PATHOGENESIS AND TREATMENT OF DIABETES MELLITUS pdf

1: Pathogenesis of type 1 and type 2 diabetes mellitus.

This perspective examines aspects of the pathogenesis of type 2 diabetes and its treatment, considering also future needs if we are going to reverse what is clearly the most damaging consequence of obesity.

Advanced Search SIXTEEN million individuals in the United States with type 2 diabetes mellitus and an additional 30â€”40 million with impaired glucose tolerance result in health care costs exceeding billion dollars annually 1. Treatment is predominantly directed at microvascular and macrovascular complications 2. In type 1 diabetes mellitus the relationship between glycemic control and microvascular complications has been well established 3. The relationship between tight glycemic control and microvascular disease in type 2 diabetes mellitus appears to be established in the recently completed United Kingdom prospective diabetes study 4 , 5. Despite the morbidity and mortality associated with retinopathy, nephropathy, and neuropathy, cardiovascular disease remains the leading cause of death in type 2 diabetes mellitus 6 , 7. Consequently, the treatment of confounding risk factors of obesity, hypertension, and hyperlipidemia assumes major importance and must be coordinated with good glycemic control for reduction in total mortality in type 2 diabetes mellitus 6 â€” Based on the emerging relationship between the degree of glycemic control and microvascular complications as well as the contribution of hyperglycemia in the development of macrovascular disease, it is the purpose of this review to summarize the current state of knowledge to provide a rational basis for the treatment of type 2 diabetes mellitus. Classification of type 2 diabetes mellitus The definition of type 2 diabetes mellitus, previously termed noninsulin-dependent diabetes mellitus, was recently modified by the American Diabetes Association. Several criteria may be used independently to establish the diagnosis: Fasting glucose values are preferred for their convenience, reproducibility, and correlation with increased risk of microvascular complications. Individuals with impaired fasting glucose and IGT are considered to be at high risk for the development of diabetes and macrovascular disease 13 , Although one third of these patients will eventually develop diabetes, dietary modification and exercise can lower the risk of progression from impaired glucose tolerance to type 2 diabetes; and may also prevent the development of IGT in nondiabetic individuals at high risk Pharmacological agents may also be of benefit in limiting the progression from IGT to diabetes 13 , Pathophysiology of type 2 diabetes mellitus Type 2 diabetes mellitus is a heterogeneous disorder with varying prevalence among different ethnic groups. In the United States the populations most affected are native Americans, particularly in the desert Southwest, Hispanic-Americans, and Asian-Americans 1. The primary events are believed to be an initial deficit in insulin secretion and, in many patients, relative insulin deficiency in association with peripheral insulin resistance 16 , Initiation of the insulin response depends upon the transmembranous transport of glucose and coupling of glucose to the glucose sensor. The induction of glucokinase serves as the first step in linking intermediary metabolism with the insulin secretory apparatus. This defect is improved by the sulfonylureas 21 , Later in the course of the disease, the second phase release of newly synthesized insulin is impaired, an effect that can be reversed, in part at least in some patients, by restoring strict control of glycemia. An impairment in first phase insulin secretion may serve as a marker of risk for type 2 diabetes mellitus in family members of individuals with type 2 diabetes mellitus 26 â€” 30 and may be seen in patients with prior gestational diabetes However, impaired first phase insulin secretion alone will not cause impaired glucose tolerance.

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2: # Diabetes Mellitus Pathophysiology # Food Recipes For Diabetics

Abstract. Type 1 diabetes mellitus (T1DM), also known as insulin-dependent diabetes, is a chronic disease caused by autoimmune (type 1a) or spontaneous (type 1b) destruction of pancreatic beta cells, resulting in insulin deficiency.

