



Ain Shams University  
Faculty of Specific Education

Home Economics Department

# **Effect of Some Plants and Herbs on Blood Sugar in Diabetic Rats**

**BY**

**Sabah Afify Ebrahim Ahmed**

*B.Sc. Home Economics (1999)  
Home Economics Dept., Fac. Of Specific Education,  
Ain Shams University*

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Ain Shams University

**Dr. Mostafa Mohamed Said**

Prof. of Biochemistry  
National Organization for Drug Control and Research

**Dr. Eveleen Said Abdalla**

Assoc. Prof. Nutrition & Food Science  
Dept. of Home Economics  
Faculty of Specific Education  
Ain Shams University

**Dr. Safwat Hassan Ali**

Assoc. Prof. Biochemistry  
Dept. of Biochemistry  
Faculty of Agriculture  
Ain Shams University

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# **EFFECT OF SOME PLANTS AND HERBS ON BLOOD SUGAR IN DIABETIC RATS.**

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## **ABSTRACT**

The present study was aimed to investigate the prophylactic and curative effect of ginseng root and guar seeds on blood glucose and evaluate the changing in the antioxidants activity in normal and diabetic rats. 64 female albino rats divided in to 8 groups (8 animals each). Groups 1,2 were normal and diabetic, they were fed on balanced diet. Groups 3,4 were served as curative groups with ginseng root (500-mg/kg b.w) and guar seeds (10-gm/kg b.w) respectively after induction of diabetes. Groups 5,6,7,8 were treated as prophylactic groups: group 5,6 (normal and diabetic) treated with ginseng root (500-mg/kg b.w), while group 7,8 (normal and diabetic) treated with (10 gm/ kg b.w) guar seeds before and after induction of diabetes.

Prophylactic study revealed that ginseng root decreased significantly blood glucose by 52.8 % ( $P < 0.01$ ), TG 42.7 % ( $P < 0.05$ ), MDA 56.3 % ( $P < 0.01$ ) and increased L- Ascorbic Acid by 50.7 % ( $P < 0.01$ ) in diabetic rats. While, guar seeds decreased blood glucose 45.2 % ( $P < 0.05$ ), TG 50.6 % ( $P < 0.001$ ), MDA 58.8 % ( $P < 0.01$ ) and increased GSH 31.9 % ( $P < 0.01$ ), L- Ascorbic Acid 32.4 % ( $P < 0.01$ ). While, in curative study ginseng root decreased T.lipids by 37.3 % ( $P < 0.001$ ), TG 60.6 % ( $P < 0.001$ ), MDA 34.8 % ( $P < 0.05$ ) and increased GSH 30.5 % ( $P < 0.05$ ) and L-Ascorbic Acid 106.9 % ( $P < 0.01$ ). Also, Guar seeds decreased blood glucose 33.4 % ( $P < 0.01$ ), T.lipids 25.5 % ( $P < 0.01$ ), TG 41.8 % ( $P < 0.05$ ), MDA 44.9 % ( $P < 0.05$ ), and increased GSH 26.6 % ( $P < 0.05$ ) and L- Ascorbic Acid 105.2 % ( $P < 0.01$ ).

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# Abbreviations

A.C	After Christ
B.C	Before Christ
CAT	Catalase
EDTA	Ethylene Diamine Tetra Acetic Acid
GAD	Glutamic Acid Deccboxylase
GSH	Glutathione
H <sub>2</sub> O <sub>2</sub>	Hydrogen Peroxide
Hb	Haemoglobin
HDL-C	High Density Lipoprotein-Cholesterol
HLA	Human Leukocyte Antigen
IAA	Insuline Auto-Antibodies
ICA	Iselt Cell Antibodies
IDDM	Insuline Dependent Diabetes Mellitus
L-AsA	L-Ascorbic Acid
LDL-C	Low Density Lipoprotein-Cholesterol
MDA	Malondialdehyde
NIDDM	Non Insulin Dependent Diabetes Mellitus
NODCAR	National Organization for Drug Control and Research
O <sub>2</sub> <sup>•</sup>	Superoxide Anion
O <sub>2</sub>	Oxygen
<sup>1</sup> O <sub>2</sub>	Singlet Molecular Oxygen Species
ROS	Reactive Oxygen Species
STZ	Streptozotocin
TG	Triglycerides
TBA	Thiobarbituric Acid
TCA	Trichloro Acetic Acid
VLDL-C	Very Low Density Lipoprotein-Cholesterol

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# **INTRODUCTION**

Diabetes mellitus is one of the major metabolic diseases, which affects large number of people around the globe (**Nandini et al., 2003**). It is a major healthy, socioeconomic problem and may occur in children, adult and elderly people, it is a widely hereditary disease. It caused disorders of carbohydrates, fats and proteins metabolism result inability of the body to properly use and store glucose. Diabetes mellitus is a complex metabolic disease, which causes damage complications, and still with patients for all life as it considered chronic disease, the nutrition care for diabetic patients has been very important to reduce the complications (**Abdalla, 2005**). Diabetes mellitus is a metabolic disorder and all the disturbance occurred is due to insulin deficiency. There is a greater incidence of type II non-insulin dependent diabetes mellitus (**NIDDM**) than type I **insulin** dependent diabetes mellitus (**IDDM**) (**Ramachandran, 1995**).

