

## DIABETES MELLITUS AND ITS MANAGEMENT

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### ABSTRACT

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia due to insulin deficiency, resistance, or both. It is categorized primarily into type 1 and type 2 diabetes, with type 2 being the more prevalent form globally and in India. India is witnessing an alarming increase in diabetes cases, influenced by urbanization, sedentary lifestyles, and dietary changes. The disease is further complicated by socio-economic disparities, geographic variability, and lack of nationwide epidemiological data. Insulin plays a pivotal role in glucose metabolism, and its dysfunction leads to a wide range of systemic effects and complications. Diagnosis involves tests such as fasting blood glucose, random glucose measurement, and oral glucose tolerance tests. The burden of diabetes is further compounded by gestational and secondary diabetes, with significant health implications. Early symptoms include frequent urination, increased thirst, weight loss, and fatigue. Effective management requires early detection, lifestyle modification, public awareness, and structured government policies. Global best practices such as those implemented in the US, UK, Australia, and the UAE demonstrate that comprehensive public health initiatives can improve diabetes care. India must adopt similar grassroots efforts to mitigate this growing public health challenge.

**KEYWORDS:** Diabetes mellitus, glucose, diabetes, insulin.

### INTRODUCTION

Diabetes mellitus is a group of metabolic diseases characterized by high blood sugar (glucose) levels, which result from defects in insulin secretion, or action, or both. Diabetes mellitus, commonly referred to as diabetes (and in this article will be referred to as "diabetes"), was first identified as a disease associated with "sweet urine," and excessive muscle loss in the ancient world. Elevated levels of blood glucose (hyperglycemia) lead to spillage of glucose into the urine, hence the term sweet urine. Normally, blood glucose levels are tightly controlled by insulin, a hormone produced by the pancreas. Insulin lowers the blood glucose level. When the blood glucose elevates (for example, after eating food), insulin is released from the pancreas to normalize the glucose level. In patients with diabetes, the absence or insufficient production of insulin causes hyperglycemia. Diabetes is a chronic medical condition, meaning although it can be controlled, it lasts a lifetime.<sup>[1]</sup>

### Current Scenario

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease. In 2000, India (31.7 million) topped the world with the highest number of

people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively. According to Wild et al. the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India, while China (42.3 million) and the United States (30.3 million) will also see significant increases in those affected by the disease. India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country. Many influences affect the prevalence of disease throughout a country, and identification of those factors is necessary to facilitate change when facing health challenges. So what are the factors currently affecting diabetes in India that are making this problem so extreme?<sup>[2]</sup>

The aetiology of diabetes in India is multifactorial and includes genetic factors coupled with environmental influences such as obesity associated with rising living standards, steady urban migration, and lifestyle changes. Yet despite the incidence of diabetes within India, there are no nationwide and few multi-centric studies conducted on the prevalence of diabetes and its

complications. The studies that have been undertaken are also prone to potential error as the heterogeneity of the Indian population with respect to culture, ethnicity, socio-economic conditions, mean that the extrapolation of regional results may give inaccurate estimates for the whole country.<sup>[3]</sup>

There are, however, patterns of diabetes incidence that are related to the geographical distribution of diabetes in India. Rough estimates show that the prevalence of diabetes in rural populations is one-quarter that of urban population for India and other Indian continent countries such as Bangladesh, Nepal, Bhutan, and Sri Lanka. Preliminary results from a large community study conducted by the Indian Council of Medical research (ICMR) revealed that a lower proportion of the population is affected in states of Northern India (Chandigarh 0.12 million, Jharkhand 0.96 million) as compared to Maharashtra (9.2 million) and Tamil Nadu (4.8 million).

**FACTORS RESPONSIBLE FOR DIABETES:** Glucose is a simple sugar found in food. Glucose is an essential nutrient that provides energy for the proper functioning of the body cells. Carbohydrates are broken down in the small intestine and the glucose in digested food is then absorbed by the intestinal cells into the bloodstream, and is carried by the bloodstream to all the cells in the body where it is utilized. However, glucose cannot enter the cells alone and needs insulin to aid in its transport into the cells. Without insulin, the cells become starved of glucose energy despite the presence of abundant glucose in the bloodstream. In certain types of diabetes, the cells' inability to utilize glucose gives rise to the ironic situation of "starvation in the midst of plenty". The abundant, unutilized glucose is wastefully excreted in the urine.<sup>[5]</sup>

Insulin is a hormone that is produced by specialized cells (beta cells) of the pancreas. (The pancreas is a deep-seated organ in the abdomen located behind the stomach.) In addition to helping glucose enter the cells, insulin is also important in tightly regulating the level of glucose in the blood. After a meal, the blood glucose level rises. In response to the increased glucose level, the pancreas normally releases more insulin into the bloodstream to help glucose enter the cells and lower blood glucose levels after a meal. When the blood glucose levels are lowered, the insulin release from the pancreas is turned down.

