International Journal of Biology Research
ISSN: 2455-6548
Impact Factor: RJIF 5.22
www.biologyjournal.in
Volume 3; Issue 1; January 2018; Page No. 202-207

Understanding the types and causes of diabetes mellitus

Juliana Maina Wanjiru
Murang'a University, Kenya

Abstract
Statistics show that the prevalence of diabetes among different populations around the globe has been gradually going up in the last few decades. The trend is indeed worrying, so much that the number of diabetes patients is estimated to hit 36 million by 2030. This paper aimed at providing essential information for public education on diabetes mellitus. The paper accomplished this goal by conducting a literature review of various articles on types and different causes of diabetes mellitus. These articles were sources from prominent academic databases such as EBSCO, Science Direct, CINAHL, Google Scholar, and MEDLINE. The existing literature cite two types of diabetes, including Type I diabetes and Type II diabetes. Type I diabetes is generally associated with the decrease in the amount of insulin that is being released by the pancreas while Type II diabetes is associated with insulin resistance, insulin insufficiency, and glucagon excess. Both types have different detectable abnormalities. It is imperative that people understand the condition in general and the different factors that cause it. For Type I diabetes, the causes range from autoimmune beta cell destruction to genetic susceptibility, virus infections, poor infant-feeding practices, and environmental factors. On the other hand, the causative factors for Type II diabetes include hypertension, increase in BMI (body mass index), physical inactivity, unfavorable blood lipids, dietary habits, and genetics. To increase the survival rate in the context of diabetes, knowledge of these factors for both types is crucial especially for proper management of the disease. With this knowledge, it is possible for an individual to determine the type of diabetes that they or another person has and that can be life-saving during emergencies.

Keywords: type I diabetes, type II diabetes, insulin

1. Introduction
Diabetes is a pancreas (insulin producing gland) disorder brought about by high levels of glucose in blood. The pancreas, located between and the spine and stomach plays a crucial role in digestion process. The role of pancreas in the process of digestion is evident in its releasing of insulin (a hormone) into the blood (Wannamethee *et al*., 2001; Diamond *et al*., 2003) [31, 5]. The hormone produced by the pancreas assists the blood to carry glucose to all body cells. In some situations, the insulin does not function as intended, thereby leading to abnormal glucose transfer to the body cells. As a result, glucose accumulates in the blood. With this increased stay of glucose in the blood, the blood sugar level might get too high and this results into diabetes (Diamond *et al*., 2003) [5].

With such a disease as diabetes, it is imperative that the patient learns about this illness and how to manage it. Such an undertaking is significant in ensuring that, in circumstances where diabetic patients experience complications from this disease and are not near a hospital or a medically trained professional, they can respond to such situations in an effective manner. Diamond *et al*., (2003) [5] argues that, in such a case, the knowledge the diabetic patients have on diabetes might be the only thing that will save their life. It is also vital to note that diabetes is a lifestyle illness and the routine or day-to-day undertakings of an individual with diabetes is of great concern, especially in relation to having adequate knowledge about this disease.

1.1 Research Questions
This study aimed to address the following research questions:
1. What are the various types of diabetes?
2. What factors are responsible for the occurrence of diabetes?

2. Methodology
This study focused on the review of the existing literature. This goal was accomplished by executing a search on five academic websites that host reputable, peer reviewed research articles. These academic websites were MEDLINE, EBSCO, CINAHL, Science Direct, and Google Scholar. These search engines were selected owing to their reliability for hosting dependable academic resources such as peer-reviewed articles, books, periodicals, and reports among others. The search strategy employed a mix of key words like "Patients," "Diabetic," "Insulin," "Type II diabetes," "Type I diabetes," "Mellitus," "Insipidus," and "Causes." The inclusion criteria focused on articles that relate to the topic of research and are study articles. The exclusion criteria involved articles that do not relate to the study topic and are systematic reviews. The relevant articles were then subject to a thorough review as presented in the subsequent section (Literature review section).

