expectations can also serve a perpetuating role by providing positive reinforcement for restrictive eating and weight loss.

3. Complications of Eating Disorders

a) Physiological Complications

No other psychiatric disorder manifests as many physiological complications as eating disorders (Tables 1 and 2), although the majority of complications resolve

quickly upon symptom recovery. Most complications are foreseeable considering the maladaptive behaviours in which these patients engage; the effects of starvation and purging behaviours cause the most suffering. Individuals with subclinical eating disorders present with many of the same medical complications as clinical eating disorders, but the severity of the complications depends on the extent of the eating disturbance.

Gastrointestinal complications, cardiac abnormalities and fluid and electrolyte abnormalities occur frequently in individuals with eating disorders and usually result from laxative or diuretic abuse and self-induced vomiting. Renal abnormalities are very common in anorexia nervosa, being reported in up to 70% of patients (Brotman et al., 1986; Sharp and Freeman, 1993). Neuroendocrine abnormalities are also quite common, with ammenorrhea resulting from deficiencies in gonadal steroid hormones being a mandatory diagnostic criteria for anorexia nervosa. The deficiency of gonadal steroid hormones can result in low bone mass leading to extremely high risk for osteoporosis (Bachrach et al., 1990; Sharp and Freeman, 1993). Osteoporosis is one of the only medical complications of eating disorders not rapidly reversed by weight regain.

Table 1: Medical Complications of Bulimia Nervosa

Gastrointestinal	**Fluid and Electrolyte**
Salivary gland hypertrophy	Dehydration
Esophageal and gastric perforation	Alkalosis
Esophagitis	Acidosis
Gastric rupture	Hypochloremia
Gastric and duodenal ulcer	Hypokalemia
Pancreatitis	Hyponatremia
Constipation	**Endocrine**
Cathartic colon	Irregular menses
Bleeding	**Dental**
Steatorrhea	Enamel erosion
Protein-losing gastroenteropathy	
Cardiovascular	
Hypotension	
Arrhythmias	
Ipecac toxicity	

Table 2: Medical Complications of Anorexia Nervosa

Gastrointestinal	**Bones**
Impaired taste	Osteoporosis
Delayed gastric emptying	**Metabolic**
Constipation	Fasting hypoglycemia
Cardiovascular	Abnormal temperature regulation
Hypotension	**Hematologic**
Arrhythmias	Anemia
Mitral valve prolapse	Leukopenia
Cardiomyopathy	**Musculocutaneous**
Orthostasis	Hair loss
Peripheral edema	Lanugo hair
Bradycardia	Dry skin
Renal	
Elevated blood urea nitrogen	
Decreased glomerular filtration rate	
Endocrine	
Amenorrhea/irregular menses	
Low thyroid	
High plasma cortisol	

Mitchell et al., 1987; Robinson et al., 1988; de Zwaan and Mitchell, 1993; Mitchell, 1995; Riad et al., 1991; Goldbloom and Kennedy, 1995; Brotman et al., 1986; Sharp and Freeman, 1993

It is clear that eating disorders are associated with a vast number of medical complications, some of which are life threatening. The complications can be severe and complex and are typically related to purging and pathological eating behaviours. While they participate in the same unhealthy behaviours, but to a lesser degree of severity, individuals with subclinical eating disorders are at risk for many of the same medical complications.

b) Psychological Complications

Depression, anxiety disorders, poor social adjustment, and obsessive-compulsive disorders are common in individuals with eating disorders (Garner et al., 1990; Halmi et al., 1991). In most cases, these psychiatric symptoms develop secondary to the eating disorder and improve as eating disorder symptoms improve (Garner et al., 1990; Rastam et al., 1992; Mitchell and Groat, 1984). In anorexia nervosa, depression and obsessive thoughts can result from neurotransmitter disturbances that are caused by extreme

weight loss. In bulimia nervosa, depression, social withdrawal, and isolation may result from stress and embarrassment over loss of control of eating habits (Steere et al., 1990). Anxiety symptoms in eating disorders stem from the core features of these disorders; namely, a fear of eating certain foods, a fear of social situations involving food and a fear of having one's body exposed to scrutiny (Steere et al., 1990).

Disordered eating can have devastating consequences, both physically and emotionally. The consequences frequently play a role in actually perpetuating the eating disorder. Intensive treatment is often necessary to address the complications as well as the underlying issues of the disorder.

4. Treatment of Eating Disorders

a) Clinical Eating Disorders

The treatment of bulimia nervosa and anorexia nervosa consists of medical nutrition therapy, medical management, psychotherapy and antidepressant medications as indicated. A multidisciplinary team made up of a psychiatrist, a psychologist, a physician and a dietitian is usually responsible for the care of an individual with an eating disorder. The initial goal of treatment is nutritional rehabilitation, followed by the long-term goal of diagnosing and resolving associated psychological, family, social and behavioural problems (APA, 1993).

Medical nutrition therapy (MNT) is a crucial component in the treatment of eating disorders as chaotic eating patterns and self induced starvation often perpetuate the disorder. MNT is usually under the responsibility of the dietitian. Weight restoration is the initial treatment goal for the anorexic patient, followed by weight stabilization and maintenance. Patients with bulimia nervosa need to resume normal and relaxed eating behaviours, avoid restrictive practices and tolerate being at a weight higher than they desire. Most patients have misconceptions about nutrition, energy metabolism and dieting that may perpetuate the disorder itself. A dietitian's nutritional guidance and education is required for nutritional rehabilitation, as well as patient empowerment to maintain new healthy eating behaviours.

Psychotherapy of eating disorders encompasses a number of different therapies including cognitive behavioural therapy, pharmacotherapy, family therapy, interpersonal therapy, supportive-expressive therapy, and psychoeducation. The treatment of bulimia nervosa has been well studied, with research convincingly showing cognitive behavioural therapy (CBT) as the most effective treatment (Fairburn et al., 1986; Garner et al., 1993; Fairburn et al., 1991b). CBT may be even more effective for bulimia nervosa when combined with antidepressant medication (Agras et al., 1992; Mitchell et al., 1990). Relatively few controlled studies have been done on the treatment of anorexia nervosa. CBT and behavioural therapy have proven useful for patients with anorexia nervosa (Russell et al., 1987), as has family therapy for adolescent patients with the disease (Channon et al., 1989; Crisp et al., 1991). Long-term psychodynamic psychotherapy is used for some patients, but has not been well studied and there is little documentation of its effectiveness (APA, 1993).

CBT is based on the theory that the characteristic beliefs about shape and weight are of primary importance in maintaining the behavioural disturbances of the eating disorder; it is problem oriented and focuses on the present and future. Psychoeducation is usually the first step of CBT, followed by a combination of behavioural and cognitive techniques to help patients recognize the connections between their dysfunctional thoughts and maladaptive behaviours. CBT helps patients critically evaluate their attitudes about shape, weight and other cognitive distortions such as low self-esteem and extreme perfectionism. This mode of treatment is usually conducted on a one-on-one basis and consists of self-monitoring, stimulus control, assertiveness training, relaxation training, education about the disorder, problem solving, cognitive restructuring, learning coping skills and relapse prevention.

Family therapy has been effective in the treatment of adolescents with anorexia nervosa (Russel et al., 1987; Crisp et al., 1991). It is probably most effective when used after weight restoration and in combination with other treatments (Crisp et al., 1991). Therapy that involves family members can reduce hostile blaming, improve direct communications, provide mutual support, foster healthy autonomy, resolve feelings of guilt and clarify dysfunctional patterns. This therapy is based on the assumption that

the family may be ineffective in helping the patient eliminate her symptoms; it is not based on the assumption that anorexia nervosa is caused by the family.

Psychodynamic therapy is a long-term therapy that uses the relationship between the patient and the therapist as the primary treatment tool (Herzog, 1995). Therapy can last for up to 10 years, although some individuals improve after 1 to 2 years. Psychodynamic therapy provides the opportunity for therapeutic discussions and the correction of distorted cognitions. The goals of this type of therapy are to restore the capacity to feel and care, develop modes of feeling and expressing power and dependency and develop coping strategies that are more adaptive than current eating behaviours.

b) Subclinical Eating Disorders

The treatment of subclinical eating disorders remains fairly unstudied to date. These disorders are not associated with medical complications to the same extent that clinical eating disorders are. Research indicates, however, that 30% to 45% of individuals with subclinical eating disorders will subsequently develop clinical eating disorders. Therefore treatment of subclinical disorders could prevent the life-threatening complications of anorexia nervosa and bulimia nervosa. Psychoeducation has the potential to be an inexpensive and easily provided treatment for those displaying subclinical disordered eating.

Psychoeducation provides information about the nature of eating disorders in order to encourage attitudinal and behavioural changes. It is usually provided in a group format and allows participants to refine existing coping strategies and to develop competencies by being provided with information of how to do so. Emphasis is placed on personality and behavioural deficits rather than on eating patterns directly.

In a review of cognitive behavioural treatments for bulimia nervosa, Garner (1987) reported that 53% of studies included psychoeducation as part of therapy. There have only been a few studies that have investigated the sole use of psychoeducation in the treatment of eating disorders. Connors et al. (1984) showed that a 12 session, group psychoeducation program for bulimia nervosa resulted in significant improvements in psychological functioning and a 70% reduction in binge/purge episodes. Wolchik et al.

(1986) found similar improvements in women with bulimia nervosa who received a 7 week psychoeducation program. Olmsted et al. (1991) found that the group psychoeducation was not only significantly more cost-effective than intensive individual CBT, but was equally effective as CBT at decreasing eating disorder symptomatology in bulimic individuals who tended to be less pathological. Kaminski and McNamara (1996) conducted the only study to date that evaluated an intervention for subclinical eating disorders. Their 8-session intervention resulted in significant improvements in body satisfaction and self-esteem and significantly decreased use of potentially dangerous methods of weight management (ie. purging, skipping meal, fasting, diet pills, laxatives and diuretics) in subclinical bulimic women.

5. Measures of Eating Disorders Symptomatology and Related Measures

a) Eating Disorder Symptomatology

Assessing eating disorder symptoms provides useful information for research and in clinical practice. There are a number of approaches to assessing symptoms, with the most frequently used methods being semi-structured clinical interviews, self-monitoring and self-report questionnaires. The advantages of clinical interviews include the ability to arrive at a diagnosis and allowing for clarification of patient responses (Fairburn and Cooper, 1993). The disadvantages, however, include the fact that they take an hour or more to administer, a trained interviewer is required and they are not suitable when anonymous or group administration is required. Self-monitoring requires patients to record their food intake, weight control behaviours, and thoughts and feelings. It provides more accurate information about eating behaviours than retrospective assessment tools, but self-monitoring may influence the very behaviours being monitored. Self-report questionnaires are widely used to assess eating disorder symptoms. They have the advantage of being relatively economical, brief, easily administered and objectively scored. The major disadvantage of self-report questionnaires is that they can be less accurate than interview methods when ambiguous behaviours such as binge eating are assessed and they cannot be used to diagnose an eating disorder (Fairburn and Belin et al., 1994). It has been recommended that self-

reports be supplemented by symptom frequency data derived from an interview or a symptom checklist (Garner, 1995).

The two most commonly used self-report instruments for eating disorders include the Eating Disorder Inventory (EDI) and the Eating Attitudes Test (EAT). The EDI assesses specific psychological characteristics and symptoms common to disordered eating, while the EAT assesses global symptoms of eating disorders (Garner and Garfinkel, 1979; Garner et al., 1983). The EDI has 64 questions that make up 8 subscales including three eating disorder psychopathology subscales (Drive for Thinness, Bulimia and Body Dissatisfaction) and five general psychopathology subscales (Maturity Fears, Interoceptive Awareness, Interpersonal Distrust, Ineffectiveness and Perfectionism). Coefficient alphas for the EDI subscales range from .69 to.93, indicating good internal consistency (Garner, 1991). Additionally, the EDI has good test-retest reliability over a 3 week time period with coefficients all above .81 except for the Maturity Fears subscale (.65) (Wear and Pratz, 1987). The subscales also correlate well with the EAT, clinician rating and the Restraint Scale which measures dieting behaviour (Herman and Polivy, 1975; Garner et al., 1983; Williamson et al., 1995). Furthermore, the subscales can effectively differentiate between individuals with eating disorders and normal controls (Gross et al., 1986; Garner et al., 1983) and a total score above 40 has been shown to identify those at risk for eating disorders (Sundgot-Borgen, 1993).

Factor analysis of the EDI has been conducted in both clinical samples (those with clinical eating disorders) and non-clinical samples (college females without eating disorders). An eight-factor pattern corresponding to the original eight subscales was found in a sample of females with eating disorders (Welch et al., 1990). However, in non-clinical patient samples, researchers have not consistently found an eight-factor solution (Welch et al., 1988). Welch et al. (1988) found a three factor solution with one factor made of the perfectionism subscale, one factor comprised of the drive for thinness, bulimia and body dissatisfaction subscales and a third factor made up of the ineffectiveness and interpersonal distrust subscales. Also using a non-clinical sample, Williams et al. (1986) found an eight-factor solution similar to the eight original

subscales and Klemchuk et al. (1990) found a six-factor solution. In sum, factor analysis has confirmed the appropriateness of the eight subscales of the EDI for those with eating disorders, but there is conflicting evidence as to the effectiveness of the 8 subscales in non-clinical populations. In non-clinical samples, research does support two fairly stable factors of the EDI, a factor that measures specific eating disorder behaviours (drive for thinness, bulimia and body dissatisfaction) and a perfectionism factor (Williamson et al., 1995)

There are two versions of the EAT, the EAT-40 and the EAT-26, both of which are frequently used in research and clinical practice. Garner et al. (1982) found the additional questions on the EAT-40 to be redundant, although it is still frequently used. Garner et al. (1979) also found the EAT to have an alpha reliability coefficient of .94, indicating very good internal consistency. Carter and Moss (1984) reported a test-retest reliability coefficient of .84 over a 2-3 week time period. Additionally, the EAT is moderately correlated with the Mizes Anorectic Cognitions Questionnaire (MAC), the Bulimic Investigatory Test (BITE) and the 3 eating disorder symptoms subscales on the EDI (Gross et al., 1986; Henderson and Freeman, 1987; Williamson et al., 1995). The EAT is sensitive to therapeutic changes and reflects changes in symptomatology (Fairburn et al., 1986: Garner et al., 1993). Furthermore, this questionnaire can differentiate between eating disorder groups and can identify those at risk for disordered eating (Gross et al., 1986; Garner and Garfinkel, 1979). A score above 30 on the EAT-40 and above 20 on the EAT-26 have been identified as cut-off scores for detecting potential cases of eating disorders (Garner et al., 1982).

The EDI and the EAT are both widely used for screening and assessing treatment efficacy. For screening purposes, it is recommended that the questionnaires be used as the first component of a two part screening process; the second component being an interview to confirm assessment of eating behaviours (Garner, 1991). Without an interview, a diagnosis cannot be made. In assessing treatment efficacy, the EAT and the EDI should be administered together as they assess slightly different aspects of eating disorders (Gross et al., 1986). Additionally, a symptom checklist should be used to clarify the use of specific eating and purging behaviours.

b) Self-Esteem

Low self-esteem increases the risk of eating disorder development and is associated with eating disorder symptomatology (Kendler et al., 1991). In those with established eating disorders, improvements in self-esteem are associated with improvements in eating disorder symptomatology, although the direction of this association is not known (Garner et al., 1993; Wolchik et al., 1986). Measuring self-esteem is very useful since it provides information about patient quality of life, risk of developing disordered eating and response to treatment.

The Rosenberg Self-Esteem Scale (RSE), a 10 item self-report questionnaire, is the most widely used measure of self-esteem and has received substantial support as a unidimensional and single-factor scale of global self-esteem (Blascovich and Tomaka, 1991). It is a popular tool since it is very easy to complete and simple to score. Although originally designed as a Guttman-type scale, it is typically scored using a four point Likert scale scoring format. Wylie (1989) reported test-retest reliability coefficients of .85 and .63 over a 2 week and 7 month period, respectively. The RSE also has high internal reliability; Lewis (1982) and Fleming and Courtney (1984) reported Cronbach's alphas of .87 and .88, respectively. Additionally, the RSE is associated with many self-esteem related constructs. For example, Byrne et al. (1983) reported the RSE to be significantly correlated with the Self-Esteem Inventory – General Self Subscale (r=.604), and Fleming and Courtney (1984) reported it to be significantly correlated with general self-regard (r=.78), social confidence (r=.51), and school abilities (r=.35). The RSE is one of the most valid measures of global self-esteem and is sensitive to therapeutic changes in eating disorder treatment (Byrne, 1983; Kaminski and McNamara, 1996; Wolchik et al., 1986).

c) Depression

Depression is also strongly associated with eating disorder symptomatology. In most cases, depression occurs concurrently with the eating disorder and tends to improve as the eating disorder improves (Walters and Kendler, 1995; Rastam, 1992;

Wolchick et al., 1986). The Beck Depression Inventory (BDI) is one of the most widely used instruments to assess depression. It is a 21 item self-report questionnaire that is easy to administer, simple to score and is considered the best screening instrument for depression (Riley, 1995)

The psychometric properties of the BDI were reviewed in depth by Beck et al. (1988) who concluded that the BDI has high internal consistency, good test-retest reliability and high concurrent validity with other measures of depression. The test-retest stability of the BDI is quite good, naturally better in non-psychiatric than in psychiatric populations. The test-retest reliability over a 2-week time period ranges from .74 to .87 (Ambrosini et al., 1991). Studies on the internal consistency of the BDI have reported mean coefficient alphas of .86 and .81 for psychiatric and non-psychiatric populations, respectively (Beck et al., 1988). The ability of the BDI to differentiate between normal control subjects, patients with medical illnesses and depressed individuals is well established, as is its ability to differentiate subtypes of depression (Beck et al., 1988).

The BDI is commonly used to screen for depression and to assess treatment efficacy in individuals with eating disorders (Wolchik et al., 1986; Fairburn et al., 1991b). Individuals with disordered eating commonly score above average on the BDI (Riley, 1995). The above average scores may reflect the high prevalence of depression in the eating disorder population or the endorsement of the two appetite/weight items, two items concerning fatigue/sleep and an item on interest in sex (Williamson et al., 1985; Garfinkel et al., 1983).

d) Emotional Distress

General emotional distress is a useful indicator of psychiatric symptoms and overall clinical change or improvement. The Brief Symptom Inventory (BSI) is a 53 item self-report inventory that has received strong support as a unidimensional construct of general psychological distress or discomfort (Piersma et al., 1994). The BSI has 9 primary symptom subscales and three global indices of distress. Researchers have

demonstrated that the global symptom index is the most valid index; little is gained by using the separate subscales (Piersma et al., 1994).

The BSI is the shortened form of the more widely used Symptom Checklist-90-Revised (SC-90-R). The BSI correlates extremely well with the SC-90-R which is commonly used to investigate distress in individuals with eating disorders (Derogatis, 1993; Williamson et al., 1985; Fairburn et al., 1991b). Although no studies of its use in eating disorders have been reported, the BSI has been used in a number of patient populations (Derogatis, 1993). The BSI demonstrates good test-retest reliability over a 2-week time span, with the GSI index demonstrating a stability coefficient of .90 (Derogatis and Melisaratos, 1983). Additionally, the BSI correlates well with other tools that assess psychological status and demonstrates good predictive validity in a number of patient populations (Derogatis, 1993; Derogatis and Melisaratos, 1983).

