

# Effects of Educational Interventions for Children and Adolescents with Type I Diabetes Mellitus

*Thesis*

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***Pediatrics***

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## ABSTRACT

Type 1 diabetes (T1D) is one of the most frequent chronic diseases of childhood. Its management in children should include regular assessment, careful monitoring of glycemic control and educational training on disease management.

***The aim of this work:*** evaluation of the quality of the current education programme for diabetic children and their parents at Diabetes Endocrine and Metabolism Pediatric Unit (DEMPU).

***Patients and Methods:*** The present study was an observational longitudinal study conducted on 100 cases of T1D admitted in DEMPU inpatient section from period October 2011 to April 2012. Using a questionnaire that covered all aspects of the programme solved before and after attendening 5 days education sessions.

***Results:*** marked increase in diabetic knowledge after attending education programme, as 95% of interviewed parents knew how to prevent hypoglycemia at night, 97% of them knew that diabetic parent were not responsible for affection of their children with diabetes, 94% of the studied children and their parents reported that their schools were aware about the child disease and 92% of them allow their children to share in school activities and there was no significant difference between different social classes in understanding education programme. Linear regression analysis showed that the only factor which has an effect on HbA1c was total post education score.

***Conclusion:*** the efficient points of the education programme at DEMPU were identified and included knowledge about nature of T1D, role of family history in developing T1D, awareness of symptoms of hypoglycemia, how to prevent hypoglycemia during sports and at night, school awareness of the disease and sharing school activities, while the non efficient point in the education programme was defective carbohydrate counting.

**Key Words:**

**Education, Type I Diabetes, Evaluation.**

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## List of Abbreviations

<b>ACR</b>	: Albumin /Creatinine Ratio
<b>ACEI</b>	: Angiotensin Converting Enzyme Inhibitor
<b>ADA</b>	: American Diabetes Association
<b>ANOVA</b>	: Analysis of Variance between groups
<b>BG</b>	: Blood Glucose
<b>BMA</b>	: Body Mass Index
<b>BPA</b>	: Bisphenol A
<b>CHO</b>	: Carbohydrate
<b>CHD</b>	: Congenital heart disease
<b>DCCT</b>	: Diabetes Control and Complications Trial
<b>DEMPU</b>	: Diabetes, Endocrine and Metabolism ,Pediatric Unit
<b>DEXA</b>	: Dual Energy X-ray Absorptiometry
<b>DIAMOND</b>	: Diabetes Mondiale
<b>DHC</b>	: Diabetes Health Care
<b>DSME</b>	: Diabetes Self-Mangment Education
<b>DKA</b>	: Diabetic Ketoacidosis
<b>FFAs</b>	: Free Fatty Acids
<b>GAD</b>	: Glutamic Acid Decarboxylase
<b>GFR</b>	: Glomerular Filtration Rate
<b>G6PD</b>	: Glucose 6 phosphate dehydrogenase
<b>GI</b>	: Glycemic Index
<b>GL</b>	Glycemic Load
<b>HbA1c</b>	: Glycated Hemoglobin
<b>HDL</b>	: High Density Lipoprotein
<b>HLA</b>	: Human Leucocytes Antigen
<b>HRQOL</b>	: Health related quality of life

<b>HNF-4<math>\infty</math></b>	: Hepatocyte Nuclear Factor 4
<b>IAs</b>	: Insulin Auto Antibodies
<b>ICA</b>	: Islet Cell Antibodies
<b>IOM</b>	: Institute of Medicine
<b>ISPAD</b>	: International Society of Pediatric and Adolescent Diabetes
<b>IZS</b>	: Insulin Zinc Suspension
<b>LDL</b>	: Low Density Lipoprotein
<b>JAMA</b>	: Journal of the American Medical Association
<b>MCQ</b>	: Multiple Choice Question
<b>MNT</b>	: Medical Nutrition therapy
<b>MODY</b>	: Maturity onset diabetes of the youth
<b>MJA</b>	: Medical Journal of Australia
<b>NCHP</b>	National Center of Health and Population
<b>NOD</b>	: Non Obese Diabetic Mice
<b>NKS</b>	: Nutrition knowledge survey
<b>NPH</b>	: Neutral protamine Hagedome Insulin
<b>PDR</b>	: Proliferative Diabetic Retinopathy
<b>PAS</b>	: polyglandular autoimmune syndrome
<b>SDS</b>	: Standard Deviation Score
<b>SMBG</b>	: Self Monitoring Blood Glucose
<b>SPSS</b>	: Statically Package for the Social Science
<b>T1D</b>	: Type 1 diabetes
<b>TG</b>	: Triglycerides
<b>UK</b>	: United Kingdom
<b>UKPDS</b>	: United Kingdom of Pediatric Diabetes Society
<b>USA</b>	: United State of America
<b>VSD</b>	: Ventricular Septal Defect
<b>WHO</b>	: World Health Organization

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## INTRODUCTION

**D**iabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels (*ADA, 2012*).

Diabetes poses a major threat to global health, healthcare structures and national economic. Diabetes mellitus (DM) affects nearly 3.9 million individuals in Egypt; currently ranking the 10<sup>th</sup> worldwide in terms of diabetes prevalence. Egypt is expected to jump to 8<sup>th</sup> position by 2025. So in order to maintain and improve their health related quality of life, it should be a public goal (*IDF, 2010*).

