8-2007

The Impact and Efficacy of Diabetes Education Programs among Adults.

Mireille Hamdan
East Tennessee State University

The Impact and Efficacy of Diabetes Education Programs among Adults

---

A thesis

Presented to

the faculty of the Department of Family and Consumer Science

East Tennessee State University

In partial fulfillment

of the requirements for the degree

Master of Science in Clinical Nutrition

---

by

Mireille Hamdan

August 2007

Committee:

Elizabeth Lowe, Chair

Amelia Brown, PhD

Alison Schaefer

Keywords: Diabetes mellitus, education program, blood sugar, Hemoglobin A1c test.

ABSTRACT

The Impact and Efficacy of Diabetes Education Programs among Adults

by

Mireille Hamdan

The purpose of this study was to measure the impact of diabetes education classes in increasing knowledge and promoting healthy lifestyle behaviors. Thirty-three subjects participated in the classes between October 2004 and October 2005. Only six subjects agreed to participate in this study. Changes in knowledge after the classes were measured by a survey test one year following the completion of classes. Survey responses were analyzed using percentages. Subject's Hgb A1C and weights were also collected to measure the direct impact of education on participants' blood glucose management. Overall, participants were very knowledgeable of diabetes symptoms, complications, carbohydrate counting, and serving sizes after the nutrition intervention.

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to my great instructor, Beth Lowe, for her role in the completion of this project. The time she spent in proofreading and reviewing is invaluable. I appreciate her guidance and encouragement both in scholastic and other matters to help me finish my master's degree program successfully. Thank you also to my committee members, Dr. Amelia Brown and Alison Schaefer, for their time and expertise.

Thank you to the Health Resource Center staff and Susan McAuley who supported me and helped me work with their patients. I would also like to thank all of my family for their genuine care; specifically my mother for her great emotional support. Finally, a very special thanks goes to my husband, Dr. Lucien Abboud. I feel lucky to have met someone so loving and supportive. I appreciate each of you, and I value the important and unique part that you have played in my life.

CONTENTS

LIST OF TABLES

CHAPTER 1

INTRODUCTION

Diabetes is a metabolic disorder in which there is little or no control of blood sugar through inadequate insulin production (Type 1 or insulin dependent diabetes) or decreased cellular sensitivity to insulin (Type 2 or non-insulin dependent diabetes). Insulin is needed to convert sugar into energy for daily life. Both genetics and environmental factors, such as obesity and lack of exercise, play important roles in the occurrence of this metabolic disorder (1).

Diabetes has become increasingly common in the United States. In 2002, the number of Americans with diabetes was estimated of 18.2 million, yet only 13 million people were diagnosed (2). Approximately 8.7 % of people 20 years or older have diabetes, and the percentage rises to 18.3 % for people 60 years or older (3). In 2002, the cost of diabetes care in the United States was estimated at $132 billion (4). Direct medical expenditures related to diabetes in 2002 was estimated at $91.8 billion which was divided into $23.2 billion for diabetes care, $24.6 billion for chronic complications accompanied with diabetes, and $44.1 billion for general medical conditions. Indirect costs, totaling $40.8 billion, were associated with lost productivity including premature death and disability. Statistics also indicated that 51.8% of direct medical expenditures were incurred by people 65 years and older (4).

The risk of death is higher among people having diabetes due to the complications associated with the disease. Diabetes increases the risk of kidney disease, blindness

(retinopathy), heart disease, stroke, high blood pressure, and nervous system disease (neuropathy) (2). Studies indicate that diabetes prevention or delay is possible by consuming healthier diets and getting physical activity (5).

Statement of the Problem

The purpose of this study was to measure the impact of diabetes education classes in increasing knowledge and promoting healthy lifestyle behaviors. Diabetes afflicts more than 18 million people in the United States. It is a main cause of kidney failure and blindness and is implicated as a major cause of heart disease and stroke (6). Subjects with Type 2 diabetes (non insulin-dependent diabetes mellitus), or adult onset diabetes, represent up to 90 % of all diabetes cases. It is most common in adults over age 40, and it is strongly associated with obesity, inactivity, and a family history of diabetes; 90% of Type 2 diabetics are obese. The increasing incidence of Type 2 diabetes demands effective out-patient programs for people with diabetes in order to improve their quality of life (6).

Significance of the Problem

Diabetes mellitus contributes to a considerable increase in morbidity and mortality rates every year. The risk for death among people with diabetes is about two times that of people without diabetes (2). The cost of treating diabetes and its complications or compensating for disability resulting from diabetes is high; $132 billion was spent in the year 2002. The direct cost of medical care is $13,243 / person /year with diabetes compared with $2560 per person without diabetes (4). The problem is significant, but

many solutions are available beginning with dietary intervention and increasing the level of physical activity (5).

Hypothesis

Participation in a diabetes education program will result in significantly better glycemic and weight control for participants in a diabetes education program.

Null-hypothesis

There will be no significant difference in weight and glycemic control after participating in a diabetes education program.

Limitations

Limitations of this study include:

1. Lack of consistency due to the nutrition programs being presented by two different instructors, unexpected absence of the participants in classes, and participants dropping out of the program for a variety of reasons.
2. Subjects could improve glycemic control independently of weight loss.
3. There was no control of motivational state of the participants.
4. The effect of medications or insulin was not taken into consideration among participants. Some subjects could be taking both insulin and oral medications; others will be taking either one of them.
5. Small group sample size.

Assumptions

Assumptions of this study include:

1. Patients will provide honest answers to the survey questions.
2. The researcher assumed that the changes in Hemoglobin A1C test and in the body weight were only due to the quality of the diet and not affected by any other physiological changes.

Definition of Terms

Metabolic Syndrome- defined as abdominal obesity and insulin resistance accompanied with an increased risk for cardiovascular disease and Type 2 diabetes. The risk factors include elevated low-density lipoprotein, decreased high-density lipoprotein, elevated triglyceride, hypertension, and impaired glucose tolerance (7).

Obesity- is an increase in fat cells proportion in the viscera and subcutaneous tissues of the body resulting in a body mass index (BMI) greater than 30. (7).

Keto-acidosis- incomplete oxidation of fat in the body in which ketone bodies build up in the blood, resulting from an insufficient availability of insulin to allow proper action of glucose for energy (7).

Type 1 diabetes- known as insulin dependent diabetes mellitus, results from absolute insulin deficiency secondary to β-cell destruction and leading to hyperglycemia. Persons with Type 1 diabetes are dependent on exogenous insulin (7).

Type 2 diabetes- known as non-insulin dependent diabetes mellitus, results from a combination of cellular insulin resistance and insulin deficiency. The endogenous insulin

level may be normal or elevated but inadequate to overcome the decreased tissue sensitivity to insulin (7).

Gestational diabetes - any degree of glucose intolerance recognized during second and third trimester of pregnancy (7).

Hypoglycemia- defined as low blood glucose level < 70mg/dl which can be a result of low food intake or an excessive insulin administration (7).

Hyperglycemia- defined as high blood glucose levels (7).

Hemoglobin A1C- HgbA1C reflects blood glucose levels over the past 120 days (7).

Lipolysis- the breakdown of fat stored in fat cells. During this process free fatty acids are released and ketones are produced (7).

Insulin resistance- tissue sensitivity to endogenous and exogenous insulin is decreased due to an impaired biologic response (7).