C. EATING DISORDERS AND TYPE 1 DIABETES

Type 1 diabetes and eating disorders are both relatively common conditions; therefore, it is expected that they would co-exist by chance with a reasonable frequency. There are some theoretical reasons to expect that eating disorders may be over-represented in the type 1 diabetic population, however little evidence is available to document this phenomenon. Nevertheless, there is significant risk associated with subclinical and clinical eating disorders in individuals with type 1 diabetes, but research is lacking in the area of prevention and treatment of disordered eating in these individuals.

1. Risk Factors Associated with Type 1 Diabetes

In the majority of cases, the diagnosis of type 1 diabetes occurs before that of an eating disorder, suggesting that diabetes may be a risk factor for disordered eating (Friedman et al., 1998). Individuals with type 1 diabetes may be at increased risk because of factors specific to diabetes or because of factors involved with coping with a chronic illness. One risk factor involves the weight changes that commonly occur around the time of diagnosis of type 1 diabetes. Individuals commonly lose a significant amount of weight immediately prior to diagnosis. Once insulin therapy is initiated and metabolic control is established, the weight is usually regained (Steel et al., 1990). These weight fluctuations can trigger decreased self-esteem and increased body dissatisfaction, both of which can lead to an increased likelihood of disordered eating behaviours (Stice and Agras, 1998). Steel et al. (1990) demonstrated an increase in body dissatisfaction in women within the first year of diagnosis of type 1 diabetes. Adolescent girls with type 1 diabetes tend to have a greater body mass index than their peers (Pietilainen et al., 1995). This tendency to be heavier can also lead to low self-esteem, weight preoccupation and dangerous attempts at weight loss.

A second risk factor for eating disorders involves the traditional nutritional management of diabetes. Individuals with type 1 diabetes are commonly taught to avoid specific foods, to maintain a strict scheduling of meals and snacks and to limit weight gain. This can be seen as a form of chronic dietary restraint which has been

consistently associated with binge eating (Polivy, 1996). Rigid medical and nutritional advice can create a situation in which perfectionistic, unrealistic attitudes towards eating, blood glucose and weight can flourish, rendering individuals particularly susceptible to disordered eating.

Individuals with type 1 diabetes also have access to a very convenient weight loss method: reducing or omitting required insulin doses. Reducing required insulin doses results in hyperglycemia, dehydration, and weight loss. Approximately 12-37% of women with type 1 diabetes omit insulin to control weight (Friedman et al., 1998; Fairburn et al., 1991a; Rydall et al., 1997; Peveler et al., 1994; Rodin et al., 1991; Stancin et al., 1989). The use of insulin reduction or omission to control weight is not confined to those who have clinical eating disorders, but is commonly found in women with subclinical eating disorders (Fairburn et al., 1991a). Access to such a convenient and readily available weight loss technique has the potential to perpetuate an eating disorder.

A chronic medical illness such as type 1 diabetes can pose a challenge to psychological well-being, potentially increasing vulnerability to disordered eating. Diabetes has been noted to significantly interfere with adolescent development by causing feelings of being different, low self-esteem, and exacerbating dependence/independence conflicts with family (Jacobson et al., 1986; Tattersall and Lowe, 1981). Patton et al. (1986) reported that physical illness predisposes to anorexia nervosa. The diagnosis of a chronic illness could be seen as a traumatic life event that could act as a precipitating factor for disordered eating in those who do not have adequate coping skills or adequate support (Ward et al., 1995).

Certain family dynamics may also contribute to eating disorder development in vulnerable individuals with type 1 diabetes. Research has shown a linear relationship between disordered eating status and dysfunctional family dynamics in individuals with type 1 diabetes (Maharaj et al., 1998). Maharaj et al. (1998) demonstrated that girls with type 1 diabetes and severe eating disturbances report more impaired communication and mistrust in the accessibility and responsiveness of both parents to their needs, as compared to girls with no eating disturbances. Girls with mild eating

disturbances reported levels of impaired communication and mistrust in between that of the highly disturbed and non-disturbed groups. In addition, girls with eating disturbances and their mothers described their families as conflictual, lacking in support and cohesion and having inadequate organization and structure in planning activities and responsibilities. Furthermore, the families of girls with disordered eating also emphasized independence, indicating that these families set high standards for achievement or independence, but fail to provide adequate support and structure. These dysfunctional family dynamics may create an environment that could foster the development of eating disorders, although it is unclear at this time whether the dynamics described are a consequence of or a predisposing factor to eating disturbances.

2. Prevalence of Eating Disorders in Type 1 Diabetes

There are many factors associated with type 1 diabetes that can increase susceptibility to eating disorders; however, recent studies suggest that the prevalence of eating disorders in the type 1 diabetic population is equivalent to that in the general population (Powers et al., 1990; Robertson and Rosenvinge, 1990). Some studies have reported a higher prevalence of subclinical eating disorders among women with type 1 diabetes (Rosmark et al., 1986; Steel et al., 1989), although these findings have not been confirmed by more recent studies (Marcus et al., 1992; Peveler et al., 1992). Noteworthy is the fact that bulimia nervosa or bulimia-like disorders have accounted for the vast majority of disordered eating diagnoses in individuals with type 1 diabetes. No study to date has had adequate statistical power to demonstrate conclusively that there is not an increased risk for eating disorders within the diabetic population (Daneman et al, 1998).

3. Complications Associated with Co-Existing Eating Disorders and Type 1 Diabetes

Eating disorders are associated with significant medical complications. In individuals with type 1 diabetes, eating disorders may pose an even greater health risk.

A number of physiological and psychological complications can result due to the impact of disturbed eating on metabolic control and self-esteem.

a) Physiological Complications

Although the prevalence of eating disorders may not be increased in the type 1 diabetic population, the coexistence of the two conditions is a serious concern. More than half of individuals with co-existing type 1 diabetes and disordered eating omit insulin to produce hyperglycemia and weight loss (Rodin et al., 1991). Studies have consistently demonstrated that the combination of eating disorders and type 1 diabetes is associated with poor metabolic control, recurrent episodes of severe hypoglycemia, and diabetic ketoacidosis (Affenito et al., 1997a; Marcus et al., 1992; Steel et al., 1989; Wing et al., 1986; Fairburn et al., 1991a). It is especially noteworthy that the same associations have been found with subclinical eating disorders (Affenito et al., 1997a). Affenito et al. (1997b) found subclinical and clinical eating disorders to be associated with elevated serum triglycerides, cholesterol and total lipid concentrations. Additionally, subclinical and clinical eating disorders are associated with increased risk of microvascular complications of diabetes, including painful neuropathy (Affenito et al., 1997a; Rydall et al., 1995; Rydall et al., 1997). Although the direction of the association between poor metabolic control and eating disorders has not been demonstrated, most researchers speculate that disordered eating directly affects metabolic control which then leads to long-term complications. Disordered eating is associated with poor adherence to diabetes self care routines such as blood and urine testing (Affenito et al., 1997a; Littlefield et al., 1992; LaGreca et al., 1990). The complications associated with eating disorders are likely attributable not only to purging behaviours and erratic caloric intake, but to insulin misuse and noncompliance to the other aspects of the diabetes treatment regimen.

b) Psychological Complications

Depressive symptoms and anxiety disorders are common in people with eating disorders and in people with diabetes mellitus (Lustman, 1992; Grubb et al., 1993;

Steere et al., 1989). However, there has been little research on the psychological status of individuals with co-existing disordered eating and type 1 diabetes. Case reports of individuals with disordered eating and type 1 diabetes indicate that depression is very common (Copeland et al., 1995, Mannucci et al., 1997; Peveler and Fairburn, 1992; Peveler and Fairburn, 1989). The interaction between eating disorders, diabetes mellitus, depression and metabolic control is likely very complex. In individuals with diabetes, depression is associated with poor metabolic control as well as with poor adherence to diabetes self-care routines (Mazze et al., 1984; Lustman et al., 1986; Littlefield et al., 1992). In eating disorder, depression is likely a result of the unhealthy eating behaviours. The co-existence of diabetes and disordered eating is associated with poor adherence to diabetes self-care routines, poor metabolic control and depression. Depression could be caused by disordered eating, poor metabolic control and/or poor adherence to diabetes self-care routines. Alternatively, poor metabolic control and poor adherence to diabetes self-care routines could be caused by the depression. Further research is required to clarify these complex interactions.

4. Treatment of Co-Existing Eating Disorders and Type 1 Diabetes

a) Clinical Eating Disorders

Eating disorders in individuals with diabetes can have serious consequences including poor blood glucose control, diabetic complications and emotional disturbances. Although little research has been conducted on the treatment of eating disorders in people with diabetes, it should be quite similar to that provided to non-diabetic individuals. Treatment should include nutrition therapy, medical management, psychotherapy and psychiatric medications as indicated. The initial goal of treatment is nutritional rehabilitation, including stabilization of blood glucose (ie. absence of severe hypo- and hyper-glycemic episodes), weight restoration, and stabilization of eating behaviours and (Peveler and Fairburn, 1989). The long-term goal is to diagnose and help resolve associated psychological, family, social and behavioural problems, as well as to achieve optimal glycemic control (APA, 1993). A close liaison between the eating disorder treatment team and the diabetes care team is essential. It is usually

desirable to have one person undertake the overall responsibility of the patient's medical care during treatment for the eating disorder (Peveler and Fairburn, 1989). When treatment is completed, it is helpful for the team treating the patient to give the diabetic care team advice on continuing management to minimize relapse risk.

There are a number of psychotherapies available for eating disorder treatment including pharmacotherapy, family therapy, interpersonal therapy, supportive-expressive therapy, and psychoeducation, although none have been systematically assessed for use with individuals with diabetes. Case reports suggest that CBT, family therapy, interpersonal therapy, group therapy and antidepressant medications may be useful in individuals with co-existing type 1 diabetes and disordered eating (Pitel et al., 1998; Copeland et al., 1995; Hillard et al., 1983; Fairburn and Peveler, 1992; Peveler and Fairburn, 1989; Peveler, 1995). A number of difficulties with the psychological treatment of individuals with type 1 diabetes have been reported. One such difficulty is engaging the patient in therapy (Peveler and Fairburn, 1992). It is possible that eating disorders in people with type 1 diabetes may be particularly intractable. It is also possible that significant mood and personality disorders are more common in diabetic patients, making treatment of eating disorders more difficult (Peveler, 1995).

Therapy requires a number of modifications for individuals with type 1 diabetes. First, in addition to monitoring their own thoughts and eating habits, they must also monitor their insulin injections and blood glucose. Second, there is usually a degree of discrepancy between the modifications to eating habits advocated for the management of the eating disorder and the dietary advice often given for the management of diabetes. There is evidence to suggest that strict dieting predisposes to bulimia nervosa; therefore, treatment is aimed at lessening the patient's wish to diet and promoting a more flexible approach to eating. However, the traditional dietary management of diabetes includes total avoidance of certain foods and strict scheduling of meals and snacks. It may be necessary to find a compromise between the two extremes. This usually involves accepting a slightly raised GHb level and using multiple daily insulin injections.

b) Subclinical Eating Disorders

Subclinical disordered eating is considered relatively unimportant when it occurs in people without diabetes because the consequences are not usually life-threatening. However, individuals with subclinical eating disorders are at significant risk for developing more serious disorders. Additionally, the co-existence of subclinical disordered eating and type 1 diabetes is associated with a number of severe medical complications. Unfortunately, there are no treatment recommendations for these individuals. Implementing the same treatments for subclinical conditions as for clinical conditions may represent "over-treatment" for some individuals. Group psychoeducation has proven effective in non-diabetic individuals with mild to moderate eating disorders as well as subclinical eating disorders. Only one study to date has evaluated group psychoeducation for individuals with co-existing subclinical disordered eating and type 1 diabetes. Olmsted et al. (1998) reported significant improvements in eating disorder symptomatology in a group of adolescent girls who received a 6-session group psychoeducation program; the control group did not demonstrate any such improvements. The results concerning metabolic control are pending. Further studies are required to document the effectiveness of group psychoeducation for this critical population.

5. Measures of Eating Disorder Symptomatology in Diabetes

It is important to have valid eating disorder assessment tools to use with individuals with diabetes. The two most commonly used self-report measures for eating disorder symptomatology are the EAT and the EDI. Although neither questionnaire has been specifically validated for use with diabetic populations, both questionnaires have been widely used in diabetic populations (Steel et al., 1989; Robertson et al., 1990; Rosmark et al., 1986). The EAT-40 has been used more commonly in individuals with diabetes than the EAT-26. Some of the questions on the EDI and EAT may demand particular answers from diabetic individuals, potentially resulting in false positive scores; therefore, specific questions are removed from each of the indices prior to analysis (Steel et al., 1989; Robertson et al., 1990; Rosmark et al., 1986). Cantwell and

Steel (1995) also suggest adding a question about insulin misuse to either questionnaire in order to obtain additional information concerning disordered eating behaviours.

Six questions are normally removed from the EAT-40 for analysis for individuals with diabetes, resulting in the EAT-34. Three questions are usually removed from the EDI for analysis in individuals with diabetes. All three of these questions are in the drive for thinness subscale. As a result of the removal of questions, the established cut-off scores for these questionnaires are more conservative for use in diabetic populations. A score above 18 instead of 30 on the EAT-40 has been suggested as a cut-off to detect potential cases of eating disorders in individuals with type 1 diabetes (Steel et al., 1989). Cantwell and Steel (1996) suggest that the cutoff could be raised to 24 to increase specificity. There have been no studies assessing a cut-off for the EDI for use with diabetic individuals.

D. SUMMARY OF LITERATURE

Eating disorders and type 1 diabetes mellitus are both serious conditions, each with diverse etiological factors, severe consequences and complex treatment approaches. When the two disorders co-exist, the consequences can be especially devastating and difficult to treat. Subclinical disordered eating has the potential to progress to more serious forms of disordered eating; when it occurs in individuals with type 1 diabetes the consequences can be quite serious. Therefore, the treatment of subclinical disordered eating in individuals with type 1 diabetes is very important, yet there are very few treatment recommendations for this group. Psychoeducation has the potential for being a cost-effective treatment for those with type 1 diabetes, although only one study has documented its effectiveness. This study will further evaluate the effectiveness of group psychoeducation in individuals with co-existing type 1 diabetes and subclinical disordered eating.

CHAPTER THREE
EXPERIMENTAL DESIGN AND METHODOLOGY

A. ETHICAL APPROVAL

The present study received ethical approval from the University of Alberta, Faculty of Medicine and Oral Health Sciences Research Ethics Board. The Certificate of Approval from the University of Alberta is provided in Appendix A. Participants provided informed consent before participation in the screening and before participation in the intervention component of the study (see Appendix B and C).

B. EXPERIMENTAL DESIGN

The present study was conducted at the University of Alberta, Department of Agricultural, Food and Nutritional Sciences and Department of Medicine, Edmonton, Alberta, Canada. It was a pre-test/post-test design involving a treatment group and a control group (Figure 1). Participants were women with co-existing type 1 diabetes and subclinical disordered eating. They were assigned to the treatment group or the control group in a random manner based on the dates that they were recruited. The treatment group received a 6-session group psychoeducation program, while the control group did not receive any treatment. Measurements were taken at pre-, post-, and 1 month post-intervention in the treatment and control groups, and at 6 months post-intervention in the treatment group only. For ethical reasons as well as to ensure adequate recruitment, the control group's participation in the study was terminated after the 1 month post-intervention measurements, at which time they received the intervention.

Four major variables related to eating disorders were measured at each time point. These included metabolic control, diabetes treatment adherence, eating disorder symptomatology and general psychopathology. Metabolic control was analyzed as 2 sub-variables, fructosamine and hemoglobin A_{1c} (HbA_{1c}). Diabetes treatment adherence was assessed using a self-report questionnaire called the Self-Care Inventory

(SCI). Eating disorder symptomatology was analyzed as 3 sub-variables, the Eating Attitudes Test (EAT-34), eating disorder symptoms and insulin omission. This information was obtained using the EAT-34, the sum of 3 subscales on the Eating Disorder Inventory (EDI) and the sum of 2 questions addressing insulin omission. General psychopathology was analyzed as 3 sub-variables, self-esteem, depression, and general emotional distress. General psychopathology was measured using 3 separate self-report questionnaires, the Rosenberg Self-Esteem Scale (RSE), the Beck Depression Inventory (BDI) and the Brief Symptom Inventory (BSI).

Group	Pre-Intervention	Post-Intervention	1 Month Post-Intervention	6 Months Post-Intervention
Treatment Group (n=8)				
Control Group (n=6)				

Figure 1 – Experimental Design

C. SCREENING COMPONENT

The intention was to screen at least 100 women with type 1 diabetes in order to find 20 women who had subclinical disordered eating who wanted to take part in intervention component of the study. The sample size of 18 was calculated based on a pilot study that investigated the prevalence of eating disorder symptomatology (using the total EDI score) in the University Hospital Metabolic Center - Type I Intensive Diabetes Management Clinic. To obtain a 95% confidence level, 9 subjects per group (total n=18) were thought to be required (see Appendix D).

1. Participants

Participants had to meet certain criteria with respect to gender, age and years of diabetes in order to be eligible for the screening component of the study. Only women

were investigated in this project because women are at much higher risk for developing eating disorders than men. This may be even more pronounced in those with diabetes, as researchers have found that men with type 1 diabetes do not report omitting or reducing insulin for weight control (Fairburn et al., 1991a; Polonsky et al., 1994; Rydall et al., 1997). Additionally, it would not be appropriate to include men and women in the same psychoeducation group program because of the potential consequences on group dynamics, group discussions and personal disclosures.

The second eligibility criteria was related to age. Initially, only individuals between the ages of 16 and 35 were going to be recruited. A small age range would ensure a homogeneous sample. The upper limit was increased to 50 years of age in order to increase subject enrollment.

The final eligibility criteria for screening involved duration of diabetes. Participants were required to have had type 1 diabetes for more than one year. The risk of eating disorder development increases substantially during the first year of diagnosis of diabetes. Steel et al. (1990) demonstrated that women experience a significant increase in body dissatisfaction during this first year. Additionally, the first year of diagnosis can be quite traumatic, being diagnosed with a chronic illness requiring daily lifestyle changes. In order to ensure that the participants were experiencing the same issues associated with diabetes and the same risk of eating disorder development, they were required to have had diabetes for more than 1 year.

2. Recruitment

Recruitment efforts began in April 1997 and ended in November 1998. Volunteers for screening were recruited through a number of methods including community announcements and direct contact through the University Hospital Metabolic Centre (University Hospital, Edmonton, AB). Specific recruitment methods included the following:

- Attending the University Hospital Metabolic Center - Type I Intensive Diabetes Management Clinic every Monday to distribute questionnaires

- Attending the University Hospital Metabolic Center - 4 Day Education Program for individuals with type 1 diabetes once a month
- Letters to 60 patients of the University Hospital Metabolic Center - Type I Intensive Diabetes Management Clinic (see Appendix E)
- Public service announcements on two local Edmonton television stations and in the Edmonton Journal
- Three advertisements in the Edmonton Journal, Life Section
- Two announcements in the Reach Magazine (Canadian Diabetes Association Newsletter for Northern Alberta)
- An article in the Edmonton Examiner (Free Edmonton Community Newspaper)
- Flyers posted at all Diabetes Clinics in Edmonton
- Flyers posted at the Canadian Diabetes Association Main Office and at various locations on the University of Alberta Campus
- Letters to 31 women from the Type 1 Diabetes Registry who lived in Edmonton and had previously agreed to be contacted about research studies (See Appendix F)

The public service announcements, newspaper advertisements and posters contained the same information with only slight modifications in format and wording (see Appendix G).