3. Literature Review
3.1 Types of Diabetes
According to Joanne and Morton (2010) [9]; Valdes *et al*.,

(2007) [26] Lyssenko *et al*., (2008) [12], and Stumvoll *et al*., (2005) [21], there are two types of diabetes including diabetes insipidus and diabetes melitus. Diabetes insipidus occurs when there is a production of an abundant amount of urine as a result of deficiency in the production of an antidiuretic hormone (ADH) by a part of the brain known as pituitary gland (Joanne & Morton *et al*., 2010) [9]. Diabetes mellitus results from a disorder within the pancreas, usually a failure to produce adequate amounts of insulin. As aforementioned, the hormone release by the pancreas ensures that different body cells (especially fat and muscle cells) take up sugar (in form of glucose) from the blood. More specifically, this hormone facilitates the crossing over of this glucose across the cell membranes (Lyssenko *et al*., (2008) [12].

With a reduction in pancreatic production of insulin, glucose accumulates and its level thus goes up in the blood. Glucose is often lost in the urine when it reaches higher concentration. Joanne and Morton (2010) [9] inform that regular soft drinks, cakes, and other sweets account for the majority of cases of excessive sugar in the blood. They are thus responsible for most instances of diabetes mellitus. According to Valdes *et al*., (2007) [26], Lyssenko *et al*., (2008) [12], and Stumvoll *et al*., (2005) [21], Diabetes mellitus occurs in two forms namely Type I and Type II diabetes. They are also referred to as juvenile onset diabetes or Insulin Dependent Diabetes Mellitus and adult onset diabetes or Non-Insulin Dependent Diabetes Mellitus respectively.

3.1.1 Type I Diabetes

Lyssenko *et al*., (2008) [12] assert that Type I diabetes often develops suddenly in younger people aged between 8 and 12 years, often at the onset of puberty. However, this form of diabetes mellitus can occur at any age. The role of genetics in the occurrence of this disease is evidenced by the fact that it is known to run in certain families (Valdes *et al*., 2007) [26]. Glucose in blood gets to abnormally high levels, leading to hospital admission especially after meals. In such cases, the pancreas' ability to produce insulin fails totally (Stumvoll *et al*., 2005) [21]. The survival of the patient becomes complicated in the absence of insulin, which makes this type of diabetes to be known as insulin-dependent diabetes mellitus.

Considering the seriousness of Type I diabetes, it would be imperative to comprehend what really causes it so that its onset can be prevented in future. In many instance, this illness often starts with a disorder in the body that causes the immune system to attack and destroy insulin-producing cells (Valdes *et al*., 2007; Joanne & Morton, 2010) [26, 9]. In response to this attack (and destruction), the pancreas itself initiates the production of certain proteins that stimulate an even more serious and furious attack. In the end, the organ loses its ability to produce insulin, marking the beginning of the disease's clinical stage. The need for early treatment is elevated in this case because it helps put a halt or at least manage the immune-linked destruction.

Treatment and/or management of Type I diabetes is through medical nutrition therapy where the patient is injected with insulin 2-6 times daily (Valdes *et al*., 2007) [26]. Another alternative is to use an insulin pump that dispenses insulin into the body at regular intervals with greater amounts administered immediately after meals. Complimentary dietary measures are recommended where, for instance, the patient ought to take food rich in fiber and with low fat as this helps reduce the body's insulin requirement. Others measures in this respect include taking three meals a day as well as occasional snacks well-spaced throughout the day. In the same vein, it is also crucial to observe a regulated fat and protein intake so that there are no glucose swings. With an insufficiency of glucose, there is a likelihood of hypoglycemia as insulin will act on any insulin available, however little the amount there may be. A good diet for a Type I diabetes patient should have complex carbohydrates besides polyunsaturated fiber and dietary fiber (Joanne & Morton, 2010) [9]. Such a diet should be able to supply enough oxygen that balances with the body's energy needs. Too much animal fat and glycemic index carbohydrates are also discouraged.