References If insulin production and secretion are altered by disease, blood glucose dynamics will also change. If insulin production is decreased, glucose entry into cells will be inhibited, resulting in hyperglycemia. The same effect will be seen if insulin is secreted from the pancreas but is not used properly by target cells. If insulin secretion is increased, blood glucose levels may become very low hypoglycemia as large amounts of glucose enter tissue cells and little remains in the bloodstream. Following meals, the amount of glucose available from carbohydrate breakdown often exceeds the cellular need for glucose. Excess glucose is stored in the liver in the form of glycogen, which serves as a ready reservoir for future use. When energy is required, glycogen stores in the liver are converted into glucose via glycogenolysis, elevating blood glucose levels and providing the needed cellular energy source. The liver also produces glucose from fat fatty acids and proteins amino acids through the process of gluconeogenesis. Glycogenolysis and gluconeogenesis both serve to increase blood glucose levels. Thus, glycemia is controlled by a complex interaction between the gastrointestinal tract, the pancreas, and the liver. Multiple hormones may affect glycemia. Insulin is the only hormone that lowers blood glucose levels. The counter-regulatory hormones such as glucagon, catecholamines, growth hormone, thyroid hormone, and glucocorticoids all act to increase blood glucose levels, in addition to their other effects. Type 1 Diabetes The underlying pathophysiologic defect in type 1 diabetes is an autoimmune destruction of pancreatic beta cells. Following this destruction, the individual has an absolute insulin deficiency and no longer produces insulin. Autoimmune beta cell destruction is thought to be triggered by an environmental event, such as a viral infection. Genetically determined susceptibility factors increase the risk of such autoimmune phenomena. The onset of type 1 diabetes is usually abrupt. It generally occurs before the age of 30 years, but may be diagnosed at any age. Most type 1 diabetic individuals are of normal weight or are thin in stature. Since the pancreas no longer produces insulin, a type 1 diabetes patient is absolutely dependent on exogenously administered insulin for survival. People with type 1 diabetes are highly susceptible to diabetic ketoacidosis. Because the pancreas produces no insulin, glucose cannot enter cells and remains in the bloodstream. To meet cellular energy needs, fat is broken down through lipolysis, releasing glycerol and free fatty acids. Glycerol is converted to glucose for cellular use. Fatty acids are converted to ketones, resulting in increased ketone levels in body fluids and decreased hydrogen ion concentration pH. Ketones are excreted in the urine, accompanied by large amounts of water. The accumulation of ketones in body fluids, decreased pH, electrolyte loss and dehydration from excessive urination, and alterations in the bicarbonate buffer system result in diabetic ketoacidosis DKA. Untreated DKA can result in coma or death. Spontaneous hypoglycemia in adults is of two principal types: Diabetes Complications The major cause of the high morbidity and mortality rate associated with Chronic Complications of Diabetes Late clinical manifestations of diabetes mellitus include a number of pathologic changes Diabetes Cardiovascular complications Cardiovascular disease risk is increased in patients with type 1 diabetes Complications of Insulin Therapy Hypoglycemic reactions, the most common complication of insulin therapy Diabetic Nephropathy As many as cases of end-stage renal disease occur each year among diabetic people in the United States Diabetic Neuropathy Diabetic neuropathies are the most common complications of diabetes affecting Primary treatment goals for diabetes patients include the achieving of blood glucose levels Many patients with type 1 diabetes are initially diagnosed with the disease following a hospital admission for DKA. In a known diabetic patient, periods of stress or infection may precipitate DKA. More often, however, DKA results from poor daily glycemic control. Patients who remain severely hyperglycemic for several days or longer due to inadequate insulin administration or excessive glucose intake are prone to developing DKA. Most type 2 diabetes patients are overweight, and most are diagnosed as adults. The genetic influence in type 2 diabetes is greater than that seen with type 1. Although the genetic predisposition to type 2 diabetes is strong, no single genetic defect has been found. In addition to genetic influences, acquired risk factors for type 2 diabetes