3. Procedures

The screening component of the study involved the completion of a consent form (Appendix B), the EDI and a general demographic information questionnaire; however, procedures differed slightly between those individuals who were part of the University Hospital Metabolic Center and those who were from the community at large. The first difference was that patients of the University Hospital Metabolic Centre were required to complete four additional questionnaires that were not used in this study (Independent Study entitled, Assessment of Interventions to Deal with Identified Psychosocial Coping Abnormalities in Subjects with insulin dependent diabetes

mellitus). The second difference included the fact that an endocrinologist at the University Hospital Metabolic Centre, also a researcher on this project, had full access to her patients' questionnaire results.

The specific procedures for the patients of the University Hospital Metabolic Centres weekly Type I Intensive Diabetes Management Clinic were as follows. On the clinic days, subject eligibility was determined from patient charts and then eligible subjects were approached in the waiting room before their appointment. At this time the project was explained and the subjects were asked if they would like to participate by completing the questionnaire package. Subjects were told that the information from the questionnaires would help their endocrinologist individualize their diabetes care plan. Questionnaires were completed in the waiting room with the researcher present.

In addition to the Type 1 Intensive Diabetes Management Clinic, the University Hospital Metabolic Centre conducts a 4 Day Education Program once a month for individuals with type 1 diabetes. During the monthly education program, patients were given information by the researcher about the screening project and were given the choice to participate. They were also informed that their endocrinologist would be accessing their results. Those who showed interest were given a questionnaire package to complete that evening. The packages were retrieved the next day by the researcher. Since the researcher was not present during the completion of the questionnaires, subjects were given the opportunity to ask questions the next day and to complete the questionnaires at that time, if necessary. Additionally, because it was not possible to confirm eligibility prior to the education program, eligibility was determined after the questionnaires were completed. The questionnaire results of those who did not meet the eligibility criteria were treated in a similar fashion to all other results (ie. given to endocrinologist), but not included in the study results.

All other participants were either patients of the University Hospital Metabolic Centre who were contacted by letter, individuals from the Type 1 Diabetes Registry (contacted by letter) or from the community at large. All of these participants contacted the researcher by telephone. At this time the inclusion criteria was confirmed and an appointment was made for questionnaire completion. All subjects completed the

questionnaires at the Agriculture/Forestry Centre (University of Alberta, Edmonton, AB) except those who were patients from the University Hospital Centre Metabolic Clinic; these individuals completed the questionnaires at the clinic. The researcher was present while patients completed the questionnaires.

All participants who were interested in their questionnaire results were telephoned for discussion of questionnaire results. The published norms were used to quantify questionnaire results and additional information or referrals were given as requested.

4. Screening Instruments

The following assessment tools were used during screening to identify individuals with potential subclinical disordered eating who would be eligible for the study:

a) Eating Disorder Inventory (EDI)

The total EDI score and the three eating disorder symptom subscales (Body Dissatisfaction, Bulimia and Drive for Thinness) were used to identify individuals with subclinical disordered eating. In addition, the five general psychopathology subscales (Interpersonal Distrust, Interoceptive Awareness, Maturity Fears, Perfectionism, Ineffectiveness) were used to study the difference between those identified with subclinical disordered eating and those identified with normal eating behaviours.

Test materials were purchased from Psychological Assessment Resources Inc. (Odessa, Florida) and included item booklets and answer sheets. Information on administration, scoring, norms, reliability and validity was obtained from the EDI-2 Professional Manual (Garner, 1991). The EDI has 64 questions that subjects rate on a 6-point likert scale as to how much the statement applies to them (1=never, 2=rarely, 3 = sometimes, 4 = often, 5 = usually, 6 = always). For example, "I think my stomach is too big". Scoring was as follows. Responses for each item were weighted from 0 to 3. A score of 3 was assigned to the response farthest in the "symptomatic" direction (ie. "always" or "never" depending on whether the item was keyed in the positive or

negative direction). The next adjacent responses were assigned a 2 and a 1, respectively. A score of 0 was assigned to the three responses farthest in the "asymptomatic" direction. Three items in the Drive for Thinness subscale are thought to be affected by diabetes and were removed prior to analysis (see Table 3). Subscale scores were computed by summing the score of all items within that specific subscale.

The total EDI score provides general information concerning the symptoms and psychopathologies commonly associated with anorexia nervosa and bulimia nervosa. The three eating disorder symptom subscales provide detailed information related to specific eating disorder symptoms. The Drive for Thinness subscale assesses the pursuit for thinness. Items within this subscale assess excessive concern with dieting, preoccupation with weight and fear of weight gain (Garner, 1991). The Bulimia subscale assesses the tendencies to think about and engage in bouts of uncontrolled overeating (Garner, 1991). The Body Dissatisfaction subscale assesses unhappiness with overall shape and size of particular regions of the body (ie. stomach, hips, thighs and buttocks) that are usually of greatest concern in those with eating disorders (Garner, 1991). The five general psychopathology subscales assess psychological traits relevant to eating disorders. The Interpersonal Distrust subscale assesses reluctance to form close relationships, while the Interoceptive Awareness measures confusion in recognizing and accurately responding to emotional states. The Maturity Fears subscale assesses the desire to retreat to the security of childhood and the Perfectionism subscale assesses the extent to which one believes that personal achievements should be superior. Finally, the ineffectiveness subscale assesses feelings of general inadequacy, insecurity, worthlessness and emptiness.

The norms used to interpret the specific subscales are based on the scores of 770 female college students without eating disorders in first and second year psychology classes at the University of Toronto (Garner et al., 1983). A cut-off of ≥ 40 was used to identify individuals with potential subclinical disordered eating.

Table 3 – Items Removed from the EDI for Use with Individuals with Type 1 Diabetes

1	I eat sweets and carbohydrates without feeling nervous
7	I think about dieting
11	I feel extremely guilty after overeating

b) Insulin Omission

Since insulin omission or reduction is a common behaviour in individuals with type 1 diabetes and disordered eating, two questions were designed to assess insulin omission thoughts and behaviours. This information was used to compare the women identified with subclinical disordered eating and those identified with normal eating behaviours. Two questions were added to the end of the EDI and were as follows: "I have thoughts of omitting insulin to control my weight" and "I have omitted insulin to control my weight". Respondents answered the two questions on a six point likert scale ranging from "always" to "never". Possible scores on each question ranged from 0 to 4. An answer of "always" received a score of 4, "usually" a score of 3, "often" a score of 2, "sometimes" a score of 1, and "rarely" and "never" a score of 0. The scores on the two questions were added together to receive a total score out of 8 for insulin omission.

c) Demographics

A demographic questionnaire was used to assess age, self-reported weight and height, highest level of education completed, duration of diabetes, date and value of last HbA_{1c} value and frequency of self-blood glucose monitoring (Appendix H).

D. INTERVENTION COMPONENT

1. Participants

The screening component of the project detected individuals with potential subclinical disordered eating. These individuals had to meet additional criteria that confirmed subclinical disordered eating and secured eligibility for the intervention component of the study. For the purposes of this research project, an individual who experiences subclinical disordered eating demonstrates the same unhealthy eating

attitudes and behaviours as individuals with anorexia nervosa or bulimia nervosa, but to a lesser degree. Individuals with subclinical disordered eating were identified as follows:

1) Total EDI score ≥ 40

+

2) At least one out of three elevated EDI eating disorder symptom subscales:
   a) Drive for Thinness subscale score ≥ 7 (≥73 percentile of college females) **OR**
   b) Bulimia subscale score ≥ 2 (≥77 percentile of college females) **OR**
   c) Body Dissatisfaction score ≥ 15 (≥76 percentile of college females)

+

3) EAT score ≥ 17

To ensure that participants could truly be characterized as individuals with subclinical disordered eating, the inclusion and exclusion criteria were very specific. Sundgot-Borgen (1993) found a total EDI score ≥ 41 to effectively detect those with potential clinical eating disorders from a sample of 448 girls between the ages of 12 and 35 years who did not have diabetes. Since questions are removed from the EDI for use with individuals with diabetes, this cut-off score is more conservative for individuals with diabetes, therefore, a score ≥ 40 was used to detect subclinical disordered eating in individuals with type 1 diabetes. The EDI is comprised of 8 subscales, 3 are specific eating disorder symptoms and 5 are general psychopathologies. To ensure that the elevated total EDI score was due to higher than normal eating disorder symptomatology, eligible study participants had to have at least one of the three eating disorder symptom subscales elevated. It has been recommended that the EDI and EAT be administered together as they assess different aspects of disordered eating. An EAT-34 score of ≥ 18 (90 percentile) effectively detected clinical eating disorders in a sample of 138 women between 16 and 25 years of age who had type 1 diabetes (Steel et al., 1989). Since our aim was to identify individuals with subclinical disordered eating, not to identify clinical eating disorders, a score of ≥ 17 on the EAT-34 was used.

In order to further ensure that individuals recruited for the study had subclinical disordered eating, individuals with potential clinical eating disorders were excluded. The current diagnostic criteria for bulimia nervosa include the occurrence of bingeing and inappropriate compensatory behaviours at least twice a week for 3 months (APA, 1994). Individuals who reported, on the EDI Symptom-Checklist, bingeing and the use of any compensatory behaviours twice a week for 3 months (on average) were considered to have potential bulimia nervosa. They were excluded from the study and referred to a psychiatrist for further assessment and treatment. Additionally, individuals with a body mass index (BMI) < 20 were excluded. One of the diagnostic criteria for anorexia nervosa is a body weight below that expected for age and height. Excluding those with a BMI < 20 would ensure that no study participants were below a healthy body weight.

Finally, eligible subjects had to be able to participate fully in all the psychoeducation sessions. Individuals who could not read, were not fluent in English or had to miss more than one session of the program were excluded from the study. Additionally, individuals who sought psychological treatment, including medications, during the course of the study were excluded. Pregnancy and major complications of diabetes, including blindness and organ transplantation, were also criteria for exclusion as they could involve issues beyond the scope of the program curriculum.

2. Procedures

All screening consent forms had a question as to whether the individual would like to be contacted regarding a related study. Individuals who consented to being contacted in addition to meeting the criteria for the intervention component of the study (ie. reported potential subclinical disordered eating on EDI) were telephoned and given information about the research project. Depending on the timing, they were invited to attend either an information meeting with other eligible subjects or an individual appointment to receive further details about the project and sign consent forms (Appendix C). Pre-defined dates were set for recruitment of the treatment group and the control group. As participants were recruited they were assigned to each group

based on when they were recruited; however both groups began participation at similar dates to ensure that time of year was not a confounding factor (see Figure 2). Recruiting each group sequentially allowed for the recruitment of enough participants to satisfy a minimum group size to run a psychoeducation program, while avoiding an extensive waiting period before beginning the program. To ensure effective group dynamics, a minimum of four subjects was required to run a psychoeducation program.

April 1997 - February 1998	Screening and treatment subject recruitment
March 1998 – May 1998	First psychoeducation program
March 1998 – May 1998	Screening and control subject recruitment
June 1998 – August 1998	Screening and treatment subject recruitment
August 1998 – October 1998	Second psychoeducation program
September 1998 – November 1998	Screening and control subject recruitment

Figure 2: Timeline for Subject Recruitment and Psychoeducation Program

The specific procedures for data collection were as follows. During the pre-intervention assessment visit participants received laboratory requisitions for blood tests and were asked to go to the lab within one week. They also had their height and weight assessed and completed the following questionnaires: SCI, EAT, EDI, EDI-Symptom Checklist, RSE, BDI, and BSI. The EDI, EAT, EDI – Symptom Checklist, height and weight were used to confirm eligibility for the study. Although the participants had previously completed the EDI, it was administered again during the initial visit to confirm eligibility criteria since the screening could have taken place up to 10 months earlier. The psychoeducation program took place in the Agriculture/Forestry Centre and started within 1 week of the treatment group's pre-intervention measurements. The programs were 6 weeks long and the post-intervention measurements for the treatment subjects were conducted within 1 week of program completion. Treatment subjects came into the university approximately 4 weeks and 6 months after the post-intervention assessment visit for the 1 month post- and 6 months post-intervention measurements, respectively. The control subjects came into the university

approximately 6 weeks and 10 weeks after the pre-intervention assessment visit in order to complete their post- and 1 month post-intervention measurements, respectively. The post-, 1 month post- and 6 months post-intervention assessment visits involved distributing blood requisitions and having participants complete the following questionnaires in the presence of the researcher: SCI, EAT, EDI, RSE, BDI, and BSI. Subjects were asked to go to the lab within the next week. During most visits, assessments were completed individually, without other subjects present. There were some instances when subjects completed the assessments in the presence of other subjects, but interaction was kept to a minimum. All assessment visits took place the Agriculture/Forestry Centre (University of Alberta, Edmonton, AB).

At the completion of data collection, all participants were sent a comprehensive summary of individual and group results (See Appendix I). Participants were encouraged to contact the researcher for clarification of results either by telephone or in person.

3. Instruments used to Confirm Subject Eligibility

a) EDI – Symptom Checklist

The EDI – Symptom Checklist was used to ensure that participants were not suffering from bulimia nervosa. The checklist is a structured, self-report, fill in the blank questionnaire that provides detailed information relevant to the diagnosis of eating disorders, such as information about frequency of bingeing and purging behaviours. The checklist was purchased from Psychological Assessment Resources Inc. (Odessa, Florida).

b) Anthropometrics

Height and weight were measured pre-intervention to determine if participants met the BMI eligibility criteria. Height was determined, without shoes, to the nearest 0.1 cm using either a standiometer or a set square and a flat wall. Participants turned their back to the standiometer or the wall to have their height assessed. In the case of the set square, a mark was made at the participant's height, they stepped away and then

their height was measured. Weight was measured to the nearest 0.1 kg using a medical balance beam scale (Healthometer, Continental Scale Corporation, Bridgeview, IL). Participants were weighed with light clothing. BMI was determined with the following equation: weight (kg) ÷ height $(m)^2$.

E. DEPENDENT MEASURES

Four major variables related to disordered eating were measured pre-, post- and 1 month post-intervention in the control and treatment groups, as well as 6 months post-intervention in the treatment group only. The subjects who completed the group psychoeducation program also completed program evaluation forms. Measurements included:

1. Metabolic Control

Metabolic control was assessed using fructosamine and HbA_{1c}. All blood collection was conducted at a public collection site by a trained technician (Dynacare-Kasper Medical Laboratories (DKML), Edmonton, AB). At every assessment visit, one 4.5 ml SST tube was collected for fructosamine analysis and one 5 ml EDTA tube was collected for HbA_{1c} analysis. After blood draw, the SST tubes were centrifuged, and the serum was aliquoted on site and placed in storage at -4° C. Serum samples were then picked up by the researcher and transported on ice in a biohazard container in accordance with biohazard transport regulations. They were stored at –20° C in the Nutrition and Metabolism Laboratories at the University of Alberta for fructosamine analysis. The EDTA tubes for HbA_{1c} analysis were sent via DKML courier to the DKML main laboratory (Edmonton, AB).

a) Fructosamine

At two time points during the study, August 1998 and April 1998, serum samples were send to Calgary Laboratory Services (Calgary, AB) for fructosamine analysis. The longest period of time that a blood sample was in storage before being

sent to Calgary was 6 months. The procedures for sending the samples to Calgary were as follows. The tubes were placed in ziplock bags with approximately 5 per bag. Bags were then placed, surrounded with dry ice, in a styrofoam box that was then put into a cardboard box. The box was labeled and shipped overnight to Calgary and analyzed the next morning.

Serum fructosamine was determined by a kinetic reduction test with nitroblue tetrazolium (NBT) (Boehringer Mannheim Diagnostics). The kit was designed to measure fructosamine from human serum or plasma (heparinized or EDTA). The kinetic reduction test involved combining fructosamine with NBT in an alkaline medium, causing the NBT to be reduced to formazan. A Boehringer Mannheim Diagnostics Hitachi 717 Chemistry Analyzer was used to determine fructosamine concentration in sample. Fructosamine results were not adjusted for serum protein concentrations. The reference range for fructosamine is 200-285 umol/L (Calgary Laboratories Services, Calgary, AB).

b) Hemoglobin A_{1c} (HbA_{1c})

GHb analysis was determined by the VARIANT Hemoglobin A_{1c} Program (Bio-Rad Laboratories). The program utilizes the principles of ion exchange high performance liquid chromatography (HPLC) for the automatic separation of HbA_{1c}. The program was designed to measure HbA_{1c} in human whole blood. The program has been certified by the National Glycohemoglobin Standardization Program, indicating that the program has documented traceability to the Diabetes Control and Complications Trial's (DCCT) reference method. This indicates that the test results using the VARIANT A_{1c} program are comparable to those reported in the DCCT. The non-diabetic reference range for HbA_{1c} for the laboratory was 4.3% to 6.1% (DKML, Edmonton, AB). The 1998 Clinical Practice Guidelines for the Management of diabetes in Canada state that individuals with diabetes should strive for HbA_{1c} values less than 7% (Meltzer et al., 1998).

2. Diabetes Treatment Adherence

a) Self-Care Inventory (SCI)

The SCI was administered to assess changes in perceived adherence to recommendations for diabetes care. The SCI was obtained from the author, A. LaGreca (University of Miami, USA), who granted permission to use the questionnaire. The SCI consists of 14 diabetes self-care tasks (ie. blood glucose testing, administering correct insulin doses, eating meals on time) that subjects rate on a 5 point scale indicating how well they feel they have followed the prescribed regimen during the past month (1 = never do it, 2 = sometimes follow recommendations; mostly not, 3 = follow recommendations about 50% of the time, 4 = usually do this as recommended; occasional lapses, 5 = always do this as recommended without fail, NA = cannot rate this item/not applicable). As recommended by the author (LaGreca, unpublished), the overall adherence score was calculated by taking an average of items 1,2,5,6,7,8, and 13. If subjects chose NA for any of the items, the item was omitted and the score was calculated by taking an average of the remaining items. The mean adherence score of 3.54 reported for a sample of 71 adults with type 1 diabetes was used as reference point for interpretation (LaGreca, unpublished). A higher overall adherence score indicates higher perceived adherence.

3. Eating Disorder Symptomatology

a) Eating Attitudes Test (EAT)

The EAT assesses changes in global disordered eating attitudes. A copy of the questionnaire and scoring procedures can be found in Garner and Garfinkel's (1979) article. The EAT is a 40 item self-report questionnaire, but 6 questions may demand particular answers from individuals with diabetes and are removed from the index prior to analysis; therefore it is renamed the EAT-34 (see Table 4). Each question is answered on a 6-point likert scale similar to that of the EDI (1 = never to 6 = always). Respondents rate whether each item applies "always", "very often", "often", "sometimes", "rarely", or "never". For example, "Like eating with other people".