#### **TYPES OF DIABETES**

There are two major types of diabetes, called type 1 and type 2. Type 1 diabetes was also called insulin dependent diabetes mellitus (IDDM), or juvenile onset diabetes mellitus. In type 1 diabetes, the pancreas undergoes an autoimmune attack by the body itself, and is rendered incapable of making insulin. Abnormal antibodies have been found in the majority of patients with type 1 diabetes. Antibodies are proteins in the blood that are

part of the body's immune system. The patient with type 1 diabetes must rely on insulin medication for survival.<sup>[6]</sup>

While it is said that type 2 diabetes occurs mostly in individuals over 30 years old and the incidence increases with age, we are seeing an alarming number patients with type 2 diabetes who are barely in their teen years. In fact, for the first time in the history of humans, type 2 diabetes is now more common than type 1 diabetes in childhood. Most of these cases are a direct result of poor eating habits, higher body weight, and lack of exercise.<sup>[8]</sup>

Regarding age, data shows that for each decade after 40 years of age regardless of weight there is an increase in incidence of diabetes. The prevalence of diabetes in persons 65 to 74 years of age is nearly 20%. Type 2 diabetes is more common in certain ethnic groups.

Diabetes can occur temporarily during pregnancy. Significant hormonal changes during pregnancy can lead to blood sugar elevation in genetically predisposed individuals. Blood sugar elevation during pregnancy is called gestational diabetes. Gestational diabetes usually resolves once the baby is born. However, 25-50% of women with gestational diabetes will eventually develop diabetes later in life, especially in those who require insulin during pregnancy and those who are overweight after their delivery. Patients with gestational diabetes are usually asked to undergo an oral glucose tolerance test about 6 weeks after giving birth to determine if their diabetes has persisted beyond the pregnancy.<sup>[9]</sup>

#### **SYMPTOMS**

The early symptoms of untreated diabetes are related to elevated blood sugar levels, and loss of glucose in the urine. High amounts of glucose in the urine can cause increased urine output and lead to dehydration. Dehydration causes increased thirst and water consumption. The inability to utilize glucose energy eventually leads to weight loss despite an increase in appetite. Some untreated diabetes patients also complain of fatigue, nausea and vomiting. Patients with diabetes are prone to developing infections of the bladder, skin, and vaginal areas. Fluctuations in blood glucose levels can lead to blurred vision. Extremely elevated glucose levels can lead to lethargy and coma (diabetic coma).

#### **DIAGNOSED**

The fasting blood glucose (sugar) test is the preferred way to diagnose diabetes. It is easy to perform and convenient. After the person has fasted overnight (at least 8 hours), a single sample of blood is drawn and sent to the laboratory for analysis.

When fasting a blood glucose stays above 100mg/dl, but in the range of 100-126mg/dl, this is known as impaired fasting glucose (IFG). While patients with IFG do not have the diagnosis of diabetes, this condition carries with it its own risks and concerns, and is addressed elsewhere.

### THE ORAL GLUCOSE TOLERANCE TEST

Though not routinely used anymore, the oral glucose tolerance test (OGTT) is a gold standard for making the diagnosis of type 2 diabetes. It is still commonly used for diagnosing gestational diabetes. With an oral glucose tolerance test, the person fasts overnight (at least 8 but not more than 16 hours). Then first, the fasting plasma glucose is tested. After this test, the person receives 75 grams of glucose (100 grams for pregnant women). There are several methods employed by obstetricians to do this test, but the one described here is standard. Usually, the glucose is in a sweet- tasting liquid that the person drinks. Blood samples are taken at specific intervals to measure the blood glucose.

For the test to give reliable results, the person must be in good health (not have any other illnesses, not even a cold). Also, the person should be normally active (not lying down, for example, as an inpatient in a hospital) and should not be taking medicines that could affect the blood glucose. For 3 days before the test, the person should have eaten a diet high in carbohydrates (150- 200 grams per day). The morning of the test, the person should not smoke or drink coffee.

### EVALUTING THE RESULT OF ORAL GLUCOSE TOLERANCE TEST

Glucose tolerance tests may lead to one of the following diagnoses.