More often than not, the hormone imbalance resulting out of inadequacy of insulin may lead to other issues such as ketosis, which is a culmination of body fat being mobilized and released to liver cells. The condition occurs because this fat is largely concentrated in ketone bodies that are forced into urine once they increase significantly in blood (Stumvoll *et al*., 2005) [21]. This and other series of events have the potential to initiate a chain reaction that might in the end bring about dehydration, coma, an imbalance in ions, and sometimes death. This is particularly common in patients with poorly controlled/managed Type I diabetes.

3.1.2 Type II Diabetes

This is brought about by an imbalance between two very important mechanisms: insulin secretion and insulin sensitivity. Clinical studies have shown that for this disease, the earliest detectable abnormality is impairment of the body's response potential to insulin (Lyssenko *et al*., 2008; Joanne and Morton, 2010) [12, 9]. Several issues demonstrate this impairment, among them being in relation to the working of the skeletal muscle, the adipose tissues, and the liver (Stumvoll *et al*., 2005; Valdes *et al*., 2007) [21, 26]. In reaction to this impairment, the pancreas increases its production of insulin in an attempt to counter the impaired insulin action. In this respect, the level of glucose is maintained by the compensatory hyperinsulinemia within normal range. In individuals with a higher risk of developing diabetes, there is an eventual decline in the function of the beta cells which in turn impairs glucose tolerance and subsequently overt diabetes mellitus (Stumvoll *et al*., 2005) [21].

A number of risk factors have been associated with the risk of the development of this type of diabetes. Examples of this factors that have been uncovered in clinical research include hypertension, physical inactivity, poor dietary habits/patterns, smoking, family history, body mass index, and low education among others. Additionally, more recent findings indicate that genes also play a role as a risk factor for Type II diabetes (Valdes *et al*., 2007; Lyssenko *et al*., 2008; Stumvoll *et al*., 2005) [12, 26, 21].

3.2 Causes of Diabetes

3.2.1 Causes of Type I diabetes

The disease is caused by insulin insufficiency, usually a condition brought about by the destruction of pancreatic beta cells that produce insulin. The cells are attacked by the body's

immune system. This is in essence an abnormality since under normal circumstances the body is supposed to protect against infections through identification and destruction of bacteria, viruses, and other foreign substances that might harm the body. While the destruction of these cells takes place over a long period of time, the disease's symptoms develop over a short period of time. The disease is mostly common among young adults and children, hence the name juvenile diabetes.

The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) (2011) [17] explains of another kind of Type I diabetes, called latent auto immune diabetes in adults (LADA), that also develops slowly. The disease is usually diagnosed after 30 years of age. In LADA, the immune system starts to destroy the beta cells (NIDDK, 2011) [17]. Even though people with LADA can still produce insulin at the time of diagnosis, eventually they get to a position where they need an insulin pump or insulin shots to control the blood glucose levels (NIDDK, 2011) [17].

Genetic Susceptibility

Heredity can be used to determine persons likely to develop the Type I diabetes. The genes are from the biological parents to a child. These genes carry with them instructions on how proteins are made which are needed for the cells of the body to function well. Many interactions between genes are believed to influence protection from and susceptibility to Type 1 diabetes. It has been noted key genes vary from one population to another. Such variations that affect a significant percentage (more than 1percent) of the population are called gene variants (NIDDK, 2011) [17]. Gene variants associated with the risk of this disease are those linked to the production of certain proteins found on white blood cells. These proteins, called human leukocyte antigens, are responsible for the identification of any foreign bodies or materials that may be present in the body. An abnormality occurs when they wrongly recognize body cells (beta) as foreign material, leading to initiation of attack on these cells.

Autoimmune Destruction of the Beta Cells

As already discussed, the onset of Type I diabetes involves destruction of beta cells by white blood cells. The process starts long before the symptoms of the disease can be noticed and goes on even after diagnosis (Almdal *et al*., 2008) [1]. Importantly, diagnosis happens after destruction of most of these cells. After this destruction, the patient needs a dosage to be administered daily in order to survive. Current scientific research is seeking a way of halting this process and maintaining the functioning of beta cells.