Macrovascular diseases- diseases related to large blood vessels, includes peripheral vascular disease, cardiovascular disease, and coronary artery disease (7).

Microvascular disease- disease related to the small blood vessels like retinopathy and nephropathy (7).

Postprandial blood glucose- blood glucose level after eating (7)

Fasting blood glucose- defined as the level of blood glucose in the morning before eating (7).

Impaired Glucose tolerance (IGT) - called pre-diabetes, defined as an elevated blood glucose level, two hours after a meal, greater than or equal to 140 and less than 200mg/dl (7).

Hypertension- is an elevated blood pressure level exceeding 140/90 mmHg (7).

Hyperglycemic Hyperosmolar State (HHS) - is characterized by high blood glucose levels with absence of ketosis and dehydration (7).

CHAPTER 2

REVIEW OF LITERATURE

Diabetes is one of the most important clinical and public health problems facing the world today (8, 9). Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both (10). The development of diabetes may be due to pathogenic processes, ranging from the autoimmune destruction of the beta-cells of the pancreas causing insulin deficiency to cellular disturbances resulting in insulin resistance (10, 11). Insulin deficiency may be due to inadequate secretion or diminished tissue response to insulin in the complex pathway of hormone action; these result in disturbances in carbohydrate, fat, and protein metabolism (11, 12).

Classification of Diabetes

The majority of diabetes cases fall into one of four categories: Type 1, Type 2, gestational diabetes, or pre-diabetes (10).

Type 1 Diabetes

This type was known previously as insulin-dependent diabetes, or juvenile-onset diabetes. The primary cause of Type 1 diabetes is pancreatic beta-cell destruction leading to absolute deficiency of insulin secretion (13, 14) and resulting in hyperglycemia. Symptoms of hyperglycemia include excessive urination, excessive thirst, dehydration, electrolyte disturbance, and keto-acidosis. Type 1 diabetes may be caused by an

autoimmune pathologic process occurring in the pancreatic islets or by genetic markers. These markers could be islet cell auto-antibodies (ICAs), auto-antibodies to insulin (IAAs), or auto-antibodies to glutamic acid decarboxylase which is a protein on the surface of beta-cells (8, 14). People with Type 1 diabetes require exogenous sources of insulin to control their blood sugar levels. It develops most often in children and young adults and accounts for about 5% to 10% of diagnosed diabetes in the United States. Patients are rarely obese when they present with this type of diabetes, and they are typically at high risk for keto-acidosis due to poor glycemic control (13, 14).

Type 2 Diabetes

This type is known as non-insulin-dependent diabetes, Type 2 diabetes, or adult-onset diabetes. The cause is a combination of insulin resistance and inadequate compensatory insulin secretion response resulting in hyperglycemia (14, 15). This form of diabetes is frequently undiagnosed in the early stages. Insulin resistance is first demonstrated in target tissues, mainly muscle tissues and the liver. Initially there is a compensatory increase in insulin secretion. This process will maintain normal glucose concentrations, but as the disease progresses, insulin production gradually decreases. Hyperglycemia begins with an elevation of the postprandial blood glucose due to insulin resistance at the cellular level, and it is followed by an elevation in fasting glucose concentrations (14, 16, 17). As insulin secretion decreases; hepatic glucose production and pre-prandial blood glucose increase (7, 14). The risk of developing Type 2 diabetes increases with age, obesity, and lack of physical activity. The obesity is not only measured by the traditional weight criteria, but it is also associated with an increased percentage of body fat distributed predominantly in the

abdominal region (14, 17). In addition, Type 2 diabetes is associated with a strong genetic predisposition (7, 18).

Gestational Diabetes

This type is discovered during pregnancy and usually disappears when the pregnancy is over. Women who have had gestational diabetes have a grater risk of developing Type 2 diabetes later in their lives (7, 15, 19).

Pre-Diabetes

A pre-diabetes stage includes the impaired fasting glucose (IFG) and impaired glucose tolerance (IGT). Impaired fasting glucose represents a metabolic stage of impaired glucose homeostasis intermediate between normal and diabetes. Impaired glucose tolerance is associated with increased macro-vascular disease. This stage of impaired glucose homeostasis or pre-diabetes is not a category of diabetes mellitus but it indicates a high risk for future diabetes (7, 10, 14).

Diagnostic Criteria for Diabetes Mellitus and Pre-Diabetes

Diabetes mellitus is diagnosed using one of three methods and must be confirmed on a subsequent day (7, 20).

- Random plasma glucose is greater than or equal to 200 mg/dl.
- Fasting plasma glucose is greater than or equal to 126 mg/dl.
- Two-hour postprandial plasma glucose is greater than or equal to 200 mg/dl during a 75 g oral glucose tolerance test (OGTT).

Impaired fasting glucose (IFG) is diagnosed when fasting glucose levels are greater than or equal to 100 mg/dl but less than 126 mg/dl (14, 20). Impaired glucose tolerance is diagnosed when two-hour oral glucose tolerance test (OGTT) values are greater than or equal to 140mg/dl but less than 200 mg/dl. So, the normal fasting glucose is less than 100mg/dl and normal OGTT is less than 140 mg/dl (14, 20).

Hemoglobin A1C

Hemoglobin A1C test (HgbA1C test) is also called the diabetic control index and glycosylated or glycated hemoglobin. It is a test that indicates the quantity of glucose in the blood during the preceding period of two to four months. A routine blood sugar test reveals how close to normal the glucose level is at the time of the test (21, 22).

The hemoglobin A1C test measures the percentage of hemoglobin bound to glucose. Hemoglobin is a protein found in the blood cells. Hemoglobin and glucose, found in red blood cells (RBC), bind to the A1C form of hemoglobin in a process called glycosylation. The amount bound reflects how much glucose has been in the blood during the past average 120 day lifespan of RBC. This can be measured by several methods such as electrophoresis, chromatography, and immunoassay which are all based on the separation of hemoglobin bound to glucose from that without glucose (22). The American Diabetes Association recommends that this test should be done during the first diagnosis of diabetes, again after the beginning of the treatment, and than repeated every six months; if the person does not meet the treatment goals, it should be repeated every three months (21, 22). According to Edelman et al. and Peters et al. the following categories were defined based on HgbA1C results for persons without prior diagnosis of diabetes: normal- HgbA1C <6%;

high risk for diabetes- HgbA1C 6%-6.99%; new diabetes- HgbA1C≥ 7% (23, 24). Hemoglobin A1C is a better screening test for diabetes than random glucose. The mean serum glucose was 100mg/dl ± 22 mg/dl for persons without prior diagnosed diabetes and with a HgbA1C between 6% and 7%. For those with HgbA1C test of 7 % the mean random glucose was 153mg/dl ±62mg/dl (24). So, the measurement of HgbA1C is essential for early diagnosis and long-term monitoring of blood glucose in patients with diabetes (24).

Complications and Risk Factors of Diabetes

Obesity is one of the greatest risk factors for Type 2 diabetes. Particularly, abdominal obesity is one of the factors leading to plurimetabolic abnormalities known as insulin resistance syndrome (25, 26). Insulin resistance at the adipocyte level leads to lipolysis and an elevation in the circulating free fatty acids. An increase in free fatty acids will cause a further decrease in insulin sensitivity at the cellular level that will impair pancreatic insulin secretion and at the same time increase hepatic glucose production (14, 16). In addition an increase in free fatty acids in the blood will increase the blood pressure and triglycerides which are two other risk factors for insulin resistance syndrome and lead to cardiovascular disease (26, 27).