Education is the keystone for diabetes care and structured diabetes self-management education (DSME) is the key to a successful outcome (*ISPAD, 2011*). The Diabetes Control and Complication Trial (DCCT) provided unequivocal evidence that intensification of management reduces microvascular complications and that intensification requires effective diabetes self-management. Most importantly, effective self-management requires frequent and high levels of educational input and continuing support (*Funnell, 2007*).

It is widely accepted that diabetes cannot be successfully managed without behavioral modification (*Northam, 2006*). Health professionals need to understand that education per se with acquisition of knowledge is unlikely to alter behavior particularly in those individuals where diabetes appears to be an overwhelming difficulty. There is, therefore, a need for training the diabetes team not only in the principles of teaching and structured education, but also in behavioral change management including counseling techniques (*ISPAD, 2009*).

## AIM OF WORK

The aim of this work is to evaluate the effectiveness of the current educational interventions for children and adolescents with type 1 diabetes applied at the Diabetes Endocrine Metabolic Pediatric Unit (DEMPU), Children's Hospital Cairo University. Specifically, it addresses the following research questions: Evaluation of the quality of education programme and defining the points of strength and weakness of this programme.

## CHAPTER I

### Type 1 Diabetes

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels (**ADA, 2012a**).

The vast majority of cases of diabetes fall into two broad etiopathogenetic categories. In one category, type 1 diabetes (T1D), the cause is an absolute deficiency of insulin secretion. Individuals at increased risk of developing this type of diabetes can often be identified by serological evidence of an autoimmune pathologic process occurring in the pancreatic islets and by genetic markers. In the other, much more prevalent category, type-2 diabetes, the cause is a combination of resistance to insulin action and an inadequate compensatory insulin secretory response table (1) (**ADA, 2004a**).

#### **Diagnosis of Diabetes in Childhood and Adolescence (*IDF, 2011*):**

- Diabetes in children usually presents with characteristic symptoms such as polyuria, polydipsia, blurring of vision, and weight loss, in association with glycosuria and ketonuria.
- In its most severe form, ketoacidosis or rarely a non-ketotic hyperosmolar state may develop and lead to stupor, coma and in absence of effective treatment, death.

- In the absence of symptoms or presence of mild symptoms of diabetes, hyperglycemia detected incidentally or under conditions of acute infective, traumatic, circulatory or other stress may be transitory and should not in itself be regarded as diagnostic of diabetes.

### **Criteria for the Diagnosis of Diabetes in Childhood and Adolescence (*ISPAD, 2011*):**

- Symptoms of diabetes plus casual plasma glucose concentration  $\geq 11.1$  mmol/l (200 mg/dl)\*. Casual is defined as any time of day without regard to time since last meal
- Fasting plasma glucose  $\geq 7.0$  mmol/l ( $\geq 126$  mg/dl) †. Fasting is defined as no caloric intake for at least 8 hours
- 2 hour post load glucose  $\geq 11.1$  mmol/l ( $\geq 200$  mg/dl) during an OGTT. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water or 1.75 g/kg of body weight to a maximum of 75 g.
- HbA1c  $\geq 6.5$ . However, there are difficulties with assay standardization and individual variation in the relationship between blood glucose and HbA1c, which may outweigh the convenience of this test.
- Prediabetes includes Impaired Glucose Tolerance (IGT) and Impaired Fasting Glycaemia (IFG)  
IGT: 2 hour post load plasma glucose 7.8-11.1 mmol/l (140-199 mg/dl)  
IFG: plasma glucose 5.6-6.9 mmol/l (100-125 mg/dl).

## **Different Types of Diabetes:**

**Table (1):** Etiologic classification of diabetes mellitus.

<b>A. Type 1-D:</b> Which is characterized by destructive lesion of pancreatic $\beta$ -cells by an autoimmune mechanism or unknown cause.	<b>B. Type II-DM:</b> This is characterized by combination of decreased insulin secretion and decreased insulin sensitivity (insulin resistance).
<b>C. Genetic defects of <math>\beta</math>-cell function</b> 1. Chromosome 12, HNF-1 $\alpha$ (MODY3) 2. Chromosome 7, glucokinase (MODY2) 3. Chromosome 20, HNF-4 $\alpha$ (MODY1) 4. Chromosome 13, insulin promoter factor- (IPF-1; MODY4) 5. Chromosome 17, HNF-1 $\beta$ (MODY5) 6. Chromosome 2, NeuroD1 (MODY6) 7. Mitochondrial DNA mutation 8. Chromosome 7, KCNJ11 (Kir6.2) 9. Others	<b>D. Genetic defects in insulin action</b> 1. Type A insulin resistance 2. Leprechaunism 3. Rabson-Mendenhall syndrome 4. Lipotrophic diabetes 5. Others
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<b>K. Gestational Diabetes.</b>	

MODY: Maturity onset diabetes of the young; HNF-4 $\alpha$ : Hepatocyte Nuclear Factor 4; NeuroD1: Neurogenic differentiation 1; KCNJ11 (Kir6.2): inward-rectifier potassium ion channel gene.

(ADA, 2012b)

The differentiation between type 1, type 2 and monogenic diabetes has important implications for both therapeutic decisions and educational approaches. Regardless of the type of diabetes, however, the child who