Oxidants free radicals have been implicated in the pathogenesis of type I diabetes mellitus. In addition, diabetic patients have significant defects of antioxidants protection, and it is believes that the metabolic disorders in type I diabetes mellitus may be due to increase cellular oxidative stress and reduce antioxidants protection (**El-Missiry and El-Gindy, 2000**). Diabetes mellitus induces an increasing of oxidative stress, which is postulated to play a significant role in the development of complication. Possible sources of oxidative stress in diabetes include increase generation of reactive oxygen species by auto-oxidation of glucose, decrease tissue concentration of low-molecular weight antioxidants, and impaired activities of antioxidant enzymes (**Renu, 2003**).

Human know curative technique with natural herbs and plants for thousands of years and there are many studies, which are recommended to back to nature and use the natural herbs and plants to treat many of diseases. In many countries it is traditional

to use medicinal plants to control diabetes. The antihyperglycemic effect of several plant extracts and herbal formulation, which are used as antidiabetic remedies, has been confirmed (**Pari et al., 2001**). Historical records reveal that in traditional medical systems, a disease similar to type II diabetes was treated with plant extracts for example, the root of panax ginseng, or Asian ginseng, has been used clinically to treat type II diabetes. Result of in vitro and in vivo animals studies and clinical trails demonstrated that the root of panax ginseng and root of other ginseng species (e.g., *Panax quinquefolius*, or American ginseng) possess antihyperglucemic activity. However, most in vivo animal studies have used type I, not type II, diabetic models (**Attele, et al., 2002**).

An increasing amount of data now suggest that dried leguminous seeds as classes are among the foods that cause the smallest change in blood glucose concentration (**Abdalla, 1988**). Whether this is due to ther fairly content, to the nature of the starch they contain, or perhaps to inhibitors of carbohydrate absorption is not clear. Previous studies have been shown that guar crispbread reduces the glycaemic response to carbohydrate (**Jenkins et al., 1979**). On the other hand whole seed diet of guar in short term study reduced blood glucose in normal and well as diabetic guinea pigs (**Srivastava et al., 1987**). Also guar crispbread added to the diabetic diet in short-term studies reduced urinary glucose cutout (**Jenkins et al., 1977**).

### **The aim of the study:**

The present study was aimed to investigate the prophylactic and curative effect of root of *Panax ginseng* and guar's seeds on blood glucose level and evaluate the changing in the antioxidants activity in normal and diabetic rats.

# **Review of Literature**

## **Definition of diabetes mellitus:**

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia associated with absolute or relative deficiencies in insulin secretion or function. Lack of insulin, whether absolute or relative, affects the metabolism of carbohydrates, proteins, fats and causes a significant disturbance of water and electrolytes homeostasis (**Macloed, 1986; WHO, 1994; Bennett, 1994; Paul, 1997**). It affects more than 30 million people around the globe (**WHO, 1985**). Diabetes mellitus is usually irreversible and although patients can have a reasonably normal lifestyle, its late complications resulted in reduced life expectancy and considerable uptake of health resources (**Paul, 1997**).

## **Diabetes mellitus in history:**

The first reference on diabetes was recorded in the Papyrus Ebers in 1500 B.C, found at Luxor in Egypt at 1872 (**Chang and Diani, 1985; Abdalla, 2005**). A medical prescription on how to stop polyurea was included. The Greek term “diabetes” meant “to run through a siphon” was first introduced by Aerates of Cappadocia (81-138 A. C). During the tenth century, diabetes was thoroughly studied by the arabian physician Ibn-Sina (980-1037), he noticed the abnormal appetite, sweetness of urine, gangrene and loss of sexual function among diabetes. Cullen (1709-1790) introduced the adjective “mellitus”. Cawley (1778) was the first one associate diabetes with the pancreas. He described multiple calculi and marked destruction of pancreatic tissue at autopsy in a patient, who had died from diabetes. Hedon (1893) observed that diabetes was absent in pancreatectomized dog following transplantation of partially resected pancreas. This

finding suggested the presence of internal rather than external secretion of the pancreas. In (1869) langerhans described yellow areas of about 0.1 to 0.2 mm in the rabbit pancreas. Laguesse (1893) named them the islets of Langerhans and suggested that they were concerned with the internal pancreatic secretory function involved in carbohydrate metabolism. Moreover Banting and Best (1921) demonstrated the extensive lowering of blood and urinary sugar in pancreatectomized dogs had given pancreatic extract, they called the pancreatic hormone causing this effect of “insulin”.