- **Normal response:** A person is said to have a normal response when the 2-hour glucose level is less than 140 mg/dl, and all values between 0 and 2 hours are less than 200 mg/dl.
- **Impaired glucose tolerance:** A person is said to have impaired glucose tolerance when the fasting plasma glucose is less than 126 mg/dl and the 2-hour glucose level is between 140 and 199 mg/dl.
- **Diabetes:** A person has diabetes when two diagnostic tests done on different days show that the blood glucose level is high.
- **Gestational diabetes:** A woman has gestational diabetes when she has any two of the following: a 100g OGTT, a fasting plasma glucose of more than 95 mg/dl, a 1- hour glucose level of more than 180 mg/dl, a 2-hour glucose level of more than 155 mg/dl, or a 3-hour glucose level of more than 140 mg/dl.<sup>[15]</sup>

### WHY IS BLOOD SUGAR CHECKED AT HOME

Home blood sugar (glucose) testing is an important part of controlling blood sugar. One important goal of diabetes treatment is to keep the blood glucose levels near the normal range of 70 to 120 mg/dl before meals and under 140 mg/dl at 2 hours after eating. Blood glucose levels are usually tested before and after meals, and at bedtime. The blood sugar level is typically determined by pricking a fingertip with a lancing device and applying the blood to a glucose meter, which reads the value. There are many meters on the market, for example, Accu- Check Advantage, One Touch Ultra,

Sure Step and Freestyle. Each meter has its own advantages and disadvantages (some use less blood, some have a larger digital readout, some take a shorter time to give you results, etc). The test results are then used to help patients make adjustments in medications, diets, and physical activities.<sup>[14]</sup>

### Hemoglobin A1c

To explain what an A1c is, think in simple terms. Sugar sticks, and when it's around for a long time, it's harder to get it off. In the body, sugar sticks too, particularly to proteins. The red blood cells that circulate in the body live for about 3 months before they die off. When sugar sticks to these cells, it gives us an idea of how much sugar is around for the preceding 3 months. In most labs, the normal range is 4-5.9 %. In poorly controlled diabetes, its 8.0% or above, and in well controlled patients it's less than 7.0%. The benefits of measuring A1c is that it gives a more reasonable view of what's happening over the course of time (3 months), and the value does not bounce as much as finger stick blood sugar measurements. There is a correlation between A1c levels and average blood sugar levels as follows.

Table-01.

A1c(%)	Mean blood sugar (mg/dl)
6	135
7	170
8	205
9	240
10	275
11	310
12	345

### ACUTE COMPLICATION OF DIABETES

Severely elevated blood sugar levels due to an actual lack of insulin or a relative deficiency of insulin. Abnormally low blood sugar levels due to too much insulin or other glucose- lowering medications. Diabetic ketoacidosis can be caused by infections, stress, or trauma all which may increase insulin requirements. In addition, missing doses of insulin is also an obvious risk factor for developing diabetic ketoacidosis. Urgent treatment of diabetic ketoacidosis involves the intravenous administration of fluid, electrolytes, and insulin, usually in a hospital intensive care unit.

### Hypoglycemia

means abnormally low blood sugar (glucose). In diabetes, the most common cause of low blood sugar is excessive use of insulin or other glucose- lowering medications, to lower the blood sugar level in diabetic patients in the presence of a delayed or absent meal. When low blood sugar levels occur because of too much insulin, it is called an insulin reaction. Sometimes, low blood sugar can be the result of an insufficient caloric intake or sudden excessive physical exertion.<sup>[10]</sup>

### CHRONIC COMPLICATION OF DIABETES

These diabetes complications are related to blood vessel

diseases and are generally classified into small vessel disease, such as those involving the eyes, kidneys and nerves (microvascular disease), and large vessel disease involving the heart and blood vessels (macrovascular disease). Diabetes accelerates hardening of the arteries (atherosclerosis) of the larger blood vessels, leading to coronary heart disease (angina or heart attack), strokes, and pain in the lower extremities because of lack of blood supply (claudication). For more information, please read the following articles: Stroke, Angina, and Heart Attack.<sup>[16]</sup>

### Eye Complications

The major eye complication of diabetes is called diabetic retinopathy. Diabetic retinopathy occurs in patients who have had diabetes for at least 5 years. Diseased small blood vessels in the back of the eye cause the leakage of protein and blood in the retina. Disease in these blood vessels also causes the formation of small aneurysms (microaneurysms), and new but brittle blood vessels (neovascularization). Spontaneous bleeding from the new and brittle blood vessels can lead to retinal scarring and retinal detachment, thus impairing vision.