Two different categories of diabetes complications are defined: acute complications and long-term complications (10).

Acute Complication

The acute complications related to diabetes are hypoglycemia, diabetic keto-acidosis, and hyperglycemia. With insulin therapy it is extremely difficult to have good glycemic control. Sometimes blood glucose will drop suddenly to a level lower than or equal to 70 mg/dl causing hypoglycemia that must be treated immediately. Symptoms of hypoglycemia include sweating, trembling, difficulty concentrating, and lightheadedness. With progression to severe hypoglycemia the diabetic will be not able to self-treat due to mental confusion, and sometimes if left untreated this may result in a loss of consciousness (14, 28).

Prolonged hyperglycemia can lead to two types of acute metabolic complications: diabetic keto-acidosis (DKA) and hyperosmolar hyperglycemic state (HHS). Keto-acidosis is caused by inadequate insulin to use glucose for energy. It is a life-threatening complication, but reversible; it is characterized by severe disturbances in carbohydrate, protein, and fat metabolism (7, 15). In contrast, hyperosmolar hyperglycemic state is caused by an excessive amount of blood glucose, with an absence of ketosis and profound dehydration (7, 28).

Long-Term Complications

The most significant diabetes issues are the irreversible long-term complications. These include macrovascular diseases or diseases of large blood vessels and microvascular diseases that involve the small blood vessels (16).

Macrovascular disease includes coronary heart disease, peripheral vascular disease, and cerebrovascular disease. Adults with diabetes have rates of heart disease and stroke two to

four times higher than those without diabetes (29, 30). In fact, the development of metabolic syndrome or insulin resistance syndrome will cause Type 2 diabetes and subsequent macrovascular diseases. This metabolic syndrome is characterized by abdominal obesity (waist circumference in men > 102 cm and in women >88 cm), and associated with dyslipidemia where serum triglycerides are at least 150 mg/dl and high density lipoprotein (HDL) cholesterol is <40mg/dl, hypertension (blood pressure > 130/ 80 mm Hg) and glucose intolerance (7, 20).

Microvascular diseases include retinopathy (eye disease), nephropathy (kidney disease), and neuropathy or nerve damage due to chronic high blood glucose. Diabetic retinopathy is the leading cause of blindness especially in adults after the age of 20 years. After a long period of having diabetes, all people with Type 1 diabetes and 60% of Type 2 diabetics have some degree of retinopathy caused by this disease (14, 31). Diabetes is also a common cause of end-stage renal disease in which 43 % of the cases require dialysis or kidney transplantation (14, 32). In people with diabetes nerve damage occurs over time and may lead to numbness, pain, and weakness in the hands, arms, feet, legs, digestive tract, heart, and sex organs (14, 32).

Self-Management Education Program

The treatment of diabetes mellitus requires counseling on lifestyle modifications and nutritional management (33). The increased marketing and availability of food has contributed to the U.S. obesity epidemic and thus a higher occurrence of Type 2 diabetes (34, 35). Consumers have been confused concerning correct portion sizes (34), and diet therapy is very important to reduce risk factors such as overweight. Diabetes education

should be an integral part of treatment of diabetes mellitus. The desired outcome of the diabetes education is self-care knowledge and the empowerment of the patient in making therapeutic decisions. Self-management programs have been widely reported to help the people managing symptoms of diabetes. Such programs are separated from the clinical patient care but work in collaboration with health care professionals (36). In other words, diabetes self-management is training individuals to manage their diabetes, help optimize metabolic control, prevent acute and chronic complications, and improve quality of life while keeping costs at acceptable levels (37). In these programs specialists like registered dietitians and nurses teach, counsel, make dietary recommendations, and suggest strategies for self-management for the people with diabetes. Successful diabetes care programs are patient centered and establish guidelines and specific responsibilities for each member of the team. The focus, at the same time, is on specific outcomes and the way to achieve these outcomes (9). Medical treatment of diabetes is used to lower blood glucose to normal levels and hemoglobin A1C level to less than 7% (29, 38). The education program should focus on the importance of constant monitoring, regular glucose testing and quarterly review of Hemoglobin A1C. In order to achieve these goals the educational program should focus on the importance of weight loss, reducing the risk of dyslipidemia, and controlling carbohydrate intake (33). Basically the weight loss begins by reducing the total energy and reducing the fat intake to less than 30% of energy intake. The reduction of saturated fat intake (<10% of energy intake), cholesterol intake (< 300mg/day), and trans-unsaturated fatty acid are also recommended to reduce the risk of dyslipidemia. A high fiber diet with a daily intake of 3g of soluble fiber can decrease the total cholesterol by about 5mg/dl or 2% reduction (7, 33, and 39). The nutrition counseling includes the total

amount of carbohydrate intake and the type of sugar intake (glucose, fructose, sucrose, lactose) that will affect the weight loss and the level of glucose in the blood (9). Portioning skills are also taught to emphasize the importance of weight loss (34).

Success of the self-management program can be attributed to helping patients set reasonable goals and empowering them with the appropriate knowledge and skills to achieve their goals (21). The long-term outcome of the program depends on increasing the frequency of testing hemoglobin A1C, decreasing anthropometric measures, and increasing knowledge (21).

CHAPTER 3

DESIGN AND METHODOLOGY

Participants

Participants who attended education classes for diabetes between October 2004 and October 2005 included 33 subjects having diabetes with an age range from 19 to 60 years. These participants were enrolled in the "Caring For Your Diabetes" program sponsored by Mountain States Health Alliance (MSHA). The subjects were employees of the MSHA hospital system or their dependents. The education classes were offered free of charge to all employees and dependents with diabetes and were held at the Health Resource Center in the Johnson City Mall. Participants volunteered to be a part of the education program, and as a result of participation they received free medications and supplies for the treatment of their diabetes. All subjects agreed to participate in the study by signing a release form for the Health Resource Center (HRC).

Instrumentation

The only instrument used in this research was a survey developed by the principal investigator, reviewed by the research committee, and validated by seven MSHA employees with different levels of education. The survey questions were developed and based upon the education materials given in classes. All the questions were intended to measure the degree of understanding of the participants in managing their diabetes (Appendix A).

Data Collection Procedure

Permission to conduct this study was received from The Research Board of MSHA and the East Tennessee State University Institutional Review Board. The participants were employees or dependents of employees in the hospital system of Mountain State Health Alliance (MSHA) who had completed a series of three educational classes. They were trained to manage their diabetes by decreasing their body weight and changing the type of food they consumed. An informed consent, a HIPPA authorization, the survey and two self-addressed stamped envelopes were mailed to the participants. After three weeks, a follow up survey was mailed to those who had not previously responded. After receiving completed surveys, HIPPA authorizations and the informed consents, all the data were collected and analyzed. The collected data from the participants' records at the HRC included the results of the Hemoglobin A1C tests and the changes of the body weight.

Data Analysis

All the survey results, the hemoglobin A1C tests, and body weight changes were gathered. Survey answer percentages and weight changes percentages were calculated and can be found in the tables in Chapter 4. Hgb A1C and weight changes of each subject were recorded, analyzed, and graphed.