include obesity, advancing age, and an inactive lifestyle. The underlying pathophysiologic defect in type 2 diabetes does not involve autoimmune beta-cell destruction. Rather, type 2 diabetes is characterized by the following three disorders: Increased tissue resistance to insulin generally occurs first and is eventually followed by impaired insulin secretion. The pancreas produces insulin, yet insulin resistance prevents its proper use at the cellular level. Glucose cannot enter target cells and accumulates in the bloodstream, resulting in hyperglycemia. The high blood glucose levels often stimulate an increase in insulin production by the pancreas; thus, type 2 diabetic individuals often have excessive insulin production hyperinsulinemia. Over the years, pancreatic insulin production usually decreases to below normal levels. In addition to hyperglycemia, type 2 diabetic patients often have a group of disorders that has been called "insulin resistance syndrome" or syndrome X. Obesity contributes greatly to insulin resistance, even in the absence of diabetes. In fact, weight loss is a cornerstone of therapy for obese type 2 diabetic patients. Insulin resistance generally decreases with weight loss. Obesity also may explain the dramatic increase in the incidence of type 2 diabetes among young individuals in the United States in the past 10 to 20 years. Type 2 diabetes usually has a slow onset and may remain undiagnosed for years. Approximately half of those who have type 2 diabetes are unaware of their disease. Unfortunately, the insidious nature of the disease allows prolonged periods of hyperglycemia to begin exerting negative effects on major organ systems. By the time many type 2 diabetic patients are diagnosed, diabetic complications have already begun. Type 2 diabetic patients do not require exogenous insulin for survival since they still produce insulin. However, insulin injection is often an integral part of medical management for type 2 diabetes. Unlike type 1 diabetic patients, individuals with type 2 diabetes are generally resistant to DKA because their pancreatic insulin production is often sufficient to prevent ketone formation. Severe physiologic stress may induce DKA in those with type 2 diabetes. Long periods of severe hyperglycemia may result in hyperosmolar nonketotic acidosis. Hyperglycemia results in the urinary excretion of large amounts of glucose, with attendant water loss. If fluids are not replaced, the dehydration can result in electrolyte imbalance and acidosis. It usually develops during the third trimester and significantly increases perinatal morbidity and mortality. As with type 2 diabetes, the pathophysiology of gestational diabetes is associated with increased insulin resistance. People with IFG have increased fasting blood glucose levels but usually have normal levels following food consumption. Those with IGT are normoglycemic most of the time but can become hyperglycemic after large glucose loads. IGT and IFG are not considered to be clinical entities; rather, they are risk factors for future diabetes. The pathophysiology of IFG and IGT is related primarily to increased insulin resistance whereas endogenous insulin secretion is normal in most patients.

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3: Diabetes Pathophysiology

In Type 1 diabetes, the pancreas cannot synthesize enough amounts of insulin as required by the body. The pathophysiology of Type 1 diabetes mellitus suggests that it is an autoimmune disease, wherein the body's own immune system generates secretion of substances that attack the beta cells of the pancreas.