### Kidney damage

Kidney damage from diabetes is called diabetic nephropathy. The onset of kidney disease and its progression is extremely variable. Initially, diseased small blood vessels in the kidneys cause the leakage of protein in the urine. Later on, the kidneys lose their ability to cleanse and filter blood. The accumulation of toxic waste products in the blood leads to the need for dialysis. Dialysis involves using a machine that serves the function of the kidney by filtering and cleaning the blood. In patients who do not want to undergo chronic dialysis, kidney transplantation can be considered.

### MEDICATION FOR THE TYPE 2 DIABETES

All the information listed below applies to patients who are not pregnant or breastfeeding. At present the only recommended way of controlling diabetes in these situations is by diet, exercise and insulin therapy. You should refer to your doctor if you are on these medications and are considering becoming pregnant, or if you have become pregnant while taking these medications.

Based on what is known, medications for type 2 diabetes are designed to.

Increase the insulin output by the pancreas.

- Decrease the amount of glucose released from the liver.
- Increase the sensitivity (response) of cells to insulin.

When selecting therapy for the treatment of type 2 diabetes, consideration should be given to.

- The magnitude of change in blood sugar control that each medication will provide.

- Other coexisting medical conditions (hypertension high cholesterol, etc.)

### Medications that increase the insulin output by the pancreas - sulfonylureas and meglitinides

**SULFONYLUREAS** - Sulfonylureas are medications that increase insulin output from the pancreas to lower blood glucose levels. Older drugs include chlorpropamide and tolbutamide, while newer ones are glyburide (DiaBeta), glipizide (Glucotrol), and glimepiride (Amaryl). They are effective but can cause hypoglycemia and should be avoided in patients with sulfa allergies.

**Meglitinides - repaglinide (Prandin) and nateglinide (Starlix):** Meglitinides, such as repaglinide (Prandin) and nateglinide (Starlix), also stimulate insulin release but through a different potassium channel. They are short-acting, taken 30 minutes before meals, and peak within one hour. Although they can cause hypoglycemia, the risk is lower compared to sulfonylureas.

### Starlix

Nateglinide (Starlix) has essentially the same profile of side effects and interactions as Prandin. The major benefit of Starlix is that the starting dose of 120mg does not need to be adjusted upward, but rather remains constant. These medications are also relatively safe to use in people with impaired kidney function. For more, please read the drug information pamphlet on nateglinide (Starlix).

### Thiazolidinediones

(pioglitazone, rosiglitazone)

With the introduction of this new class of drug in 1997, the world has watched the peroxisome proliferator activated receptor (PPAR)- $\gamma$  agonists with anticipation. The net effect of these drugs results from stimulation of a nuclear PPAR- $\gamma$  that regulates the transcription of genes culminating in an increase in insulin sensitivity.

The effects of the glitazones on cardiovascular morbidity and mortality remain a topical issue. It seems, from the literature, that pioglitazone especially has a more favourable effect on major cardiovascular outcomes. The PROactive study showed a significant reduction of 16% in the main secondary endpoints of all-cause mortality.<sup>37</sup> It is thought that the beneficial effects of the glitazones extend beyond their influence on glycaemic control through so-called pleiotropic.