The scoring procedures for the EAT were also similar to those of the EDI and were as follows. Responses for each item were weighted from 0 to 3. A score of 3 was assigned to the responses farthest in the "symptomatic" direction, while the adjacent responses were given a score of 2 and 1, respectively. A score of 0 was assigned to the three responses farthest in the "asymptomatic" direction. A cut-off score of ≥ 17 was used to identify individuals with subclinical disordered eating.

Table 4: Items Removed from the EAT for Use with Individuals with Type 1 Diabetes

9	Aware of the calorie content of foods that I eat
29	Avoid foods with sugar in them
30	Eat diet foods
32	Display self control around food
36	Feel uncomfortable after eating sweets
39	Enjoy trying new rich foods

b) Eating Disorder Inventory (EDI)

The sum of the three eating disorder symptom subscales was used to assess changes in eating disorder symptomatology throughout the study period. The three subscales were added together to minimize dependent variables. Additionally, factor analysis in samples of individuals without clinical eating disorders has not consistently demonstrated three distinct eating disorder symptom factors, but rather one eating disorder symptom factor (Welch et al., 1988). The sum of the 3 eating disorder symptom subscales provides detailed information related to eating disorder symptoms. The entire EDI was administered and scored as mentioned previously. The sum of the eating disorder symptom subscales was computed simply by adding the scores on all of the items of each of the 3 subscales.

c) Insulin Omission

Two questions were used to assess insulin omission thoughts and behaviours. The two questions were added to the end of the EAT and were as follows: "I have thoughts of reducing my insulin to control my weight" and "I have reduced my insulin to control my weight". In both cases, respondents answered the two questions on a 6

point likert scale ranging from "always" to "never". Possible scores on each question ranged from 0 to 4. An answer of "always" received a score of 4, "very often" a score of 3, "often" a score of 2, "sometimes" a score of 1, and "rarely" and "never" a score of 0. The scores on the two questions were added together to receive a total score for insulin reduction or omission.

4. General Psychopathology

a) Rosenberg Self-Esteem Scale (RSE)

The RSE was used to assess changes in global self-esteem. The RSE was developed by M. Rosenberg and can be found in the author's book (Rosenberg, 1965). Permission was not needed to use the RSE. The RSE consists of 10 items that subjects rate on a 4-point likert scale according to how much they agree with the statements (1= strongly agree, 2= agree, 3 = disagree and 4 = strongly disagree). For example: " On the whole, I am satisfied with my self". Although originally designed as a Guttman-type scale, the RSE is usually scored using a 4-point likert scale scoring format; the likert scale scoring format was used here and was as follows. The response indicating the highest self-esteem received a score of 4, and the adjacent responses received a score of 3, 2 and 1, respectively. Possible scores ranged from 10 and 40, with higher scores indicating higher self-esteem (Blascovich and Tomaka, 1991). Scoring of five of the items was reversed (items 1,2,4,6,and 7) to satisfy congruency with scoring format. A previously established cut-off was used to interpret results. A score on the RSE < 21 indicates low self-esteem (Bagely et al., 1997).

b) The Beck Depression Inventory (BDI)

The BDI was used assess changes in depression. The questionnaires were purchased from The Psychological Corporation (San Antonio, TX). Information on the administration, scoring, norms, reliability and validity were obtained from the BDI-II manual (Beck et al., 1996). The BDI is made up of 21 items, each item consisting of 4 statements that respondents pick from to indicate how they have been feeling during the past 2 weeks, including the day of testing (example: 0 = I do not feel sad, 1 = I feel sad

much of the time, 2 = I am sad all the time, 3 = I am so sad or unhappy that I can't stand it). Each item was scored from 0 to 3, with a score of 3 indicating the greatest degree of depression. The total BDI score was determined by adding the scores of each of the 21 items. Beck et al. (1996) recommends the following cut-off scores for interpretation of the BDI: 0 to 13 = minimal depression, 14-19 = mild depression, 20-28 = moderate depression and 29-63 = severe depression. These guidelines are based on the scores of 127 patients with major depressive disorder.

c) The Brief Symptom Inventory (BSI)

The BSI was used to assess changes in current emotional distress. The General Severity Index (GSI) of the BSI was used for assessment. Questionnaires were purchased from Multi-Health Systems Inc. (Toronto, ON). Information on the administration, scoring, norms, reliability and validity was obtained from the BSI Administration, Scoring and Procedures Manual (Derogatis, 1993). The BSI consists of 53 problems that subjects rate on a 5-point scale according to how much the particular problem has distressed or bothered them in the past 7 days. For example, "Faintness or dizziness". The scale ranged from "not at all" to "extremely" (0= not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit and 4 = extremely). The GSI was calculated by adding all 53 items together and dividing by 53. If any items were not completed, the sum was divided by the number of items that were actually endorsed. Higher scores indicate higher levels of distress. The norms used to interpret results are based on a sample of 480 healthy females over the age of 19, and 807 healthy females between the ages 13 to 19 (Derogatis, 1993). The norms are published as raw scores and the corresponding T scores. The authors suggest using a T score ≥ 63 on the GSI to detect cases of possible psychiatric disorders. The corresponding raw scores are ≥ 1.59 and ≥ 0.78 for adolescent and adult females, respectively.

5. Program Evaluation

All participants who took part in the group psychoeducation program completed a program evaluation form (see Appendix J). Completion of the evaluation forms was

anonymous and participants had the option of taking them home and returning the completed forms at the next visit. Subjects were asked if they would recommend this group to anyone else. They were also asked to rate 11 topics that were included in the program as "very helpful", "somewhat helpful" or "not helpful". Additionally, they were asked which aspects of the group program they found the most and least valuable. They were also asked for suggestions for future groups.

F. PSYCHOEDUCATION PROGRAM

The program was provided at two different time points (March 1998 and August 1998) by the same registered dietitian who was trained in the area of eating disorders and diabetes. The group psychoeducation program was based on a 7-week program, with 2 additional individual sessions, developed by Weiss et al. (1985) for women with bulimia nervosa. The program has been used in a number of studies to date. For example, Wolchik et al. (1986) demonstrated that the program was effective in improving eating disorder symptomatology, self-esteem, depression and body image in a group of women with bulimia nervosa. Kaminski and McNamara (1996) used a slightly modified version of the original program (8 group sessions with no individual sessions) to demonstrate similar significant improvements in a group of subclinical bulimic women. The original 7-week program was modified for use in the current research project. Modifications involved condensing the program from seven to six sessions and omitting the two individual sessions. A shorter version of the program was appropriate since the target population was only suffering from mild disordered eating, making some of the original material inappropriate. For example, the first two sessions originally contained information specific to individuals with bulimia nervosa. Detailed information on bingeing and purging was not appropriate since not all participants binged and very few purged. Additional modifications were made to the program to make it specifically targeted to individuals with type 1 diabetes. These modifications included discussions of how type 1 diabetes is related to disordered eating and the risk factors associated with having type 1 diabetes and disordered eating. Additional aspects of type 1 diabetes were discussed throughout the program when relevant. For example,

setting realistic goals for blood sugar control instead of having perfectionistic views about control, as well as how self-esteem may be linked to having type 1 diabetes.

The specific group program used here consisted of six, 1.5 hour sessions. Each session consisted of a 15 to 20 minute slide presentation on a certain topic, followed by discussion and questions pertaining to the topic. Session 5 of the program consisted of a 45 minute slide presentation on "Cultural Expectations of Thinness". Various homework activities, usually self-monitoring exercises, were given each week to be discussed during the following session. A manual developed by the researcher, based on the original program by Weiss et al. (1985), was provided at the beginning of the first session. It contained various activities, a summary of each session, and homework exercises. The lesson plan for the program was as follows:

Week 1

- what are subclinical eating disorders/ who is at risk for developing them
- complications associated with subclinical eating disorders
- risk factors for disordered eating development associated with type 1 diabetes

Week 2

- cues for eating (emotional/physical)
- healthy body weights / healthy eating habits
- emotional eating and alternative coping responses
- stress management

Week 3

- perfectionism / setting realistic goals
- nourishing ourselves without food
- self-esteem / improving self-esteem
- learning positive thought patterns

Week 4

- assertiveness
- saying "no"
- expressing anger

Week 5

- effects of the societal expectations of thinness on women
- the media's contribution to the pressures placed on women
- enhancing body image

Week 6

- conclusions and summing up

G. STATISTICAL ANALYSES OF RESULTS

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS, Version 7.5). All data were entered into SPSS files from a spreadsheet in Microsoft Excel. Prior to analysis, all entries were verified by comparing the recorded data to a printed copy of the data files entered in SPSS. All results are presented as mean ± standard deviation. All significant p-values ($p<0.05$) are indicated in bold print. Normality was analyzed using kurtosis and skewness statistics (Data not shown).

Sphericity was assessed before conducting each repeated measures analysis. The degree to which data meet the sphericity assumption is reflected in the parameter epilson (e), with a value of 1.0 indicating no violation (perfect sphericity) (Schutz and Gessaroli, 1987). As recommended by Schutz and Gessaroli (1987), reasonably exact tests were used to adjust reported p-values according to the extent to which e was less than 1.0. The Huynh-Feldt (H-F) adjusted p-values were reported if the e estimate was more than 0.75 and the Greenhouse-Geinsser (GG) adjusted p-value was used when e

estimate was less than 0.75. The Wilk's lambda statistic was used to analyze all MANOVA tests results.

1. Screening Component

The following variables were measured during screening: age, self-reported weight and height, highest level of education completed, duration of diabetes, value of last HbA_{1c} value, frequency of self-blood glucose monitoring, EDI and its subscales and insulin omission. Independent t-tests (two-tailed) were performed on all of these variables in order to compare the group of women identified as having subclinical disordered eating to those who were identified as normal. Additionally, independent t-tests (two-tailed) were performed to compare those women who agreed to take part in the intervention component of the study to those who did not.

2. Intervention Component

Three of the four major constructs, metabolic control, eating disorder symptomatology, and general psychopathology, were divided into sub-variables that could be measured, while diabetes treatment adherence was measured as only one variable. Metabolic control was measured as fructosamine and HbA_{1c}. Eating disorder symptomatology was measured as the EAT-34, eating disorder symptoms and insulin omission. General psychopathology was measured as self-esteem, depression and general emotional distress. Table 5 shows a graphical representation of the four constructs and their sub-variables.

Table 5 – Breakdown of Dependent Variables

Metabolic Control	**Diabetes Treatment Adherence**	**Eating Disorder Symptomatology**	**General Psychopathology**
Fructosamine	Diabetes treatment adherence	EAT-34	Self-esteem
HbA_{1c}		Eating disorder symptoms	Depression
		Insulin omission	General emotional distress

Metabolic control, eating disorder symptomatology and general psychopathology were analyzed by multivariate analysis of variance (MANOVA) repeated measures procedures. MANOVAs are appropriate for analyzing sets of measures that represent underlying constructs (Bray and Maxwell, 1982). Additionally, MANOVAs allow for the reduction of the total number of dependent variables (in this case n=9) to smaller sets (n= 4) ((Bray and Maxwell, 1982). Diabetes treatment adherence was the only measure used to assess adherence and was analyzed by analysis of variance (ANOVA) with repeated measures on the time factor.

Specific procedures for data analysis were as follows. Three separate 2 (Group) X 3 (Time) MANOVAs with repeated measures on the time factor were performed for metabolic control (fructosamine and HbA_{1c} treated as single constructs), eating disorder symptomatology (EAT 34, eating disorder symptoms and insulin omission treated as single constructs) and general psychopathology (self-esteem, depression and general emotional distress treated as single constructs). With alpha set to 0.05, the interaction effect and time effects were tested in order to determine whether the psychoeducation program had a differential effect on the treatment group and whether the groups changed over time. Significant MANOVA results were followed up with protected univariate statistics on the dependent variables. (Using the Bonferonni adjustment, the experiment-wise alpha of the overall MANOVA was divided by the number of dependent variables in the follow-up univariate analysis). This is the suggested post hoc procedure for significant MANOVAs (Bray and Maxwell, 1982; Schutz and Gessaroli, 1987). Contrasts helped to explain significant differences. These contrasts were set to compare the change between the two groups pre-intervention and post-intervention, and post-intervention and 1 month post-intervention, as well as the change in time from pre-intervention to post-intervention and post-intervention to 1 month post-intervention.

A 2 (Group) X 3 (Time) ANOVA with repeated measures on the time factor was performed on diabetes treatment adherence. Again the alpha was set to 0.05, and the interaction effect and time effects were analyzed. Contrasts helped to explain significant differences and were set to compare the change between the two groups pre-

intervention and post-intervention, and post-intervention and 1 month post-intervention, as well as the change in time from pre-intervention to post-intervention and post-intervention to 1 month post-intervention.

To investigate whether changes in the treatment group were maintained from 1 month post-intervention to 6 months post-intervention, three separate one way repeated measures MANOVAs (with time of assessment as the repeated measure) were performed for metabolic control, eating disorder symptomatology, and general psychopathology. With alpha set to 0.05, the time effect was tested in order to determine if there was a change over the time period between 1 month and 6 months post-intervention. Significant MANOVA results were followed up with protected univariate statistics on the dependent variables. (Using the Bonferonni adjustment, the experiment-wise alpha of the overall MANOVA was divided by the number of dependent variables in the follow-up univariate analysis). An additional one way repeated measures ANOVA (with time of assessment as the repeated measure) was performed on diabetes treatment adherence. Alpha was again set to .05 and the time effect was tested between 1 month and 6 months post-intervention.

CHAPTER FOUR
RESULTS

A. PARTICIPANT RECRUITMENT

Approximately 173 women with type 1 diabetes who met the screening entry criteria received direct information about the research study either by letter, through the University Hospital Metabolic Centre or by telephoning the researcher concerning posters or advertisements. Ninety-one individuals completed the screening questionnaire, representing a 53% response rate. Thirty-one subjects were identified with potential subclinical disordered eating. Due to difficulty recruiting, a subject with an EDI score of 39 was considered to have potential subclinical disordered eating because she also had elevated scores on all three eating disorder subscales of the EDI, as well as a score of 17 on the EAT. Of the 31 subjects identified with potential subclinical disordered eating, 18 agreed to take part in the study, 9 were not interested, and 4 lived too far away to commute to the university. Upon confirmation of the study inclusion criteria (ie. confirmed subclinical disordered eating), 14 of the 18 potential participants met the entry criteria and 4 did not (3 were classified as having normal eating behaviours and 1 had symptoms consistent with bulimia nervosa).

The 14 eligible participants were assigned to either the treatment group (n=8) or the control group (n=6). All of the control subjects completed the required measurements (pre-, post- and 1 month post-intervention measurements). All of the treatment subjects completed the psychoeducation program and the first three measurements (pre-, post- and 1 month post-intervention measurements); however, 3 treatment subjects did not complete the 6 months post-intervention measurements (2 subjects began taking anti-depressant medication after the 1 month post-intervention measurements, and 1 subject unexpectedly moved out of the country between the 1 month post- and 6 months post-intervention measurements). All 6 control subjects were interested in participating in the psychoeducation program upon completion of the study; however 2 did not take part in the program because of schedule conflicts.

Having too small a sample size to run multivariate analysis of variance (MANOVA) was a concern. However, for MANOVAs, it is recommended to have a minimum sample size of [p(k-1) + j], where p is the number of dependent variables per MANOVA (largest p=3), k is the number of repeated observations (largest k=3), and j is the number of groups (largest j=2). Therefore, the total sample size had to be at least 8 ([3(3-1) + 2] = 8) (Schutz and Gessaroli, 1987). The total sample size was 14, thereby exceeding the minimum sample size.

B. SCREENING COMPONENT

Of the 91 subjects who completed screening questionnaires, 35% had EDI scores indicative of potential subclinical disordered eating (total EDI score >40) and 15% met the study entry criteria of confirmed subclinical disordered eating and took part in the study. Table 6 and 7 represent summaries of self-reported demographic, anthropometric, general diabetes-related information and disordered eating information of the women who were screened. Comparisons are made between the study participants (ie. confirmed subclinical disordered eating) and the individuals who were identified with normal eating attitudes and behaviours. Data are not presented on 14 subjects, 13 of whom had elevated scores on the EDI but did not wish to take part in the project further so their disordered eating status was not confirmed, and 1 subject who had bulimia nervosa. Data are also not presented on self-reported HbA_{1c} because only 41% of the screening subjects reported a HbA_{1c} value.

Table 6: General Characteristics of Women Screened

Variable	Subclinical Disordered Eating (n=14)	Normal Eating Attitudes and Behaviours (n=63)	P Value
Age	31.9 ± 9.4 [a]	28.4 ± 10.6	0.264
BMI (kg/m^2) [b]	27.3 ± 4.6	24.7 ± 3.9 [c]	0.066
Years of Education	14.6 ± 2.9	13.8 ± 2.5 [d]	0.288
Frequency of Daily Blood Glucose Monitoring	4.25 ±3.9	3.1 ± 1.4 [e]	0.286
Age of Onset of Diabetes (Years of Age)	16.3 ± 9.3	12.4 ± 8.2	0.126
Years of Diabetes	15.6 ± 9.6	16.0± 10.7	0.902

[a] Results presented as mean ± standard deviation
[b] BMI based on self-reported height and weight (actual BMI of subclinical disordered eating group was 28.6 ± 3.1 kg/m^2)
[c] Missing 5 cases (n=58 in normal eating behaviours group)
[d] Missing 2 cases (n=61 in normal eating behaviours group)
[e] Missing 4 cases (n=59 in normal eating behaviours group)

Table 7: Eating Disorder Symptomatology of Women Screened

Variable	Normal Result [a]	Subclinical Disordered Eating (n=14)	Normal Eating Attitudes and Behaviours (n=63)	P Value
Total EDI	< 40	71.6 ± 23.5 [b]	22.6 ± 12.0	**<0.001**
Body Dissatisfaction Subscale	≤ 12	20.6 ± 6.1	8.5 ± 6.3	**<0.001**
Drive for Thinness Subscale	≤ 6	7.2 ± 2.5	1.5 ± 2.4	**<0.001**
Bulimia Subscale	≤ 1	6.1 ± 6.0	0.95 ± 1.6	**0.007**
Interpersonal Distrust Subscale	≤ 2	6.3 ± 6.2	1.8 ± 3.0	**0.020**
Interoceptive Unawareness Subscale	≤ 3	5.5 ± 6.2	1.2 ± 1.9	**0.022**
Maturity Fears Subscale	≤ 3	5.6 ± 5.3	2.2 ± 2.0	**0.034**
Perfectionism Subscale	≤ 6	9.7 ± 5.1	4.3 ± 4.1	**0.002**
Ineffectiveness	≤ 2	10.5 ± 7.9	2.1 ± 3.3	**0.002**
Insulin Omission [c]	N/A	2.8 ± 2.5	0.33 ± 0.99 [d]	**0.003**

[a] For EDI subscales, ≤ mean of a sample of 770 healthy college female students, is considered normal
[b] Results presented as mean ± standard deviation
[c] Possible scores for insulin omission range from 0 to 8, with 8 indicating the most pathological score
[d] 1 case missing (n=62 for normal eating behaviours group)
N/A = Not available

There were no significant differences between the women with subclinical disordered eating and those with normal eating attitudes and behaviours with respect to any of the following variables: age, years of education, frequency of daily blood glucose monitoring, age of onset of diabetes and years of diabetes. There was a trend for the women with subclinical disordered eating to have a higher self-reported BMI than those with normal eating attitudes and behaviours, although this did not reach statistical significance (p=0.066). The actual BMI of the study participants was 28.6 ± 3.1 kg/m^2.