Diabetes Epidemiology Genetics Pathogenesis Experimental Models Healthcare Abstract Type 1 diabetes mellitus T1DM , also known as insulin-dependent diabetes, is a chronic disease caused by autoimmune type 1a or spontaneous type 1b destruction of pancreatic beta cells, resulting in insulin deficiency. It is generally diagnosed in children before 20 years of age and is oftentimes fatal. This review will discuss the epidemiology of T1DM, including its incidence and prevalence, related temporal trends and risk factors for development. Furthermore, pathogenesis and immune system involvement of the disease will be evaluated, with a particular focus on cells of the adaptive and innate immune systems. Finally, an overview of past, present and future treatments for T1DM will be discussed. From , the incidence of T1DM in children ages 14 and younger was reported to be 0. Furthermore, it was observed that the prevalence of the disease increased as age increased in most groups. The investigators attributed the variation between ethnic groups to genetic and environmental differences Maahs, West, Lawrence et al. From , the average annual increase in the occurrence of the disease was 2. Firstly, T1DM is prevalent in youth 20 years of age and younger. The incidence rates of the disease increase from birth and peak at age The prevalence of T1DM decreases after puberty and stabilizes in adulthood. Secondly, it has been found that female and male children are equally affected with T1DM. However, certain studies have shown that males are disproportionally affected in regions with a high prevalence of T1DM while females are disproportionally affected in regions with a low prevalence of the disease Maahs, West, Lawrence et al. The authors indicated that the prevalence of T1DM is highest in non-Hispanic white populations and lowest in Navajo groups. Moreover, Redondo et al. While some diseases are due to single gene mutations, many autoimmune diseases result from an unlucky combination of many gene variants. Transcription of the hormone is controlled by binding of the transcription factor Purl to the INS promoter region element called VNTR variable number of tandem repeats Pugliese, Thus, the distribution of T1DM susceptibility alleles and haplotypes partially explains differences in worldwide incidences of the disease Ghazarian, Diana, Simoni et al. Hence, it is likely that external factors contribute to the onset of the disease as well. Furthermore, it has been found that the incidence of T1DM differs in Western and Eastern Germany, despite similar genetic backgrounds in both areas Ikegami, Noso, Babaya et al. Therefore, it is possible that a combination of genetics and environmental factors induce T1DM. Epidemiological studies have found associations between T1DM and socio-economic status SES , dietary and nutritional habits, and pathogen exposure. Socioeconomic Factors In a study, Patterson et al. Westernized countries place a strong emphasis on cleanliness and invest in education and medical care e. High levels of hygiene limit the survival, proliferation, and spread of pathogens. An untrained immune system may respond inappropriately to self, increasing the frequency of autoimmune diseases, including T1DM Ghazarian, Diana, Simoni et al. A study by Elliott et al. Secondly, dietary gluten has been associated with the development of T1DM. A study of 1, children with T1DM parents found that the consumption of gluten before 3 months of age increases islet autoantibody risk Grieco, Vendrame, Spagnuolo et al. Hence, countries with greater levels of sunlight and enhanced Vitamin D synthesis show a lower incidence of the disease. It should be noted, however, that the above studies on diet are controversial, with other research showing no effect. By mimicking sequence homology of a self-peptide, a pathogen-derived peptide may trigger an immune response against self-tissue in the host organism known as molecular mimicry Grieco, Vendrame, Spagnuolo et al. Furthermore, viral infections may provoke inflammation and destruction of host cells, causing release of autoantigens and activation of autoreactive T cells called bystander activation of T cells Grieco, Vendrame, Spagnuolo et al. Significant inflammation may induce stress in the endoplasmic reticulum ER , resulting in protein denaturation and presentation of new autoantigens known as antigen spreading Grieco, Vendrame, Spagnuolo et al. Secondly, human enteroviruses, including polioviruses; echoviruses; and rhinoviruses, are associated

with T1DM. Enteroviruses are generally transmitted through consumption of contaminated food and drink. Pathogenesis of T1DM can be categorized into two stages. By presenting self-antigens to autoreactive T cells, APCs can remove damaged cells. In inflamed tissues, however, signals that encourage APC maturation are prematurely generated. However, they can cause extensive local damage when uncontrolled. Hence, DCs contribute to the onset of the disease. It has been hypothesized that NK cells may promote or protect against T1DM depending on anatomical location and chemical environment. Under normal circumstances, T cells i. It is not entirely certain why or how the signal is issued. Understanding the development of rogue T cells will better allow scientists and clinicians to treat T1DM Wagner, Moreover, animal models are crucial for developing therapeutic interventions for diabetes patients. They can be used to investigate T1DM under different conditions. By studying the effects of different environmental factors on the development of T1DM, researchers can attain a stronger understanding of the underlying mechanisms of the disease. In , a clinical trial tested the glycaemic control of patients who use continuous subcutaneous insulin infusion and patients who use intensive insulin injections Ikegami, Noso, Babaya et al. It was found that glycaemic control was higher in patients who used continuous subcutaneous insulin infusion compared to patients who used intensive insulin injections. Furthermore, it was discovered that less insulin was needed to achieve a strict control level in patients who used continuous subcutaneous insulin infusion Ikegami, Noso, Babaya et al. It is clear that clinical trials are frequently used to investigate multiple aspects of T1DM.