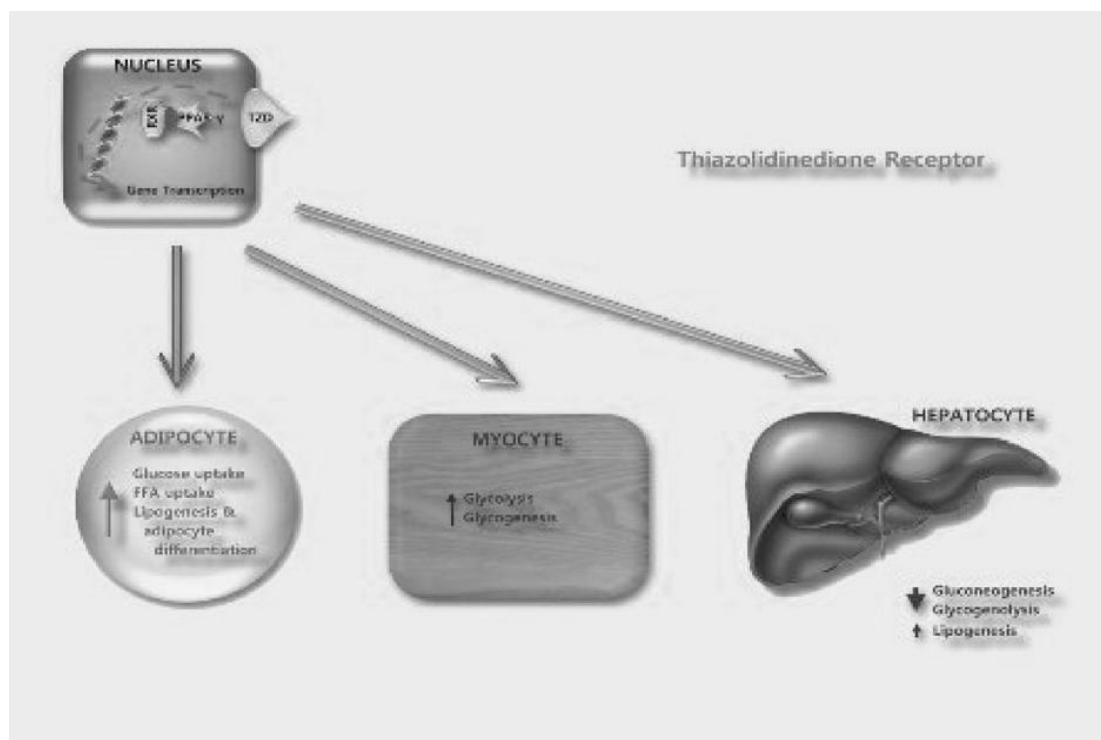


Fig-01.

#### ***Medications that decrease the amount of glucose produced by the liver***

A class of drugs called biguanides has been used for many years in Europe and Canada. In 1994, the FDA approved the use of metformin (Glucophage) for the treatment of type 2 diabetes in the United States. Glucophage is unique in its ability to decrease glucose production from the liver. Briefly, because metformin does not increase insulin levels, when used alone, it does not usually cause hypoglycemia.

#### ***Medications that increase the sensitivity of cells to Insulin***

At present in the United States, the class of drugs known as thiazolidinediones lowers blood glucose by improving target cell response to insulin (increasing the sensitivity of the cells to insulin). Troglitazone (Rezulin) was the first of this type of compound introduced in the United States. Because of severe toxic liver effects, troglitazone has been taken off the market. Sister compounds are now available with a better safety profile. These drugs include pioglitazone (Actos) and rosiglitazone (Avandia). As an aside, Actos and Avandia have an added benefit of changing cholesterol patterns in diabetes. HDL (or good cholesterol) increases on these medications, and triglycerides often decrease, while there is some controversy regarding what happens to bad cholesterol (LDL) levels, and a suggestion that Actos may be superior in LDL reduction as a side benefit. In this population that is already at an increased risk for heart disease, a benefit in cholesterol profile is a beneficial outcome.

#### ***Medications that decrease the absorption of carbohydrates from the intestine***

Before being absorbed into the bloodstream, carbohydrates must be broken down into smaller sugar particles, such as glucose, by enzymes in the small intestine. One of enzymes involved in breaking down carbohydrates is called alpha glucosidase. By inhibiting this enzyme, carbohydrates are not broken down as efficiently and glucose absorption is delayed.<sup>[27]</sup>

#### ***Precose***

The name of the alpha glucosidase inhibitor available in the United States is Precose. In clinical trials with over 700 patients, the use of acarbose (Precose) showed a reduction in hemoglobin A1c values (a well known measurement of 3 month average blood sugars) significantly greater than the placebo (no treatment). Precose is taken three times a day at the beginning of meals. The dosage varies from 25 mg to 100 mg with each meal. The maximum recommended dose is 100 mg three times a day. At doses greater than this, reversible liver abnormalities may be seen. Because of its mechanism of action, Precose has significant gastrointestinal side effects. Abdominal pain, diarrhea, and gas are common and are seen in up to 75% of patients taking Precose. For this reason, Precose is administered using a low initial dose that is increased over weeks depending on the patient's tolerance.

#### ***New medications that effect glycemic control***

##### ***Symlin (pramlintide)***

Symlin is the first in a new class of injected antihyperglycemic medications for use in patients with type 2 or type 1 diabetes treated with insulin.

Pramlintide, the active ingredient in Symlin, is a synthetic analog of human amylin, a naturally occurring neuroendocrine hormone synthesized from pancreatic beta cells that contributes to glucose control during the postprandial period. Amylin, similar to insulin, is absent or deficient in patients with diabetes. When used with insulin, this compound can help patients achieve improved glycemic control with additional benefits that cannot be realized with insulin alone.

Symlin is taken just prior to meals, three times a day. It is given in injection form and is indicated for.

- Type 2 diabetes, as an adjunct treatment in patients who use mealtime insulin therapy and have failed to achieve desired glucose control despite optimal insulin therapy, with or without a concurrent sulfonylurea agent and/or metformin.

Type 1 diabetes, as an adjunct treatment in patients who use mealtime insulin therapy and who have failed to achieve desired glucose control despite optimal insulin therapy.