The group identified as having subclinical disordered eating had significantly higher scores on the EDI and all of its subscales than the group identified as having normal eating attitudes and behaviours. Similarly, the women with subclinical

disordered eating had significantly greater scores on insulin omission than the women identified with normal eating attitudes and behaviours.

The mean EDI subscale scores of the subclinical disordered eating group were all above the mean of the normative sample. Conversely, the mean EDI subscale scores of the normal eating behaviour group were all below the mean of the normative sample, with the exception of the Ineffectiveness subscale score which was only marginally above the mean of the normative sample.

C. INTERVENTION COMPONENT

1. Preliminary Analyses

Thirteen subjects were identified with potential subclinical disordered eating upon screening, but did not wish to participate in the project. A preliminary analysis was conducted to compare the subjects who participated in the study (n=14) and those who did not (n=13) (data not shown). The two groups were compared with respect to self-reported demographic, anthropometric, general diabetes-related information (age of onset of diabetes, duration of diabetes and frequency of blood glucose testing) and disordered eating information (total EDI, the 8 EDI subscales and insulin omission). There were no significant differences between the two groups on any of the variables. There was a trend for the women who took part in the study to have more years of education (p=0.075) and to score higher on the perfectionism subscale of the EDI (p=0.077), but these differences did not reach significance.

An additional preliminary analysis was conducted to identify any pre-intervention differences between the treatment group and the control group (data not shown). There were no significant differences between the treatment group and the control group with respect to demographic, actual BMI, general diabetes-related information, fructosamine, HbA_{1c}, diabetes treatment adherence, eating disorder symptoms, EAT-34 score, insulin omission or depression. The control group had significantly greater general emotional distress at baseline than the treatment group (p=0.009). There was a trend for the control group to have significantly greater EAT-34 (p=0.075) and depression scores (p=0.075) at baseline, but these differences did not reach significance.

a) Main Results

The main results include comparisons between the treatment group and the control group over the three assessment points, pre-, post- and 1 month post-intervention.

a) Metabolic Control

Table 8 depicts the means and standard deviations for the metabolic control sub-variables (fructosamine and HbA_{1c}) for the treatment group and the control group at each of the three assessment points, pre-, post- and 1 month post-intervention. Table 9 summarizes the repeated measures MANOVA results for metabolic control.

Table 8: Mean Scores for Metabolic Control Sub-Variables

		Treatment Group (n=8)			Control Group (n=6)		
Variable	**Normal Result**	**Pre-**	**Post-**	**1 Month Post-**	**Pre-**	**Post-**	**1 Month Post-**
Fructosamine (umol/L)	200 - 285	269.1± 63.7[a]	310.5 ± 39.8	319.3 ± 64.0	281.8 ± 35.9	338.2 ± 36.7	309.5 ± 87.1
HbA_{1c} (%)	≤ 7.0	8.2 ± 1.2	8.3 ± 1.1	8.2 ± 1.2	8.1 ± 0.8	8.3 ± 0.9	8.3 ± 0.6

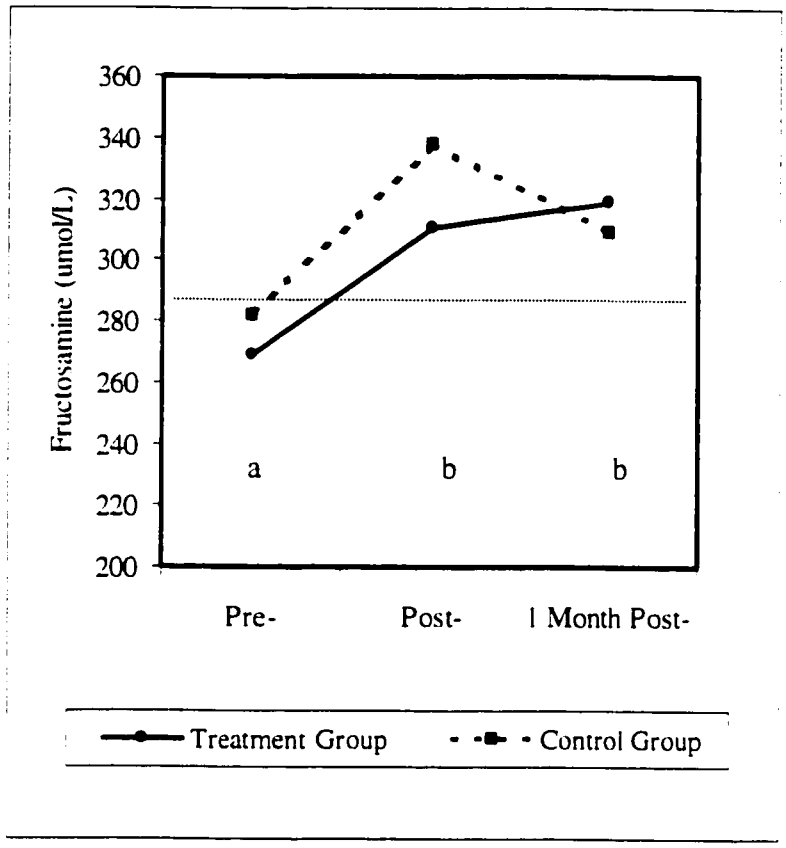
[a] Results presented as mean ± standard deviation

Table 9: Repeated Measures MANOVA Summary Results for Metabolic Control

	P Value
Time Effect	0.037
Time X Group Effect	0.855

The overall MANOVA results for metabolic control revealed a significant time effect (p=0.037) but no significant interaction effect (p=0.855). The follow-up univariate F tests helped to explain the changes over time by revealing that there was a significant time effect for fructosamine (p=0.021) but not for HbA_{1c} (p=0.282). The contrasts further explained the time effect by showing that the fructosamine values of the groups as a whole significantly increased between the pre-intervention time point and the post-intervention time point (p=0.008), but did not significantly change between the post-intervention time point and the 1 month post-intervention time point (p=0.523) (Figure 3).

Figure 3: Mean Fructosamine Values at Pre-Intervention (Pre-), Post-Intervention (Post-) and 1 Month Post-Intervention (1 Month Post-)



a,b: time points with dissimilar letters differ significantly (p=0.008)
Standard deviations can be found in Table 8
Note: Points on or below the line indicate normal fructosamine levels (200–285 umol/L)

b) Diabetes Treatment Adherence

Table 10 depicts the means and standard deviations for diabetes treatment adherence for the treatment group and the control group at each of the three assessment points, pre-, post- and 1 month post-intervention. Table 11 summarizes the repeated measures ANOVA results for diabetes treatment adherence.

Table 10: Mean Diabetes Treatment Adherence Scores

		Treatment Group (n=8)			Control Group (n=6)		
Variable	**Normal Result**	**Pre-**	**Post-**	**1 Month Post-**	**Pre-**	**Post-**	**1 Month Post-**
Diabetes Treatment Adherence	≥ 3.54	3.14 ± 0.99 [a]	3.07 ± 0.78	3.20 ± 0.90	3.14 ± 0.85	3.04 ± 0.84	2.91 ± 0.87

[a] Results presented as mean ± standard deviation

Table 11: Repeated Measures ANOVA Summary Results for Diabetes Treatment Adherence

	P Value
Time Effect	0.710
Time X Group Effect	0.424

The ANOVA results for diabetes treatment adherence demonstrated no significant time effect (p=0.710) or interaction effect (p=0.424).

c) Eating Disorder Symptomatology

Table 12 shows the means and standard deviations for the eating disorder symptomatology sub-variables (EAT-34 score, eating disorder symptoms and insulin omission) for the treatment group and the control group at each of the three assessment points, pre-, post- and 1 month post-intervention. Table 13 summarizes the repeated measures MANOVA results for eating disorder symptomatology.

Table 12: Mean Scores for Eating Disorder Symptomatology

		Treatment Group (n=8)			Control Group (n=6)		
Variable	**Normal Result**	**Pre-**	**Post-**	**1 Month Post-**	**Pre-**	**Post-**	**1 Month Post-**
EAT-34	< 17	22.5 ± 5.3	19.9 ± 7.8	20.6 ± 10.3	30.8 ± 10.6	33.0 ± 8.6	33.8 ± 9.6
Eating Disorder Symptoms	N/A	35.4 ± 7.8	29.9 ± 6.1	32.1 ± 6.2	32.0 ± 11.9	28.0 ± 11.2	33.2 ± 9.2
Insulin Omission [b]	N/A	2.0 ± 2.1	1.8 ± 2.1	1.8 ± 1.8	2.3 ± 2.1	2.3 ± 2.2	2.8 ± 1.7

[a] Results presented as mean ± standard deviation
[b] Possible scores for insulin omission range from 0 to 8, with 8 indicating the most pathological score
N/A = not available

Table 13: Repeated Measures MANOVA Summary Results for Eating Disorder Symptomatology

	P Value
Time Effect	0.325
Time X Group Effect	0.762

The overall MANOVA for eating disorder symptomatology was not significant, with neither the time effect (p=0.324) nor the interaction effect (p=0.762) approaching significance.

d) General Psychopathology

Table 14 depicts the means and standard deviations for the general psychopathology sub-variables (self-esteem, depression and general emotional distress) for the treatment group and the control group at each of the three assessment points, pre-, post- and 1 month post-intervention. Table 15 summarizes the repeated measures MANOVA results for general psychopathology.

Table 14: Mean Scores for General Psychopathology Sub-Variables

		Treatment Group (n=8)			Control Group (n=6)		
Variable	**Normal Result**	**Pre-**	**Post-**	**1 Month Post-**	**Pre-**	**Post-**	**1 Month Post-**
Self-Esteem	> 21	26.1 ± 4.5 [a]	26.9 ± 4.9	26.6 ± 4.7	23.0 ± 4.4	21.2 ± 4.9	20.8 ± 6.1
Depression	≤ 13	18.3 ± 10.8	11.0 ± 7.6	11.0 ± 11.2	29.5 ± 10.5	23.3 ± 12.8	26.7 ± 12.3
General Emotional Distress	≤ 0.78	1.00 ± 0.59	0.66 ± 0.45	0.70 ± 0.54	1.89 ± 0.45	1.37 ± 0.44	1.50 ± 0.65

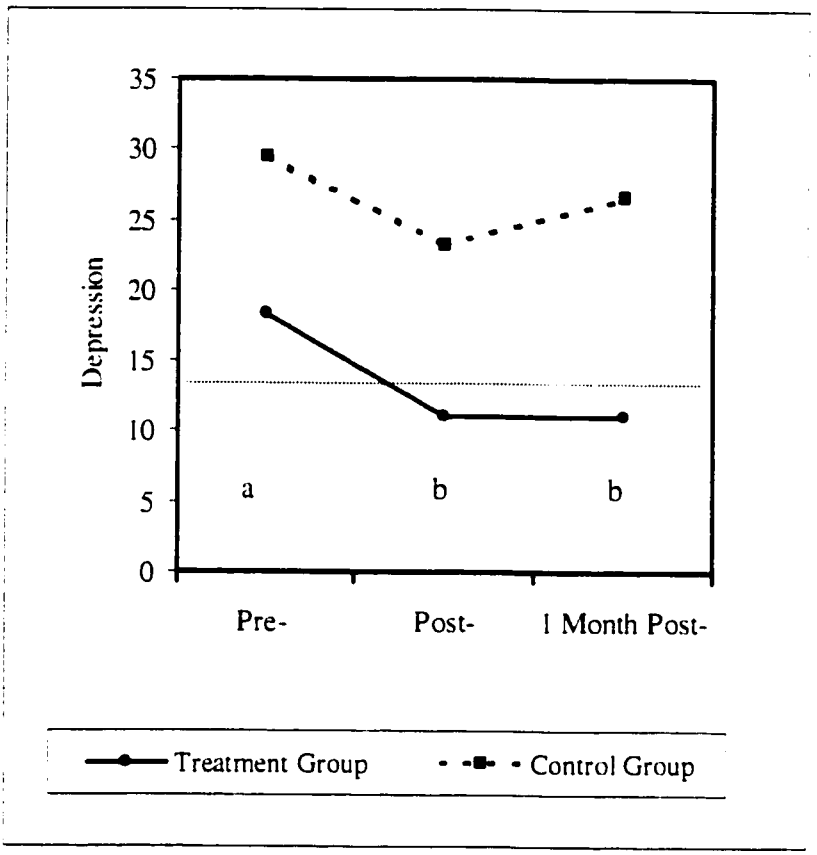
[a] Results presented as mean ± standard deviation

Table 15: Repeated Measures MANOVA Summary Results for General Psychopathology

	P Value
Time Effect	0.006
Time X Group Effect	0.590

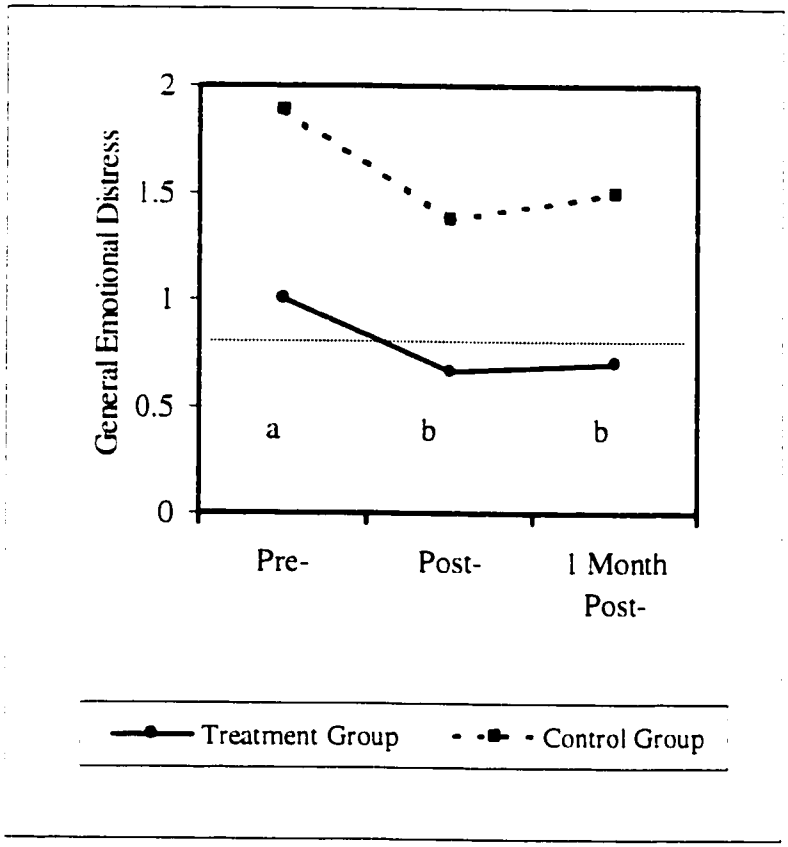
The MANOVA results for general psychopathology revealed that there was a significant time effect (p=0.006) but no significant interaction effect (p=0.590). The univariate follow-up F test revealed a significant time effect for depression (p=0.006) and general emotional distress (p<0.001), but not for self-esteem (p=0.676). The contrasts helped to explain these changes over time by revealing that the groups experienced a significant improvement in depression (p=0.003) and general emotional distress (p=0.001) between the pre-intervention time point and the post-intervention time point, but no significant changes between the post-intervention time point and the 1 month post-intervention time point with respect to depression (p=0.360) and general emotional distress (p=0.260) (Figure 4 and 5).

Figure 4: Mean Depression Scores at Pre-Intervention (Pre-), Post-Intervention (Post-) and 1 Month Post-Intervention (1 Month Post-)



a,b: time points with dissimilar letters differ significantly (p=0.003)
Standard deviations can be found in Table 14
Note: Points on or below the line indicate minimal depression, while points above the line indicate the presence of depression.

Figure 5: Mean General Emotional Distress at Pre-Intervention (Pre-), Post-Intervention (Post-) and 1 Month Post-Intervention (1 Month Post-)



a.b: time points with dissimilar letters differ significantly (p=0.001)
Standard deviations can be found in Table 14
Note: Points on or below the line indicate no general emotional distress, while points above the line indicate distress.

3. Follow-up Analysis

Only 5 of the original 8 treatment subjects completed the entire study period up to the 6 months post-intervention time point. An analysis was conducted to compare the subjects who completed all of the study requirements (n=11) and those who did not (n=3). There were no significant differences between the two groups with respect to the baseline measures (data not shown). Nevertheless, the previously presented 2 (Group) X 3 (Time) repeated measures analyses on all major variables were recalculated with only the participants who completed the entire study requirements (n=5 instead of n=8 for the treatment group). The same results were found for all variables except metabolic control (data not shown). A significant time effect was demonstrated for depression and general emotional distress between the pre-intervention and post-intervention time points, but not between the post- and 1 month post-intervention time points. No significant time effect was demonstrated for metabolic control.

The follow-up analyses on each variable were conducted using the 5 treatment subjects who completed the final 6 months post-intervention measurements. Paired comparisons are made within the treatment group between the 6 months post-intervention measurements and the 1 month post-intervention measurements.

a) Metabolic Control

Table 16 depicts the means and standard deviations for the metabolic control sub-variables (fructosamine and HbA_{1c}) at the 1 month and 6 months post-intervention time points.

Table 16: Mean Follow-up Scores for Metabolic Control Sub-Variables in the Treatment Group (n=5)

Variable	Normal Result	1 Month Post-Intervention	6 Months Post-Intervention
Fructosamine (umol/L)	200 -285	335.8 ± 72.4 [a]	359.4 ± 40.8
HbA_{1c} (%)	≤ 7.0	8.4 ± 1.2	8.5 ± 1.8

[a] results presented as mean ± standard deviation

The one way repeated measures MANOVA for metabolic control revealed no significant time effect (p=0.789) between the 1 month and 6 months post-intervention time points.

b) Diabetes Treatment Adherence

Table 17 depicts the means and standard deviations for diabetes treatment adherence at the 1 month and 6 months post-intervention time points.

Table 17: Mean Follow-up Diabetes Treatment Adherence Scores in the Treatment Group (n=5)

Variable	Normal Result	1 Month Post-Intervention	6 Months Post-Intervention
Diabetes Treatment Adherence	≥ 3.54	2.88 ± 0 .67 [a]	2.71 ± 0.84

[a] results presented as mean ± standard deviation

The results of the one way repeated measures analysis for diabetes treatment adherence revealed no significant time effect (p=0.288) between the 1 month and 6 months post-intervention time points.

c) Eating Disorder Symptomatology

Table 18 depicts the means and standard deviations for the eating disorder symptomatology sub-variables (EAT-34 score, eating disorder symptoms and insulin omission) at the 1 month and 6 months post-intervention time points.

Table 18: Mean Follow-up Scores for Eating Disorder Symptomatology Sub-Variables in the Treatment Group (n=5)

Variable	Normal Result	1 Month Post-Intervention	6 Months Post-Intervention
EAT-34	< 17	18.6 ± 7.9 [a]	21.6 ± 9.0
Eating Disorder Symptoms	N/A	33.6 ± 1.5	31.4 ± 3.4
Insulin Omission [b]	N/A	1.4 ± 1.3	0.6 ± 0.9

[a] results presented as mean ± standard deviation
[b] Possible scores for insulin omission range from 0 to 8, with 8 indicating the most pathological score
N/A = not available

The one way repeated measures MANOVA for eating disorder symptomatology revealed no significant time effect (p=0.365) between the 1 month and 6 months post-intervention time points.

d) General Psychopathology

Table 19 depicts the means and standard deviations for the general psychopathology sub-variables (self-esteem, depression and general emotional distress) at the 1 month and 6 months post-intervention time points.