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4: # Diabetes Mellitus Pathophysiology # Ways To Prevent Diabetes

The pathophysiology of type 2 diabetes mellitus is characterized by peripheral insulin resistance, impaired regulation of hepatic glucose production, and declining Î²-cell function, eventually leading toÎ² -cell failure.

My spouse has diabetes and because does we pay for all your his diabetic supplies drugs sometimes not generics also hospital goes to. If a cure were ever found might just take that magic pill or shot or whatever scenario may be and wow! No more drain on financial institution account! But then those who make insulin lancets sugar meters many others. Diabetes Mellitus Pathophysiology Make protein foods and vegetables your sutures. Protein foods include - free range chicken turkey grass-fed beef fish seafood etc. One third to two-thirds of home plate will get some amino acids. The most your plate should be different vegetables limit potatoes corn peas bean legumes. Creates stable blood sugar levels and allows the physique to function more efficently. Diabetes Mellitus Pathophysiology Restoring the balance is essential in improving quite a few health disparities seen our own country. Possibilities people going hungry from a country that in general is encountering the curse of selection. Its like dying of dehydration on a raft part way through a river lake. In case the lake were instead an ocean the dehydration would be understandable mainly because ocean water is undrinkable. But how can this happen in a huge fresh water lake? Do you know of we carry extra weight around my waist whilst others starve? It doesnt seem right. This is the other side of the curse of abundance disparity. The curse shortens our own lives by both providing too much to some and providing too little to others. And the lack of patient compliance is making their usefulness unethical. Diabetes Mellitus Pathophysiology We appear forward to individuals or Us magazine running the cover story Im back! Clearly it has been coming for your time. Diabetes Mellitus Pathophysiology Now which have read these tips the rest is a great deal you. Handling the complex array of dietary choices medicines and lifestyle factors that influence the lifetime of diabetes could be extremely daunting but a person are take the tips you just read on board you can live a long happy life despite diabetes.

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5: # Diabetes Mellitus Pathophysiology # Treatment For Diabetic Coma

INTRODUCTION. Type 2 diabetes mellitus is characterized by hyperglycemia, insulin resistance, and relative impairment in insulin secretion. It is a common disorder with a prevalence that rises markedly with increasing degrees of obesity [].

