# My Genetic Predisposition to Diabetes

## Introduction

Genetic predisposition refers to the likelihood of a person developing a particular disease due to that person’s genetic makeup. This is as a result of variations that are often inherited from a parent. Variations in genetic increases the likelihood of disease development but do not cause the disease directly thus certain individuals with predisposing genetic changes will get the disease while others will not even within the same family (Anjos, 2004). In this study am going to discuss the genetic predisposition to diabetes. This disorder has been chosen because of the serious adverse health effects that it has on patients. Diabetes causes abnormal metabolism of glucose that could result into various complications in the long term including: peripheral, cardiovascular, ocular, renal, vascular and neurologic abnormalities (Hansen, 2003). It is therefore important to note that diabetes is a source of psychological, social and financial burden amongst populations across the world.

Diabetes mellitus is heterogeneous disorder that is evident by persistent hyperglycemia and caused by a combination of genetic and environmental risk factors. Certain types of diabetes are inherited directly such as diabetes caused by mutations in the mitochondrial DNA maturity onset diabetes in the young. Present studies suggest that genetics play a significant role in the development of both type 1 and type 2 diabetes. According to Anjos (2004), first degree relatives are six times riskier of developing type 1 diabetes than unrelated individuals. Present studies have identified 20 regions of the genome that could be involved in the genetic susceptibility to type 1 diabetes.

Type 1 diabetes (T1D) is caused as a result of autoimmune destruction of the beta cells of the pancreas. Present studies have suggested that the HLA region of chromosome 6 contains various genes that are believed to be responsible for immune response. They include DQ, HLA-DR and DP. The HLA class II genes (IDDMI) is strongly associated with type 1 diabetes contributing to approximately 40-50% of the hereditary risk for type 1 diabetes (Anjos, 2004). However, present studies have indicated that genes in extended class 1 and central class 1 may also increase the risks of type 1 diabetes. Mutation in these classes of genes results into beta cells within the pancreas being destroyed thus stop producing insulin. Lack of secretion of insulin results into poor glycemic control and high blood sugar in the body. In type 2 diabetes, candidate genes have been studied and are believed to be involved in the pancreatic beta cell function, glucose metabolism, action of insulin or other metabolism that increases the risk of the disease.

Common candidate genes for type 2 diabetes include ABCC8, CALPN10, PPARγ and KCNJ1. Peroxisome proliferator-activated receptor-γ (PPARγ) is important in the metabolism of lipid and adipocyte and research suggest that one form of the gene reduces the sensitivity of insulin thus increases the risk of type 2 diabetes (Hansen, 2003). The ABCC8 *and* KCNJ1 plays an important role in ATP-sensitive potassium channel, an important physiological processes in the regulating and release of hormones such as glucagon and insulin within the beta cell. Mutation processes in these genes interfere with the activities of potassium channel hence affect insulin secretion and eventually leads to the development of type 2 diabetes. As stated by Hansen (2003), current treatments for type 2 diabetes target the KCNJ1*,* PPARγ and ABCC8*.* This results into pharmacogenetic implications for maintaining better control of blood sugar. This implies that genetic testing is very crucial not only in determining the risk for developing type 2 diabetes but also for guiding its treatment regimes.

Management of diabetes is important since there is no known cure for diabetes. The most important thing in management of diabetes is by addressing the first barrier. While different gene-environment interact in different populations, the risk of type 2 diabetes continue to increase. It is therefore important for more epidemiological research to be done in the future so that specific risks associated with particular genetic variants are identified. As stated by Hansen (2003), when it comes to the genetic testing for any of the genetic predisposed diabetes, customized model should be applied since there are variations in gene-environment interactions. In this respect, a medical history would also be important in understanding the risk factors especially for children who inherit these genes. Family history provides information that is important in understanding the probability or chances of a child inheriting certain genes that are either associated with typ1 or type 2 diabetes. Creating a medical tree when gathering family history is vital in evaluating predisposition to the disease. The challenge of a medical tree is that parents must be tested so that their genetic traits may be identified. Gathering information about genetics of parents would be very difficult especially when the parents are deceased.

In conclusion, Scientists have linked various gene mutations to the risk of developing both type 1 and type 2 diabetes. However, it is important to acknowledge that not everybody who carries the gene mutation will get the disease. What scientists have discovered is that gene mutation predisposes individuals to high risk of diabetes since such mutations interact with the environment. Risk of diabetes is increased when these genes interact with the environment. Mutation in genes that regulate insulin production and how glucose is produced increases the risk of diabetes. They include; CAPN10, KCNJ1*,* PPARγ and ABCC8, TCF7L2, GCGR among others*.* Knowing ones risk of type 2 diabetes is therefore important in initiating earlier prevention measures including genetic testing.

## References

1. Anjos, S., Polychronakos, C. (2004). Mechanisms of genetic susceptibility to type 1 diabetes: beyond HLA*. Mol Genet Metab, Vol. 81* (187-195).
2. Hansen, L. (2003). Candidate genes and late-onset type 2 diabetes mellitus. Susceptibility genes or common polymorphisms? *Dan Med Bull* Vol. 50 (320-346).