CHAPTER 4

RESULTS

The Sample

Participants in this study attended education classes for diabetes between October 2004 and October 2005 at the Health Resource Center. Thirty-three surveys were mailed. Only six participants (18%) completed the survey and agreed to be part of the study by returning the informed consent and the HIPPA forms. The weight changes and Hgb A1C were collected only for those subjects.

Data Analysis

The testing instrument included 11 questions (Appendix A). Seven items had answers limited to true or false; four items were multiple choice questions. The subjects were instructed to leave the question blank if they did not feel comfortable answering; therefore, non-response was considered as incorrect. All the survey questions were analyzed using percentages. The results of these questions are listed in Table 1, with both the number and percentages of respondents for each question.

Define Type 2 diabetes

The first question addressed subjects' knowledge of Type 2 diabetes. All participants had the correct answer on this question (Table 1).

Carbohydrate

The second question was a true or false addressing the carbohydrate metabolism and the amount of calories it produces. All six respondents answered this question correctly.

Question number nine defined carbohydrate choices to help participants count their carbohydrates for each meal. Only four subjects answered correctly (66.6%), (Table 1).

Fiber Intake

The third question addressed the role of a high fiber diet in controlling blood sugar. Four respondents answered correctly (66.6%), (Table 1).

Obesity

The fourth question indicated the relationship between carbohydrate, protein, and fat intake and increase in body fat. All six subjects had the correct answer (Table 1).

Symptoms and Complications

Question five identified the most common signs of diabetes: frequent urination and excessive thirst. All participants were able to recognize these symptoms (Table 1).
The sixth question addressed the relationship between long-term blood sugar management and diabetes complications. All the respondents answered this question correctly (Table 1). Question ten dealt specifically with symptoms of low blood sugar. All participants identified the most common symptoms: feeling shaky and sweaty (Table 1).

Blood Sugar/ Hgb A1C

The seventh question addressed the critical lower limit of blood sugar below which symptoms of low blood sugar will appear. All participants recognized 70mg/dl is the critical point. Question eight measured knowledge of expected Hgb A1C results. Only 50% of participants answered correctly (Table 1). If half of blood sugar results are in the recommended range the expected Hgb A1C should be about 6%.

Serving Size

Question 11 tested the knowledge of participants in estimating serving sizes for carbohydrate counting. Five subjects had the correct answer (83%), (Table 1).

Table 1. Survey question participant responses

	Survey n= 6 No., (%)	
Questions	Correct	Incorrect
1) Define Type 2 Diabetes	6 (100%)	0
2) Carbohydrate	6 (100%)	0
3) Fiber Intake	4 (66.6%)	2 (33.3%)
4) Obesity	6 (100%)	0
5) Symptoms	6 (100%)	0
6) Complications	6 (100%)	0
7)Blood Sugar	6 (100%)	0
8) Hgb A1C	3 (50%)	3 (50%)
9) Carbohydrate Choices	4 (66.6%)	2 (33.3%)
10) Symptoms of Blood Sugar	6 (100%)	0
11) Serving Sizes	5 (83%)	1 (16.6%)

Weight Changes and Hemoglobin A1C

After analyzing the survey questions, HgbA1C and weight changes of participants were collected. Percent weight change was calculated for each subject (Table 2). Subjects one and six lost weight compared to subjects two, three, four, and five who gained weight after starting the classes (Table 2). In fact, subject two gained 21 pounds or an 8.8% weight gain within a year of completion of education classes. Subject five gained 25 pounds or a 10% weight gain within 15 months.

Table 2. Survey and weight change results

Subject	% Wt Change	Wt change in pounds	Survey score (11 questions)
1	7%	-13	10 (91%)
2	8.80%	21	9 (82%)
3	2.90%	8	10 (91%)
4	1.80%	4	9 (82%)
5	10%	25	10 (91%)
6	0.60%	-1	11 (100%)

The survey scores in Table 2 showed that subjects had good knowledge in diabetes management. But the weight gain of subject two and five indicates that the application of their knowledge was minimal even though they answered correctly most of the questions in the survey. Subject six had a 100% score on his survey, yet lost only one pound and had an

increase in HgbA1C. There was no significant correlation between weight change and subject survey score.

Hgb A1C and weight change were also analyzed in graphs to show the relationship between both variables for each subject. Table 3 shows Hgb A1C and weight changes for each subject.

Table 3. Weight and Hgb A1C changes for subjects

Subject Number	Weight change (pound)	Hgb A1C change	Hgb A1C initial	Hgb A1C second	Hgb A1C third	Hgb A1C fourth	Hgb A1C fifth	Hgb A1C sixth
1	-13	-2.2	7.8	5.3	5.6			
2	21	-3.4	11.2	9.9	6.7	7.8		
3	7	0.1	5.9	6.1	6	5.7	6.1	6
4	4	0.1	7.9	7.2	7.2	7.4	8.7	8
5	25	0.4	5.5	5.3	5.5	5.9		
6	-1	0.4	6.3	6.2	5.9	6.7		

Appendix B shows HgbA1C changes of subject one from June 2005 until July 2006. HgbA1C decreased from 7.8 to 5.6. At the same time Figure 1 (Appendix B) shows weight changes from 180 pounds in June 2005 to 167 pounds in October 2006 for this subject.

Subject two showed a HgbA1C decrease from 11.2 in August 2005 to 6.7 in January 2006 accompanied by 1.5 pound initial weight loss (Appendix C, Figure 2). After October subject two gained 22 pounds until February 2007 (Figure 2).

Figure 3 shows the fluctuating HgbA1C of subject three (Appendix D). The lowest HgbA1C was 5.7 in March 2006. Once weight started increasing (Figure 3) Hgb A1C increased to 6.1 in the same month. Subject four shows the highest HgbA1C 8.7 was in November 2006; at the same time weight reached the maximum level of 226 pounds in November 2006 (Appendix E).

The relationship between HgbA1C and weight changes was very obvious for subjects five and six. Actually, Figure 5 shows increase in Hgb A1C to 5.9 along with an increase of weight from 245 pounds to 270 pounds. Figure 6 shows fluctuation in HgbA1C from 6.3 in March 2005 to 5.9 in January 2006, then 6.7 in September 2006. HgbA1C changes were accompanied by weight changes from 150lbs in April 2005 to 144 pounds in August 2005 and back to 150 pounds in July 2006 (Figure 6).

CHAPTER 5

DISCUSSION, CONCLUSIONS, AND RECOMMENDATIONS

Discussion

Overall, the survey responses indicated a good knowledge of diabetes management among subjects participating in education classes for diabetes. Although only six subjects completed the surveys, they answered most of the questions correctly with an average percentage correct of 89%. Only half of the subjects' responses were correct regarding HgbA1C knowledge. One participant expressed his confusion regarding HgbA1C ranges by giving two answers for this question. Another subject was unable to specify the grams of carbohydrate in one carbohydrate choice; at the same time he considered high fiber diet to increase his blood glucose. A possible explanation would be the time lapse in completing the education classes that made it difficult to remember a lot of details.

Also, the data collected regarding HgbA1C and weight changes indicated a positive relationship between the variables. Data indicated as weight decreased, HgbA1C decreased. Some of the participants lost weight, and others gained weight; for some this resulted in HgbA1C increase.