Interesting, research has indicated the possibility of insulin being responsible for the aforementioned immune attacks. The attacks happen when the immune system responds to insulin the same way it would to an antigen or foreign substance. In responding to these antigens, large quantities of antigens are produced. Thus, testing for antigen quantity in individuals could help identify people who are at risk of Type I diabetes.

Environmental factors

Environmental factors also gain relevance in the current conversation for they potentially contribute to the development of the disease. Indeed, toxins, food, and viruses among others have been identified in this context although their specific role is yet to be determined and illuminated. Some theories have it that environmental factors play a significant role in triggering auto-immune beta cell destruction (Almdal *et al*., 2008) [1]. Others postulate that this role is more 'on-going' than otherwise and it continues even after diagnosis.

Viruses and infections

Diabetes cannot be caused by just a virus. People can be diagnosed with Type I diabetes after or during a viral infection and this suggests there is a link between these two. During the winter, there is an onset of Type I diabetes when the viral infections increase. Some of the viruses that have been associated with the disease include cytomegalovirus, mumps, rubella, adenovirus, and coxsackievirus B (Almdal *et al*., 2008) [1]. The manner in which these viruses affect pancreatic beta cells has been discussed extensively by many scientists who believe they have the potential to trigger an autoimmune response specifically in susceptible persons. Attempts are being made to establish which specific viruses cause Type I diabetes so that a vaccine can be developed to counter the virus, hence prevent the disease (Almdal *et al*., 2008) [1].

Infant feeding practices

It has been suggested by several studies that dietary factors have a hand as far as the risk for developing the disease is concerned. For instance, it has been shown that children who are breastfed and receive adequate vitamin D supplements are at a lower risk for developing the disease. Besides, it is believed that early exposure to cereal proteins and cow milk may increase the risk (NIDDK, 2011) [17]. There is need for more research to determine the effect that infant feeding habits have on the risk for Type I diabetes (NIDDK, 2011) [17].

3.2.2 Causes of Type II Diabetes

Body Mass Index (BMI)

It has been shown that high body mass index is a valid risk factor for this disease (Almdal *et al*., 2008) [1]. These researchers showed that in both men and women, there is a strong association that has been discovered between Type II diabetes and obesity. Mention of obesity in the current conversation is particularly important because obese people are more likely to develop insulin resistance and Type II diabetes than non-obese individuals. It is also believed that obesity results from large amounts of glycerol (released by adipose tissues) as well as hormones and non-esterified fatty acids (Wild and Byrne, 2006) [28]. Insulin resistance coupled with a beta cell dysfunction leads to a general reduction in insulin secretion. Type II diabetes thus develops as the body loses its ability to control glucose levels in blood. As to the role of genes in the development of this disease, genes interact differently with other environmental factors, sometimes leading to obesity which then plays a role as a risk factor. Wild and Byrne (2006) [28] assert that close to 90% of Type II diabetes patients have their weights at above 120% of the desirable weight. Nevertheless, it should be noted that there are contentions on the measurement of BMI in different communities. One such example is the suggestion by some researchers to have the BMI level of Asians lower than that of

the Americans. Reason for this is the difference in visceral adipose tissue and relative height between these communities.

Lipids

Researchers such as Mesinger *et al*. (2008) and Taskinen (2003) [24] have discovered that unfavorable blood lipids are a risk factor for Type II diabetes. There is documented evidence of the existence of an inverse relationship between the risk of this disease and HDL cholesterol. In women, low levels of HDL cholesterol have been found to be a significant risk factor for the disease. Research has also shown that low plasma HDL cholesterol and high levels of plasma triglyceride are constituted in a pre-diabetic state called insulin resistance syndrome (Taskinen, 2003) [24]. This suggests that HDL cholesterol levels and non-fasting triglycerides indicate the level to which the insulin is being resisted. Many studies have shown that high body mass index is associated with relatively higher cholesterol levels as well as unfavorable lipid patterns. Besides, high body mass index has also been found to be associated with high triglyceride concentrations and low concentration of HDL cholesterol. It is also believed that changes in LDP (low density lipoprotein), cholesterol, and triglycerides are associated with changes in body mass index over time.