Table 19: Mean Scores for General Psychopathology Sub-Variables in the Treatment Group (n=5)

Variable	Normal Result	1 Month Post-Intervention	6 Months Post-Intervention
Self-Esteem	> 21	27.6 ± 4.7 [a]	28.4 ± 6.2
Depression	≤ 13	11.8 ± 12.1	15.8 ± 13.9
General Emotional Distress	≤ 0.78	0.78 ± 0.61	0.85 ± 0.73

[a] results presented as mean ± standard deviation

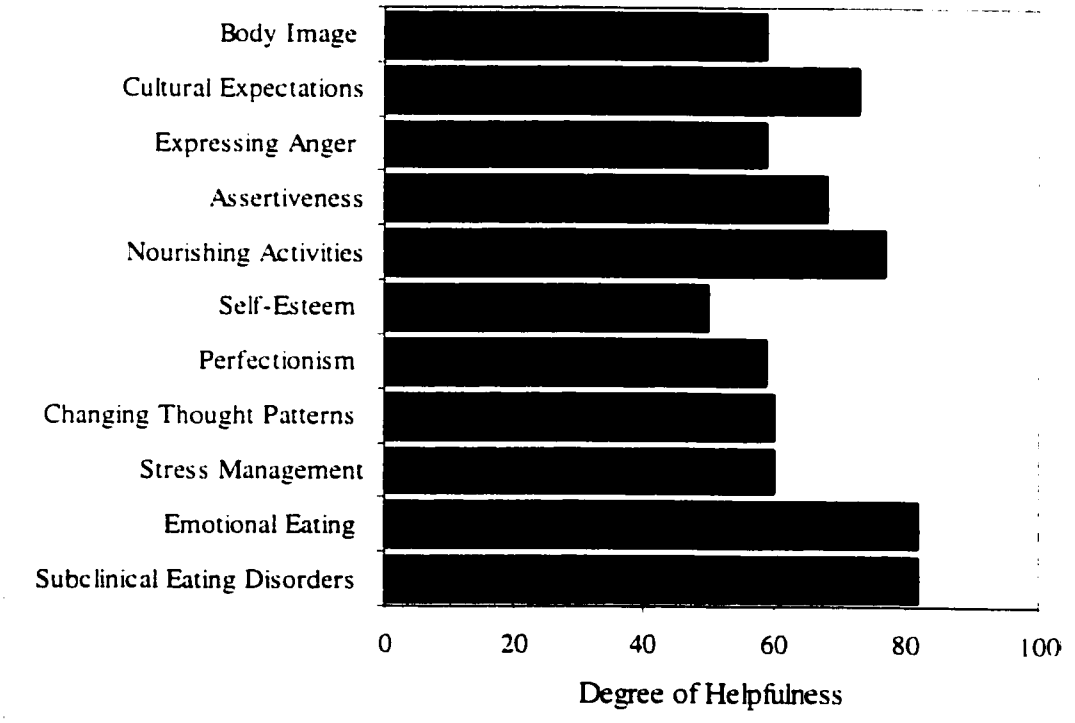
The results of the one way repeated measures MANOVA for general psychopathology demonstrated no significant time effect (p=0.411) between the 1 month and 6 months post-intervention time points.

Note: Wilfley et al. (1993) suggest that the individuals who drop out of study follow-up assessments should be assigned follow-up scores equal to those at pre-test. This protects results from the bias that could be introduced by the tendency of non-improving individuals to drop out of study assessments. This was done for the 3 treatment subjects who did not complete the 6 months follow-up assessments. The results presented above remained unchanged when the replacement values were used for the 3 subjects who did not complete 6 months follow-up assessments.

D. PROGRAM EVALUATION

Eleven women completed the group psychoeducation program, 8 treatment subjects and 3 control subjects. The program had excellent attendance, with only 2 subjects missing 1 session each. When asked the question "Would you recommend this group to anyone else?", 9 answered "yes" and 2 did not answer the question. Participants were also asked to rate each of the 11 topics covered in the program in terms of how helpful they were, with a score of 2 indicating very helpful, 1 indicating somewhat helpful and 0 indicating not helpful. Figure 6 represents the average score for each of the topics covered in the program.

Figure 6: Program Evaluation: Average Score on Each Topic Included in the Program

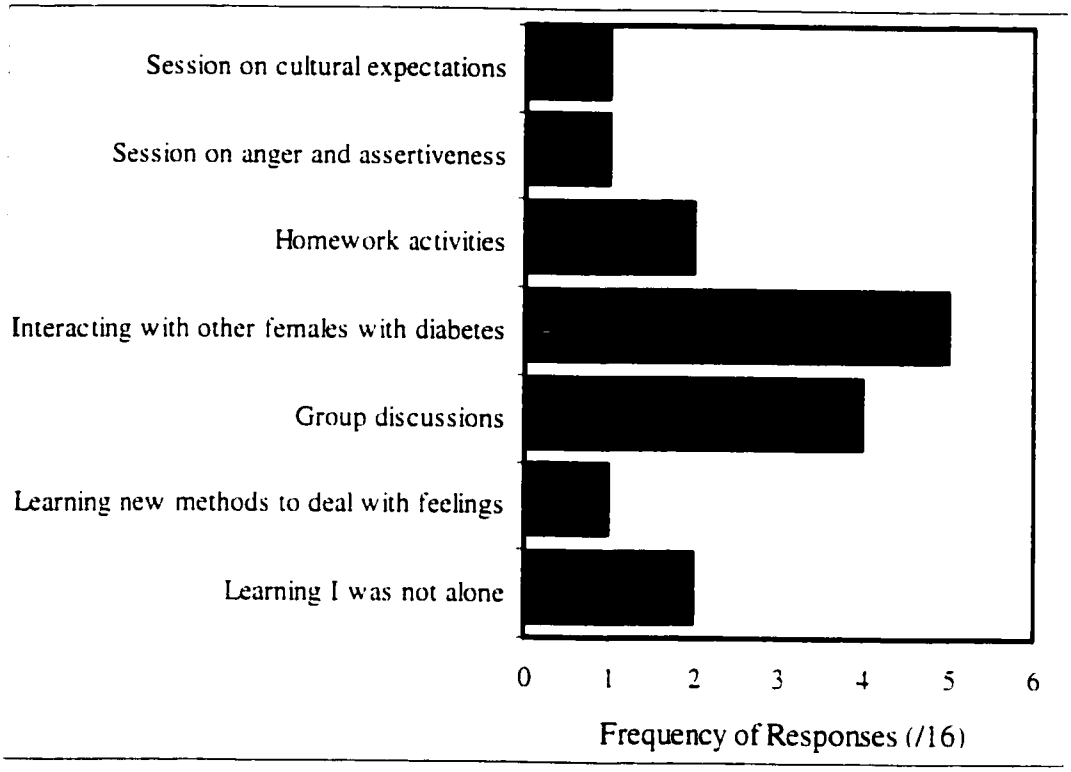


0 = not helpful, 1 = somewhat helpful, 2 = very helpful

All of the topics included in the psychoeducation program received a score of 1 or greater. The information on nourishing activities, emotional eating and subclinical eating disorders obtained the highest scores in terms of how helpful they were, while the information of self-esteem received the lowest score of the 11 topics.

Figure 7 summarizes the responses to the question "What aspects of the group did you find the MOST valuable?". There were 16 responses to this question on the 11 evaluation forms.

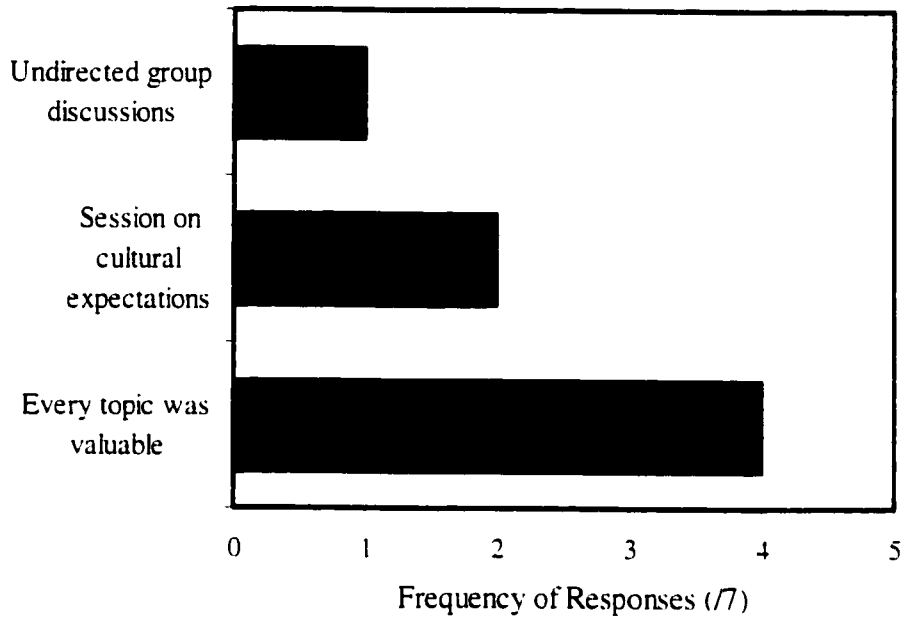
Figure 7: Program Evaluation: Perceived MOST Valuable Aspects of Group Program



Having the opportunity to interact with other females with diabetes was the most frequently reported positive aspect of the program (n=5), with group discussions being the second most frequently reported response (n=4).

Figure 8 shows the responses to the question "What aspects of the group did you find the LEAST valuable?". There were 7 responses to this question on the 11 evaluation forms.

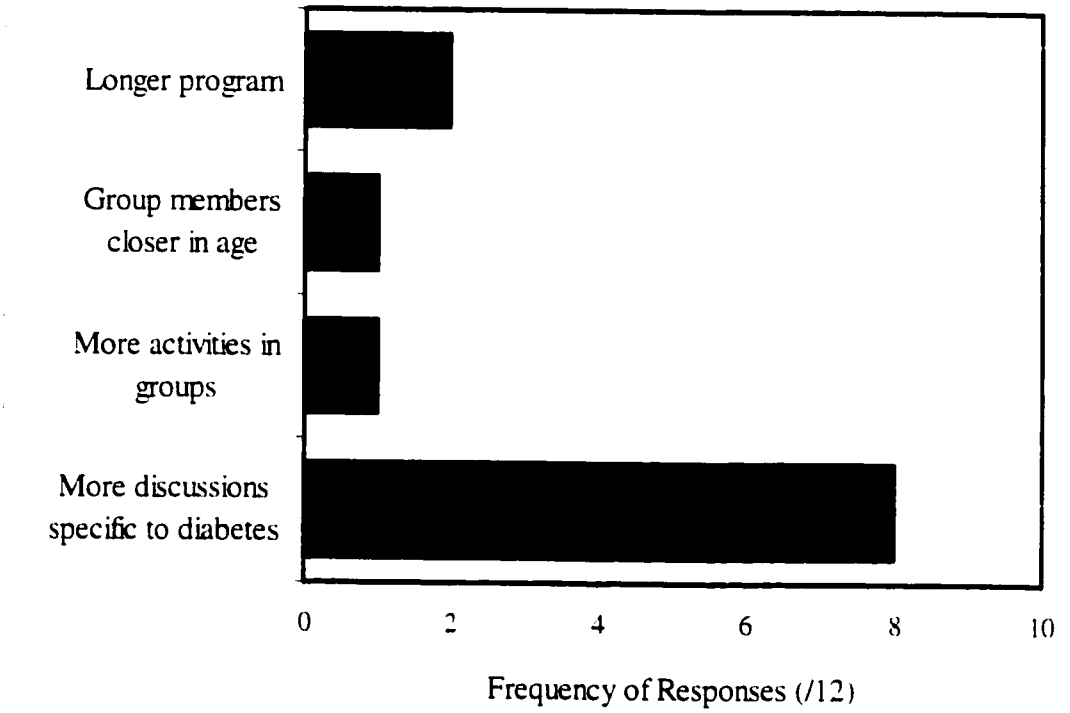
Figure 8: Program Evaluation: Perceived LEAST Valuable Aspects of Group Program



When asked what the least valuable aspect of the group program was, the most frequently reported response was that all aspects of the program were valuable (n=4). Two subjects found the session on cultural expectations to be the least valuable aspect of the program.

Figure 9 shows the combined responses to the questions "What topic would you like to see added to the group?" and "What suggestions do you have for future groups?". There were a total of 12 responses to these questions.

Figure 9: Program Evaluation: Suggestions for Future Groups



The most frequently reported suggestion for future groups was to have more discussions on topics specific to diabetes (n=8). Two participants suggested that program could have been longer than 6 weeks.

E. SUMMARY OF RESULTS WITH RESPECT TO THE STUDY HYPOTHESES

Hypotheses: A group psychoeducation program will result in the following effects in individuals with type 1 diabetes and subclinical disordered eating:

a) increased metabolic control

b) increased diabetes treatment adherence

c) decreased eating disorder symptomatology

d) decreased general psychopathology

The hypotheses were rejected, as there were no significant differences over time between the treatment group and the control group on any of the variables.

CHAPTER FIVE
DISCUSSION

A. MAJOR FINDINGS

The major finding of this research project was that there were no differences between the treatment group and the wait-list control group over time with respect to metabolic control, diabetes treatment adherence, eating disorder symptomatology or general psychopathology. This indicates that the 6-week group psychoeducation program was no more effective than the wait-list control group in treating women with type 1 diabetes and subclinical disordered eating. The second major finding was that both the treatment group and the wait-list control group experienced significant improvements in depression and general emotional distress between the pre-intervention time point and the post-intervention time point. These results suggest that both the psychoeducation program and the wait-list control group may have been somewhat therapeutic as slight improvements were demonstrated in both groups.

B. PARTICIPANT RECRUITMENT

A total of 173 women with type 1 diabetes, who were between the ages of 16 and 50 years, obtained information about the study by mail, posters, advertisements or in-person during clinic visits. Ninety-one women completed the screening questionnaire, representing a 53% response rate. This response rate is comparable to other screening studies that used similar recruitment strategies to those used in the present study. Stancin et al. (1989) had a 65% response rate when women with type 1 diabetes were contacted by letter or in-person during clinic visits to complete a screening questionnaire. Response rates are substantially lower when recruitment is conducted by mail instead of in-person. Krenz et al. (1993) had a response rate of 49% when men and women with diabetes were recruited by mail out screening questionnaires. Rodin et al. (1991) and Fairburn et al. (1991) found response rates of

85% and 90%, respectively, when recruiting participants solely through direct contact during clinic visits.

The number of participants in the actual study (n=14) was smaller than what was originally calculated as necessary to document a difference in total EDI score (n=18). Total recruitment may have been more successful had all potential participants been contacted in-person during clinic visits, but this was not feasible within the realms of the project. Additionally, recruiting subjects from diabetes clinics as well as from the community allowed for a sample that was more representative of the actual population of women who have subclinical disordered eating and type 1 diabetes.

C. SCREENING COMPONENT

The purpose of screening was not to document the prevalence of disordered eating, but to find individuals eligible for the study. However, since the screening sample was randomly chosen, it is valid to compare the prevalence of disordered eating in our sample to previously reported estimates of disordered eating among women with type 1 diabetes. Thirty-five percent of the women screened for disordered eating had EDI scores above the cut-off used to identify potential subclinical disordered eating. In a similar screening project, Olmsted et al. (1998) screened a sample of 212 girls with type 1 diabetes between the ages of 12 and 19 and found 59% to have mild disordered eating attitudes and behaviours. The age range in our study was 16 to 50 years of age and could explain why the prevalence of potential disordered eating was considerably lower than 59%.

In order to confirm that the women identified with potential subclinical disordered eating could actually be truly classified as such, they had to meet additional criteria with respect to eating disturbances. Of the total sample screened, 15% (n=14) demonstrated actual subclinical disordered eating. However, 14% (n=13) of the women screened chose not to have their disordered eating confirmed and as a result, the proportion of women who had actual subclinical disordered eating may have been substantially higher than what was found.

Although there has been a large variation in the reported prevalence of disordered eating in women with type 1 diabetes, the prevalence of subclinical disordered eating demonstrated in the present study is comparable to that demonstrated in similar screening studies, suggesting that screening with the EDI and EAT is effective. Affenito et al. (1997a) reported subclinical eating disorders (assessed by interview) in 14% of a sample of women between the ages of 18 and 46 years. Cantwell and Steel (1996) found 20% of women between the ages of 17 and 30 years to have either subclinical or clinical eating disorders (assessed by questionnaire and semi-structured interview). When Rydall et al. (1997) investigated the prevalence of disordered eating in girls between the ages of 12 and 18 years, 19% had moderately disordered eating (assessed by interview). Pollock et al. (1995) reported that the prevalence of "eating problems" (as assessed by interview) was 11.4% in a sample of girls with a mean age of 21 years.

The women identified as having subclinical disordered eating scored significantly higher on the EDI and all of its subscales than the women with normal eating attitudes and behaviours. There were no differences between the two groups with respect to age, years of education, frequency of daily blood glucose monitoring, age of onset of diabetes or years of diabetes. This suggests that none of these factors are associated with disordered eating. This is consistent with previous findings by Affenito et al. (1997a) who screened women between the ages of 18 and 46 years, but inconsistent with findings by Maharaj et al. (1998) who screened girls between the ages of 11 and 19 years. Maharaj et al. (1998) found that girls with highly disturbed eating behaviours had an older age on onset of diabetes than girls with mildly disturbed eating behaviours, who had an older age of onset of diabetes than girls with normal eating behaviours. The inconsistency in the findings is likely due to the different age groups that were studied. Older age of onset of diabetes may be a risk factor for eating disorder development in adolescent girls, but not in adult women who have had type 1 diabetes for many years.

There was a trend for the group with subclinical disordered eating to have a higher self-reported BMI than the group of women with normal eating behaviours,

although this difference was not statistically significant. Research has shown that obesity is a risk factor for the development of disordered eating because it increases the risk of dieting (Fairburn et al., 1997). It is therefore not surprising that the women with subclinical disordered eating reported that they were slightly heavier than did the women with normal eating behaviours did. Previous research on measured BMI has not demonstrated a significant difference in BMI between women with type 1 diabetes and disordered eating and those with normal eating behaviours (Affenito et al., 1998; Wing et al., 1986).

Women with subclinical disordered eating scored higher on insulin omission than women with normal eating attitudes and behaviours. This indicates that women with subclinical disordered eating think about and/or actually reduce their insulin to control their weight more frequently than do women with normal eating behaviours. This is consistent with previous findings that insulin omission is associated with disordered eating attitudes and behaviours (Polonsky et al., 1994; Biggs et al., 1994). It is also consistent with previous research that demonstrated that individuals with clinical eating disorders demonstrate more insulin omission than those without an eating disorder (Rodin et al., 1991; Affenito et al., 1997a). Contrary to our findings however, Affenito et al (1997a) found no difference in the occurrence of insulin omission between women with subclinical disordered eating and those with normal eating behaviours (Affenito et al., 1997a). However, the same research group found that individuals with subclinical disordered eating reported more past insulin misuse (as opposed to present misuse) than healthy controls (Affenito et al., 1997a). More research is necessary to document the occurrence of insulin misuse in individuals with subclinical disordered eating.