Unfortunately, the small number of participants in the study did not allow further statistical analysis to show the exact correlation between variables. Some of the participants in the study experienced severe health problems and as a result they stopped exercising that may have led to gaining weight. Others were on oral diabetic medication and they started on insulin that could have resulted in lower HgbA1C readings.

Other possible explanations for the differences in how subjects responded to the program and to changing their eating habits include: 1) availability and type of food that subjects can afford; 2) lack of compliance to recommendations; 3) one-time education classes could not ensure the knowledge, the participant's stage of change, or motivation to comply (21, 34).

Conclusions

Coordinating this study with the Health Resource Center provided a way to reach all participants who attended diabetes education classes from October 2004 to October 2005. The subjects participating in this study were successful in demonstrating good knowledge of diabetes even though the study participation rate was very low in comparison to 33 mailed surveys. The absence of direct contact with subjects made the participation rate lower then expected even though all 33 subjects are still enrolled in "Caring For Your Diabetes" program with the Health Resource Center.

Also, the data showed a positive relationship between HgbA1C and weight changes of all six subjects. But the direct impact of these subjects' knowledge on their lifestyle changes was not conclusive because some of them had actually gained weight. This weight change could be a result of noncompliance in diet and in carbohydrate counting because more than a year had passed since completing the education classes.

Recommendations

Reinforcement of the information presented in education classes one year earlier is essential to promote sustained behavioral change among participants. Follow-up phone

calls, support groups, and email correspondence could help refresh subjects' knowledge and provide motivation. Offering programs like "Caring For Your Diabetes" can be very beneficial to help participants control their lifestyle changes and blood sugar to prevent any long-term complications. In addition, long-term studies should be encouraged to analyze the long-term effect of the education classes. Programs that offer free medications and decreased insurance rate by offering education classes can motivate participants to better manage their diabetes. Also, education classes for diabetes can be implemented in a variety of settings in the community to increase awareness of diabetes and with some degree of coordination it can be delivered to varying age or management groups to reinforce the benefits of a healthy lifestyle.

BIBLIOGRAPHY

1. Bailes, Barbara K. Diabetes mellitus and its chronic complications. Association of Perioperative Registered Nurses Journal, 2002.
2. American Diabetes Association. National Diabetes Fact Sheet, 2002. Available at: http://www.diabetes.org/diabetes-statistics/national-diabetes-fact-sheet.jsp. Accessed November 05, 2004.
3. National Diabetes Information Clearinghouse (NDIC); A Service of the National Institute of Diabetes and Digestive and Kidney Disease (NIDDK), NIH; Diabetes. Available at: http://diabetes.niddk.nih.gov/dm/pubs/statistics/index.htm#7. Accessed November 05, 2004
4. American Diabetes Association. Economic Cost of Diabetes in the U.S. in 2002. Available at: http://care.diabetesjournals.org/cgi/content/full/26/3/917 Accessed November 06, 2004.
5. National Diabetes Education Program. Diabetes Prevention. Available at: http://www.ndep.nih.gov/diabetes/prev/prevention.htm Accessed November 06, 2004.
6. National Institute of Diabetes and Digestive and Kidney Disease (NIDDK). Clinical Alert: Diet and Exercise Dramatically Delay Type 2 Diabetes; Diabetes Medication Metformin Also Effective. Available at: http//:nlm.nih.gov.ezproxy.etsu.edu:2048/databases/alerts/diabetes01.htm. Accessed September 28, 2004.

7. Franz J.M., Medical Nutrition Therapy For Daibetes Mellitus and Hypoglycemia of Nondiabetic Origin. In: *Mahan K, Escott-Stump S. Krause's, Food Nutrition and Diet Therapy*. 11th edition. Philadelphia, Pennsylvania: Curtis Center; 2004: 792-837.
8. Jabbour SA, Goldstein BJ. Improving disease management with new treatments for type 2 diabetes mellitus. Clinic of Geriatrics, 2000; 8: 44-56.
9. Venters J, Hunt A, Pope J, Molaison E. Are patients with diabetes receiving the same message from dietitians and nurses? The Diabetic Educator, 2004; 30: 2.
10. National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. Diabetes. 1979; 28:1039-1057.
11. Atkinson MA, Maclaren NK: The pathogenesis of insulin dependent diabetes. New England Journal of Medicine. 1994; 331:1428-1436.
12. Moore H, Summerbell C, HooperL Cruickshank K, Vyas A, Johnstone P, Ashton V, Kopelman P. Dietary advice of Type 2 diabetes mellitus in adults. In: *The Cochrane Library*. Chichester, UK: John wiley & Sons, Ltd. Issue 3, 2004.
13. Schott M, Schatz D, Atkinson M, Krischer J, Mehta H, Vold B, Maclaren N: [GAD.sub.65] Auto-antibodies increase the predictability but not the sensitivity of islet cell and insulin auto-antibodies for developing insulin dependent diabetes mellitus. Journal of Autoimmunity. 1994; 7:865-872.
14. American Diabetes Association. The expert committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 2003; 26: 5-16.
15. Olefsky JM, Kolterman OG, Scarlett JA: Insulin action and resistance in obesity and noninsulin-dependent type II diabetes mellitus. American Journal of Physiology, 1982. 243: 15-30.

16. Bergman RN, Alder M: Free fatty acids and pathogenesis of type 2 diabetes mellitus. Trends Endocrinology Metabolism. 2000; 11: 351.

17. American Diabetes Association: Type 2 diabetes in children and adolescent. Diabetes Care. 2000; 23:391

18. Albright A. et al: American College of sports medicine position stand: exercise and Type 2 diabetes, Medical Scientific Sports Exercise. 2000; 32: 1345.

19. Gabbe S, Graves C, Steven G, Cornelia G. Management of diabetes mellitus complicating pregnancy. Obstetrics and Gynecology. 2003; 102: 857-868.

20. American Diabetes Association. Standards of medical care for patients with diabetes mellitus. Diabetes Care. 2002; 25: 33.

21. Maizlish N, Shaw B, Hendry K. Glycemic control in diabetic patients served by community health center. American Journal of Medical Quality. 2004; 19:4

22. Grant T, Soriano Y, Marantz, Nelson I et al. Community –Based screening for cardiovascular disease and diabetes using Hgb A1c. American Journal of Preventive Medicine. 2004; 26: 271-275.

23. Edelman D, Edwards L, Olsen M, Dudley T, Stat M, Harris A, Blackwell D, Oddone E. Screening for diabetes in an outpatient clinic population. Journal of General Internal Medicine. 2002; 17: 23-28.

24. Peters A, Davidson M, Schriger D, Hasselblad V. A clinical approach for the diagnosis of diabetes mellitus: An analysis using glycosylated hemoglobin levels. Meta- analysis Research Group on the Diagnosis of Diabetes Using Glycated Hemoglobin Levels. JAMA. 1996; 276: 1246-1252.

25. Groop L, Orho-Melander M. The dysmetabolic syndrome. Journal of Internal Medicine. 2001; 250: 105-200.

26. Okosun I, Dever A. Abdominal obesity and ethnic differences in diabetes awareness, treatment, and glycemic control. Obesity Research. 2002; 10: 1241-1250.

