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## Etiology and Pathophysiology of Diabetes Mellitus

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### ABSTRACT

Diabetes mellitus is taken from the Greek word diabetes, meaning siphon - to pass through and the Latin word mellitus meaning sweet. A review of the history shows that the term "diabetes" was first used by Apollonius of Memphis around 250 to 300 BC. Ancient Greek, Indian, and Egyptian civilizations discovered the sweet nature of urine in this condition, and hence the propagation of the word Diabetes Mellitus came into being.

### KEYWORDS

metabolic disease, endocrinopathies, prolonged hyperglycemia, human leukocyte antigen (HLA).

### INTRODUCTION

Diabetes mellitus (DM) is a metabolic disease, involving inappropriately elevated blood glucose levels. DM has several categories, including type 1, type 2, maturity-onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary causes due to endocrinopathies, steroid use, etc. The main subtypes of DM are Type 1 diabetes mellitus (T1DM) and Type 2 diabetes mellitus (T2DM), which classically result from defective insulin secretion (T1DM) and/or action (T2DM). T1DM presents in children or adolescents, while T2DM is thought to affect middle-aged and older adults who have prolonged hyperglycemia due to poor lifestyle and dietary choices. The pathogenesis for T1DM and T2DM is drastically different, and therefore each type has various etiologies, presentations, and treatments.

In the islets of Langerhans in the pancreas, there are two main subclasses of endocrine cells: insulin-producing beta cells and glucagon secreting alpha cells. Beta and alpha cells are continually changing their levels of hormone secretions based on the glucose environment. Without the balance between insulin and glucagon, the glucose levels become inappropriately skewed. In the case of DM, insulin is either absent and/or has impaired action (insulin resistance), and thus leads to hyperglycemia.

T1DM is characterized by the destruction of beta cells in the pancreas, typically secondary to an autoimmune process. The result is the absolute destruction of beta cells, and consequentially, insulin is absent or extremely low. T2DM involves a more insidious onset where an imbalance between insulin levels and insulin sensitivity causes a functional deficit of insulin. Insulin

resistance is multifactorial but commonly develops from obesity and aging.

The genetic background for both types is critical as a risk factor. As the human genome gets further explored, there are different loci found that confer risk for DM. Polymorphisms have been known to influence the risk for T1DM, including major histocompatibility complex (MHC) and human leukocyte antigen (HLA). T2DM involves a more complex interplay between genetics and lifestyle. There is clear evidence suggesting that T2DM is has a stronger hereditary profile as compared to T1DM. The majority of patients with the disease have at least one parent with T2DM. Monozygotic twins with one affected twin have a 90% likelihood of the other twin developing T2DM in his/her lifetime. Approximately 50 polymorphisms to date have been described to contribute to the risk or protection for T2DM. These genes encode for proteins involved in various pathways leading to DM, including pancreatic development, insulin synthesis, secretion, and development, amyloid deposition in beta cells, insulin resistance, and impaired gluconeogenesis regulation. A genome-wide association study (GWAS) found genetic loci for transcription factor 7-like 2 gene (TCF7L2), which increases the risk for T2DM. Other loci that have implications in the development of T2DM include NOTCH2, JAZF1, KCNQ1, and WFS1. MODY is a heterogeneous disorder identified by non-insulin-dependent diabetes diagnosed at a young age (usually under 25 years). It carries an autosomal dominant transmission and does not involve autoantibodies as in T1DM. Several genes have implications in this disease, including mutations to hepatocyte nuclear factor-1-alpha (HNF1A) and the glucokinase (GCK) gene, which occurs in 52 to 65 and 15 to 32 percent of MODY cases, respectively. The genetics of this disease are still unclear as some patients have mutations but never develop the disease, and others will develop clinical symptoms of MODY but have no identifiable mutation.

Gestational diabetes is essentially diabetes that manifests during pregnancy. It is still unknown why it develops; however, some speculate that HLA antigens may play a role, specifically HLA DR2, 3, and 4. Excessive proinsulin is also thought to play a role in gestational diabetes, and some suggest that proinsulin may induce beta-cell stress. Others believe that high concentrations of hormones such as progesterone, cortisol, prolactin, human placental lactogen, and estrogen may affect beta-cell function and peripheral insulin sensitivity. Several endocrinopathies, including acromegaly, Cushing syndrome, glucagonoma, hyperthyroidism, hyperaldosteronism, and somatostatinomas, have been associated with glucose intolerance and diabetes mellitus, due to the inherent glucogenic action of the endogenous hormones excessively secreted in these conditions. Conditions like idiopathic hemochromatosis are associated with diabetes mellitus due to excessive iron deposition in the pancreas and the destruction of the beta cells.

A patient with DM has the potential for hyperglycemia. The pathology of DM can be unclear since several factors can often contribute to the disease. Hyperglycemia alone can impair pancreatic beta-cell function and contributes to impaired insulin secretion. Consequentially, there is a vicious cycle of hyperglycemia leading to an impaired metabolic state. Blood glucose levels above 180 mg/dL are often considered hyperglycemic in this context, though because of the variety of mechanisms, there is no clear cutoff point. Patients experience osmotic diuresis due to saturation of the glucose transporters in the nephron at higher blood glucose levels. Although the effect is variable, serum glucose levels above 250 mg/dL are likely to cause symptoms of polyuria and polydipsia. Insulin resistance is attributable to excess fatty acids and proinflammatory cytokines, which leads to impaired glucose transport and increases fat breakdown. Since there is an inadequate response or production of insulin, the body responds by inappropriately increasing glucagon, thus further contributing to hyperglycemia. While insulin resistance is a component of T2DM, the full extent of the disease results when the patient has inadequate production of insulin

to compensate for their insulin resistance. Chronic hyperglycemia also causes nonenzymatic glycation of proteins and lipids. The extent of this is measurable via the glycation hemoglobin (HbA1c) test. Glycation leads to damage in small blood vessels in the retina, kidney, and peripheral nerves. Higher glucose levels hasten the process. This damage leads to the classic diabetic complications of diabetic retinopathy, nephropathy, and neuropathy and the preventable outcomes of blindness, dialysis, and amputation, respectively.

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