#### **Byetta (exenatide)**

Byetta (exenatide) is a new medication on the market that has its origins in an interesting place--the Gila monster's saliva. Scientists studying this small lizard noted it could go a long time without eating. They found a substance in its saliva that slowed stomach emptying, thus making the lizard feel fuller longer. This substance was similar in nature to a gut hormone found in humans

known as GLP-1. Thus, the studies began. Ultimately, after modifying this hormone, exenatide (with the trade name Byetta) was developed. Byetta is the first in a new class of drugs for the treatment of type 2 diabetes called incretin mimetics. Byetta has been shown to have many of the same effects on sugar regulation as GLP-1, so it mimics the body's natural physiology for self-regulating blood sugar.

#### **Combination Medications**

**Glyburide/metformin (Glucovance),** **rosiglitazone/metformin (Avandamet),** and **glipizide/metformin (Metaglip)** are 3 relatively new combination pills that are on the market to treat diabetes. Glucovance combines glyburide with metformin in varying doses. Avandamet is a combination of varying doses of Avandia and metformin. And Metaglip is a combination pill containing glipizide and metformin in varying strengths. While they work well, I personally like to give patients individual medications, until I know what doses are working, and then switch to a combination pill once the patient has been stable on the doses of individual medications for a period of time.

#### **TREATMENT OF DIABETES WITH INSULIN**

Insulin is the mainstay of treatment for patients with type 1 diabetes. Insulin is also important in type 2 diabetes when blood glucose levels cannot be controlled by diet, weight loss.

**Table:-2.**

<b>Name of Insulin</b>	<b>Onset of Action</b>	<b>Peak Effect After Injection</b>
Humalog and Novolog//Very Short Acting	5-15 minutes	30-60 minutes
Regular/Short Acting	30 minutes	2-5 hours
NPH/Intermediate Acting	1-2.5 hours	8-14 hours
Lente/Intermediate Acting	1-2.5 hours	8-12 hours
Ultra Lente/Long Acting	4-6 hours	10-18 hours
Lantus	2-3 hours	Stable from 2-3 hours to @20 hours
Combinations - 75/25, 70/30, 50/50	30 minutes	7-12 hours

For example, a patient may take an injection of Lente in the morning and evening to provide a baseline of insulin throughout a 24-hour period. In addition, the same patient may take an injection of Humalog just before meals to cover the increase in carbohydrate load after eating.

#### **Different methods of delivering insulin**

Not only is the variety of insulin preparations available growing, so are the methods for administering insulin.

#### **Pre-filled insulin pens**

In the past, insulin was available only in an injectable form. This involved carrying syringes (which a few decades ago were made of glass and required sterilization), needles, vials of insulin, and alcohol swabs. Needless to say, patients often found it difficult to

take multiple shots a day, and as a result, good blood sugar control was often compromised. Many pharmaceutical companies are now offering discreet and convenient methods of insulin delivery. Both Novo Nordisk and Lilly have an insulin pen delivery system. This system is similar to an ink cartridge in a fountain pen. A small pen-sized device holds an insulin cartridge (usually containing 300 units). Cartridges are available in the most widely used insulin formulations, such as those listed in the table above. The amount of insulin to be injected is dialed in by turning the bottom of the pen until the required number of units is seen in the dose-viewing window.

#### **Insulin pump**

The most recently available advance in insulin delivery is the insulin pump. In the United States, MiniMed, Deltec and Disetronic market the insulin pump. An

insulin pump is composed of a pump reservoir similar to that of an insulin cartridge, a battery-operated pump, and a computer chip that allows the user to control the exact amount of insulin being delivered. Currently, pumps on the market are about the size of a beeper. The pump is attached to a thin plastic tube (an infusion set) that has a soft cannula (or needle) at the end through which insulin passes. This cannula is inserted under the skin, usually on the abdomen. The cannula is changed every 2 days. The tubing can be disconnected from the pump while showering or swimming. The pump is used for continuous insulin delivery, 24 hours a day. The amount of insulin is programmed and is administered at a constant rate (basal rate). Often, the amount of insulin needed over the course of 24 hours varies depending on factors like exercise, activity level, and sleep. The insulin pump allows for the user to program many different basal rates to allow for this variation in lifestyle. In addition, the user can program the pump to deliver a "bolus" during meals to cover the excess demands of carbohydrate ingestion.