27. Cryer PE, Fisher J, Shanon H. Hypoglycemia in diabetes. Diabetes Care. 2003; 26: 1902.

28. American Diabetes Association. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care. 2002; 25: 202.

29. Lynne D. Diabetes disease management in managed care organizations. Disease Management. 2004; 7: 47-60.

30. American Diabetes Association. Vital statistics, Diabetes 2001: Alexandria VA. American Diabetes Association, 2001.

31. American Diabetic Association. Diabetic Retinopathy. Diabetes Care. 2002; 25: 90.

32. National Diabetes Information Clearinghouse. Diabetic Neuropathies: The nerve damage of diabetes, 2002. Available at: http://diabetes.niddk.nih.gov/dm/pubs/neuropathies/index.htm. Accessed November 29, 2004.

33. Van de Laar F, Lucassen P, Tigchelaar J et al. Fat intake in patients newly diagnosed with Type 2 diabetes: a 4-year follow-up study in general practice. British Journal of General Practice. 2004; 54: 177-182.

34. Christensen N, Williams P, Pfister R. Cost savings, and clinical effectiveness of an extension service diabetes program. 2004; 17: 171.

35. Wright HS, Mitchell DC, Mickle SJ, Goldman JD, Cook A. Food commonly eaten in the U.S., 1989-1991, and 1994-1996: are portion sizes changing? Journal American Dietetic Association. 2003; 103: 41-47.

36. Warsi A, Wang P, LaValley M, Avorn J, Solomon D. Self-management education programs in chronic disease. Arch Intern Med. 2004; 164: 1641-1649.

37. Nebel IT, Klemn T, Fasshauer M et al. Comparative analysis of conventional and an adaptive computer-based hypoglycemia education programs. Patient Education and Counseling. 2004; 53: 315-318.

38. American Diabetes Association. Tests of glycemic control in diabetes. Diabetes Care. 2001; 24: 80-82.

39. Franz MJ, Bantle JM, Beebe CA, et al. American Diabetes Association position statement: evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care. 2002; 25: 148-198.

APPENDICES

APPENDIX A

TESTING INSTRUMENT

Diabetes Knowledge Assessment

Directions: Read each question and decide which choice best completes the statement or answers the question. Indicate your answer by circling the appropriate letter.

1. With type 2 diabetes, the pancreas secretes too little insulin or insulin that does not work right.

True False

2. Carbohydrate has 4 calories per gram and turns into blood sugar.

True False

3. A high fiber diet may increase your blood sugar.

True False

4. Excess calories from carbohydrate, protein, or fat can increase body fat.

True False

5. Frequent urination and excessive thirst are signs of diabetes.

True False

6. Long-term blood sugar management has little impact on diabetes complications.

True False

7. Low blood sugar is defined greater than 70 mg/dl.

True False

8. If at least half (50%) of your blood sugar (glucose) levels are in the recommended target range your A1C test should be:

a. About 6%

b. About 7%

c. About 8%

9. One carbohydrate choice contains:

a. 5 grams of carbohydrates.

b. 15 grams of carbohydrates.

c. 25 grams of carbohydrates.

10. Symptoms of low blood sugar include:

a. Feeling shaky or sweaty

b. Dry skin.

c. Feeling energetic.

11. A tennis ball is equal in size to _____ serving of fresh fruit (apple, orange, peach, etc):

a. one

b. one-half

c. 1 oz

APPENDIX B

HgbA1C AND WEIGHT CHANGES OF SUBJECT 1

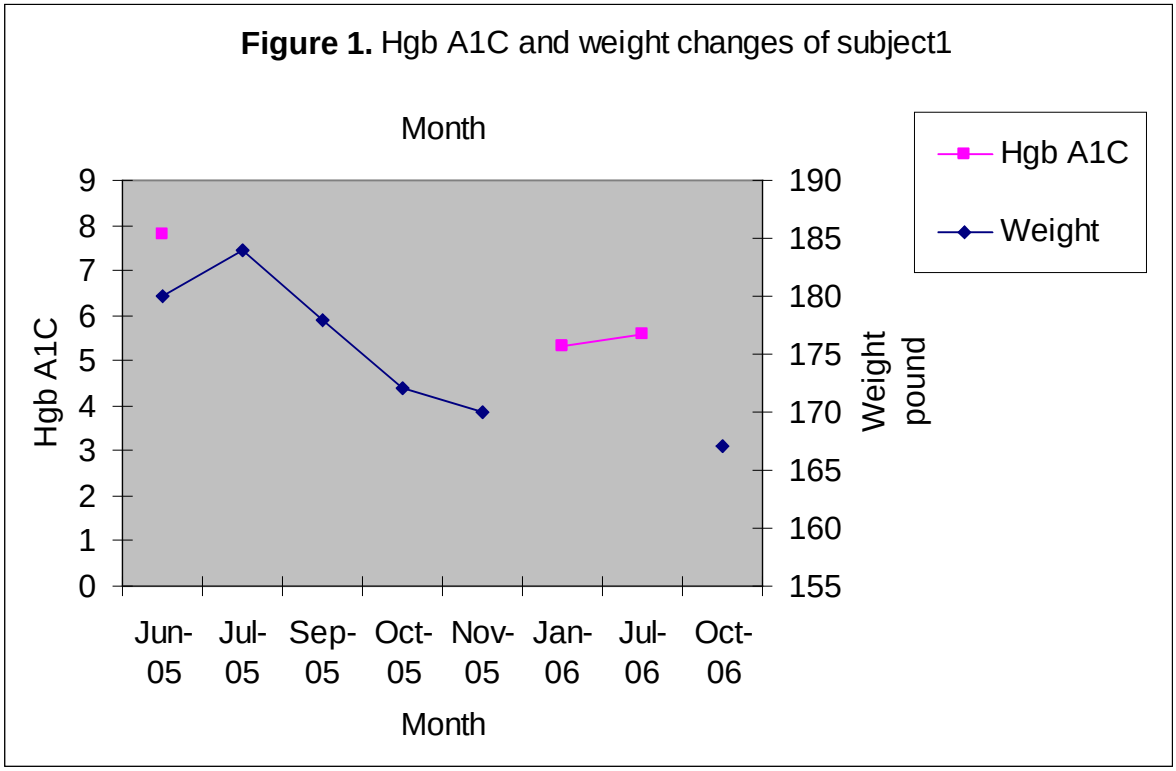
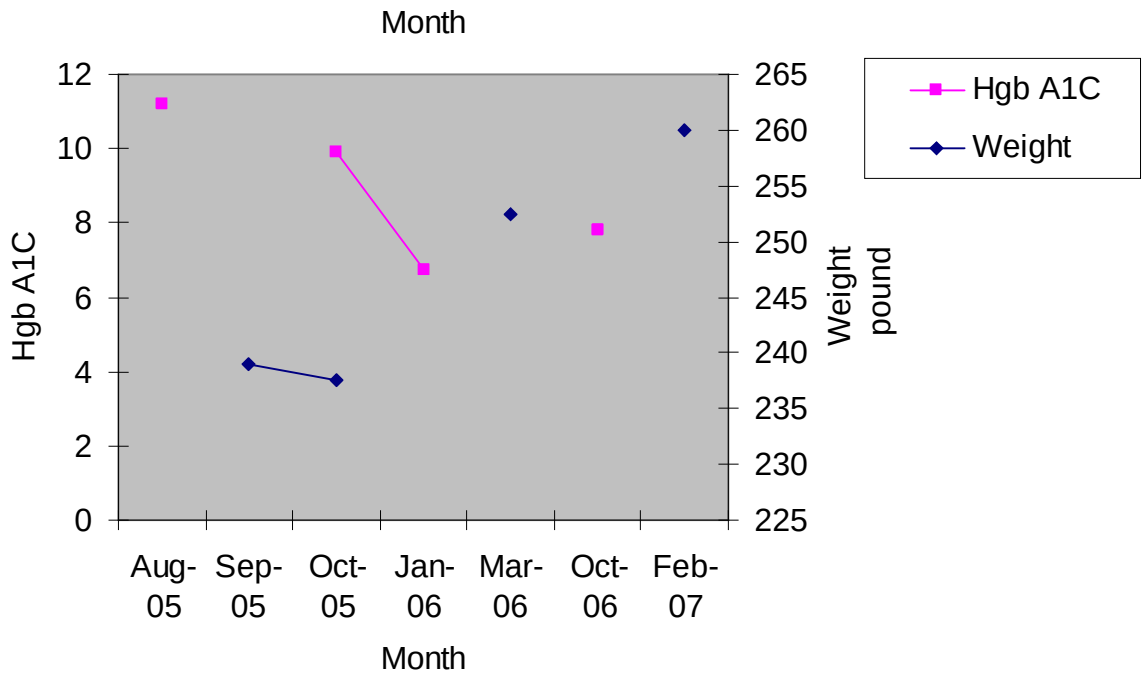