Check new design of our homepage! Detailed Information on the Pathophysiology of Diabetes Mellitus Studies conducted on the pathophysiology of diabetes mellitus suggested that abnormal metabolism of insulin hormone is the primary cause for the development of this complex syndrome. Even though the etiologies and triggering factors of the three types of diabetes mellitus are different, they cause nearly the same symptoms and complications. HealthHearty Staff Last Updated: Apr 22, Diabetes mellitus DM or simply diabetes, is a chronic health condition in which the body either fails to produce sufficient amounts of insulin or it responds abnormally to insulin. Commonly referred to as a syndrome, diabetes is classified into three types, namely, Type 1 diabetes, Type 2 diabetes, and Gestational diabetes. The ultimate outcome for all three types of diabetes is high blood glucose level. The pathophysiology of diabetes mellitus is very complex, as this ailment is characterized by different etiologies while sharing similar signs, symptoms, and complications. Pathophysiology The pathophysiology of all types of diabetes is related to the hormone insulin, which is secreted by the beta cells of the pancreas. In a healthy person, insulin is produced in response to the increased level of glucose in the bloodstream, and its major role is to control glucose concentration in the blood. What insulin does is, allowing the body cells and tissues to use glucose as a main energy source. Also, this hormone is responsible for conversion of glucose to glycogen for storage in the muscles and liver cells. This way, sugar level is maintained at a near stable amount. In a diabetic person, there is an abnormal metabolism of insulin hormone. The actual reason for this malfunction differs according to the type of diabetes. Whatever the cause is, the body cells and tissues do not make use of glucose from the blood, resulting in elevated blood glucose a typical symptom of diabetes called hyperglycemia. This condition is also exacerbated by the conversion of stored glycogen to glucose, i. Over a period of time, high glucose level in the bloodstream can lead to severe complications, such as eye disorders, cardiovascular diseases, kidney damage, and nerve problems. In Type 1 diabetes, the pancreas cannot synthesize enough amounts of insulin as required by the body. Consequently, the pancreas secretes little or no insulin. Type 1 diabetes is more common among children and young adults around 20 years. In case of Type 2 diabetes mellitus, the insulin hormone secreted by the beta cells is normal or slightly lower than the ideal amount. However, the body cells are not responding to insulin as they do in a healthy person. Since the body cells and tissues are resistant to insulin, they do not absorb glucose, instead it remains in the bloodstream. Thus, the Type 2 diabetes is also characterized by elevated blood sugar. It is commonly manifested by middle-aged adults above 40 years. The third type of diabetes is called Gestational diabetes. As the term clearly suggests, it is exhibited by pregnant women. Over here, high level of blood glucose is caused by hormonal fluctuations during pregnancy. Usually, the sugar concentration returns to normal after the baby is born. However, there are also instances, in which it remains high even after childbirth. This is an indication for increased risks of developing diabetes in the near future. As already mentioned, the symptoms and effects of all the three forms of diabetes are similar. The noticeable symptoms include increased thirst polydipsia , increased urination polyuria , and increased appetite polyphagia. Other diabetes signs and symptoms include excessive fatigue, presence of sugar in the urine glycosuria , body irritation, unexplained weight loss, and dehydration. Elevated blood sugar and glycosuria are interrelated; when sugar amount in the blood is abnormally high, the reabsorption by proximal convoluted tubule is reduced, thereby retaining some glucose in the urine. As per statistics, Type 2 diabetes is the most commonly occurring type, in comparison to the other two forms of diabetes mellitus. Early and correct detection of the diabetes is necessary to prevent severe health effects. After diagnosis, the physician prescribes appropriate medication for treatment of diabetes, which may include insulin injections or oral insulin medicines, depending upon the type of diabetes mellitus. In addition to the therapeutic intervention, healthy lifestyle modifications, especially in terms of diet and exercises are recommended for effective management of diabetes symptoms and long-term

effects. Since it is a global health issue, studies regarding the diabetes mellitus pathophysiology are currently in progress in order to minimize its associated health effects, and also, to treat it effectively.

6: Detailed Information on the Pathophysiology of Diabetes Mellitus

Understanding the pathophysiology of type 2 diabetes mellitus and determining optimal management strategies are critical health care priorities because of the high morbidity and mortality associated with the disease.1 The treatment goal for all patients with diabetes is to prevent its short- and long-term.