Hypertension

In a different case control study, it has been determined that the progression of hypertension is actually an independent predictor of this disease (Tsal *et al*., 2004) [25]. There are several causes that are believed to be the reason for the association between the hypertension and Type II diabetes. A common pathophysiological pathway explaining the association between the occurrence of Type II diabetes and blood pressure is endothelial dysfunction. Based on evidence emanating from the findings of many studies, it has been inferred that a strong association exists between the onset of Type II diabetes and endothelia dysfunction (Movahed *et al*., 2010) [15].

The onset of Type II diabetes has also been found to be related to inflammation markers such as C-reactive proteins (Hu *et al*., 2004) [7]. According to Blake and colleagues (2003) [3], the proteins are also related to the increase in blood pressure. Based on these facts, it can be inferred that inflammation is another factor explaining the association between Type II diabetes (and blood pressure) occurrence and the metabolic syndrome (Ridker *et al*., 2003) [19, 3]. Finally, it is also imperative to mention that a potential link exists between Type II diabetes incidence and blood pressure.A wide body of literature also suggests the existence of a relationship between blood pressure and body mass index and the risk of developing Type II diabetes (Wild, & Byrne, 2006) [28]. However, different studies show that there is a correlation between blood pressure and BMI as an increase in BMI leads to an increase in blood pressure.

Smoking

Many studies have shown that smoking is a significant risk factor that may contribute to the development of Type II diabetes (Yeh *et al*., 2010; Willi *et al*., 2007; Nagaya *et al*., 2008) [30, 29, 16]. For instance, one meta-analysis conducted by Willi *et al*. (2007) [29] revealed that the risk of developing the disease is higher among smokers (as opposed to non-smokers). Nagaya *et al*. (2008) [16] also found out that this risk was higher in heavy smokers as compared to former smokers and light smokers. It has also been established that the risk remains quite high for non-smokers for the first three years after they quit smoking (Yeh *et al*., 2010) [30]. Smoking has negative results on these patients. It leads to the inadequate response of compensatory insulin secretion and insulin resistance. These effects could be due to components of the cigarette smoke and nicotinic effect on the beta cells. In relation to smoking, research suggests heavy smokers with increased systematic inflammation who gain significant weight after quitting smoking are at an in increased risk of developing Type II diabetes (Yeh *et al*., 2010) [30]. However, in the long run (with time), there is a reduced risk for the disease among individuals who quit smoking.

Physical inactivity

Physical inactivity has been uncovered as a potential risk factor for the development of the disease under discussion. (Almdal *et al*., 2008) [1]. in both men and women, it was determined that the prolonged watching of television increased the risk of developing diabetes. Fretts and colleagues (2009) contribute to the current conversation by presenting that vigorous and moderate physical activity significantly reduces this risk. Evidence emanating from clinical trials suggests that the onset of the disease can be prevented or delayed effectively by incorporating physical activity in one's life (Ramachandran *et al*., 2006) [18]. These physical activities were discovered to improve the insulin sensitivity of an individual leading to a reduction in the resistance of the insulin thus indirect benefits on the body mass and composition (Kay, & Fintarone, 2006) [10].

Low education

In previous studies it was discovered that there was an association between the educational level of an individual to their rate of being at risk of developing Type II diabetes (Valdes *et al*., 2009; Dasgupta *et al*., 2010) [4]. Valdes *et al*., (2009) established that a significant predator of the Type II diabetes was low educational levels (Valdes *et al*., 2009). In a research carried out by National Population Health Survey, it was discovered that people whose educational levels went up to high school diploma faced a higher risk of developing diabetes as opposed to those with degrees and other higher education. In a research executed by Dasgupta *et al*., (2010) [4], the researchers discovered that obese and least educated individuals leading inactive lifestyles were at a greater risk of developing the disease than more educated individuals leading active lifestyles. According to Valdes *et al*., (2009) and Dasgupta *et al*., (2010) [4], educational attainment plays a crucial role in reducing an individual's risk for diabetes development since such an individual is fairly informed on how to live a healthier life.