Figure 1. Hgb A1C and weight changes of subject1

APPENDIX C

HgbA1C AND WEIGHT CHANGES OF SUBJECT 2

Figure 2. HgbA1C and weight changes of subject 2



APPENDIX D

HgbA1C AND WEIGHT CHANGES OF SUBJECT 3

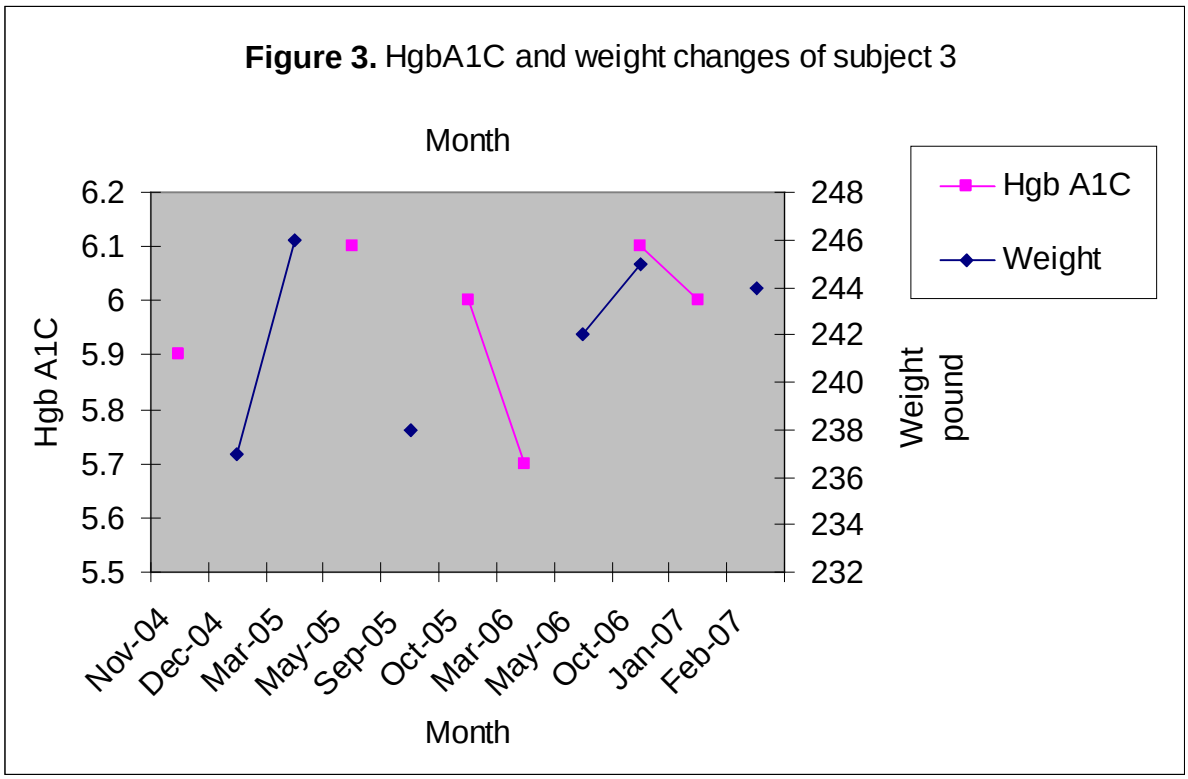
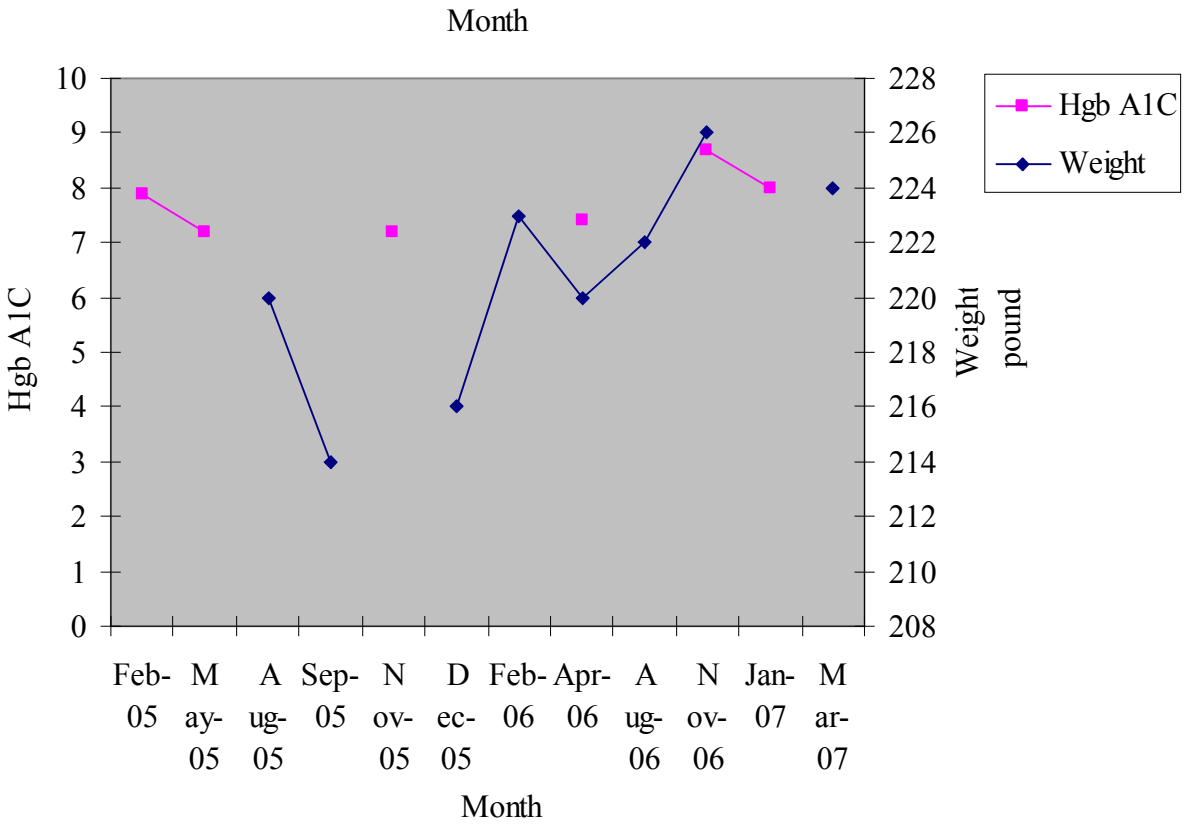