D. INTERVENTION COMPONENT

There were minimal differences between the treatment group and the control group with respect to baseline variables. The only variable that was different between the two groups at baseline was general emotional distress, with the control group being

significantly higher than the treatment group. However, both groups were above the cut-off that is used to indicate the presence of current emotional distress. The assignment of participants to the treatment group and the control group was successful at minimizing group differences, although it did not involve complete randomization. Differences between groups at baseline were managed by the statistical analyses that involved comparing changes within the groups over time, as opposed to differences between groups over time.

When the treatment group and the wait-list control group were compared over the three time points, pre-, post- and 1 month post-intervention, the results revealed no significant group by time interactions for any of the variables. This indicates that there were no differences in how the treatment group and the wait-list control group changed over time. This is in contrast to previous studies that documented group psychoeducation as being more effective than a wait-list control group and a no-treatment control group in the treatment of disordered eating in individuals without diabetes (Kaminski and McNamara, 1996; Wolchick et al., 1986). It is also in contrast with the only study to date to evaluate group psychoeducation for the treatment of individuals with type 1 diabetes (Olmsted et al., 1998). This study by Olmsted et al. (1998) demonstrated that group psychoeducation was more effective than a no-treatment control group for the treatment of young girls considered at risk for developing subclinical or clinical disordered eating (Olmsted et al., 1998).

There are several explanations as to why the group psychoeducation program evaluated in the present study was not more effective than a wait-list control group for the treatment of women with subclinical disordered eating and type 1 diabetes. Individuals with diabetes who develop disordered eating may develop a particularly intractable form of the disease that is not readily amenable to treatment (Peveler and Fairburn, 1992). Although only case reports have been published, treatment of individuals with co-existing type 1 diabetes and clinical eating disorders has proven very difficult and outcome is often poor (Pitel et al., 1998; Peveler and Fairburn, 1992; Hillard and Hillard, 1984; Szmukler, 1984; Malone and Armstrong, 1985; Peveler and Fairburn, 1989). Olmsted et al. (1998) conducted the only controlled study to date on

the treatment of individuals with type 1 diabetes and disordered eating, although the subjects had more mild disordered eating than the women in the present study and were considered at risk for developing subclinical or clinical eating disorders. Nonetheless, Olmsted et al. (1998) found group psychoeducation to be effective in ameliorating disturbed eating attitudes and behaviours compared to a no-treatment control group. Group psychoeducation may be effective for individuals with type 1 diabetes who are at risk for disordered eating, but who are not experiencing significant disordered eating attitudes and behaviours. More individualized and intensive treatment approaches may be necessary for individuals suffering from actual subclinical disordered eating. The original psychoeducation program developed by Weiss et al. (1985) for individuals without diabetes who had bulimia nervosa, included 7 group sessions and 2 individual sessions. Modifications were made to the program to adapt it for the population of women being studied here. These modifications included eliminating the 2 individuals sessions and combining 2 group sessions to make the program 6 instead of 7 sessions in length. Perhaps if a longer program had been used, that also incorporated 2 individual sessions, the psychoeducation program would have been more effective.

Another explanation as to why the group program was not more effective than the wait-list control group could have been due to the presence of significant general psychopathology. It has been suggested that affective disorders and personality disturbances, which are more common among individuals with co-existing diabetes and eating disorders than among individual without diabetes, could minimize treatment effectiveness in this population (Peveler and Fairburn, 1992). The women in the present study were experiencing some psychopathology prior to commencing the study. Both groups were experiencing depression and emotional distress at baseline, indicated by mean Beck Depression Inventory (BDI) scores above 13 and mean Brief Symptom Inventory (BSI) scores above or equal to 0.78. The mean scores on the Rosenberg Self-Esteem Scale (RSE) were within normal ranges, indicating normal self-esteem. Although the women were experiencing depression at baseline, the mean scores on the BDI were similar to the scores reported in individuals without diabetes who had disordered eating and were successfully treated with group psychoeducation (Wolchik

et al., 1986; Davis et al., 1997). The scores on the RSE were also similar to those previously reported for individuals successfully treated with group psychoeducation (Wolchik et al., 1986). The BSI has not been used to date for the evaluation of psychoeducation so comparisons cannot be made. Although some research has demonstrated that the disordered eating accompanied with general psychopathology results in a poorer prognosis (Mitchel and Groat, 1984), most evidence suggests that general psychological disturbances at pre-treatment have little effect on eating disorder prognosis (Garner et al., 1990; Wonderlich et al., 1997; Rossiter et al., 1993). Conversely, evidence to date suggests that personality disorders are a strong negative prognostic factor in eating disorder treatment (Johnson et al., 1990; Rossiter et al., 1993). Although the women in the present study were experiencing some general psychopathology at baseline, this likely did not interfere with the success of the group psychoeducation program. It is possible that some women were suffering from personality disorders that could have minimized treatment effectiveness, but this was not assessed at any time during the study period.

A third explanation as to why group psychoeducation was not more effective than the wait-list control group could have been due to the fact that the women in the present study were considerably older than those previously studied in group psychoeducation research. The average age of the women in the present study was 31.9 years of age, whereas in previous studies evaluating group psychoeducation the average age of the participants has ranged from 18.3 to 25.2 years (Kaminski and Mcnamara, 1996; Wolchik et al., 1986; Connors et al., 1984). In the study by Olmsted et al. (1998) which demonstrated that group psychoeducation was effective for treating girls with type 1 diabetes and mild disordered eating, the age range was 12 to 19 years (average age not reported), whereas the age range in the present study was 21 to 44 years. Connors et al. (1984) reported a negative association between age and improvements in eating disorder symptomatology in response to group psychoeducation. The authors suggest that younger individual's beliefs are less entrenched, making them more susceptible to change in response to group psychoeducation.

Although there was no differential effect demonstrated between the treatment group and the control group, the two groups as a whole changed significantly over time with respect to several variables. Both the treatment group and the control group experienced a significant decline in short-term metabolic control as indicated by an increase in fructosamine from the pre-intervention time point to the post-intervention time point. This decline in short-term metabolic control was maintained at the 1 month post-intervention time point. There was no change, however, in long-term metabolic control reflected in a constant HbA_{1c} over the 3 time points, a total time period of 2.5 months. Both groups experienced significant improvements in depression and general emotional distress from pre-intervention to the post-intervention and the improvements were maintained for 1 month. Diabetes treatment adherence and eating disorder symptomatology did not change over the study period.

Fructosamine increased in both groups immediately post-intervention. It is not surprising that metabolic control would decline during the initial course of eating disorder treatment as therapy frequently involves liberalization of meal plans, avoiding dietary restraint and learning to eat according to hunger cues (Peveler and Fairburn, 1992). It would take a number of months for an individual to re-establish normal eating behaviours once liberalization of eating habits began. During the initial stages of eating disorder treatment, the goal of "tight" glycemic control should be temporarily replaced with the goal of "adequate" glycemic control and restoration of normal eating habits (Hillard and Hillard, 1984; Peveler and Fairburn, 1989). However, it is surprising that the subjects demonstrated an increase in fructosamine with no accompanying changes in eating disorder symptomatology. Changes in eating disorder symptoms would have been expected if changes in short-term metabolic control resulted from a liberation of dietary restraint. It is possible that small changes in disordered eating did occur, but were not detected with the assessment tools used here, as self-report questionnaires of eating disorder symptoms are known for being less accurate than interview-based tools (Williamson et al., 1995). It is also surprising that both groups experienced the same changes in fructosamine since the control group received no information about eating habits, although they did report on the frequency of bingeing and purging behaviours,

which is a form of self-monitoring. Self-monitoring is frequently used in the treatment of eating disorders and has been shown to result in positive behavioural change (Haynes, 1983; Agras et al., 1989; Peveler and Fairburn, 1992). Glycated hemoglobin did not change over the course of the study, indicating that the short-term changes in fructosamine were not large enough to affect long-term glycemic control. The participants were not at increased risk for long-term complications as a result of the changes that occurred during the course of the study.

Although both the treatment program and the control group experienced improvements in general psychopathology, the women in both groups did not experience any improvements with respect to eating disorder symptomatology. This is interesting because eating disorder symptomatology is closely associated with general psychopathologies such as depression, anxiety, social withdrawal and isolation. These general psychological disturbances are very common in individuals with eating disorders and frequently develop secondary to the eating disorder itself (Garner et al., 1990, Halmi et al., 1991; Rastam et al., 1992). These same psychological disturbances frequently serve to perpetuate the eating disorder. For example, stress and anxiety have been shown to precipitate bingeing (Elmore and Castro, 1990). Although most researchers have found that psychiatric symptoms improve as eating disorder symptoms improve, it is possible for the opposite to occur where improvements in psychological difficulties cause improvements in eating disorder symptoms (Mitchell and Groat, 1984; Garner et al., 1990; Rastam et al., 1992; Steere et al., 1990). It is surprising that the women in the present study experienced significant improvements in depression and distress without any changes eating disorder symptomatology. The changes in general psychopathology were probably unrelated to eating disorder symptomatology. It is possible that if the program had been longer, changes in eating disorder symptoms would have accompanied the changes in general psychopathology seen in the treatment group. It is also possible that there were small changes in eating disorder symptoms that were not detected with the self-report questionnaires used in the study.

Changes in diabetes treatment adherence and long-term metabolic control also did not accompany the changes in general psychopathology. Diabetes treatment

adherence and metabolic control are strongly associated with depression, anxiety and quality of life (Littlefield et al., 1992; Mazze et al., 1984). It is suspected that the relationship that exists between these variables in one in which improvements in diabetes treatment adherence and metabolic control cause improvements in depression and not vice versa (Mazze et al., 1984; Littlefield et al., 1992). This would explain why there were no improvements in adherence and metabolic control accompanying the improvements in depression and distress. The improvements in depression and distress were probably unrelated to metabolic control or diabetes treatment adherence. It is possible that the changes in diabetes treatment adherence did occur, but the measure of diabetes treatment adherence was not able to detect any differences in adherence. It is well known that measures of diabetes adherence can be unreliable because many patients misjudge their own behaviour (McNabb, 1997).

Although the only improvements demonstrated in this study were related to general psychopathology, the improvements were valuable in this group of women who were depressed and suffering from general emotional distress at the beginning of the study. The mean scores for the treatment group indicated that they were not experiencing depression or distress at the post- and 1 month post-intervention time points. The control group was experiencing depression and distress at the post and 1 month post-intervention time points, although they had experienced significant improvements.

In spite of the fact that group psychoeducation was not shown to be more effective than a wait-list control group, there seemed to be similar therapeutic factors associated with the wait-list control group and the psychoeducation program because positive changes with respect to general psychopathology were demonstrated in both groups. The therapeutic aspect of the group psychoeducation program was probably largely due to the supportive nature of interacting with other women with type 1 diabetes who all experienced similar struggles with eating. Research on support groups for eating disorders have demonstrated greater improvements in depression and social attitudes than in disordered eating behaviours and attitudes (Rathner et al., 1993). Although unexpected, it is not altogether surprising that the women in the control group

improved over time. Women with type 1 diabetes displaying disordered eating represent only a small fraction of the entire diabetes population. We found that many women felt isolated and alone in their struggles with disordered eating attitudes and behaviours. Prior to signing consent forms an information sheet was discussed with the potential participants. At this time, the definition of subclinical disordered eating was discussed and the research participants were told that there were other women taking part in the study who had similar experiences as they had. It was also explained that, whether or not they were assigned to the treatment group or the control group, they would receive the psychoeducation program. This information session could have affected the women's feelings of isolation, helplessness and motivation. The women in the control group were required to come to the university 3 times to complete the questionnaire package. Mitchell et al. (1996) suggest that assessment questionnaires and interviews may in themselves be therapeutic for some patients. The questionnaires completed by the research participants involved reporting frequencies of specific disordered eating behaviours as well as reporting on emotions and feelings. This could be seen as a form of self-monitoring which is frequently used in the treatment of eating disorders and has been shown to positively effect behaviours (Agras et al., 1989; Peveler and Fairburn, 1992). During the assessment visits, research subjects developed a relationship with the researcher who would later facilitate the group psychoeducation program. This relationship could have given the subjects feelings of security in knowing that they had access to a health professional who understood their struggles concerning eating attitudes and behaviours.

Similar improvements in control groups or minimal intervention groups have been reported in a small number of studies involving eating disorder treatment (Mitchell and Groat, 1984; Thackwray et al., 1993). Thackwray et al. (1993) used a non-specific self-monitoring control group in evaluating the effectiveness of two treatments for bulimia nervosa. The control group consisted of therapist contact and attention, self-monitoring and instilling positive expectancies in the subjects. The therapist reviewed self-monitoring forms with the subjects, but maintained a non-directive role, offering no suggestions for change. The subjects in this control group experienced immediate

improvements in binge-purge frequency and depression immediate post-intervention, but the changes were not maintained at the 6 month follow-up. The authors suggest that presenting for treatment, interacting with a therapist and engaging in self-monitoring can produce immediate beneficial psychological changes.

It is possible that the changes documented in the wait-list control group and the group psychoeducation program can be attributed to taking part in either group, but this conclusion cannot be made based on the evidence in this study. There is a possibility that the women in the study would have spontaneously improved whether or not that had taken part in the psychoeducation program or the control group. It was interesting that positive changes occurred in the wait-list control group, but in order to attribute the changes to the actual factors involved in being in that group, another control group would have been needed. This control group would be required to receive very little information about their disordered eating status, to not receive the treatment program upon completion of the study, to not complete self-monitoring types of assessment questionnaires and to not develop a relationship with the health professional running the psychoeducation program. This type of evaluation would have been very difficult to complete.

In conclusion, a 6-session group psychoeducation program was no more effective than a wait-list control group in the treatment of subclinical disordered eating in adult women with type 1 diabetes. Although group psychoeducation may be effective in women with clinical and subclinical eating disorders without diabetes and adolescent girls with type 1 diabetes who are at risk for eating disorders, adult women with type 1 diabetes and subclinical disordered eating may need more intensive treatment. Although improvements in depression and general emotional distress were documented in the treatment group, the control group also experienced the same improvements. Group psychoeducation and a wait-list control group that involves self-monitoring may both be somewhat therapeutic in women with these two co-existing disorders, although further research is need to confirm this finding.

E. FOLLOW-UP ANALYSIS

The follow-up analysis revealed that there were no changes in the treatment group from 1 month post-intervention to 6 months post-intervention. This indicates that the improvements in general emotional distress and depression seen immediately post-intervention in the treatment group were maintained for 6 months and that no improvements occurred with respect to diabetes treatment adherence and eating disorder symptomatology during the 6 months after the treatment program ended. It is noteworthy that fructosamine did not change between the 1 month post- and 6 months post-intervention time points in the treatment group. However, this does not indicate that the increase in fructosamine demonstrated post-intervention was maintained for 6 months. Only 5 treatment subjects completed the 6 month follow-up assessment and the analysis of these 5 subjects did not reveal an increase in fructosamine levels between the pre- and post-intervention time points. It is unclear whether the increase in fructosamine demonstrated between the pre and post-intervention time points for the entire treatment group was maintained for 6 months. Glycated hemoglobin remained unchanged at 6 months follow-up, indicating that metabolic control remained the same throughout the follow-up period.

Although the wait-list control group and the treatment group experienced the same immediate post-intervention changes, there is no way to determine if the changes in the control group were maintained for 6 months because 6 month follow-up data were not obtained for this group. Previous studies that have documented positive change in minimal intervention control groups have found these changes to be more short-lived than changes seen in treatment groups (Thackwray et al., 1993). A differential effect between the two groups may have been demonstrated had the 6 month follow-up data been obtained for the control group.

F. PROGRAM EVALUATION

Although the results of the present study do not demonstrate that group psychoeducation is more effective than a wait-list control group for the treatment of individuals with type 1 diabetes and subclinical disordered eating, the program participants perceived the program as being very valuable. Every participant who answered the question concerning whether or not they would recommend the program to someone else responded "yes". Additionally, each topic included in the program was considered at least "somewhat helpful" and none were considered "not helpful". The program had excellent attendance, indicating that the participants found the weekly sessions useful. Research has demonstrated that individuals who find group therapy to be meeting their needs are less likely to drop out of treatment (McKisack and Waller, 1996). The participants reported the following aspects of the group program as being the most valuable: the session on cultural expectations of thinness, the session on anger and assertiveness, the homework activities, interacting with other females with diabetes, group discussion, learning new methods to deal with feelings and learning that they were not alone in their struggles. The most valuable part of the group program seemed to be related to the supportive nature of interacting in a group setting with other women with type 1 diabetes who all experienced similar problems. There were only 2 aspects of the program that participants reported were not valuable. These included when the group discussions were undirected or slightly off topic and the session on cultural expectations of thinness. This is interesting since one subject reported the session on cultural expectations of thinness on women as being the most valuable aspect of the program.

The participants gave several suggestions for future groups and these included having a program longer than 6 weeks, having more discussions specific to diabetes, having more group activities and having group members closer in age. These are very useful suggestions that could easily be incorporated into the program. The psychoeducation program may have been more effective had it been longer and had there been more information specific to diabetes. The psychoeducation program was

adapted from a program developed for individuals without diabetes (Weiss et al., 1985). Modifications were made to adapt it for individuals with diabetes, but more modifications could be made. Group members occasionally requested basic information concerning diabetes management and meal planning. This was not meant to be a focus of the program, as it was assumed that the group members would have basic knowledge concerning these topics. Additionally, the focus of the program developed by Weiss et al. (1985) is intended to be on enhancing personal competencies rather than directly on maladaptive eating patterns. Nevertheless, another session could be added to the beginning of the program to provide information on nutrition and diabetes management.

It is important to discuss the suggestion concerning having groups members closer in age. The initial aim of this project was to recruit women who were between the ages of 16 and 35 years to ensure somewhat homogeneous groups; however, due to recruitment difficulties, the age range had to be extended to increase subject enrollment. There are a limited number of women with type 1 diabetes who suffer from subclinical disordered eating and as a result it may not be feasible to run group programs with women of a smaller age range.

Although the group psychoeducation program was not empirically shown to be more effective that the wait-list control group, the women who participated in the program evaluated it very positively and found it useful. They enjoyed attending each weekly meeting and even requested further meetings upon completion of the program.

CHAPTER SIX
CONCLUSIONS

A. CONCLUSION

Although research has convincingly demonstrated that subclinical disordered eating is associated with many adverse complications, individuals with co-existing type 1 diabetes and subclinical disordered eating rarely, if ever, receive treatment for eating disturbances. This is the only study to date to investigate the treatment of adult women with co-existing subclinical disordered eating and type 1 diabetes. Group psychoeducation was not found to be more effective than a wait-list control group. It is possible that adult women with subclinical disordered eating need more intensive treatment than what was offered in the 6-session group psychoeducation program. Noteworthy is the fact that those who took part in the program experienced significant improvements with respect to depression and general emotional distress which were maintained over 6 months. However, the therapeutic effects demonstrated in the treatment group cannot be attributed solely to the group psychoeducation program, as the wait-list control group experienced the same improvements. Nonetheless, the women who participated in the group program evaluated it very positively and considered the experience to be very valuable. Due to the small size of the sample used in this study, the results must be regarded as preliminary. Further research is required to determine definitively whether or not group psychoeducation is an effective treatment for women with type 1 diabetes and subclinical disordered eating.