Modern therapy of diabetes began with the demonstration of **Banting and Best** in 1922 that an extract of beef pancreatic could successfully lower blood glucose level in human diabetics which was considered as a marker for the first use pancreatic antidiabetic principle, insulin, in the treatment of diabetes mellitus.

### **Classification of Diabetes Mellitus:**

Diabetes mellitus is not a single entity but rather a heterogeneous group of disorders in which there are distinct genetic patterns as well as, other etiologic and pathophysiologic mechanisms that lead to impairment of glucose tolerance (**Paul, 1997**). There are different forms of diabetes primary and secondary diabetes:

#### **Primary Diabetes:**

Attributed to inability of the pancreas to produce sufficient insulin; also substances in the blood which inhibit or interfere with the action of insulin and production of an abnormal form of insulin, and so primary diabetes could be classified into two main types:

1. Type I: Insulin dependent diabetes mellitus (IDDM).
2. Type II: Non-insulin dependent diabetes mellitus (NIDDM)

## **Secondary Diabetes:**

This type is secondary to known cause. **Anderson and Geil (1994)** enumerate many causes such as:

- 1- Malnutrition related diabetes mellitus.
- 2- Pancreatic disease.
- 3- Hormonal: as growth hormone in acromegaly.
- 4- Insulin structure abnormality.
- 5- Genetic disorders.

### **1. Type I insulin dependent diabetes mellitus (IDDM):**

IDDM recently, Type I diabetes formerly was known as Juvenile-onset diabetes; Ketosis-prone diabetes. It is the most common in children and young adults (**Abdalla, 2005**). Autoimmune destruction of pancreatic  $\beta$ -cells is the most common cause of IDDM (**Bennett, 1994**), however, IDDM can be a manifestation of several different pathogenetic processes that produce marked deficits in insulin secretion and hence in its action as shown in Fig (1). The deficiency in insulin action lead to a variety of metabolic consequences, the manifestations of which are severe hyperglycemia and its related symptoms (polyuria, thirst, polyphagia, and weight loss), with poorly regulated lipolysis that lead to ketosis and ketonuria. Consequently, the necessity for insulin treatment is not only to control the hyperglycemia and its related symptoms, but also to prevent spontaneous occurrence ketoacidosis.

### **Factors for IDDM:**

In the etiology of IDDM, three factors appear to interact to produce insulin deficiency, genetic factors, immunoligical factors and environmental factors. These factors are present in different proportions and in some cases one of them predominates (**Yoon, 1990 and Myers et al., 1998**).

### **a. Genetic :**

The risk that IDDM will develop before the age of 20 years is about 6% for a patient with IDDM and 5% for a child whose father has IDDM. The overall risk for the child of a mother with IDDM is 2-3% but varies according to the age of the mother at diagnosis and at childbirth. Many studies have shown that IDDM is strongly genetically linked and associated with Human Leukocyte Antigen system (HLA) on chromosome 6 (**Undlien et al., 1997**). It is possible that a combination of HLA with other genetic factors such as the insulin gene on chromosome 11 (**Bell et al., 1984 and Bennett et al., 1995**), and the cytotoxic T-lymphocyte antigen gene on chromosome 2 may either enhance or decelerate the IDDM process. Acquired factors such as viral infection, chemical toxic and/or autoimmunity are capable of causing diabetes only in the genetically susceptible host (**Felig and Bergman, 1995**).

### **b. Immunological :**

The first clue, the involvement of an immune mechanism in the pathogenesis of diabetes came from the association between IDDM and other endocrine deficiencies of autoimmune etiology as those of the adrenal or thyroid glands (**Christy et al., 1977**). Patients with IDDM were found to have a remarkable increase in the prevalence of other organ-specific antibodies in their sera (**Maccuish et al., 1974**).

Cell-mediated autoimmunity directed against the endocrine pancreas was evidenced by **Nerup et al. (1971)**. Lymphocytes from diabetic subjects have adherence and cytotoxicity against cultured insulinoma cells. These changes in lymphocyte function are accompanied by alteration in the circulating lymphocyte population; increases in certain T-lymphocyte population have been seen in acute IDDM in both men (**Jackson et al., 1982**) and rat (**Poussier et al., 1982**).