### **Inhalation**

Another promising route of insulin administration is through inhalation. Inhaled insulin is currently being tested but has not been approved by the United States Food and Drug Administration (FDA). Many devices are available that allow for other medications to be used in this manner, the best example of which is asthma therapy. Insulin is not absorbed through the bronchial tubes (airways), and must reach the air sacs at the end of the bronchial tubes (alveoli) to be absorbed. Once at the alveoli, insulin can be absorbed and enter the bloodstream. Currently, powdered inhalers and nebulizers are being studied to determine which delivery system is the most reliable. The safety of inhaled insulin still needs to be established before a product for consumer use can be made available. Trials are currently underway to establish the safety of inhaled insulin. These trials are well into phase III, meaning that human subjects have already used inhaled insulin and the results are promising. Inhaled insulin will likely be on the market within the next 1-2 years.

### **Intranasal, Transdermal, PILL**

Other routes for the delivery of insulin have also been tried. Intranasal insulin delivery was thought to be promising. However, this method was associated with poor absorption and nasal irritation. Transdermal insulin (skin patch delivery) has also yielded disappointing results to date. Insulin in pill form is also not yet effective since the digestive enzymes in the gut break it down.

### **The future of pancreas transplantation**

Ultimately, the goal in the management of type 1 diabetes is to provide insulin therapy in a manner that mimics the natural pancreas. Perhaps the closest therapy available at this time is a transplant of the pancreas. Several approaches to pancreatic transplantation are

currently being studied, including the whole pancreas and isolated islet cells (these groups of cells contain beta cells that are responsible for insulin production). Data available from 1995 indicates that almost 8,000 patients underwent pancreatic transplantation. Most patients undergo pancreatic transplantation at the time of kidney transplantation for diabetic kidney disease.

### **CONCLUSION**

Diabetes is a chronic condition associated with abnormally high levels of sugar (glucose) in the blood. Insulin produced by the pancreas lowers blood glucose. Absence or insufficient production of insulin causes diabetes. The two types of diabetes are referred to as type 1 (insulin dependent) and type 2 (non-insulin dependent). Symptoms of diabetes include increased urine output, thirst and hunger as well as fatigue. Diabetes is diagnosed by blood sugar (glucose) testing. The major complications of diabetes are both acute and chronic. Acutely: dangerously elevated blood sugar, abnormally low blood sugar due to diabetes medications may occur. Chronically: disease of the blood vessels (both small and large) which can damage the eye, kidneys, nerves, and heart may occur. Diabetes treatment depends on the type and severity of the diabetes. Type 1 diabetes is treated with insulin, exercise, and a diabetic diet. Type 2 diabetes is first treated with weight reduction, a diabetic diet, and exercise. When these measures fail to control the elevated blood sugars, oral medications are used. If oral medications are still insufficient, insulin medications are considered.

### **REFERENCE**

1. Joshi SR, Parikh RM. India - diabetes capital of the world: now heading towards hypertension. J Assoc Physicians India, 2007; 55: 323-4.
2. Kumar A, Goel MK, Jain RB, Khanna P, Chaudhary V. India towards diabetes control: Key issues. Australas Med J, 2013; 6(10): 524-31.
3. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. Diabetes Care, 2004; 27(3): 1047-53.
4. Whiting Dr, Guariguata L, Weil C, Shawj. IDF Diabetes atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pract, 2011; 94: 311-21.
5. Anjana RM, Ali MK, Pradeepa R, Deepa M, Datta M, Unnikrishnan R, Rema M, Mohan V. The need for obtaining accurate nationwide estimates of diabetes prevalence in India - rationale for a national study on diabetes. Indian J Med Res, 2011; 133: 369-80.
6. Zargar AH, Khan AK, Masoodi SR, Laway BA, Wani AI, Bashir MI, Dar FA. Prevalence of type 2 diabetes mellitus and impaired glucose tolerance in the Kashmir Valley of the Indian subcontinent. Diabetes Res Clin Pract, 2000; 47(2): 135-46.
7. Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, Rao PV, Yajnik CS, Prasanna