Cyclosporine A reduced the activity of the immune system, decreased autoimmune responses, and lowered the incidence of T1DM Grieco, Vendrame, Spagnuolo et al. However, renal toxicity was a common side-effect and cyclosporine A testing ceased Stiller, Dupre, Gent et al. They observed a three-fold increase in severe hypoglycemia in subjects who were extensively treated with insulin pumps during the Diabetes Control and Complications Trial DCCT. A large reduction in A1c levels was observed in adults 25 years of age or older. In contrast, A1c levels rose in controls Maahs, West, Lawrence et al. Insulin was administered parenterally and orally. The study found that parenteral insulin did not decrease progression of the disease. Similarly, oral insulin did not alter overall progression of diabetes Grieco, Vendrame, Spagnuolo et al. However, in subset of patients with high levels of insulin autoantibodies, delay in progression of T1DM was observed Type 1 Diabetes TrialNet, Future Directions for Treatment A variety of new technologies and therapies are being developed in order to treat and prevent the development of T1DM. Novel Technologies It can be argued that major advances made in T1DM care within the last quarter century have come from technology rather than biology. Technologies such as rapid glucose estimation from a drop of blood, insulin pumps and quick HbA1c results are rapidly becoming commonplace. Moreover, mathematicians, engineers, physicians, scientists and patients have combined their efforts to develop external insulin delivery devices and an "artificial pancreas. Embryonic stem cells are pluripotent i. However, biological hurdles e. Induced pluripotent iPS stem cells can be reprogrammed from normal adult skin cells and can become a multitude of cell types in the body. Genetic Engineering Genetic engineering describes the process by which a functional gene is introduced into a new tissue or organ, allowing for the expression of a new feature or characteristic. Investigators of T1DM have evaluated the likelihood of modifying islet cells to render them resistant to immune destruction prior to transplantation. Furthermore, the possibility of altering various cell types to convert them into insulin-producing cells for later transplantation in the same individual had been investigated. However, significant research is required before genetic engineering techniques can be practically applied in the treatment of T1DM Wong et al. Conclusions T1DM is a chronic disease caused by autoimmune type 1a or spontaneous type 1b destruction of pancreatic beta cells, resulting in insulin deficiency. Ethnicity, genetics and environmental influences contribute to the development of T1DM. Animal models have improved our understanding of the immunological mechanisms that underlie T1DM. Common treatments for T1DM include frequent injections of rapid-acting insulin with meals combined with daily basal insulin. Moreover, insulin pumps are widely used to treat youth with the disease. However, stronger knowledge of innate immune cell structures will aid in the discovery of a cure for T1DM in humans. Predicting type 1 diabetes. Stem cell therapy for type 1 diabetes mellitus. Nat Rev Endocrinol, 6 3 , Putative environmental factors in type 1 diabetes. Diabetes Metab Rev, 14, Pathogen recognition and innate immunity. A model of immune dysregulation. New perspectives on disease pathogenesis and treatment. Genetics of type 1 A diabetes. The value of national diabetes registries: Curr Diab Rep, 10, Can NK cells be a therapeutic target in human type 1 diabetes? Eur J Immunol, 38, â€” B cells in type 1 diabetes: Studies on cell surface antibody binding. Type I insulin-dependent diabetes mellitus and cow milk: Diabetologia, 42 3 , â€” Current concepts in epidemiology, pathophysiology, clinical care, and research. Prevention of acceleration of type 1 diabetes by viruses. Life Sci, 70, Multiplicity of the antibody response to GAD65 in Type 1 diabetes. Clin Exp Immunol, 2 , Incorporating type 1 diabetes prevention into clinical practice. Clin Diabetes, 28 2 , 61â€” Innate immunity and the pathogenesis of type 1 diabetes. Semin Immunopathol, 33, Type 1 diabetes mellitus: Etiology, presentation, and management. Pediatr Clin North Am, 52, Genetic liability of type 1 diabetes and the onset age among 22, young Finnish twin pairs: Diabetes, 52 4 , â€” Genetics and pathogenesis of type 1 diabetes: Prospects for prevention and intervention. Journal of Diabetes Investigation, 2, Annu Rev Immunol, 20, â€” Role of innate

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8: The Epidemiology, Pathogenesis, and Treatment of Type 1 Diabetes Mellitus - Inquiries Journal

Later, classifications of diabetes mellitus (mellitus is Latin for "honey- sweet") are refined, and suggested treatments vary from the removal of diuretic food to taking tepid baths.

9: Diabetes Mellitus (DM) : Causes, Pathogenesis, Symptoms, Diagnosis & Treatment - DoctorAlerts

Most research in diabetes mellitus (DM) has been conducted in animals, and their replacement is currently a chimera. As compared to when they started to be used by modern science in the 17th century, a very high number of animal models of diabetes is now available, and they provide new insights into almost every aspect of diabetes.

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