B. LIMITATIONS OF STUDY

The main limitation of the study was the small sample size. With a larger sample size, differences may have been demonstrated between the treatment group and the control group over time. Alternatively, with a larger sample size, it would be possible to more confidently suggest that group psychoeducation is not more effective

than a wait-list control group for the treatment for individuals with co-existing type 1 diabetes and subclinical disordered eating. The second limitation of the study was the reliance on self-report measures of eating disorder symptomatology for screening and assessments over time. Interview-based instruments, as opposed to self-report measures, provide more accurate and more detailed information concerning eating disorder symptomatology (Williamson et al., 1995). If interview-based instruments had been used, firm diagnoses could have been made during screening and comparisons to other studies would be easier. Furthermore, if interview-based instruments had been used, small changes in eating disorder symptomatology may have been possible to detect and differences over time may have been documented between the two groups. Another limitation of the study involved the follow-up period in the control group. It would have been interesting to comment on whether or not the initial changes seen in the control group were maintained. Previous studies that have documented positive change in minimal intervention control groups have found these changes to be short-term (Thackwray et al., 1993). The final limitation of the study involved the fact that the wait-list control group seemed to have some therapeutic effects. It would have been interesting to compare the group psychoeducation program to a control group that better represented a no-treatment condition. A control group that did not involve the completion of self-monitoring questionnaires, such as the EDI-Symptom Checklist, and did not involve a detailed discussion with the researcher concerning the disease condition and the future intervention program may have been a more effective comparison. All of the above mentioned suggestions could be implemented in future research projects that evaluate group psychoeducation.

C. FUTURE RESEARCH

This is only the second study to date to investigate treatment for disordered eating in individuals with type 1 diabetes and the first to study adult women with actual subclinical disordered eating, as opposed to adolescent girls at risk for developing eating disorders. More research is needed before a firm conclusion can be made as to

whether group psychoeducation is an effective treatment for women with type 1 diabetes and subclinical disordered eating. Further studies should focus on women of varying ages and with varying degrees of eating disturbances to determine if group psychoeducation is effective in all age groups and in all degrees of disordered eating. Future studies should evaluate a group psychoeducation program that includes individual sessions and places as emphasis on how specific issues are related to having diabetes. A session should also be incorporated into the program that covers nutrition and diabetes management. Although not consistent with the findings of the present study, group psychoeducation remains a potentially effective treatment for individuals with type 1 diabetes and subclinical disordered eating.

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APPENDIX A

ETHICAL APPROVAL

Capital Health

Regional Research Administration Office
CSB 9-122, 492-1372

Memorandum

NOTICE OF APPROVAL FOR PROPOSED RESEARCH
UNIVERSITY HOSPITALS SITE

Project Title:	A Treatment Intervention for Subclinical Eating Disorders in Individuals with Insulin Dependent Diabetes Mellitus
Project No.:	T-64
Investigator(s):	Dr. E. Toth / Stefanie Wilson
Department:	Medicine / Agricultural, Good and Nutritional Science
Division:	-
Address:	410 Agriculture Forestry Centre
Phone/FAX:	492-4267

Supporting documents:

1)	Ethical Approval	August 1997 Ethics File #2403
2)	Study Protocol	Received
3)	Funds: a) Source b) Type	Applied to Canadian Diabetes Association Toronto Grant
4)	Overhead Negotiated	N/A
5)	Account #	**Please inform this office when grant is received and if an account will need to be set up.**
6)	Contract	N/A

Project Approved February 1998

THIS APPROVAL IS VALID FOR ONE YEAR

By
Title Maureen Bain, Manager
Regional Research Administration

Copies to: Department Chair/Health Sciences Faculty
Jeanette Stead, Director, Accounting Services
Phil Heuchert, Manager Trust & Research Accounts

February 18, 1998

APPENDIX B

SCREENING CONSENT FORM

A Treatment Intervention for Subclinical Eating Disorders in Individuals with Insulin Dependent Diabetes Mellitus

Diabetes Psychosocial Screening Questionnaire

Investigators: Ellen Toth, MD, Linda McCargar RD, PhD, Stefanie Wilson RD, Msc Candidate

Consent to participate:

Date _____

I _____ agree to fill in the following psychosocial questionnaire. All of my responses will be kept confidential. I have had all of my questions concerning the study answered to my satisfaction, and I understand that I may withdraw my voluntary participation at any time.

_____ _____
subject (name) signature

_____ _____
parent or legal guardian (name) signature

_____ _____
Investigator (name) signature

_____ _____
Witness (name) signature

I agree to be contacted in the future to participate in a related study concerning the results of this questionnaire.

_____ yes _____ no

APPENDIX C

INTERVENTION COMPONENT INFORMATION SHEET AND CONSENT FORM

TITLE OF RESEARCH PROJECT: **A Treatment Intervention for Subclinical Eating Disorders in Individuals with Insulin Dependent Diabetes Mellitus**

INVESTIGATOR(S):	Ellen Toth, MD	492-5276
	Stefanie Wilson RD, BSc, MSc candidate	492-4267
	Linda McCargar RD, PhD	492-9287

Background: Subclinical eating disorders (slightly irregular eating behaviors) are common among individuals with type 1 diabetes mellitus and are associated with poor blood glucose control and increased risk of long term diabetic complications. Depression, anxiety and low self-esteem are also commonly found in individuals with subclinical eating disorders. In the non-diabetic population, cases of subclinical eating disorders raise little concern and as a result there are very few treatment recommendations for this group. It has been suggested that a group education and discussion program may be useful in subclinical cases of eating disorders in individuals with type 1 diabetes, although no studies have evaluated this hypothesis.

Purpose: The purpose of the present study is to evaluate a group psychoeducation program for individuals with type 1 diabetes who have subclinical eating disorders.

Procedures: Participants in each group will be asked to complete an initial questionnaire to determine whether they meet the qualifications for the study. Participants will be assigned to one of two groups. One group will attend the education program immediately and the other group will attend the education program in 2 ½ months time.

Participants who receive the education program immediately will be asked to complete a total of 7 questionnaires at 4 different times during the study as well as to provide a blood sample at the same 4 points in time. The other group will also complete 7 questionnaires and provide a blood sample, but only at 3 different times. The first time point will be at the start of the study, the next time point will be 6 weeks after the start of the study and the third time point will be approximately 3 months after the start of the study. The fourth time point (only for the group who receives the program immediately) will be approximately 7 ½ months from the start of the study. The following is a description of each part of the study:

2. **Questionnaires** - All of the questionnaires are in a multiple choice format.

a) The Eating Disorder Inventory - This 2 part questionnaire assesses eating disorder behaviors and thoughts.

b) The Eating Attitudes Test - This questionnaire assesses attitudes and behaviors associated with eating disorders.
c) The Beck Depression Inventory - This questionnaire measures symptoms of depression.
d) The Rosenberg Self-Esteem Scale - This questionnaire assesses self-esteem.
e) The Brief Symptom Inventory - This questionnaire assesses current emotional distress.
f) The Self-Care Inventory - This questionnaire assesses adherence to diabetes treatment regimen (blood glucose testing, ketone testing, exercise frequency).
g) Hypoglycemia Fear Index – This questionnaire assesses fear of hypoglycemia.

3. Blood Work

A fasting blood sample (5 ml each time) will be drawn from each participant to be analyzed for fructosamine (an index of blood glucose control over the previous 1 month) and hemoglobin A1C (an index of blood glucose control over the previous 3 months). All venipuncture procedures will be performed by trained lab technicians using standard procedures.

4. Group Program

The group/discussion program will consist of a discussion on self-esteem, body image, methods of weight control, societal standards of thinness, expressing anger, assertiveness and stress management. Participants will be encouraged to participate and share their own experiences with the group, but will not be obligated to do so.

Possible Benefits: Individuals with subclinical eating disorders rarely receive treatment since there are very few treatment recommendations for this group. The present research will evaluate an easily reproducible psychoeducation program, allowing for treatment recommendations to be made for individuals with diabetes and subclinical eating disorders. The program will likely improve metabolic control, decrease the risk of long-term diabetic complications and improve depression and anxiety.

Possible Risks: There should be no discomfort or adverse effects to the subjects participating in this study. Blood tests may result in minor pain and possible bruising at the time of blood draw. Qualified personnel will do the venipuncture procedures in all circumstances. Individuals in the treatment group are not obligated to participate in the group discussions so as to avoid any stressful situations

Confidentiality: Personal records relating to this study will be kept confidential. Code numbers will be used rather than names and all files will be stored in a locked file cabinet. Any report published as a result of this study will present group results only and no individual will be identified.

Time Commitment:

Education program - 6 sessions of 1 to 1 1/2 hours in length that will be held once a week for 6 weeks (total = 9 hours)

Blood Tests - 30 minutes at three different times (total =1 1/2 hours) ***first group***

OR

- 30 minutes at four different times (total =2 hours) ***second group***

Questionnaires - 45 minutes at three different times (total =2 hours and 15 minutes) ***first group***

OR

- 45 minutes at four different times (total =3 hours) ***second group***

Total Time Commitment - approximately 12 hours and 45 minutes ***first group***

OR

- approximately 14 hours ***second group***

Results: Each participant will have the opportunity to review his/her results with the researcher. These results include measures of depression, emotional distress, self-esteem, eating disorder symptoms, adherence to diabetic treatment regimen, and metabolic control.

Withdrawal From Study: You are free to withdraw from the research study at any time without jeopardy. You will be promptly informed if any knowledge gained from this or any other study becomes available which could influence your decision to continue in the study. Under all circumstances, the information gathered from this study, relevant to each participant, will be communicated to that participant in an open, yet confidential manner. It will be the participant's decision to determine participation in all stages of this study.

Any Questions? If you have any questions, please feel free to call one of the following people:

Stefanie Wilson RD, BSc, MSc candidate	492-4267
Ellen Toth, MD	492-5276
Linda McCargar RD, PhD	492-9287

Consent Form

Part 1:

Title of Project: **A Treatment Intervention for Subclinical Eating Disorders in Individuals with Insulin Dependent Diabetes Mellitus**

Principal Investigator(s): Ellen Toth MD — Phone Number: 492-5276

Co-Investigator(s): Stefanie Wilson RD — Phone Number: 492-4267
Linda McCargar — Phone Number: 492-9287

Part 2 (to be completed by the research subject):

	Yes	No
Do you understand that you have been asked to be in a research study?	☐	☐
Have you read and received a copy of the attached Information Sheet?	☐	☐
Do you understand the benefits and risks involved in taking part in this research study?	☐	☐
Have you had an opportunity to ask questions and discuss this study?	☐	☐
Do you understand that you are free to withdraw from the study at any time, without having to give a reason and without affecting your future medical care?	☐	☐
Has the issue of confidentiality been explained to you, and do you understand who will have access to your medical records?	☐	☐
Do you want the investigator(s) to inform your family doctor that you are participating in this research study?	☐	☐

Who explained this study to you? _____

I agree to take part in this study: YES ☐ NO ☐

_____
Signature of Research Subject — Printed name

_____
Signature of Parent or Legal Guardian — Printed Name

_____
Date

_____
Signature of Witness — Printed Name

_____
Signature of Investigator — Printed Name

APPENDIX D

SAMPLE SIZE CALCULATIONS

1. Main end point is the total EDI score
2. Statistical difference determined by independent t-tests
3. Difference in scores required to be statistically and clinically significant = 12
4. Standard deviations are based on results of pilot study which assessed eating disorder symptoms in a sample of insulin dependent diabetic individuals
5. alpha = .05
6. Power has been calculated at 80%
7. Assuming a two-sided test, the following formula was used:

$$n = \frac{(SD_1^2 + SD_2^2)(Z_{1-\beta} + Z_{1-\alpha/2})^2}{(X_2 - X_1)^2}$$

$$n = \frac{(6.78^2 + 11.04^2)(0.84 + 1.96)^2}{(47.2 - 59.2)^2}$$

$$n = \frac{(167.85)(7.84)}{144}$$

n = 1315.94/ 144 = 9.1 = 9 subjects per group

Method from Cheney and Boushey, 1991

APPENDIX E

LETTER TO PATIENTS OF THE UNIVERSITY HOSPITAL METABOLIC CENTRE

Dear _____:

We are conducting a research project that will survey psychological issues in type 1 diabetic individuals. I would like to ask you to make an appointment to come into the clinic to complete some psychological assessment questionnaires. I will use the results of your assessment questionnaires to individualize your treatment plan. You will have full access to your results. As this is a part of a research study, it is your choice whether or not you choose to participate. If you would like to participate, you will be required to complete 5 short questionnaires that will take a total of approximately 35 minutes.

I would like to ask you to please make an appointment with my research associate, Stefanie Wilson, to come into the clinic. She can be contacted at 492-4267. For your convenience you will be able to make appointments to come during the day or evening.

Sincerely,

Ellen Toth MD

Stefanie Wilson RD, (MSc Candidate)

APPENDIX F

LETTER TO INDIVIDUALS FROM THE TYPE 1 DIABETES REGISTRY

Dear _____:

We received your name from the Type 1 Diabetes Registry. We would like to invite you to participate in a research project we are presently conducting at the University of Alberta. This is a joint project between the Department of Medicine and the Department of Agricultural, Food and Nutritional Science. The research project will survey body image and eating attitudes in females with type 1 diabetes. If you would like to participate, you will be required to complete 1 short questionnaire that will take approximately 15 minutes. You will have full access to your results. As this is a part of a research study, it is your choice whether or not you choose to participate.

Please call Stefanie Wilson to find out more about the research study and to make an appointment to complete the questionnaire. She can be contacted at 492-4267.

Sincerely,

Ellen Toth MD

Stefanie Wilson RD. (MSc Candidate)

APPENDIX G

SAMPLE RECRUITMENT NOTICES

Advertisement in Edmonton Journal, Life Section

FREE Body Image Assessment

For individuals
- With Type 1 Diabetes
- Over 15 years old
- Female

Have your body image and eating attitudes assessed for free by researchers at the University of Alberta.

Call Stefanie
492-4267
Dept. Agricultural, Food & Nutritional Science

Public Service Announcement in Edmonton Journal, Health Briefs

Participants Sought for Study

Researchers at the University of Alberta are seeking participants for a study of the eating attitudes and behaviors of females with type 1 diabetes. Participants will complete one questionnaire and will have the option to review their results with a registered dietitian. If you are a female with type 1 diabetes and are over 15, contact Stefanie Wilson RD, University of Alberta, Dept. Agricultural, Food and Nutritional Science at 492-4267.

APPENDIX H

DEMOGRAPHIC QUESTIONNAIRE

GENERAL INFORMATION:

Name: _____ Sex: M F

Phone: _____(day) _____(evening)

Age: _____ Duration of diabetes: _____

Weight: _____ Height: _____

Highest level of education completed to this point: (check one)

Grade: 1 2 3 4 5 6 7 8 9 10 11 12

Post-secondary: 1 2 3 4 5 6 7 8 years

Other: (please specify) _____

Date of last Hgb A1c: _____

Value of last Hgb A1c: _____

How often do you test blood sugar?_____

APPENDIX I

PARTICIPANT RESULTS PACKAGE

Dear Participant:

Attached you will find the results of the assessments you completed between the dates of _____. You are provided with your score for each of the psychological assessment questionnaires as well as how to interpret those scores. You are also provided with your blood glucose control results as well as the normal ranges for each measurement. You will find the group average for each assessment. This group consists of those participants who took part in the group program with you.

As you are reading your results, please keep in mind that the questionnaires are not perfect. They only give us an estimate of how you compare to other women of your age. If you have any questions or concerns about any of this information please call me. I will be available to answer questions over the phone or by appointment.

Your final fructosamine results are not available yet. The results will be available in April 1999. If you call at that time, they will be provided to you.

I would like to take this opportunity to thank you once again for your participation in this research project.

Sincerely,

Stefanie Wilson RD
(MSc Candidate)
492-4267

Type 1 Diabetes Mellitus and Subclinical Eating Disorder Research Project

Individual Results Summary

A. PSYCHOLOGICAL ASSESSMENT QUESTIONNAIRES

1. Brief Symptom Inventory

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **above 0.78** may indicate that you experience general emotional distress. Keep in mind that this questionnaire result is very general. The questionnaire results that follow may help to explain the source of emotional distress you experience.

---

2. Beck Depression Inventory

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **between 20 and 28** may indicate that you experience moderate feelings of depression
- A score **over 28** may indicate that you experience fairly strong feelings of depression

---

3. Eating Disorder Inventory

a) Drive for Thinness

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **above 6** indicates that you may have a strong preoccupation with your weight and a strong fear of gaining weight.

b) Bulimia

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **above 2** indicates that you may have mild tendencies to think about and to engage in uncontrolled overeating
- A score **above 5** indicates that you may have strong tendencies to think about and to engage in uncontrolled overeating

c) Body Dissatisfaction

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **over 14** indicates that you may experience mild unhappiness with your overall body shape and size
- A score **over 21** indicates that you may experience a large amount of unhappiness with your overall body shape and size

---

4. Eating Attitudes Test

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **over 16** indicates that you may have some feelings around eating that are slightly unhealthy. You may also have some eating behaviors that are slightly unhealthy
- A score **over 29** indicates you may have some feelings around eating that are very unhealthy. You may also have some eating behaviors that are very unhealthy.

---

5. Self-Care Inventory

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **below 3.54** indicates that you follow the recommendations about caring for your own diabetes less than the average person with Type 1 diabetes.

---

6. Rosenberg Self-Esteem Scale

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A score **below 21** indicates that you may have lower self-esteem than average.

B. METABOLIC CONTROL ASSESSMENTS

1. Hemoglobin A_{1c}

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- A satisfactory results is below 0.084
- this test result describes your blood glucose control over the previous 2 to 3 months

---

2. Fructosamine

	Date	Result	Group Average
(Before program)			
(After program)			
(1 month after program)			
(6 months after program)			

Interpretation

- normal result is 200-285 umol/l
- this test result describes your blood glucose control over the previous 2 to 3 weeks

APPENDIX J

EVALUATION FORM

1. What aspects of the group did you find the most valuable?

2. What did you find the least valuable?

3. Please rate the following topics in terms of how helpful they were to you:

	Very Helpful	Somewhat Helpful	Not Helpful
A. Overview of Subclinical Eating Disorders	_____	_____	_____
B. Emotional Eating/Normal Eating	_____	_____	_____
C. Stress Management	_____	_____	_____
D. Changing Thought Patterns (Replacing negative monologues)	_____	_____	_____
E. Perfectionism (Replacing "I Shoulds")	_____	_____	_____
F. Self-Esteem	_____	_____	_____
G. Nourishing Activities	_____	_____	_____
H. Assertiveness	_____	_____	_____
I. Expressing Anger	_____	_____	_____
J. Cultural Expectations of Thinness	_____	_____	_____
K. Body Image	_____	_____	_____

4. What topics would you have like to see added to the group?

5. Would you recommend this group to anyone else? Why or why not?

6. What suggestions do you have for future groups?

7. Additional Comments;

Thank you very much for your comments.