Dietary pattern

The dietary habits of an individual are important in the determination of the level of risk to develop diabetes. Sun *et al*., (2010) [12] inform of a positive association between various

dietary patterns and the risk of diabetes. For instance, in a study executed by Villegas *et al.* (2007) [27], the researchers established that there exist a higher dietary glycemic patterns positively associate with an elevated risk of diabetes development as has been evidenced in several cohort studies.

Genetics

According to Amini & Janghobarn (2007) [2], the genetic components of a person actually take part in the pathogenesis process of Type II diabetes. In a different study executed by Ma *et al*., (2008) [13], the researchers concluded that if there is a positive history in the first degree relatives, then the individual stands at risk of developing Type II diabetes. This risk even goes higher if both the parents are Type II diabetics (Ma *et al*., 2008) [13]. In a study that was conducted on twins helped conclude that in monozygotic, the concordance estimate is higher for the Type II diabetes as compared to dizygotic (Diamond, 2003) [5]. Diabetes prevalence varies depending with the ethnic group that a person belongs to (Diamond, 2003) [5]. According to Swapan *et al*., (2006) [23], the prevalence of diabetes is mainly affected by the environment that these ethnic groups live under and supports the notion that the genetic factors in our bodies actually contribute to disease predisposition.

Information from several laboratories is in support of the notion that genetic factors play a certain role in the development of Type II diabetes. For instance, Swapan *et al*., (2006) [23] assert that the establishment of Type II diabetes can be achieved by the reduction of insulin sensitivity and secretion. In most cases of Type II diabetes, this deterioration is usually in parallel. According to Lyssenko *et al*., (2008) [12], there are variants in 11 genes (NOTCH2, TCF7L2, PPARG, IGF2BP2, HHEX, SLC30A8, CDKAL1, FTO, JAZF1, KCNJ11 and WFS1) which are associated closely with the risk of developing Type II diabetes in the presence of other clinical factors. Lyssenko *et al*., 2008) [12] add that variants in a majority of these genes were discovered to be directly linked to the impairment of beta-cell function.

4. Findings

This study aimed to investigate the types and causes of diabetes. Considering the findings of the existing literature, it can be noted that there are two types of diabetes including diabetes mellitus and diabetes insipidus (Joanne and Morton, 2010; Valdes *et al*., 2007; Lyssenko *et al*., 2008; Stumvoll *et al*., 2005) [9, 12, 26, 21]. As argued by Valdes *et al*., 2007 [26]; Lyssenko *et al*., 2008 [12]; Stumvoll *et al*., (2005) [21], diabetes mellitus occurs in two form including Type I diabetes and Type II diabetes. Type I diabetes is generally associated with the decrease in the amount of insulin that is being released by the pancreas while Type II diabetes is associated with insulin resistance, insulin insufficiency, and glucagon excess (Valdes *et al*., 2007; Lyssenko *et al*., 2008; Stumvoll *et al*., 2005) [12, 26, 21]. Both forms of diabetes have different detectable abnormalities. It is significant that people understand the condition in general and the different factors that cause it. For Type I diabetes, the causes range from autoimmune beta cell destruction to genetic susceptibility, virus infections, poor infant-feeding practices, and environmental factors (Almdal *et al*., 2008; NIDDK, 2011) [1, 17]. On the other hand, the causative factors for Type II diabetes include hypertension, increase in BMI (body mass index), physical inactivity, unfavorable blood lipids, dietary habits, and genetics (Almdal *et al*., 2008 [1]; Mesinger *et al*., 2008; Taskinen, 2003 [24]; Tsal *et al*., 2004 [25]; Movahed *et al*., 2010 [15]; Hu *et al*., 2004 [7]; Blake *et al*., 2003 [3]; Ridker *et al*., 2003 [19]; Wild, & Byrne, 2006 [28]; Willi *et al*., 2007 [29]; Nagaya *et al*., 2008 [16]; Yeh *et al*., 2010 [30]; Fretts *et al*., 2009 [6]; Ramachandran *et al*., 2006 [18]; Kay, & Fintarone, 2006 [10]; Valdes *et al*., 2009; Dasgupta *et al*., 2010 [4]; Sun *et al*., 2010 [22]; Amini & Janghobarn, 2007 [2]; Ma *et al*., 2008 [13]; Diamond, 2003 [5]; Swapan *et al*., 2006 [23]; Lyssenko *et al*., 2008) [12]. Diabetes is diagnosed based on the concentration and/or level of glucose in the blood. This disease occurs when the pancreas fails to produce adequate insulin as may be required by the body. To handle a diabetic patient in an emergency situation, one would need to have the knowledge of what type of diabetes they are suffering from. The knowledge of the disease's causative factors is of much importance in the prevention and even management of diabetes.