- Kumar KM, Nair JD. Diabetes Epidemiology Study Group in India (DESI). High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetologia*, 2001; 44(9): 1094–101.
8. Arora V, Malik JS, Khanna P, Goyal N, Kumar N, Singh M. Prevalence of diabetes in urban Haryana. *Australas Med J.*, 2010; 3(8): 488–94.
9. Bramley D, Hebert P, Jackson R, Chassin M. Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States. *N Z Med J.*, 2004; 117(1207): U1215.
10. Sukala WR, Page RA, Rowlands DS, Lys I, Krebs JD, Leikis MJ, Cheema BS. Exercise intervention in New Zealand Polynesian peoples with type 2 diabetes: Cultural considerations and clinical trial recommendations. *Australas Med J.*, 2012; 5(8): 429–35.
11. Khalil H, George J. Diabetes management in Australian rural aged care facilities: A cross-sectional audit. *Australas Med J.*, 2012; 5(11): 575–80.
12. Rao CR, Kamath VG, Shetty A, Kamath A. A cross-sectional analysis of obesity among a rural population in coastal southern Karnataka, India. *Australas Med J.*, 2011; 4(1): 53–57.
13. Mohan V, Deepa R. Obesity and abdominal obesity in Asian Indians. *Indian J Med Res.*, 2006; 123(5): 593–96.
14. Mohan V, Shah S, Saboo B. Current glycemic status and diabetes related complications among type 2 diabetes patients in India: data from the Alchieve study. *JAPI (Suppl)*, 2013; 61: 12–15.
15. Mohan V, Seshiah V, Sahay BK, Shah SN, Rao PV, Banerjee S. Current status of management of diabetes and glycaemic control in India: Preliminary results from the DiabCare India 2011 Study. *Diabetes.*, 2012; 61: a645–a677.
16. Rema M, Premkumar S, Anitha B, Deepa R, Pradeepa R, Mohan V. Prevalence of diabetic retinopathy in urban India: The Chennai urban rural Epidemiology Study (CurES) Eye Study. I. *Invest Ophthalmol Vis Sci.*, 2005; 46: 2328–2333.
17. Unnikrishnan RI, Rema M, Pradeep R, Deepa M, Shanthirani CS, Deepa R, Mohan V. Prevalence and risk factor of diabetic nephropathy in an urban south Indian population; The Chennai urban rural Epidemiology study (CurES-45) *Diabetes Care*, 2007; 30: 2019–2024.
18. Pradeepa R, Rema M, Vignesh J, Deepa M, Deepa R, Mohan V. Prevalence and risk factors for diabetic neuropathy in an urban south Indian population: The Chennai urban rural Epidemiology Study (CurES-55). *Diabet Med*, 2008; 25: 407–412.
19. Mohan V, Deepa R, Shanthi Rani S, Premalatha G. Prevalence of coronary artery disease and its relationship to lipids in a selected population in south India. *Journal of American College of Cardiology*, 2001; 38: 682–687.
20. Premalatha G, Shanthi Rani CS, Deepa R, Markovitz J, Mohan V. Prevalence and risk factors of peripheral vascular disease in a selected south Indian population – The Chennai urban Population Study (CuPS) *Diabetes Care*, 2000; 23: 295–1300.
21. Rastogi A, Bhadada SK, Saikia UN, Bhansali A. Recurrent diabetic myonecrosis: a rare complication of a common disease. *Indian J Med Sci*, 2011; 65(7): 311–4.
22. Iyer SN, Drake AJ 3rd, West RL, Tanenberg RJ. Diabetic muscle infarction: a rare complication of long-standing and poorly controlled diabetes mellitus. *Case Rep Med*, 2011; 2011: 407921.
23. Kumar A. Insulin guidelines: taking it forward. *Medicine Update (API India)*, 2010; 20: 127–30.
24. Unnikrishnan RI, Anjana RM, Mohan V. Importance of Controlling Diabetes Early–The Concept of Metabolic Memory, Legacy Effect and the Case for Early Insulinisation. *JAPI (Suppl)*, 2011; 50: 8–12.
25. Sui Z, Turnbull D, Dodd J. Enablers of and barriers to making healthy change during pregnancy in overweight and obese women. *Australas Med J.*, 2013; 6(11): 565–77.
26. Minnie Au, Rattigan S. Barriers to the management of Diabetes Mellitus – is there a future role for Laser Doppler Flowmetry? *Australas Med J.*, 2012; 5(12): 627–32.
27. Verma R, Khanna P, Mehta B. National programme on prevention and control of diabetes in India: Need to focus. *Australas Med J.*, 2012; 5(6): 310–5.
28. State-based diabetes prevention and control program. Centers for Disease Control and Prevention. U.S Department of Health & Human Services., 2013 Accessed on 13 Dec. 2013.
29. National Diabetes Education Program. Centers for Disease Control and Prevention. Accessed on 13 Dec. 2013.
30. Authoritative Institute of Health and Welfare (AIHW) National health priority areas., 2013 Accessed on 13 Dec. 2013.
31. Ali M, Knight A. Comparative healthcare: Diabetes Mellitus. *Australas Med J.*, 2009; 1(5): 1–9.
32. National service frameworks and strategies. National Health Services. 2011 Jul; Accessed on 13 Dec. 2013.
33. Mathew E, Ahmed M, Hamid S, Abdulla F, Batool K. Hypertension and dyslipidaemia in Type 2 diabetes mellitus in United Arab Emirates. *Australas Med J.*, 2010; 3(11): 699–706.