Figure 3. HgbA1C and weight changes of subject 3

APPENDIX E

HgbA1C AND WEIGHT CHANGES OF SUBJECT 4

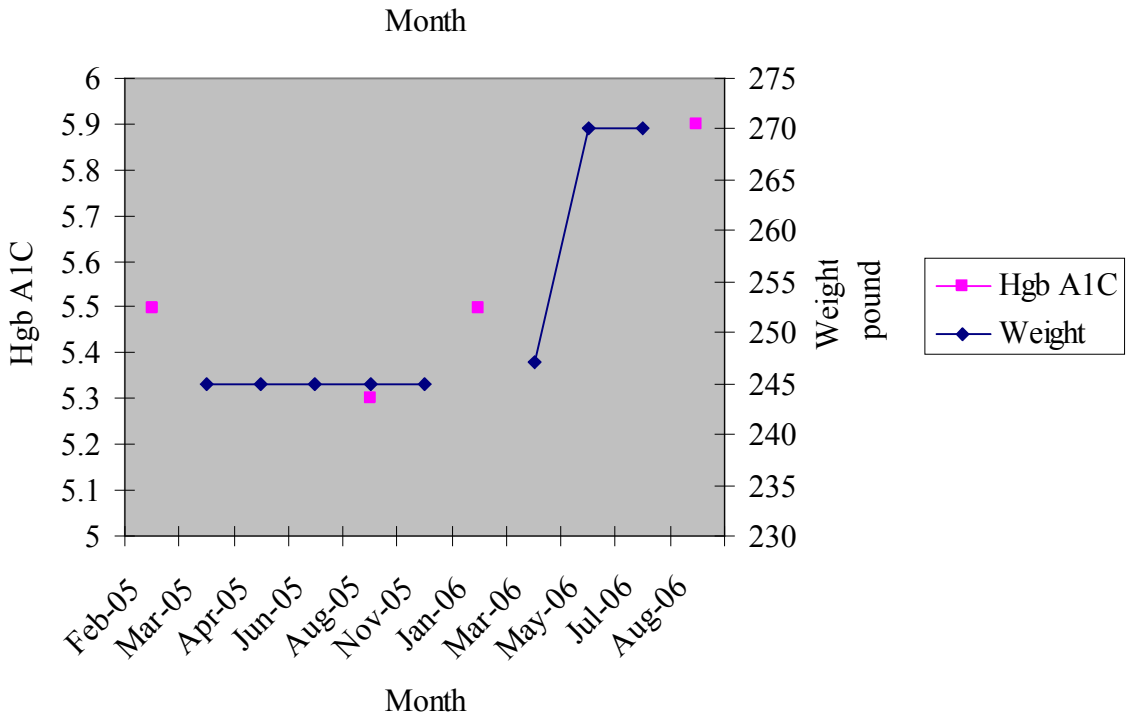
Figure 4. Hgb A1C and weight changes of subject 4



APPENDIX F

HgbA1C AND WEIGHT CHANGES OF SUBJECT 5

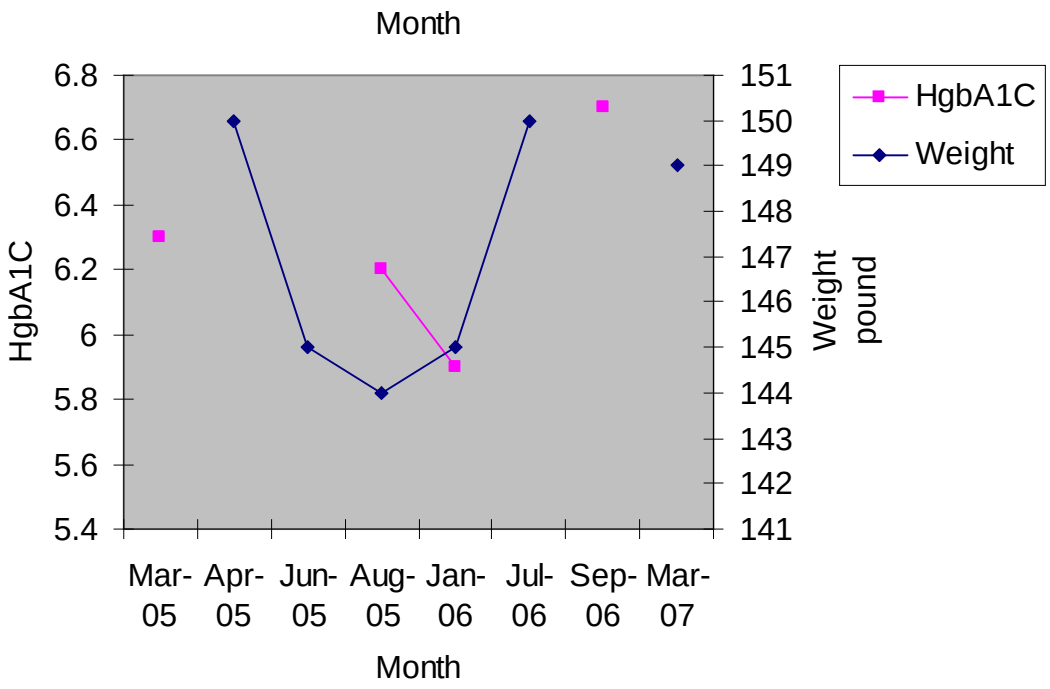
Figure 5. Hgb A1C and weight changes of subject 5



APPENDIX G

HgbA1C AND WEIGHT CHANGES OF SUBJECT 6

Figure 6. HgbA1C and weight changes of subject 6



VITA

MIREILLE HAMDAN

Personal Data: Country Of Origin: Lebanon

Date of Birth: November 12, 1980

Place of Birth: Lvov, Moscow

Professional

Experience: Clinical Dietitian, Johnson City Medical Center, Johnson City, Tennessee, 2007-current

Graduate Assistant, East Tennessee State University, Department of Family and Consumer Science, 2005-2006

Dietetic Intern, East Tennessee State University, 2005-2006

Graduate Assistant, East Tennessee State University, Department of Cross Disciplinary Studies, 2004-2005

Education: East Tennessee State University, Johnson City, Tennessee; Clinical Nutrition, M.S. 2007

Lebanese University, Lebanon; Food Science and Technology, M.S. 2003

Lebanese University, Lebanon; Agriculture Engineering, B.S. 2002

Honors: Kappa Beta Rho Chapter of Kappa Omicron Nu, National Honor Society for Family & Consumer Sciences, East Tennessee State University, 2004-2006

Gamma Beta Phi, National Honor Society, East Tennessee State University, 2004-2006