5. Conclusion

The outcomes of the existing literature provide adequate information about diabetes, which can be employed in handling diabetic individuals in emergency circumstances. Diabetes occurs in two forms including diabetes mellitus and diabetes insipidus. Diabetes mellitus can be Type I or Type II, and these are causes by various factors. Type I diabetes result from. The diagnosis of diabetes is founded on blood glucose concentration. In general, Type I diabetes is associated with a reduction in the quantity of insulin being produced by the pancreas, and is caused by factors related to genetic susceptibility, autoimmune destruction of the beta cells, environment, viruses and infections, and infant feeding practices. Type II diabetes is arises due to glucagon excess, insulin insufficiency, and insulin resistance, and its causative agents are associated with Body Mass Index (BMI), lipids, hypertension, smoking, physical inactivity, low education, dietary pattern, and genetics. Having adequate knowledge about diabetes contributes significantly to the reduction of the rates of mortality arising from this illness. A patient is not able to manage what he/she does not understand. With this understanding, a firm and strategic foundation is formed that will be a springboard for better health for a nation. The next strategic step after this study would be to learn how to manage diabetes.

6. References

1. Almdal T, Scharling H, Jensen JS, Vestergaard H. Higher prevalence of risk factors for type 2 diabetes mellitus and subsequent higher incidence in men. Eur. J Intern. Med. 2008; 19(1):40-5.
2. Amini M, Janghorban M. Diabetes and impaired glucose regulation in first degree relatives of patients with type 2 diabetes in Isfahan, Iran: Prevalence and risk factors. Rev. Diabet. Stud. 2007; 4:169-176.
3. Blake GJ, Rifai N, Buring JE, Ridker PM. Blood pressure, C-reactive protein, and risk of future cardiovascular events. *Circulation.* 2003; 108(24):2993-9.
4. Dasgupta K, Khan S, Ross NA. Type 2 diabetes in

Canada: concentration of risk among most disadvantaged men but inverse social gradient across groups in women. Diabetic Medicine. 2010; 27(5):522-31.
5. Diamond J. The double puzzle of diabetes. Nature. 2003; 423(6940):599-602.
6. Fretts AM, Howard BV, Kriska AM, Smith NL, Lumley T, Lee E. Physical activity and incident diabetes in American Indians: the Strong Heart Study. A. J Epidem. 2009; 170(5):632-9.
7. Hu FB, Meigs JB, Li TY, Rifai N, Manson JE. Inflammatory markers and risk of developing type 2 diabetes in women. *Diabetes.* 2004; 53(3):693-700.
8. Jacobsen BK, Bonaa KH, Njolstad I. Cardiovascular risk factors, change in risk factors over 7 years, and the risk of clinical diabetes mellitus type 2. The Tromso study. J Clin. Epidem. 2002; 55(7):647-53.
9. Joanne FG, Morton JF. Food Sources of Added Sweeteners in the Diets of Americans. J Amer. Die. Ass. 2010; 5(4):39-46.
10. Kay SJ, Fiatarone MA. The influence of physical activity on abdominal fat: a systematic review of the literature. Obes. Rev. 2006; 7(2):183-200.
11. Krishnan S, Rosenberg L, Palmer JR. Physical activity and television watching in relation to risk of type 2 diabetes: the Black Women's Health Study. Am. J Epidem. 2009; 169(4):428-34.
12. Lyssenko V, Jonsson A, Almgren P, Pulizzi N, Isomaa B, Tuomi T. Clinical risk factors, DNA variants, and the development of type 2 diabetes. N. Engl. J Med. 2008; 359(21):2220-32.
13. Ma XJ, Jia WP, Hu C, Zhou J, Lu HJ, Zhang R. Genetic characteristics of familial type 2 diabetes pedigrees: a preliminary analysis of 4468 persons from 715 pedigrees. CMJ. 2008; 8(36):2541-3.
14. Meisinger C, Thorand B, Schneider A, Stieber J, Doring A, Lowel H. Sex differences in risk factors for incident type 2 diabetes mellitus: The Monica Augsburg cohort study. Arch. Intern. Med. 2002; 162(1):82-9.
15. Movahed MR, Sattur S, Hashemzadeh M. Independent association between type 2 diabetes mellitus and hypertension over a period of 10 years in a large inpatient population. Clin. Exp. Hypertens. 2010; 32(3):198-201.
16. Nagaya T, Yoshida H, Takahashi H, Kawai M. Heavy smoking raises risk for type 2 diabetes mellitus in obese men; but light smoking reduces the risk in lean men: a follow-up study in Japan. Ann. Epidem. 2008; 18(2):113-8.
17. National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) Causes of Diabetes. Available at www.diabetes.niddk.nih.gov. NIH Publication, 2011, 11-5164.
18. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). Diabetologia. 2006; 49(2):289-97.
19. Ridker PM, Buring JE, Cook NR, Rifai N. C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events: an 8-year follow-up of 14 719 initially healthy American women. Circulation. 2003; 107(3):391-7.
20. Ross TA, Boucher JL, O'Connell BS. American Dietetic Association guide to diabetes medical nutrition therapy and education. United States of America: American Dietetic Association, 2005.
21. Stumvoll M, Goldstein BJ, van Haeften TW. Type 2 Diabetes: Principles of Pathogenesis and Therapy. Lancet. 2005; 365(9467):1333-46.
22. Sun Q, Spiegelman D, van Dam RM, Holmes MD, Malik VS, Willett WC. White rice, brown rice, and risk of type 2 diabetes in US men and women. Arch. Intern. Med. 2010; 170(11):961-9.
23. Swapan KD, Elbein SC. The genetic basis of type 2 diabetes. Cell Science. 2006; 2:100-31.
24. Taskinen MR. Diabetic dyslipidaemia: from basic research to clinical practice. Diabetologia. 2003; 46(6):733-49.
25. Tsai WL, Yang CY, Lin SF, Fang FM. Impact of obesity on medical problems and quality of life in Taiwan. Am. J Epidem. 2004; 160(6):557-65.
26. Valdes S, Botas P, Delgado E, Alvarez F, Cadorniga FD. Population-based incidence of type 2 diabetes in northern Spain: the Asturias Study. Diabetes Care. 2007; 30(9):2258-63.
27. Villegas R, Liu S, Gao YT, Yang G, Li H, *et al*. Prospective study of dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Arch. Intern. Med. 2007; 167(21):2310-6.
28. Wild SH, Byrne CD. ABC of obesity. Risk factors for diabetes and coronary heart disease. BMJ. 2006; 333(7576):1009-11.
29. Willi C, Bodenmann P, Ghali WA, Faris PD, Cornuz J. Active smoking and the risk of type 2 diabetes: a systematic review and met-analysis. JAMA. 2007; 298(22):2654-64.
30. Yeh HC, Duncan BB, Schmidt M, Wang NY, Brancati FL. Smoking, smoking cessation, and risk for type 2 diabetes mellitus. Ann. Intern. Med. 2010; 152(1):10-7.
31. Wannamethee SG, Shaper AG, Perry IJ. Smoking as a modifiable risk factor for type 2 diabetes in middle-aged men. Diabetes Care. 2001; 24(9):1590-5.